INEQUALITY "UNDER THE SKIN": STRESS AND THE BIODEMOGRAPHY OF RACIAL HEALTH DISPARITIES ACROSS THE LIFE COURSE

Courtney E. Boen

A dissertation submitted to the faculty at the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Sociology Department.

Chapel Hill
2017

Approved by:

Y. Claire Yang

Karolyn Tyson

Robert Hummer

Kathleen Mullan Harris

Anthony Perez

© 2017
Courtney E. Boen
ALL RIGHTS RESERVED

ABSTRACT

Courtney E. Boen: Inequality "Under the Skin": Stress and the Biodemography of Racial Health Disparities Across the Life Course
(Under the direction of Y. Claire Yang and Karolyn Tyson)

Black-White disparities in morbidity and mortality can be observed across the life course, from birth through late life. Given the persistence of racial health gaps across time and space, a wide body of literature seeks to better understand the social factors contributing to these disparities, and research suggests that racial differences in exposure to social stressors play a critical—yet largely underestimated—role in Black-White health gaps. Still, critical gaps in scientific understanding of how racial differences in exposure to stressors across the life course contribute to Black-White disparities in disease emergence and progression remain. Using two longitudinal, population-level data sets that collectively span from adolescence through late adulthood, this research examines how racial inequality patterns exposure to material conditions and psychosocial stressors to promote physiological and psychological dysregulation and ultimately affect health and disease risk. In particular, this study assesses how racial differences in exposure to stress related to criminal justice contacts, neighborhood conditions, and lifetime events and chronic strains affect Black-White inequities in pre-disease biomarkers of health and physiological function. This research thus improves scientific understanding of how racism patterns exposure to material conditions and psychosocial stressors to promote physiological and psychological dysregulation and ultimately affect health and disease risk on a population level.

ACKNOWLEDGEMENTS

I want to begin by thanking two scholars who have shepherded me since stepping foot on the Carolina campus. To Claire Yang and Karolyn Tyson: I am eternally grateful for your guidance, (constructive) criticism, collaboration, and mentorship. You are two of the best players in the game. I am also grateful to the other members of my "Dream Team" dissertation committee: Bob Hummer, Kathie Harris, and Tony Perez. Thank you for all of your energy, time, and support.

My time at Carolina—and my own intellectual growth—would not be what it was without the countless friends, peers, and fellow students who joined me for trivia games, study sessions, happy hours, debates, and drives to DC. I was blessed to be a member of, unquestionably, the fiercest, smartest, and kindest cohort to ever walk the halls of Hamilton. I will carry you Tarheels with me through all of my pursuits.

My colleagues outside of the academy also played a critical role in my development as a scholar, activist, and ally. I am particularly grateful to my friends and former coworkers in Boston who schooled me in so many ways, providing me with a deeper, fuller understanding of racism, and my own role and obligation in undoing it, than I could have learned in any book or classroom. Thank you for all that you did for me, and all that you do.

In many ways, my journey to a PhD began with a family who instilled in me a profound penchant for social justice, equity, and kindness. My parents taught me that smart, opinionated, and confident girls are not only cool, but that they are, in fact, an essential element for creating a more loving, peaceful, and productive world. I also learned early on that having sisters as best

friends is one of life's greatest blessings. To Mom, Dad, Colleen, and Molly: thank you for everything.

I want to send my love and thanks to my daughter, Riley. You have been such a big part of this journey for me, sweet girl. So much of this dissertation was written while I was thinking about you, planning for you, holding you (…literally), and loving you. You motivated me to show the world that strong, passionate, and caring women can do anything they set their minds to, with enough coffee. Thank you for bursting the seams of my heart and soul with boundless joy. I can't wait to see what you have in store for the world.

Finally, to Dan, to whom I owe everything. You should get some sort of degree for your role in this process. Your love, patience, and partnership breathed life into me when I felt burnt out, tired, and frustrated. Your support of my dreams, goals, and work has meant the world to me. I hope that I can be half the partner that you have been to me. I love you, my dear.

This dissertation examines how racism, as a system, literally and metaphorically "gets under the skin" to infest the body: its organs, systems, and cells. It provides new evidence of the role of structural racial inequality in contributing to the emergence and progression of disease and producing profound inequities in health and mortality from childhood through late life. The work presented in the pages that follow documents, with precision and specificity, how racism patterns access to resources and exposure to physiological toxins to ultimately impact health on a population level. Despite its innovations, the work presented here, in many ways, serves as an echo to what communities of color have been shouting, crying, and protesting for centuries: that racism is real; that it hurts; and that it kills. I am grateful to the many scholars and activists of color who came before me and worked alongside me for their insights, ideas, and truth, which they had the courage to voice in the face of unimaginable resistance. Still, one of my deepest

fears is that this dissertation will become a lifeless book on a shelf, rather than serving as an urgent call to action. My hope is that this work functions as a rallying cry, pleading and insisting that undoing racism becomes a scholarly, societal, and public health priority.

Now is the time.

TABLE OF CONTENTS

LIST OF TABLES xi

LIST OF FIGURES xii

CHAPTER 1: INTRODUCTION 1

Stress and Racial Health Disparities 5

Racial Inequality and the Stress Process 5

Gaps in the Literature 6

Dissertation Overview 10

Aims and Approach of the Proposed Dissertation 10

Paper 1: Race, Criminal Justice Contacts, and Health: Stress-Related Disparities in the Carceral State 12

Paper 2: Does Integration Produce Equity?: A Longitudinal Study of Neighborhood Conditions and Racial Health Inequality 12

Paper 3: Psychosocial Stress Exposure and Black-White Health Inequality in Late Life 13

Conclusions 14

REFERENCES 15

CHAPTER 2: RACE, CRIMINAL JUSTICE CONTACTS, AND HEALTH: STRESS-RELATED DISPARITIES IN THE CARCERAL STATE 22

Abstract 22

Introduction 23

Background 27

Gaps in the Literature 27

Research Questions 30

Data and Methods 31

Data and Sample 31

Measures 31

Outcomes 31

Criminal Justice Contacts 32
Other Measures 33
Analytic Strategy 33
Descriptive Analyses 33
Criminal Justice Contacts and Health 34
Incarceration and Health 34
Analytic Samples 36
Results 37
Descriptive Statistics 37
Criminal Justice Contacts and Health 37
Incarceration and Health 38
CRP 39
Depressive Symptoms 40
Discussion 41
REFERENCES 46
CHAPTER 3: DOES INTEGRATION PRODUCE EQUITY? A LONGITUDINAL STUDY OF NEIGHBORHOOD CONDITIONS AND RACIAL HEALTH INEQUALITY 59
Abstract 59
Introduction 60
Theoretical Model 63
Neighborhoods and Health 63
Mediating Mechanisms 66
Social Relationships 66
Racial Discrimination 67
Variation Across the Life Course 68
Research Questions 68
Data and Analytic Methods 69
Data 69
Measures 70
Dependent Variables 70

Key Explanatory Variables 71
Neighborhood Conditions 71
Psychosocial Mediators 72
Other Measures 73
Analytic Sample 73
Analytic Methods 73
Results 75
Descriptive Statistics 75
Multilevel Models 75
Metabolic Dysregulation 75
BMI 77
Discussion 80
REFERENCES 86
CHAPTER 4: LIFE COURSE PSYCHOSOCIAL STRESS EXPOSURE AND BLACK-WHITE HEALTH DISPARITIES IN OLD AGE 103
Abstract 103
Introduction 104
Background 105
Stress and Health 105
Structural Inequality and Stress 107
Gaps in the Literature 109
Aims of the Present Study 111
Data and Methods 112
Data and Sample 112
Measures 113
Outcomes 113
Key Explanatory Variables 114
Other Measures 115
Analytic Strategy 115
Results 117
Descriptive Statistics 117

Multivariate Models 118
Inflammation 118
Metabolic Function 120
Depressive Risk 121
Discussion 122
REFERENCES 128
CHAPTER 5: CONCLUSION 145

LIST OF TABLES

Table 2.1. Description of Measures 50

Table 2.2. Descriptive Statistics..... 52

Table 2.3. Contacts with the Criminal Justice System and Depressive Symptoms (N=5,503)..... 53

Table 2.4. Incarceration History and Log C-Reactive Protein..... 54

Table 2.5. Incarceration History and Depressive Symptoms..... 55

Table 3.1. Descriptive Statistics, Weighted (N=6,248) 92

Table 3.2. Outcome and Neighborhood Measures, by Race (N=6,248)..... 93

Table 3.3. Neighborhood Characteristics and Metabolic Dysregulation: Multilevel Regression Models, 1994-2008 94

Table 3.4. Neighborhood Fixed Effects Models, 1994-2008..... 97

Table 3.5. Neighborhood Characteristics and BMI: Multilevel Regression Models, 1994-2008 98

Table 4.1. Domain-Specific Stressor Measure Construction..... 133

Table 4.2. Descriptive Statistics by Race..... 136

Table 4.3. Stress Exposure and Log C-Reactive Protein (N=3,773) 137

Table 4.4. Stress Exposure and Metabolic Dysregulation (N=3,351) 139

Table 4.5. Stress Exposure and Depressive Symptoms (N=4,773) 141

Table 4.6. Stress Exposure and Change in Depressive Risk 2006-2012 (N=4,411) 143

LIST OF FIGURES

Figure 1.1. Conceptual Model 21

Figure 2.1. Criminal Justice Contacts and Depressive Symptoms 56

Figure 2.2. Incarceration History and C-Reactive Protein 57

Figure 2.3. Duration of Incarceration and Depressive Symptoms 58

Figure 3.1. Theoretical Model 101

Figure 3.2. Neighborhood Racial Composition and Black Reports of Discrimination 102

CHAPTER 1: INTRODUCTION

Black-White disparities in morbidity and mortality can be observed across the life course, from birth through late life. Black children are less likely than their White peers to be in good health (Flores, Olson, and Tomany-Korman 2005), and racial disparities among adults span an array of conditions and illnesses, including cardiovascular disease (Keenan and Shaw 2011) and cancer (DeLancey et al. 2008). Scholars have characterized Blacks' high rates of morbidity as "first and worst," whereby Blacks experience earlier onset of illness, greater severity of disease, and poorer survival rates than Whites (Williams et al. 2010).

In sociological research on racial health inequality, race is often treated as proxy for the real determinant of racial disparities: racism (Schnittker and McLeod 2005). An ideology and system of domination that ranks the value of racial groups as superior or inferior, racism contributes to the development and propagation of race-based attitudes, beliefs, and differential treatment of group members by both individuals and institutions (Bonilla-Silva 1997). As a system of stratification, racism operates at the institutional, interpersonal, and internalized levels to generate racial disparities in health (Chae et al. 2011; Gee, Walsemann, and Brondolo 2012a; Jones 2000). At the institutional level, racism harms health through policies and practices that restrict access to health promoting resources and opportunities, such as well-paying jobs and quality medical care, and pattern exposure to health risks and toxins, such as environmental toxins and neighborhood violence. Interpersonally mediated racism harms health through the stress and unfair treatment associated with discrimination, racial prejudice, and micro-aggressions. On the individual level, internalized racism contributes to racial health inequality

through psychological processes related to internalized racial superiority and inferiority and dimensions of racial identity. Rather than reflecting inherent biological divisions of the human species, racial disparities in health result from this broader system of racial stratification that structures access to resources, opportunities, and risks by race (Jones 2000; Mason 1994).

Given documented Black-White health disparities across a variety of outcomes, a wide body of research seeks to understand the proximal determinants of racial health inequality. The bulk of research on racial health disparities focuses on identifying and examining how racial differences in material well-being contribute to health inequality. This scholarly focus on the socioeconomic factors producing racial health disparities is warranted, as decades of research across disciplines document stark racial disparities in education, employment, income, and wealth (Bureau of Labor Statistics 2012; US Census Bureau 2012; Taylor et al. 2011). Given both stark racial disparities in socioeconomic status and well-documented linkages between socioeconomic factors and health (Link and Phelan 1995), socioeconomic factors are considered key drivers of Black-White health gaps. In fact, Phelan and Link (2015) argue that racism is a "fundamental determinant" of racial health disparities, in large part because of its role in producing stark racial differences in socioeconomic opportunities and conditions. In general, studies find that adjusting for racial differences in socioeconomic status (SES) attenuates Black-White health gaps, but disparities persist even after controlling for these factors (Franks et al. 2006; Hayward et al. 2000; Williams et al. 2010). In this way, research also suggests that other, non-socioeconomic factors and processes also play an essential role in racial health inequities.

A second, less-studied category of research on racial health disparities focuses on the psychosocial processes contributing to the racial stratification of health. Theory and research suggest that racial differences in exposure to social stressors play a critical—yet largely

underestimated—role in the Black-White health gap, net of racial differences in access to material goods and resources (Schnittker and McLeod 2005; Turner 2013a). Scholars hypothesize that Blacks' greater exposure to stress and stigmatization may contribute to disproportionate physiological deterioration and accelerated biological aging, relative to Whites (Geronimus et al. 2006; Geronimus et al. 2010a). In response to stressors, the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) respond by secreting hormones to up-regulate physiological functioning, inducing a host of immune, neuroendocrine, metabolic, and cardiovascular changes (McEwen and Stellar 1993; McEwen 1998a). While these changes are essential for protecting health against acute infections and threats in the short term, long-term activation of these systems in response to chronic stressors and strains can harm health by promoting physiological dysregulation and allostatic load (Cohen et al. 2012; Glaser and Kiecolt-Glaser 2005a; McEwen and Stellar 1993; McEwen 1998a). Studies with both animals and humans provide evidence of the harmful effects of stress on physical functioning and health, where physiological and psychological systems become ineffective and inefficient following long-term exposure to stress (Epel et al. 2006; Epel 2009; Epel and Lithgow 2014; McClintock et al. 2005). In this way, studies on the racial stratification of stress and health suggest that racial disparities in stress exposure may be another key mechanism through which racism harms health, net of SES.

In the literature, these two approaches to the study of racial health inequality—one focused on material pathways and the other centered on psychosocial processes—are most often positioned as in conflict or in opposition to one another (Chae et al. 2011; Schnittker and McLeod 2005). However, closer examination reveals that these perspectives are not mutually exclusive. In fact, research on the psychosocial processes contributing to health disparities can

elucidate the ways that macro-level structural inequality shapes access and exposure to the material conditions that pattern exposure to psychosocial stress. As Pearlin (1999: 242) argues, “many stressful experiences, it should be recognized, don't spring out of a vacuum but typically can be traced back to surrounding social structures and people's locations within them.” In other words, psychosocial stress exposure corresponds, in larger part, to individual’s locations within various systems of stratification, including those based on race, socioeconomic status, and gender.

Research confirms that stress exposure is highly patterned by race, such that Blacks and other people of color report higher levels of both discrimination-related and generalized psychosocial stress than Whites (Clarke et al. 1999; Pearlin 1999; Thoits 1995; Turner and Avison 2003; Umberson 2017). Further, research suggests exposure to stress may be one of the critical pathways through which racial differences in material conditions produce racial health inequities (Kahn and Pearlin 2006; Strenthal, Slopen, and Williams 2011), with racial differences in socioeconomic factors shaping Black-White disparities in exposure to a host of chronic stressors and strains. In this way, then, sociological research on differential exposure and vulnerability to stress can improve understanding of how broader patterns of social stratification produce health inequality, as one’s exposure and vulnerability to stress is linked to his or her position in the social structure (Aneshensel 1992; Pearlin 1989). Rather than conceptualizing material and psychosocial factors as unique, disconnected factors generating racial health disparities, an integrated approach that considers how racism patterns the material and psychosocial resources, exposures, and processes to ultimately generate racial health inequality is needed (Chae et al. 2011; Hummer 1996).

This dissertation contributes new knowledge of the role of stress in Black-White health disparities across the life course by integrating diverse and nuanced measures of stress exposure and objective markers of physiological and psychological functioning into examinations of Black-White health disparities. Using two longitudinal, population-level data sets that collectively span from adolescence through late adulthood, this research examines how racial inequality patterns exposure to material conditions and psychosocial stressors to promote physiological and psychological dysregulation and ultimately affect health and disease risk.

Stress and Racial Health Disparities

Racial Inequality and the Stress Process

Despite the overwhelming dominance of psychologists and biologists in the study of stress, sociologists also have a vested intellectual interest in the study of the stress process (Pearlin et al. 1981; Pearlin 1989). First articulated by Pearlin et al. (1981), the stress process describes how the sources, the mediators, and manifestations of stress come together to form a singular, interactive process. The sources of stress—perhaps most familiar to sociologists—include traumatic and stressful life events, financial strains, everyday and major life discrimination, and other forms of acute and chronic stress that are linked to the organization of the social world. The life events and chronic strains identified by scientists as sources of stress can be traced to the organization of social hierarchies in a society—including inequality along race, gender, and social class lines—as well as individuals locations within those systems (Pearlin 1989). The mediators of stress include the resources, behaviors, perceptions, and cognitions that individuals invoke in the face of stressors and include social supports and coping mechanisms (Pearlin et al. 1981; Pearlin 1989). Finally, the outcomes of stress include a host of psychological and biological changes that occur when the stressors overwhelm the adaptive capacity of an physiological and psychological systems (Cohen, Kessler, and Gordon 1997).

Pearlin (1981) argued that, in order to fully understand the role of stress in social inequality, research on social stress must examine how all three components of the stress process interact to form an interconnected social process.

Scholars have noted the critical importance of race and racial stratification for the stress process model (Clark et al. 1999; Pearlin 1989; Turner 2013a), with differential exposure and vulnerability to stress being key determinants of racial health disparities (Adkins et al. 2009; Jackson, Knight, and Rafferty 2010; Turner, Wheaton, and Lloyd 1995). Racism in and of itself may be perceived as inherently stressful, and it also patterns exposure to a variety of acute and chronic stressors (Clark et al. 1999). As explained by Geronimus et al. (2006: 826): "the stress inherent in living in a race-conscious society that stigmatizes and disadvantages Blacks may cause disproportionate physiological deterioration, such that a Black individual may show the morbidity and mortality typical of a White person who is significantly older."

Gaps in the Literature

Despite a growing body of literature on the role of stress in racial health inequality, critical gaps in the literature remain.

1. *Measurement of stress*

First, most studies of the health effects of stress exposure use single, individualized measures of stress. Few consider how exposure to multiple forms of psychosocial stress, including stressors in the broader social environment, individually and jointly induces individual physiological and psychological stress response (Gee, Walsemann, and Brondolo 2012b; Williams and Mohammed 2009). In fact, studies on the role of stress exposure and vulnerability in health disparities offer conflicting and inconsistent findings, which is, at least in part, attributable to the varying and incomplete measurement of stress exposure in empirical studies (Aneshensel 1992; Thoits 1995; Turner 2013a). Most research on stress exposure focuses on

either stressful life events or perceived exposure to chronic stressors. Though both forms of stress have been linked to a host of mental and physical health outcomes, when used alone, each offers an incomplete picture of how stress exposure contributes to population health disparities. Stressful life events include the acute changes in one's life that spur disruption, change, anxiety and grief; examples of these events would include events such as births of children, divorce, and deaths of family members (Thoits 1995). Though these life events have been linked to a host of outcomes, including morbidity, mortality, and psychological problems (Cohen and Williamson 1991; Thoits 1983), there are inconsistencies in how "stressful life events" are selected and measured (Aneshensel 1992). Further, some events (e.g., divorce, unemployment) are more likely to occur in some groups than in others, which could bias findings on the association between stressful life events and health (Aneshensel 1992). Further, scholars have identified a number of lower-grade, chronic stressors—including difficulty paying bills, work-family conflicts, and caring for a sick or disabled family member—that may impact mental and physical health but are not captured through the measurement of stressful, traumatic, or negative life events (Pearlin et al. 1981; Wheaton 1994). Scholars have also argued that the distinction between "acute" and "chronic" stressors is artificial, as these two forms of stressors likely co-occur and are reciprocally related and mutually reinforcing (Aneshensel 1992; Pearlin et al. 1989).

The inconsistencies in measuring stress exposure have important implications for research findings, particularly as they relate to Black-White health inequities. Primarily, by failing to fully account for racial differences in acute and chronic stress exposure, studies risk underestimating the contribution of psychosocial stressors to persistent Black-White health gaps. For example, using a checklist of recent life events, Turner and Avison (2003) found that

differences in stress exposure between Blacks and Whites were grossly underestimated. When the authors also included measures of recent life events, chronic stressors, discrimination stress, and lifetime occurrence of major and potentially traumatic life events, the estimate for the elevated stress exposure of Blacks relative to Whites was approximately 2.6 times greater than when recent life events were used alone.

In addition to using singular measures of stress exposure, most studies of the health effects of stress exposure use individualized measures of stress, such as perceived social stress and perceived racial discrimination. Few consider how stressors in the broader social environment—such as in neighborhood environments or in contacts with the criminal justice system—induce individual physiological stress response (Gee, Walsemann, and Brondolo 2012; Williams and Mohammed 2009). As a result, little is known about the health effects of macro-level structural stressors (such as policing practices or mass incarceration) or meso-level environmental stressors (such as neighborhood contexts) or the role these social stressors play in producing Black-White health disparities.

2. *Life course processes*

Second, due largely to data limitations, there is limited understanding of how the life course timing and accumulation of social stressors impacts trajectories of health with age (Williams, Neighbors, and Jackson 2003). Life course theories suggest that stress experienced early in the life course may play an especially critical role in shaping later life health (Evans and Kim 2007). In particular, research suggests that adolescence and the transition to adulthood may be especially sensitive developmental periods, during which exposure to stress may have long-lasting health impacts (Andersen and Teicher 2008; Dietz 1994; Romeo 2010). For this reason,

exposure to racism-related stress during these life course periods may be particularly critical in shaping trajectories of health.

Further, stress exposure may accumulate across the life span to accelerate physiological deterioration and biological aging (Ben-Shlomo and Kuh 2002). Individuals experience psychosocial stress across the life course. They also encounter a variety of situations and institutions that may expose them to new forms of race-related and generalized stress and contribute to the divergence of Black-White health gaps across the life course (Gee, Walsemann, and Brondolo 2012). However, few studies have tested these life course hypotheses, particularly in the context of racial health inequality.

3. *Biological mechanisms*

Finally, though most definitions of stress acknowledge the process through which the adaptive capacity of an organism is overwhelmed (Cohen, Kessler, & Gordon 1997), few studies of racial health inequality examine the physiological dysregulation that results from stress exposure. Most studies of the role of stress in health disparities examine mental health outcomes such as major depression (Ayalon and Gum 2011; Cagney et al. 2014; Drentea and Reynolds 2014; Houle 2014; Turner 2013b; Turney, Lee, and Comfort 2013; Walsemann, Gee, and Geronimus 2009). Still, other studies examine specific indicators of general health, such as disease outcomes (Williams, Neighbors, and Jackson 2005). Fewer studies assess the health effects of stress by integrating biomarkers of physiological and psychological functioning, such as markers of inflammation or metabolic risk. This reliance on general indicators of health or disease outcomes in studies of the health effects of stress is not without implications. As Turner (2013) argues, this almost exclusive focus on mental and physical disease outcomes can result in misclassification error, as 1) the diseases and disorders examined in studies have effects on other

diseases and disorders not considered in the studies, and 2) individuals who do not yet have the disease or have not yet been diagnosed with the disease are classified as "well." Further, the widespread misclassification of individuals with undiagnosed or undetected health problems may result in an underestimation of the contribution of social factors to health and disease (Aneshensel 2005). Research utilizing biomarker data to study the health effects of stress can improve understanding of the biological mechanisms linking social stress exposure to health. Further, examining how stress is implicated in the dysregulation in biological processes—including metabolic and immune function—can shed new light on how the social environment shapes the precursors to disease and offer new insights into effective disease prevention strategies.

Dissertation Overview

Aims and Approach of the Proposed Dissertation

The studies comprising this dissertation fill these gaps by integrating multidimensional measures of stress exposure and diverse measures of physiological and psychological stress response into empirical examinations of racial health disparities. Using a longitudinal data and a conceptual framework that defines both stress and health as life course processes, this research contributes new knowledge of how structural racial inequality patterns individual stress exposure, promotes physiological and psychological dysregulation, accelerates biological aging, and ultimately harms health across the life course.

A conceptual model that guides this research can be found in Figure 1.1. The aims of this research build on previous studies to understand the pathways linking racism, material conditions, stress exposure, and health across the life course. I take the position that racism patterns stress exposure both directly—with the structural disadvantages and discrimination stemming from a broader system of racial oppression being inherently stressful—and indirectly,

by patterning individual exposure to a variety of acute and chronic stressors and strains. Throughout each of the three studies presented here, I document how exposure to a diverse set of macro- (e.g., mass incarceration), meso- (e.g., neighborhood conditions), and micro-level (e.g., interpersonal discrimination) stressors are patterned by race.

I further examine how exposure to these stressors serves to up-regulate immune, metabolic, cardiovascular, and psychological changes that, over time, contribute to physiological and psychological dysfunction and increased disease and mortality risk. While prior research mainly links stress exposure to mental health outcomes, this research provides new insights into how the racial patterning of stress exposure contributes to disparities in pre-disease markers of physical and mental well-being. Given that research provides clear evidence of the association between biological and psychological markers such as C-reactive protein, blood pressure, cholesterol, and depressive symptoms with disease and mortality risk (Finch 2010a; Gruenewald et al. 2006; Saliques et al. 2010), this study improves understanding of the relevancy of the stress process model to racial health disparities by interrogating the biophysiological and psychological pathways linking racial differences in stress exposure to disparities in morbidity and mortality.

Further, research documents that psychosocial resources can buffer against or moderate physiological stress response (Yang, Boen, and Mullan Harris 2015; Yang et al. 2016). In addition to understanding how racial differences in stress exposure contribute to health disparities, this project also seeks to better understand the role of racial differences in psychosocial resources in producing disparities in biomarkers of health and physiological functioning.

Finally, guided by a longitudinal life course perspective, this research uses two population-level data sets that collectively span from adolescence through late-life to better

understand how the stress process model unfolds at various life stages to impact trajectories of health inequality. Additionally, by using measures of lifetime stress exposure, age of exposure, and duration of exposure, the three empirical chapters of this dissertation contribute new understanding of how the accumulation, timing, and duration of stress exposures across the life course impact health and contribute to Black-White health differentials.

Paper 1: Race, Criminal Justice Contacts, and Health: Stress-Related Disparities in the Carceral State

Across the criminal justice continuum, stark racial disparities can be observed. Blacks are more likely than Whites to be stopped by police, arrested, convicted, and incarcerated. Further, their encounters with criminal justice institutions and officials are characterized by high levels of stress, anxiety, and hostility. Given the large and increasing role that the criminal justice system plays in determining the life chances of Blacks in the United States, in particular, understanding the role of Black-White disparities in police encounters, arrests, convictions, and incarceration in population health inequality is critical, as research suggests that these contacts with the criminal justice system may be particularly salient sources of stress for individuals. Using longitudinal data and a variety of analytic techniques—including multivariate regression models, a unique treatment-control design, and treatment weighting procedures—this study examines the relationships between contacts with the criminal justice system and pre-disease markers of physiological and psychological stress response. I further assess the extent to which racial differences in criminal justice encounters account for Black-White disparities in health and well-being early in the life course.

Paper 2: Does Integration Produce Equity?: A Longitudinal Study of Neighborhood Conditions and Racial Health Inequality

The extant literature identifies racial residential segregation as a fundamental cause of racial health disparities (Williams and Collins 2001), with studies across disciplines linking the

conditions in racially segregated neighborhoods to disparities across a number of health outcomes, including low birth weight, depression, and cardiovascular disease (Diez-Roux et al. 1997; Williams and Jackson 2005). While research on racial residential segregation and health has boomed in recent years, critical gaps in the literature remain. This study improves understanding of the contribution of neighborhood conditions to racial disparities in health by examining how neighborhood economic conditions and racial composition jointly and uniquely relate to markers of physiological stress response from adolescence through young adulthood. By elucidating the pathways connecting neighborhoods to health, this study offers a nuanced depiction of the relationships between racism, neighborhood conditions, stress, and health and offers critical insights into the relative importance of neighborhood diversity and equity for population health.

Paper 3: Psychosocial Stress Exposure and Black-White Health Inequality in Late Life

A body of research on the age patterning of Black-White health inequality documents a divergence of health disparities with age (Shuey and Willson 2008), whereby Blacks experience earlier onset of disease and steeper increases in disease incidence with age than Whites. This finding has led scholars to hypothesize that Blacks may experience accelerated biological aging relative to Whites (Clarke et al. 2014; Geronimus et al. 2010b; Rewak et al. 2014). Given that Blacks in the U.S. are exposed to greater levels of structural disadvantage (Pager and Shepherd 2008; Taylor et al. 2011) and report higher levels of related psychosocial stress (Adkins et al. 2009; Turner, Wheaton, and Lloyd 1995; Turner 2013b) than Whites, racial differences in stress exposure may, at least in part, account for Blacks' accelerated biological aging relative to Whites. Despite a burgeoning literature on the contribution of psychosocial stress to racial health disparities, critical gaps in the literature remain. This study assesses the contribution of psychosocial stress exposure across the life course to racial disparities in markers of

physiological functioning and psychological well-being in late life. By comprehensively documenting the racial patterning of lifetime stress exposure and linking stress exposure to pre-disease markers of health, this study improves understanding of the psychosocial and biological mechanisms producing racially stratified trajectories of health at older ages.

Conclusions

Though distinct, the three studies presented here together contribute new knowledge of the role of stress in racial health disparities across the life course. Using two longitudinal, population-level data sets that collectively span the life course, this research examines how exposure to various dimensions of stress (e.g., traumatic events, chronic strains, perceived discrimination) contribute to racial differences in markers of physical health and pyschological functioning (e.g., metabolic function, inflammation, depressive symptoms) at different life stages (e.g., from adolescence through young adulthood, late life). These studies integrate diverse measures of life course stress exposure and objective markers of bodily stress response systems and utilize advanced analytic techniques to examine the biological and psychosocial pathways through which stressors generate racial disparities in disease and longevity. Findings from this study will improve scientific understanding of the role of racism in patterning stress exposure; assess the contribution of stress exposure to the racial patterning of morbidity and mortality; shed light on the biological and psychological mechanisms linking stress exposure to disease and mortality from early- through late-life; and ultimately improve preventative policy and intervention efforts aimed at achieving racial health equity.

REFERENCES

Adkins, Daniel E., Victor Wang, Matthew E. Dupre, Van den Oord, Edwin JCG and Glen H. Elder. 2009. "Structure and Stress: Trajectories of Depressive Symptoms Across Adolescence and Young Adulthood." *Social Forces* 88(1):31-60.

Andersen, Susan L. and Martin H. Teicher. 2008. "Stress, Sensitive Periods and Maturational Events in Adolescent Depression." *Trends in Neurosciences* 31(4):183-191.

Aneshensel, Carol S. 1992. "Social Stress: Theory and Research." *Annual Review of Sociology*:15-38.

Aneshensel, C. S. 2005. "Research in Mental Health: Social Etiology Versus Social Consequences." *Journal of Health and Social Behavior* 46(3):221-228.

Ayalon, Liat and Amber M. Gum. 2011. "The Relationships between Major Lifetime Discrimination, Everyday Discrimination, and Mental Health in Three Racial and Ethnic Groups of Older Adults." *Aging & Mental Health* 15(5):587-594.

Ben-Shlomo, Y. and D. Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31(2):285-293.

Bonilla-Silva, Eduardo. 1997. "Rethinking Racism: Toward a Structural Interpretation." *American Sociological Review*:465-480.

Cagney, Kathleen A., Christopher R. Browning, James Iveniuk and Ned English. 2014. "The Onset of Depression during the Great Recession: Foreclosure and Older Adult Mental Health." *Journal Information* 104(3).

Chae, David H., Amani M. Nuru-Jeter, Karen D. Lincoln and Darlene D. Francis. 2011. "CONCEPTUALIZING RACIAL DISPARITIES IN HEALTH." *Du Bois Review: Social Science Research on Race* 8(01):63-77 (http://dx.doi.org.libproxy.lib.unc.edu/10.1017/S1742058X11000166). doi: 10.1017/S1742058X11000166.

Clark, Rodney, Norman B. Anderson, Vernessa R. Clark and David R. Williams. 1999. "Racism as a Stressor for African Americans: A Biopsychosocial Model." *American Psychologist* 54(10):805-816 (https://auth.lib.unc.edu/ezproxy_auth.php?url=http://search.ebscohost.com/login.aspx?direct=true&db=pdh&AN=1999-11644-001&site=ehost-live&scope=site). doi: 10.1037/0003-066X.54.10.805.

Clarke, Philippa, Jeffrey Morenoff, Michelle Debbink, Ezra Golberstein, Michael R. Elliott and Paula M. Lantz. 2014. "Cumulative Exposure to Neighborhood Context Consequences for Health Transitions Over the Adult Life Course." *Research on Aging* 36(1):115-142.

Cohen, Sheldon, Ronald C. Kessler and Lynn U. Gordon. 1997. *Measuring Stress: A Guide for Health and Social Scientists.*Oxford University Press.

Cohen, Sheldon and Gail M. Williamson. 1991. "Stress and Infectious Disease in Humans." *Psychological Bulletin* 109(1):5.

Cohen, S., D. Janicki-Deverts, W. J. Doyle, G. E. Miller, E. Frank, B. S. Rabin and R. B. Turner. 2012. "Chronic Stress, Glucocorticoid Receptor Resistance, Inflammation, and Disease Risk." *Proceedings of the National Academy of Sciences of the United States of America* 109(16):5995-5999. doi: 10.1073/pnas.1118355109 [doi].

DeLancey, J. O., M. J. Thun, A. Jemal and E. M. Ward. 2008. "Recent Trends in Black-White Disparities in Cancer Mortality." *Cancer Epidemiology, Biomarkers & Prevention : A Publication of the American Association for Cancer Research, Cosponsored by the American Society of Preventive Oncology* 17(11):2908-2912. doi: 10.1158/1055-9965.EPI-08-0131 [doi].

Dietz, W. H. 1994. "Critical Periods in Childhood for the Development of Obesity." *The American Journal of Clinical Nutrition* 59(5):955-959.

Diez-Roux, A. V., F. J. Nieto, C. Muntaner, H. A. Tyroler, G. W. Comstock, E. Shahar, L. S. Cooper, R. L. Watson and M. Szklo. 1997. "Neighborhood Environments and Coronary Heart Disease: A Multilevel Analysis." *American Journal of Epidemiology* 146(1):48-63.

Drentea, Patricia and John R. Reynolds. 2014. "Where does Debt Fit in the Stress Process Model?" *Society and Mental Health* 5(1):16-32.

Epel, Elissa S., Jue Lin, Frank H. Wilhelm, Owen M. Wolkowitz, Richard Cawthon, Nancy E. Adler, Christyn Dolbier, Wendy B. Mendes and Elizabeth H. Blackburn. 2006. "Cell Aging in Relation to Stress Arousal and Cardiovascular Disease Risk Factors." *Psychoneuroendocrinology* 31(3):277-287.

Epel, E. S. 2009. "Psychological and Metabolic Stress: A Recipe for Accelerated Cellular Aging?" *Hormones (Athens, Greece)* 8(1):7-22.

Epel, E. S. and G. J. Lithgow. 2014. "Stress Biology and Aging Mechanisms: Toward Understanding the Deep Connection between Adaptation to Stress and Longevity." *The Journals of Gerontology.Series A, Biological Sciences and Medical Sciences* 69 Suppl 1:S10-6. doi: 10.1093/gerona/glu055 [doi].

Evans, G. W. and P. Kim. 2007. "Childhood Poverty and Health: Cumulative Risk Exposure and Stress Dysregulation." *Psychological Science* 18(11):953-957. doi: PSCI2008 [pii].

Finch, Caleb E. 2010. *The Biology of Human Longevity:: Inflammation, Nutrition, and Aging in the Evolution of Lifespans.*Academic Press.

Flores, Glenn, Lynn Olson and Sandra C. Tomany-Korman. 2005. "Racial and Ethnic Disparities in Early Childhood Health and Health Care." *Pediatrics* 115(2):e183-e193.

Franks, Peter, Peter Muennig, Erica Lubetkin and Haomiao Jia. 2006. "The Burden of Disease Associated with being African-American in the United States and the Contribution of Socio-Economic Status." *Social Science & Medicine* 62(10):2469-2478.

Gee, Gilbert C., Katrina M. Walsemann and Elizabeth Brondolo. 2012a. "A Life Course Perspective on how Racism may be Related to Health Inequities." *American Journal of Public Health* 102(5):967-974.

------. 2012b. "A Life Course Perspective on how Racism may be Related to Health Inequities." *American Journal of Public Health* 102(5):967-974.

Geronimus, Arline T., Margaret T. Hicken, Jay A. Pearson, Sarah J. Seashols, Kelly L. Brown and Tracey D. Cruz. 2010a. "Do US Black Women Experience Stress-Related Accelerated Biological Aging?" *Human Nature* 21(1):19-38.

------. 2010b. "Do US Black Women Experience Stress-Related Accelerated Biological Aging?" *Human Nature* 21(1):19-38.

Geronimus, Arline T., Margaret Hicken, Danya Keene and John Bound. 2006. "“Weathering” and Age Patterns of Allostatic Load Scores among Blacks and Whites in the United States." *Journal Information* 96(5).

Glaser, Ronald and Janice K. Kiecolt-Glaser. 2005. "Stress-Induced Immune Dysfunction: Implications for Health." *Nature Reviews Immunology* 5(3):243-251.

Gruenewald, T. L., T. E. Seeman, C. D. Ryff, A. S. Karlamangla and B. H. Singer. 2006. "Combinations of Biomarkers Predictive of Later Life Mortality." *Proceedings of the National Academy of Sciences of the United States of America* 103(38):14158-14163. doi: 0606215103 [pii].

Hayward, Mark D., Toni P. Miles, Eileen M. Crimmins and Yu Yang. 2000. "The Significance of Socioeconomic Status in Explaining the Racial Gap in Chronic Health Conditions." *American Sociological Review*:910-930.

Houle, Jason N. 2014. "Mental Health in the Foreclosure Crisis." *Social Science & Medicine* 118:1-8.

Jackson, James S., Katherine M. Knight and Jane A. Rafferty. 2010. "Race and Unhealthy Behaviors: Chronic Stress, the HPA Axis, and Physical and Mental Health Disparities Over the Life Course." *American Journal of Public Health* 100(5):933-939.

Jones, C. P. 2000. "Levels of Racism: A Theoretic Framework and a Gardener's Tale." *American Journal of Public Health* 90(8):1212-1215.

Keenan, Nora L. and Kate M. Shaw. 2011. "Coronary Heart Disease and Stroke Deaths-United States, 2006." *MMWR Surveill Summ* 60(Suppl):62-66.

Mason, David. 1994. "On the Dangers of Disconnecting Race and Racism." *Sociology* 28(4):845-858.

McClintock, Martha K., Suzanne D. Conzen, Sarah Gehlert, Christopher Masi and Funmi Olopade. 2005. "Mammary Cancer and Social Interactions: Identifying Multiple Environments that Regulate Gene Expression Throughout the Life Span." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 60(Special Issue 1):32-41.

McEwen, Bruce S. 1998. "Stress, Adaptation, and Disease: Allostasis and Allostatic Load." *Annals of the New York Academy of Sciences* 840(1):33-44.

McEwen, Bruce S. and Eliot Stellar. 1993. "Stress and the Individual: Mechanisms Leading to Disease." *Archives of Internal Medicine* 153(18):2093-2101.

Pager, D. and H. Shepherd. 2008. "The Sociology of Discrimination: Racial Discrimination in Employment, Housing, Credit, and Consumer Markets." *Annual Review of Sociology* 34:181-209.

Pearlin, Leonard I. 1989. "The Sociological Study of Stress." *Journal of Health and Social Behavior* 30(3):241-256.

Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman and Joseph T. Mullan. 1981. "The Stress Process." *Journal of Health and Social Behavior* 22(4):337-356.

Rewak, Marissa, Stephen Buka, Jennifer Prescott, Immaculata De Vivo, Eric B. Loucks, Ichiro Kawachi, Amy L. Non and Laura D. Kubzansky. 2014. "Race-Related Health Disparities and Biological Aging: Does Rate of Telomere Shortening Differ Across Blacks and Whites?" *Biological Psychology* 99:92-99.

Romeo, Russell D. 2010. "Adolescence: A Central Event in Shaping Stress Reactivity." *Developmental Psychobiology* 52(3):244-253.

Saliques, Sébastien, Marianne Zeller, Julie Lorin, Luc Lorgis, Jean-Raymond Teyssier, Yves Cottin, Luc Rochette and Catherine Vergely. 2010. "Telomere Length and Cardiovascular Disease." *Archives of Cardiovascular Diseases* 103(8):454-459.

Schnittker, Jason and Jane D. McLeod. 2005. "The Social Psychology of Health Disparities." *Annual Review of Sociology*:75-103.

Shuey, Kim M. and Andrea E. Willson. 2008. "Cumulative Disadvantage and Black-White Disparities in Life-Course Health Trajectories." *Research on Aging* 30(2):200-225.

Taylor, Paul, Rakesh Kochhar, Richard Fry, Gabriel Velasco and Seth Motel. 2011. "Wealth Gaps Rise to Record Highs between Whites, Blacks and Hispanics." *Pew Research Center, Social and Demographic Trends*.

Thoits, Peggy A. 1983. "Multiple Identities and Psychological Well-being: A Reformulation and Test of the Social Isolation Hypothesis." *American Sociological Review*:174-187.

------. 1995. "Stress, Coping, and Social Support Processes: Where are we? what Next?" *Journal of Health and Social Behavior*:53-79.

Turner, R. J. 2013a. "Understanding Health Disparities the Relevance of the Stress Process Model." *Society and Mental Health*:2156869313488121.

------. 2013b. "Understanding Health Disparities the Relevance of the Stress Process Model." *Society and Mental Health*:2156869313488121.

Turner, R. J., Blair Wheaton and Donald A. Lloyd. 1995. "The Epidemiology of Social Stress." *American Sociological Review*:104-125.

Turney, K., H. Lee and M. Comfort. 2013. "Discrimination and Psychological Distress among Recently Released Male Prisoners." *American Journal of Men's Health* 7(6):482-493. doi: 10.1177/1557988313484056 [doi].

Walsemann, K. M., G. C. Gee and A. T. Geronimus. 2009. "Ethnic Differences in Trajectories of Depressive Symptoms: Disadvantage in Family Background, High School Experiences, and Adult Characteristics." *Journal of Health and Social Behavior* 50(1):82-98.

Wheaton, Blair. 1994. "Sampling the stress universe." Pp. 77-114 in *Stress and mental health.*"Sampling the stress universe."Springer.

Williams, David R. and Selina A. Mohammed. 2009. "Discrimination and Racial Disparities in Health: Evidence and Needed Research." *Journal of Behavioral Medicine* 32(1):20-47.

Williams, David R., Selina A. Mohammed, Jacinta Leavell and Chiquita Collins. 2010. "Race, Socioeconomic Status, and Health: Complexities, Ongoing Challenges, and Research Opportunities." *Annals of the New York Academy of Sciences* 1186(1):69-101.

Williams, D. R. and C. Collins. 2001. "Racial Residential Segregation: A Fundamental Cause of Racial Disparities in Health." *Public Health Reports (Washington, D.C.: 1974)* 116(5):404-416.

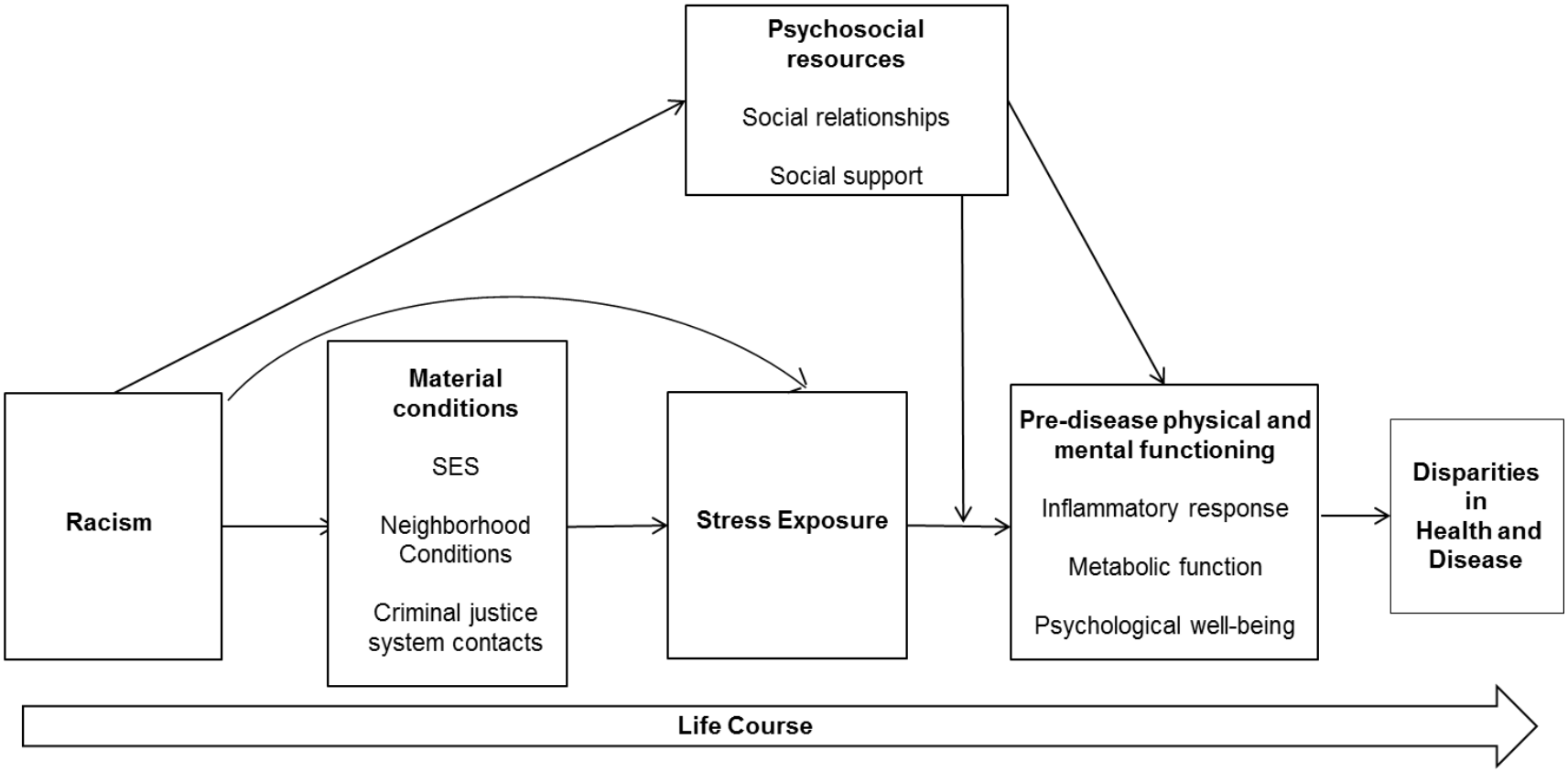
Williams, D. R. and P. B. Jackson. 2005. "Social Sources of Racial Disparities in Health." *Health Affairs (Project Hope)* 24(2):325-334. doi: 24/2/325 [pii].

Williams, D. R., H. W. Neighbors and J. S. Jackson. 2003. "Racial/Ethnic Discrimination and Health: Findings from Community Studies." *American Journal of Public Health* 93(2):200-208.

Yang, Y. C., C. Boen, K. Gerken, T. Li, K. Schorpp and K. M. Harris. 2016. "Social Relationships and Physiological Determinants of Longevity Across the Human Life Span." *Proceedings of the National Academy of Sciences of the United States of America* 113(3):578-583. doi: 10.1073/pnas.1511085112 [doi].

Yang, Y. C., C. Boen and K. Mullan Harris. 2015. "Social Relationships and Hypertension in Late Life: Evidence from a Nationally Representative Longitudinal Study of Older Adults." *Journal of Aging and Health* 27(3):403-431. doi: 10.1177/0898264314551172 [doi].

Figure 1.1. Conceptual Model



CHAPTER 2: RACE, CRIMINAL JUSTICE CONTACTS, AND HEALTH: STRESS-RELATED DISPARITIES IN THE CARCERAL STATE

Abstract

Despite a growing body of literature examining the role of the criminal justice system in social and racial inequality broadly, whether and how contacts with the criminal justice system affect individual health and contribute to racial health disparities remains relatively unexplored. Utilizing nationally representative data from the National Longitudinal Study of Adolescent to Adult Health (N=5,503) and a variety of analytic techniques—including multivariate regression models, a unique treatment-control design, and treatment weighting procedures—this study examines how contacts along the criminal justice continuum impact pre-disease, stress-related markers of physiological functioning and psychological well-being and ultimately contribute to Black-White disparities in health early in the life course. Findings reveal that, relative to individuals reporting no contact with the criminal justice system, individuals who have been stopped and/or arrested by police are at increased mental health risk. I also find that Black-White differences in criminal justice contacts account for a significant portion of racial health gaps, net of a host of potential confounders such as delinquency and family and neighborhood socioeconomic conditions. In addition, results offer evidence of a potentially casual association between incarceration and health, with formerly incarcerated individuals having higher levels of physiological inflammation and mental health risk than never-incarcerated individuals. Situating these results in a particular historical moment characterized by increasing levels of police surveillance and mass incarceration, findings from this study highlight the essential nature of the

criminal justice system in patterning population health disparities, generally, and racial health inequities, specifically.

Introduction

On the evening of February 26, 2012, Trayvon Martin—a 17 year old Black boy—was shot and killed while walking back to his father's home in Sanford, Florida after a trip to a local convenience store to buy Skittles and iced tea. George Zimmerman, the man who killed Martin, was a neighborhood watch coordinator working towards a degree in criminal justice. Despite the fact that Martin was not armed, Zimmerman was acquitted of Martin's murder on self-defense grounds. A little more than two years later and hundreds of miles away, Darren Wilson, a White police officer in Ferguson, Missouri, fired twelve shots at Michael Brown, an unarmed Black teenager who had graduated high school just eight days earlier. Eye witnesses claimed Brown had his hands in the air at the time when Wilson killed him. Despite public outcries and demands for justice, a grand jury decided not to indict Wilson, and the U.S. Department of Justice later cleared Wilson of civil rights violations in the shooting. Though distinct, these two eerily similar events in many ways reflected a disturbingly familiar, decades old problem: the disproportionate targeting and killing young Blacks by the criminal justice system. Though Trayvon Martin and Michael Brown were not the first unarmed teens to be killed by individuals acting on behalf of community safety and criminal justice institutions, many point to their deaths—and the subsequent acquittals of their killers—as sparking and crystalizing a national movement aimed at shedding light on and resisting anti-Black racism, particularly as it relates to the criminal justice system (Rickford 2016).

Though its focus extends beyond criminal justice reform, the Black Lives Matter movement, in many ways, served to bring new media and activist attention to stark racial differences in criminal justice contacts and encounters. Racial disparities exist across the

criminal justice continuum—from police stops, to arrests, convictions, and incarceration. Blacks are more likely than Whites to be stopped by police; when they are stopped, they are more likely to be arrested; when they are arrested, they are more likely to be convicted; and when they are convicted, they are more likely to face long prison sentences. Aggressive policing practices such as "stop and frisk," combined with officers' racially biased use of discretion, result in stark disparities in police encounters. For example, data from New York City's "Stop and Frisk" program revealed that, of the more than 685,000 police stops in 2011, nearly 90 percent of individuals who were stopped by police were Black or Hispanic; it is also worth noting that 88 percent of individuals stopped were not guilty of committing a crime (New York Civil Liberties Union 2016). Blacks also face higher arrest rates for various types of crimes than Whites, including drug-related crimes, and these disparities cannot be explained by racial differences in socioeconomic status, criminal offending, or residing in neighborhoods with heavy police presence (Mitchell and Caudy 2015). Further, compared to Whites charged with similar offenses, Blacks are more likely to be convicted, and they also receive sentences that are approximately 10 percent longer than Whites, even after adjusting for potential confounders such as arrest history and severity of the crime (Starr and Rehavi 2012).

Further, evidence also suggests that Blacks experience categorically different contacts with police and other criminal justice officials than Whites, categorized by higher levels of hostility, harassment, and violence (Browning et al. 1994; Brunson and Miller 2006; Brunson 2007). For example, in a study of Black young men in Chicago, Brunson (2007) found that more than 80 percent of study participants reported having been harassed by police. Other studies have also documented staggering racial disparities in reports of police harassment and misconduct (Browning et al. 1994). Blacks report high levels of frustration with the hostility they faced in

the context of police encounters, where they were often ordered to sit on lie on the pavement while being physically searched (Brunson 2007). Regarding the experience of incarceration, data from several states suggests that Black prisoners in the United States are more likely than Whites experience solitary confinement (Schlanger 2012), indicating that the time Blacks spend in prisons is quite different than White experiences with incarceration. In this way, then, Blacks not only face more exposure to criminal justice institutions than Whites, but the nature of their contacts is, in many ways, characteristically more hostile, stressful, and threatening than Whites' experiences.

In addition to stark racial disparities in criminal justice contacts, scholars have also documented the increasing role of the criminal justice system in the lives of Americans, more broadly. Research confirms an increase in aggressive policing and surveillance practices (Alexander 2012; Kraska 2007; Parenti 2000), including policing programs such as "stop and frisk," "broken windows," and police paramilitary units. Further, since the 1960s, the number of incarcerated individuals has increased eightfold, and there are currently more than 2.2 million people throughout prisons and jails in the United States (The Sentencing Project 2017). Together, these related processes—the increased use of aggressing policing and surveillance practices and rising levels of mass incarceration—have meant that the lives of an increasing number of Americans have been touched, either directly or indirectly, by the criminal justice system.

Given the documented role of the criminal justice system in determining individual life chances, particularly those of young Blacks (Pager 2003; Pettit and Western 2004; Wildeman and Western 2010), a relatively small but growing body of research has linked criminal justice contacts to health. Much of the literature in this area has focused on the relationship between

incarceration and health. In general, these studies document that, while there may be short-term protective benefits associated incarceration (Spaulding et al. 2011), the health improvements associated with improved health care access within the confines of prisons and jails generally do not translate into long-term health benefits for formerly incarcerated individuals (Wakefield and Uggen 2010). In fact, research indicates that formerly incarcerated individuals have higher rates of morbidity from an array of conditions, including hypertension (Massoglia 2008), sexually transmitted infections (Hammett, Harmon, and Rhodes 2002), and depression (Schnittker and John 2007), as well as higher rates of overall mortality (Spaulding et al. 2011).

A smaller number of recent studies have extended their analyses of the health effects of criminal justice contacts beyond incarceration to also include police stops and arrests. For example, using data from New York City, Geller et al. (2014) found that individuals who reported more contact with police also reported higher levels of anxiety and trauma. Similarly, Sewell, Jefferson, and Lee (2016) found that living in a neighborhood context characterized by aggressive policing and surveillance practices was associated with men's mental health risk.

Together, the literature on criminal justice contacts and health suggests that individuals reporting more contacts with the criminal justice system—whether in the forms of police stops, arrests, or incarceration—are at increased health risk and that the association between criminal justice contacts and health may be mediated by stress-related mechanisms. Applying Pearlin's (1981) stress process model to criminal justice contacts, the encounters and experiences with police, courts, jails, and prisons may serve as primary stressors, upregulating physiological stress response and increasing individual levels of fear and anxiety in the short-term. However, the stress related to these contacts may extend beyond the actual contact by giving rise to a host of secondary stressors, such as difficulties securing employment (Pager 2003) and battling stigma

(Schnittker 2014; Schnittker and John 2007). Together, the primary and secondary stress associated with criminal justice contacts may serve as both acute and chronic stressors in the lives of individuals, promoting physiological and psychological malfunctioning, increasing allostatic load, and raising disease and mortality risk. Still, because few studies test these claims, the role of the criminal justice system in population health disparities remains largely unexplored, leaving critical questions unanswered.

Using nationally-representative data and a variety of analytic techniques, this study aims to advance understanding of the role of the criminal justice system in population health by examining the physiological and psychological mechanisms through which police stops, arrests, convictions, and incarceration impact health risk. Further, by assessing how Black-White differences in criminal justice encounters contribute to racial health disparities, this study offers new evidence of a link between structural racial inequality, policing practices, mass incarceration, and health.

Background

Gaps in the Literature

Despite the recent up-tick in scholarly attention to the role of the criminal justice system in population health inequality, critical gaps in the literature remain. First, few studies examine how criminal justice contracts prior to incarceration impact health and disease risk. The literature identifies stress as a key mechanism underlying the association between incarceration and health (Massoglia 2008; Porter 2014; Schnittker 2014; Schnittker and John 2007), yet it remains largely unknown whether and how the primary and secondary stressors associated with police stops, arrests, and convictions impact individual health and well-being. Research documents, however, that police stops and searches, arrests, and other contacts with the criminal justice system are extremely stressful experiences for individuals, characterized by high levels of anxiety, fear, and

frustration (Brunson and Miller 2006; Brunson 2007; Geller et al. 2014; Smith, Allen, and Danley 2007). In this way, pre-incarceration contacts with the criminal justice system may serve to upregulate physiological stress response systems to ultimately impact health and contribute to racial disparities in health and well-being.

Second, because most studies assess how incarceration affects disease outcomes, questions regarding the physiological and psychological mechanisms underlying the link between criminal justice contacts and health remain. Of the studies that examine the health effects of incarceration, many utilize infectious disease outcomes by focusing on the spread of infections in jails and prisons (Braun et al. 1989; Oppong et al. 2014; Stark et al. 1997; Vescio et al. 2008), while fewer examine how incarceration, specifically, and criminal justice contacts, broadly, affect chronic disease risk. Further, most studies of the health effects of criminal justice contacts utilize measures of disease or diagnosis as outcomes, which may result in misclassification error, as individuals who do not yet have the disease or have not yet been diagnosed with the disease are classified as "well" (Turner 2013a). Research utilizing pre-disease markers of mental health and physiological functioning may improve understanding of how contacts with the criminal justice system "get under the skin" to affect health and disease risk, particularly through stress-related processes.

Third, while life course researchers have highlighted the importance of the timing and duration of exposures for health (Pavalko and Willson 2011), few studies of criminal justice contacts and health assess whether and how the age at which someone is incarcerated or the duration of incarceration may impact health. To date, only a handful of studies have examined how sentence length impacts health, and most studies indicate that exposure to incarceration matters more for health than the duration of incarceration (Schnittker and John 2007). Further,

there is a lack of studies assess whether or how the age at which one is incarcerated affects health, despite a wide body of evidence indicating that the life course timing of exposures has tremendous implications for health (Ben-Shlomo and Kuh 2002). Research suggests that the transition to adulthood may be a particularly sensitive period for health, during which life-altering experiences such as incarceration may have particularly critical effects on future trajectories of mental and physical risk (Schulenberg, Sameroff, and Cicchetti 2004), and yet this type of life course examination is absent from the literature on incarceration and health. It is quite possible, however, that whether one first incarcerated during adolescence, the transition to adulthood, or later on in life would have varying consequences for well-being. Still, questions regarding the life course timing and duration of incarceration remain to be adequately addressed, particularly as these life course processes may vary across markers of physical and mental health.

Finally, a major challenge to research on the consequences of criminal justice contacts, broadly, and incarceration, in particular, is causal inference. Even models with extensive adjustment for factors endogenous to health and imprisonment (such as criminality or drug use) do not provide convincing evidence of a causal relationship between arrest, imprisonment, release, and health (Wildeman 2011). Critics argue that many of the same measured and unmeasured factors that increase individual risk for criminal justice system involvement—such as coming from a disadvantaged family or neighborhood, having a higher propensity for delinquency and risk-taking, or having mental health problems—are also key drivers of health. In this way, any observed relationship between criminal justice contacts and health may reflect underlying differences between those reporting no contacts and those reporting a history of arrests, convictions, and incarceration, rather than reflecting any particular health effects of the

criminal justice contact (Porter 2014). Research utilizing treatment weighting procedures or treatment-control designs, while not fully able to account for endogenous factors that affect both propensity for criminal justice contacts and health, may help in obtaining less biased estimates and improve causal inference.

Research Questions

This study provides new evidence of the links between the criminal justice system and population health by examining how contacts along the criminal justice continuum—from police stops through incarceration—impact pre-disease, stress-related markers of physiological functioning and indicators of mental health and ultimately contribute to Black-White health disparities. Utilizing longitudinal data and a variety of analytic techniques—including multivariate regression models, a unique treatment-control design, and treatment weighting procedures—this study provides a robust assessment of the role of the criminal justice system in producing stress-related disparities in population health, with a particular focus on the role of criminal justice contacts in Black-White health inequities. In particular, this study addresses three overarching research questions:

1) How are contacts with the criminal justice system—including police stops, arrests, convictions, and incarceration—associated with pre-disease markers of physical and mental health?
2) Does the life course timing and duration of incarceration matter for these markers of health?
3) Are Black-White differences in criminal justice contacts determinants of racial health disparities?

Data and Methods

Data and Sample

Data for this study come from the National Longitudinal Study of Adolescent to Adult Health (Add Health) (1994-2009), which is a nationally representative, longitudinal study of U.S. adolescents. Using a school-based complex cluster sampling frame, Add Health began in 1994-95 with an in-school questionnaire administered to a nationally-representative sample of students in grades 7-12. Following the in-school questionnaire, a gender- and grade-stratified random sample of 20,745 adolescents (79% response rate) was selected for in-home interviews at Wave I. The study then followed up with a series of in-home interviews conducted in 1996 (Wave 2; 88% response rate), 2001-02 (Wave 3; 77% response rate), and 2007-08 (Wave 4; 80% response rate).

In addition to the questionnaires and interviews, Add Health also collected biological specimens from study participants at Wave IV. The collection of physical measurements, saliva samples and dried blood spots allows researchers to better understand the linkages between respondents' social lives and their cardiovascular, metabolic, and immunologic functioning

This study utilizes data from the in-home interviews at Waves I, III, and IV and the biomarker collection at Wave IV. I also utilize Census tract-level data linked to respondents' residences at Wave I and IV.

Measures

Outcomes

The outcome measures for this study include two pre-disease measures of physiological functioning and psychological well-being that have been linked to stress. C-reactive protein (CRP) is an acute phase protein produced by the liver, and elevated circulating levels of CRP indicates inflammation (Finch 2010b). Studies document a relationship between inflammation

and health risk, including prospective associations of CRP with higher rates of coronary heart disease, stroke, and mortality (Emerging Risk Factors Collaboration 2010; Harris et al. 1999; Ridker et al. 2000). Because of a skewed distribution, the measure of CRP is log transformed.

This study also utilizes a count of depressive symptoms based on the Center for Epidemiological Studies Depression Scale (CES-D). In addition to being highly predictive of future major depression (Pine et al. 1999), depressive symptoms have also been linked to declines in physical health (Kiecolt-Glaser and Glaser 2002; Penninx et al. 1998). Research suggests that depressive states can directly stimulate the production of physiological stress response systems, which in turn influences a host of diseases and conditions, including cardiovascular disease, diabetes, and cancer (Kiecolt-Glaser and Glaser 2002). Further, depressive states can also down-regulate the body's immune function, making individuals with high levels of depressive symptoms more prone to prolonged infection and delayed wound healing (Kiecolt-Glaser and Glaser 2002). Together, the outcomes of CRP and depressive symptoms offer complimentary insights into how the stress associated with criminal justice system contacts affect physiological functioning, psychological well-being, and future disease risk.

Criminal Justice Contacts

The key exposures in this study relate to contacts with the criminal justice system. First, I conduct analyses that include contacts along the criminal justice continuum, from police stops through incarceration. To do this, I construct a categorical measure where 0=no history of criminal justice contact, 1=stopped by police but not arrested, 2=arrested but not convicted, 3=convicted but not incarcerated, and 4=formerly incarcerated. These categories are mutually exclusive.

Second, I focus explicitly on the relationship between incarceration and health, where the key independent variable is incarceration history, indicated by a dummy variable where 1=ever having been incarcerated. I also utilize categorical measures indicating age at first incarceration and duration of incarceration. Age at first incarceration is indicated by a measure indicating whether the first experience of incarceration occurred before or after the age of 18 (0=never incarcerated; 1=incarcerated before age 18 years; 2=incarcerated after age 18 years). Duration of incarceration indicates whether the individual was incarcerated for more or less than one year (0=never incarcerated; 1=incarcerated for less than one year; 2=incarcerated for more than one year). Supplementary analyses with alternative operationalizations utilizing different age and duration cutoffs produced substantively similar results.

Other Measures

Given that this study assesses the roles of criminal justice contacts in racial health disparities, I include a measure of respondent race to indicate the magnitude of the racial gaps in the outcomes (1=Black). In addition to sociodemographic characteristics such as age and gender, I also include a host of baseline characteristics that may act as confounders, including family SES in adolescence, neighborhood economic disadvantage in adolescence, neighborhood racial composition in adolescence, mental health in adolescence, delinquency, whether the respondent is a repeat arrestee, and whether the respondent was arrested for a violent crime. All background covariates are measured at Wave I. I also adjust for respondent education, which is measured at Wave IV. More detail on the coding of all measures can be found in Table 2.1.

Analytic Strategy

Descriptive Analyses

I begin my analysis with descriptive analyses, paying particular attention to racial disparities in CRP, depressive symptoms, and contacts with the criminal justice system.

Criminal Justice Contacts and Health

To examine whether and how contacts along the criminal justice continuum affect markers of physical and mental well-being, I use multivariate regression models. I model log CRP using OLS regression models, and I model depressive symptoms using negative binomial regression models, as the depressive symptom outcome is an over-dispersed count variable. In these models, I regress the outcomes on the categorical measure indicating contacts with the criminal justice system, adjusting for the other covariates in a stepwise fashion. Model 1 is a model of the racial disparity in the outcomes that controls for age and gender. Model 2 builds on Model 1 by also including the measure of level of contact with the criminal justice system. Model 3 is the fully adjusted model that includes the full set of covariates. To assess the extent to which contacts with the criminal justice system help to explain racial disparities in the outcomes, I conduct Sobel-Goodman mediation tests which determine the proportion of the Black-White gaps in the outcomes mediated or "explained away" by contacts with the criminal justice system.

Incarceration and Health

In order to advance understanding of the potentially causal impact of incarceration on physical and mental health, this study also includes a multi-stage analysis of the links between incarceration history and CRP and depressive symptoms. First, I examine the associations between incarceration and the outcomes using multivariate regression models (OLS models for the continuous log CRP models and negative binomial models for depressive symptoms). This first set of models regresses the outcomes on incarceration history, age of incarceration, and duration of incarceration in a stepwise fashion while adjusting for the covariates in a traditional multivariate regression framework. I use this analytic approach to capture a baseline association between incarceration and health utilizing the analytic techniques typically employed by studies of incarceration and health.

In the second stage of the analysis, I again use OLS and negative binomial regression to determine the associations between incarceration history and the outcomes, but I limit my sample to strategic "treatment-control" groups. As discussed earlier, isolating the health effects of incarceration can be difficult because of issues related to endogeneity and selection bias. For this reason, the selection of a strategically appropriate comparison group is critical. In this second stage of the incarceration analysis, I use individuals who have been arrested and convicted, but not incarcerated, as the comparison group. The approach of using convicted but not incarcerated individuals as a comparison group in models attempting to isolate the effects of incarceration has been lauded (Massoglia and Warner 2011) and results using this approach prove promising. For example, a recent study by Apel and Sweeten (Apel and Sweeten 2010) used data on individuals convicted of a crime—only some of whom were then incarcerated—to isolate the effect of imprisonment on employment and documented a strong effect of incarceration on employment difficulties for individuals who were incarcerated. Another study by Porter (2014) used a similar approach to demonstrate higher rates of fast food consumption and a higher likelihood of smoking among formerly incarcerated individuals compared to convicted but not incarcerated survey respondents. In this stage of the analysis, I compare the outcomes of individuals who were convicted but not incarcerated to those who were incarcerated in a multivariate regression framework. Because individuals who have been convicted but not incarcerated are "closest to the incarceration decision" (Apel and Sweeten 2010: 454), they are a particularly useful control group.

While restricting the analytic sample to respondents to those who have been convicted of a crime may minimize unmeasured heterogeneity, there may still be systematic differences between those who were never incarcerated and those who have spent time in a prison or jail. In

order to further account for potential differences between the “treated” and “untreated” groups, the third stage of the incarceration analysis still utilizes the restricted “treatment-control” groups but also introduces inverse probability of treatment weighting (IPTW) with regression adjustment. IPTW requires calculating a propensity score for respondents, which is the probability of incarceration (including the timing and duration of incarceration) conditional on a set of covariates (Rosenbaum and Rubin 1983). IPTW then uses the calculated propensity scores to weight observations to create a new pseudo-population in which treatment is no longer confounded by the covariates. IPTW balances the treatment assignment across the covariates by giving more or less weight to respondents with covariate histories that are under- or over-represented in the “never incarcerated” and “formerly incarcerated” groups (Robins 1999; Robins, Hernan, and Brumback 2000). As a result, in this study, exposure to incarceration can behave as if it were randomized with respect to the observed covariates. Unlike propensity score matching strategies, which typically involve dropping a rather significant amount of unmatched observations, IPTW allows me to retain the full sample of convicted respondents used in the second stage of the analysis. In addition to IPTW, I also use a regression adjustment estimator. This strategy combines IPTW with regression modeling of the relationship between the covariates and outcomes. These models have the property of being doubly robust, such that as long as either the model for the treatment or the outcome is correctly specified, the estimate of the effect of incarceration on health will be unbiased and correctly estimated.

Analytic Samples

For the analyses that employs OLS and negative binomial regression models, analytic samples include all respondents with complete data on the variables used in the analyses, as well as valid sampling weights. Sample sizes vary by outcome and by key explanatory variable: log CRP and all criminal justice contacts (N=4,987); log CRP and incarceration (N=4,603);

depressive symptoms and all criminal justice contacts (N=5,503); and depressive symptoms and incarceration (N=5,077). It is worth noting that individuals who were incarcerated at Wave IV, when the outcomes were assessed, were not included in the analysis. Further, Add Health was not able to interview or survey individuals who dropped out prior to the initial wave of data collection, and research suggests that these individuals may be particularly susceptible to police stops, arrests, and incarceration (Lochner and Moretti 2004; Pettit and Western 2004; Thornberry, Moore, and Christenson 1985). In these ways, the results presented here may be conservative.

For the models that use the restricted "treatment-control" groups to isolate the impact of incarceration on health, I further restrict the sample to respondents who have been convicted of a crime. Excluding individuals who were missing data on the variables used in the analyses yields a total of 1,038 respondents who were ever convicted of a crime. Of these respondents, 356 (34.30 percent) were convicted but not incarcerated; 682 (65.70 percent) were convicted and incarcerated. Because of missing data on the outcomes, analytic sample sizes for these analyses vary by outcome: CRP (N=740) and depressive symptoms (N=813).

Results

Descriptive Statistics

Table 2.2 presents descriptive statistics for the outcomes and key explanatory variables by race. As seen in Table 2.1, in young adulthood, Blacks have both higher levels of CRP (p=0.016) and depressive symptoms (p<0.001) than Whites. In addition, Blacks are also more likely than Whites to have been arrested or incarcerated.

Criminal Justice Contacts and Health

While I find no associations between criminal justice contacts and CRP, I find that criminal justice contacts are associated with depressive symptoms, such that increasingly severe

contacts are related to greater mental health risk. Table 2.3 presents a summary of the results from models regressing depressive symptoms on the categorical measure indicating level of contact with the criminal justice system. First, I find that including the measure of criminal justice contact in Model 2 reduces the racial disparity in depressive risk over Model 1. Further, across both Models 2 and 3 I find that police stops, arrests, and incarceration are associated with increased depressive risk. As shown in Figure 2.1, increased levels of contact with the criminal justice system predict depressive symptoms in a progressive manner ($p<0.001$). Compared to individuals reporting no contact with the criminal justice system, individuals who have been stopped by police report higher levels of depressive symptoms. Similarly, being arrested is associated with worse mental health, compared to individuals with no contact and individuals who have been stopped but not arrested. Of all groups, formerly incarcerated individuals have the highest levels of depressive symptoms. Results of the Sobel-Goodman mediation tests revealed that racial differences in contacts with the criminal justice system explained approximately 7 percent of the Black-White disparity in depressive symptoms ($p=0.001$), net of all of the covariates included in the fully adjusted models.

Incarceration and Health

For both outcomes, the analysis of the association between incarceration and health includes three stages of analysis: multivariate regression models with full analytic sample; multivariate regression models with “convicted only” treatment-control sample; and IPTW with regression adjustment utilizing “convicted only” treatment-control sample. For each stage of the analysis, I regress the outcomes on incarceration history (Models 1, 4, and 7), age at first incarceration (Models 2, 5, and 8), and duration of incarceration (Models 3, 6, and 9).

CRP

A summary of the results from the multi-stage analysis examining the associations between incarceration and log CRP are shown in Table 2.4. Beginning with incarceration history, across all stages of analysis I find a consistent relationship between exposure to incarceration and inflammation, such that formerly incarcerated individuals have higher levels of CRP than individuals who were never incarcerated. In fact, comparing the magnitudes and statistical significance of the coefficient estimates across Models 1, 4, and 7 reveals that, as I restrict the sample to only those convicted of a crime and implement treatment weighting procedures, the association between incarceration and inflammation is actually strengthened. Results from Model 7, which includes only respondents who were convicted of a crime and utilizes IPTW strategies, shows that, compared to individuals who were convicted of a crime but never incarcerated, individuals who were both convicted and incarcerated experience higher levels of inflammation ($\beta=0.22$, $p<0.01$). Figure 2.2 visually depicts the results from Model 7 of Table 2.3 and reveals a clear disparity in CRP between formerly incarcerated individuals and individuals who were convicted but not subsequently incarcerated.

Models 2, 5, and 8 of Table 2.4 assess whether the life course timing of incarceration impacts CRP. I find marginally significant evidence that being incarcerated after the age of 18 years is associated with higher levels of inflammation in Stage 1 of the analysis ($\beta=0.19$, $p<0.1$), but by restricting my sample to only those convicted of a crime in Models 6 and 9, the association between age at first incarceration and CRP is no longer significant.

Finally, Models 3, 6, and 9 of Table 2.4 assess the extent to which duration of incarceration affects CRP. Results from these models reveal that individuals who were incarcerated for one year or more have greater inflammatory risk than individuals who were

incarcerated for less than one year. By the third stage of the analysis, the association between age of incarceration and CRP is only marginally significant.

Depressive Symptoms

A summary of the results examining the associations between incarceration and depressive symptoms is presented in Table 2.5. Consistent with the CRP analyses, within each stage of the analysis, I regress depressive symptoms on incarceration history (Models 1, 4, and 7), age at first incarceration (Models 2, 5, and 8), and duration of incarceration (Models 3, 6, and 9). In Models 1 and 4, I document a significant association between incarceration history and mental health risk, where formerly incarcerated individuals report more depressive symptoms than individuals who had never been incarcerated. However, results from Model 7, which includes the IPTW strategy, indicate no significant relationship between exposure to incarceration and depressive risk.

In Models 2, 5, and 8, I examine whether the life course timing of incarceration affects mental health risk. Results from Models 2 and 5 offer only marginal evidence that the age of incarceration matters for health. Results from Model 8, which includes the restricted sample of convicted respondents and the treatment weighting procedures, suggest that the age at incarceration does not matter for depressive risk.

Finally, the last set of models (Models 3, 6, and 9) assesses whether the duration of incarceration impacts depressive risk. Across all three sets of models, I find that longer sentences are associated with worse mental health. In each of these models, sentences over one year are associated with higher levels of depressive symptoms. Results from Model 6 of Table 2.4 are graphically represented in Figure 2.3. Compared to individuals who were convicted of a crime but not incarcerated and individuals who were incarcerated for less than one year, Figure 2.3 reveals that individuals who were incarcerated for longer than one year had the greatest number

of depressive symptoms (p=0.028). While shorter sentences are not associated with more depressive symptoms, longer sentences significantly impact mental health risk across all three stages of analysis.

Discussion

A wide and growing body of literature has interrogated the role of the criminal justice system in producing racial disparities across a number of outcomes, including Black-White disparities in employment (Pager 2003; Pager 2008), political participation (Roberts 2004; Uggen and Manza 2002), and family formation (Huebner 2005; Western and Wildeman 2009). In recent years, scholars have begun to examine the ways in which the criminal justice institutions, and racial disparities in contacts with those institutions, may be also implicated in population health inequities. Despite growing interest in this area, research has been almost exclusively limited to examining the effects of incarceration on disease outcomes or health behaviors. Little attention has been given to how the duration and life course timing of incarceration may matter for health, and few studies have provided rigorous tests for potential causality. By extending analysis to pre-incarceration contacts with the criminal justice system, integrating life course concepts such as age and duration of exposure, utilizing pre-disease markers of biological and psychological well-being, and employing quasi-experimental and treatment weighting procedure designs, this study offers new insights into the how contacts with the criminal justice system serve as stress-inducing experiences that up-regulation physiological functioning and contribute to population health disparities.

This study offers four key contributions to our understanding of the role of the criminal justice system in population health and racial health disparities. First, the results presented here indicate that incarceration, specifically, and contacts with the criminal justice system, more broadly, are important drivers of population health and health disparities. Across the several

stages of analyses I presented, which included basic regression models, a unique treatment-control sample, and treatment weighting procedures, I document a consistent relationship between incarceration and health, where formerly incarcerated individual have higher levels of inflammation and depressive symptoms than individuals who were never incarcerated. This relationship persisted even after accounting for a wide range of background characteristics and accounting for selection by combining the restricted sample with treatment weighting procedures. While concerns about selection and causality have been consistently raised in the literature on incarceration and health, my findings suggest that using robust methods to account for possible selection only strengthens the relationship between incarceration and health, which speaks to the powerful influence that incarceration has on the lives and well-being of formerly incarcerated individuals. I also find that the health impacts of the criminal justice system extend beyond incarceration to include pre-incarceration contacts, including police stops and arrest. As seen in Table 2.3 and Figure 2.1, I find a gradient in the association between level of criminal justice contact and depressive risk, with police stops showing marginal associations with mental health risk, arrests showing moderate associations with health, and incarceration having the strongest relationship with depressive symptoms. In this way, findings from this study suggest that contacts with the criminal justice system are critical determinants of population disparities of physiological and psychological well-being. It is also worth noting the present study focuses on health early in the life course, when respondents were in their teens through their early thirties. In this way, the health effects of police stops, arrests, and incarceration emerge early in life and may play an important role in producing diverging trajectories of health as these individuals age.

Second, my findings suggest that stress is a potentially powerful mechanism linking criminal justice contacts with health. The outcomes in this study included markers of physical

and psychological well-being with documented links to the body's physiological stress response system. Scholars have hypothesized that the physical and emotional environments of jails and prisons—which are characterized by solitude, isolation, fear of crime and victimization—may irreparably alter the body's ability to maintain optimal health. The stigma and damage of social relationships that extends beyond one's sentence may serve to further exacerbate physiological and psychoemotional problems. My findings also suggest that stress may be implicated in the relationship between police stops, arrests, and health, with these pre-incarceration criminal justice contacts having unique associations with mental health risk, in particular. By including measures of perceived stress and emotional well-being, future research should build on the preliminary results offered here to further assess the role of stress in linking experiences with the criminal justice system to health.

Third, while studies rarely consider whether the timing or duration of incarceration matter for health, this study extends the literature by integrating these important life course concepts. I find little evidence that the timing of incarceration matters for inflammation or depressive risk. This may because the stigma, stress, and socioeconomic disadvantages associated with incarceration occur regardless of the age at which they are experienced. I do find, however, that duration of incarceration matters, particularly for depression risk, with sentences longer than one year being particularly harmful for health. These results suggest that one's psychological state may be better able to recuperate following shorter sentences, but longer stays in correctional facilities may irreparably harm one's mental state and outlook for the future.

Finally, my analyses revealed that Blacks were more likely than Whites to report contacts with the criminal justice system, particularly in the form of arrests and incarceration, and that their higher rates of criminal justice contact put them at increased health risk. Descriptive

analyses presented in Table 2.1 show that Blacks were more likely than Whites to be arrested and convicted of a crime. Further, Sobel-Goodman mediation tests revealed that these racial differences in criminal justice contacts are critical drivers of Black-White disparities in depressive symptoms, in particular, as accounting for racial disparities in police stops, arrests, convictions, and incarceration accounted for approximately 7 percent of the Black-White gap in depressive risk. Together, these findings indicate that the criminal justice system is not only a driver of social and racial inequality in a general sense, but that it is also an essential determinant of racial health disparities. While the literature suggests that Blacks' interactions with the criminal justice system are more hostile, threatening, and violent than Whites' (Browning et al. 1994; Brunson and Miller 2006; Brunson 2007), supplementary analyses including interaction terms for race-by-level of contact and race stratified models did not indicate any evidence of differential vulnerability. Further, I did not find any evidence that racial differences in incarceration alone helped to explain away Black-White differences in the outcomes. This may because of relatively small samples or because incarceration is still a relatively rare experience, particularly this early in the life course. Future research should expand incarceration-related analyses to examine how indirect experiences with incarceration, such as the incarceration of a family member, may be implicated in racial health disparities.

This study is not without limitations. First, while my incarceration analyses include a variety of techniques aimed at improving causal inference, the analysis of pre-incarceration contacts and health uses multivariate regression models, which are more vulnerable to issues of selection, endogeneity, and confounding. Because this study was among the first to examine how police stops, arrests, and convictions impact pre-disease markers of physiological and psychological functioning, this was a first step aimed at establishing a baseline association. More

research in this area is needed. Second, because of concerns related to sample size, I restricted my analyses to only Black and White survey respondents. However, the processes described in this study extend to other racial and ethnic groups. Again, this is an area of needed research. Finally, as longitudinal biomarker data becomes available, our ability to make causal claims about how criminal justice contacts affect health will only be improved.

Recent media and activist attention to the targeting and killing of young Black boys, girls, men, and women by police has served to raise public awareness and discourse about the role of the criminal justice system in generating social and racial inequality. While these acts of state violence against Blacks have been a recent target of media and activist attention, findings from this study also indicate, however, that even those contacts that do not result in shootings, death, or other forms of acute physical harm have lasting health effects. In many ways, these contacts may serve as chronic stressors in the lives of individuals, increasing levels of ongoing stress, anxiety, stigma, and fear. As such, it is time that we consider the criminal justice system not only a driver of social and racial inequality broadly, but an essential determinant of population health inequality and racial health inequities.

REFERENCES

Alexander, Michelle. 2012. *The New Jim Crow: Mass Incarceration in the Age of Colorblindness.*The New Press.

Apel, Robert and Gary Sweeten. 2010. "The Impact of Incarceration on Employment during the Transition to Adulthood." *Social Problems* 57(3):448-479.

Ben-Shlomo, Y. and D. Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31(2):285-293.

Braun, M. M., Benedict I. Truman, Barbara Maguire, George T. DiFerdinando, Gary Wormser, Raymond Broaddus and Dale L. Morse. 1989. "Increasing Incidence of Tuberculosis in a Prison Inmate Population: Association with HIV Infection." *Jama* 261(3):393-397.

Browning, Sandra L., Francis T. Cullen, Liqun Cao and Renee Kopache. 1994. "Race and Getting Hassled by the Police: A Research Note." *Police Stud.: Int'L Rev.Police Dev.* 17:1.

Brunson, Rod K. 2007. "“police Don'T Like Black People”: African-american Young Men's Accumulated Police Experiences." *Criminology & Public Policy* 6(1):71-101.

Brunson, Rod K. and Jody Miller. 2006. "Gender, Race, and Urban Policing the Experience of African American Youths." *Gender & Society* 20(4):531-552.

Emerging Risk Factors Collaboration. 2010. "C-Reactive Protein Concentration and Risk of Coronary Heart Disease, Stroke, and Mortality: An Individual Participant Meta-Analysis." *The Lancet* 375(9709):132-140.

Finch, Caleb E. 2010. *The Biology of Human Longevity:: Inflammation, Nutrition, and Aging in the Evolution of Lifespans.*Academic Press.

Geller, Amanda, Jeffrey Fagan, Tom Tyler and Bruce G. Link. 2014. "Aggressive Policing and the Mental Health of Young Urban Men." *American Journal of Public Health* 104(12):2321-2327.

Hammett, T. M., M. P. Harmon and W. Rhodes. 2002. "The Burden of Infectious Disease among Inmates of and Releasees from US Correctional Facilities, 1997." *American Journal of Public Health* 92(11):1789-1794.

Harris, Tamara B., Luigi Ferrucci, Russell P. Tracy, M. C. Corti, Sholom Wacholder, Walter H. Ettinger, Harley Heimovitz, Harvey J. Cohen and Robert Wallace. 1999. "Associations of Elevated Interleukin-6 and C-Reactive Protein Levels with Mortality in the Elderly." *The American Journal of Medicine* 106(5):506-512.

Huebner, Beth M. 2005. "The Effect of Incarceration on Marriage and Work Over the Life Course." *Justice Quarterly* 22(3):281-303.

Kiecolt-Glaser, Janice K. and Ronald Glaser. 2002. "Depression and Immune Function: Central Pathways to Morbidity and Mortality." *Journal of Psychosomatic Research* 53(4):873-876.

Kraska, Peter B. 2007. "Militarization and policing—Its Relevance to 21st Century Police." *Policing* 1(4):501-513.

Lochner, Lance and Enrico Moretti. 2004. "The Effect of Education on Crime: Evidence from Prison Inmates, Arrests, and Self-Reports." *The American Economic Review* 94(1):155-189.

Massoglia, Michael and Cody Warner. 2011. "The Consequences of Incarceration." *Criminology & Public Policy* 10(3):851-863.

Massoglia, M. 2008. "Incarceration as Exposure: The Prison, Infectious Disease, and Other Stress-Related Illnesses." *Journal of Health and Social Behavior* 49(1):56-71.

Mitchell, Ojmarrh and Michael S. Caudy. 2015. "Examining Racial Disparities in Drug Arrests." *Justice Quarterly* 32(2):288-313.

New York Civil Liberties Union. 2016. "NYPD Stop and Frisk Data.", Retrieved January 22, 2016. (http://www.nyclu.org/content/stop-and-frisk-data).

Oppong, Joseph R., Libbey Kutch, Chetan Tiwari and Sonia Arbona. 2014. "Vulnerable Places: Prison Locations, Socioeconomic Status, and HIV Infection in Texas." *The Professional Geographer* 66(4):653-663.

Pager, Devah. 2003. "The Mark of a Criminal Record." *American Journal of Sociology* 108(5):937-975.

------. 2008. *Marked: Race, Crime, and Finding Work in an Era of Mass Incarceration.*University of Chicago Press.

Parenti, Christian. 2000. *Lockdown America: Police and Prisons in the Age of Crisis.*Verso.

Pavalko, Eliza K. and Andrea E. Willson. 2011. "Life course approaches to health, illness and healing." Pp. 449-464 in *Handbook of the sociology of health, illness, and healing.*"Life course approaches to health, illness and healing."Springer.

Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman and Joseph T. Mullan. 1981. "The Stress Process." *Journal of Health and Social Behavior* 22(4):337-356.

Penninx, Brenda W., Jack M. Guralnik, Luigi Ferrucci, Eleanor M. Simonsick, Dorly J. Deeg and Robert B. Wallace. 1998. "Depressive Symptoms and Physical Decline in Community-Dwelling Older Persons." *Jama* 279(21):1720-1726.

Pettit, Becky and Bruce Western. 2004. "Mass Imprisonment and the Life Course: Race and Class Inequality in US Incarceration." *American Sociological Review* 69(2):151-169.

Pine, Daniel S., Elizabeth Cohen, Patricia Cohen and Judith Brook. 1999. "Adolescent Depressive Symptoms as Predictors of Adult Depression: Moodiness Or Mood Disorder?" *American Journal of Psychiatry*.

Porter, L. C. 2014. "Incarceration and Post-Release Health Behavior." *Journal of Health and Social Behavior* 55(2):234-249. doi: 55/2/234 [pii].

Rickford, Russell. 2016. "Black Lives Matter Toward a Modern Practice of Mass Struggle." 25(1):34-42.

Ridker, Paul M., Charles H. Hennekens, Julie E. Buring and Nader Rifai. 2000. "C-Reactive Protein and Other Markers of Inflammation in the Prediction of Cardiovascular Disease in Women." *New England Journal of Medicine* 342(12):836-843.

Roberts, Dorothy E. 2004. "The Social and Moral Cost of Mass Incarceration in African American Communities." *Stanford Law Review*:1271-1305.

Robins, James M. 1999. "Association, Causation, and Marginal Structural Models." *Synthese* 121(1):151-179.

Robins, James M., Miguel A. Hernan and Babette Brumback. 2000. "Marginal Structural Models and Causal Inference in Epidemiology." *Epidemiology*:550-560.

Rosenbaum, Paul R. and Donald B. Rubin. 1983. "The Central Role of the Propensity Score in Observational Studies for Causal Effects." *Biometrika* 70(1):41-55.

Schlanger, Margo. 2012. "Prison Segregation: Symposium Introduction and Preliminary Data on Racial Disparities." *Mich.J.Race & L.* 18:241.

Schnittker, Jason. 2014. "The Psychological Dimensions and the Social Consequences of Incarceration." *The Annals of the American Academy of Political and Social Science* 651(1):122-138.

Schnittker, J. and A. John. 2007. "Enduring Stigma: The Long-Term Effects of Incarceration on Health." *Journal of Health and Social Behavior* 48(2):115-130.

Schulenberg, John E., Arnold J. Sameroff and Dante Cicchetti. 2004. "The Transition to Adulthood as a Critical Juncture in the Course of Psychopathology and Mental Health." *Development and Psychopathology* 16(04):799-806.

Sewell, Abigail A., Kevin A. Jefferson and Hedwig Lee. 2016. "Living Under Surveillance: Gender, Psychological Distress, and Stop-Question-and-Frisk Policing in New York City." *Social Science & Medicine* 159:1-13.

Smith, William A., Walter R. Allen and Lynette L. Danley. 2007. "“Assume the Position... You Fit the Description” Psychosocial Experiences and Racial Battle Fatigue among African American Male College Students." *American Behavioral Scientist* 51(4):551-578.

Spaulding, A. C., R. M. Seals, V. A. McCallum, S. D. Perez, A. K. Brzozowski and N. K. Steenland. 2011. "Prisoner Survival Inside and Outside of the Institution: Implications for Health-Care Planning." *American Journal of Epidemiology* 173(5):479-487. doi: 10.1093/aje/kwq422 [doi].

Stark, K., U. Bienzle, R. Vonk and I. Guggenmoos-Holzmann. 1997. "History of Syringe Sharing in Prison and Risk of Hepatitis B Virus, Hepatitis C Virus, and Human Immunodeficiency Virus Infection among Injecting Drug Users in Berlin." *International Journal of Epidemiology* 26(6):1359-1366.

Starr, S. B. and M. M. Rehavi. 2012. "Racial Disparity in Federal Criminal Charging and its Sentencing Consequences." University of Michigan Law School, Program in Law and Economics Working Paper Series, Working Paper No. 12-002.

The Sentencing Project. 2017. *Trends in U.S. Corrections.* Washington, D.C.: .

Thornberry, Terence P., Melanie Moore and RL Christenson. 1985. "The Effect of Dropping Out of High School on Subsequent Criminal Behavior." *Criminology* 23(1):3-18.

Turner, R. J. 2013. "Understanding Health Disparities the Relevance of the Stress Process Model." *Society and Mental Health*:2156869313488121.

Uggen, Christopher and Jeff Manza. 2002. "Democratic Contraction? Political Consequences of Felon Disenfranchisement in the United States." *American Sociological Review*:777-803.

Vescio, M. F., B. Longo, S. Babudieri, G. Starnini, S. Carbonara, G. Rezza and R. Monarca. 2008. "Correlates of Hepatitis C Virus Seropositivity in Prison Inmates: A Meta-Analysis." *Journal of Epidemiology and Community Health* 62(4):305-313. doi: 10.1136/jech.2006.051599 [doi].

Wakefield, Sara and Christopher Uggen. 2010. "Incarceration and Stratification." *Annual Review of Sociology* 36:387-406.

Western, Bruce and Christopher Wildeman. 2009. "The Black Family and Mass Incarceration." *The Annals of the American Academy of Political and Social Science* 621(1):221-242.

Wildeman, Christopher and Bruce Western. 2010. "Incarceration in Fragile Families." *The Future of Children* 20(2):157-177.

Wildeman, C. 2011. "Invited Commentary: (Mass) Imprisonment and (Inequities in) Health." *American Journal of Epidemiology* 173(5):488-491. doi: 10.1093/aje/kwq420 [doi].

Table 2.1. Description of Measures

MEASURES	DESCRIPTION
Outcomes	
C-reactive protein (log)	Continuous measure of log C-reactive protein, an indicator of inflammation, at Wave IV
Depressive symptoms	Count of reported depressive symptoms as indicated by Center for Epidemiological Studies Depression Scale (CES-D) at Wave IV
Key explanatory variables	
Level of criminal justice contact	Categorical measure indicating respondent history of contact with the criminal justice system; 0=no contact (reference); 1=stopped by police; 2=arrested; 3=convicted; 4=convicted (categories are mutually exclusive)
Incarceration history	Binary measure indicating history of incarceration; 1=respondent has been incarcerated and released prior to Wave IV
Age at first incarceration	Categorical measure indicating age when respondent was first incarcerated; 0=never incarcerated; 1=first incarcerated when less than 18 years old; 2=first incarcerated when 18 years or older
Duration of incarceration	Categorical measure indicating length of incarceration experience; 0=never incarcerated; 1=incarcerated for less than one year; 2=incarcerated for one year or longer
Other measures	
Race	Dummy measure indicating racial disparity in the outcomes; 1=Black
Age	Continuous age in years at Wave I
Gender	Dummy measure indicating respondent gender; 1=female
Family SES	Standardized scale of family SES in adolescence at Wave I; comprised of parental education and total household income
Completed education	Categorical measure of respondent completed education; 1=<high school; 2=high school; 3=some college; 4=BA degree or higher
Neighborhood racial composition	Percent of neighborhood residents who are non-White at Wave I
Neighborhood economic disadvantage	Scale of neighborhood economic deprivation at Wave I; comprised of proportion of residents who are unemployed, proportion of residents over the age of 25 years without a high school degree, proportion of families living in poverty, and proportion of families receiving public assistance
Depressive symptoms in adolescence	Count of reported depressive symptoms as indicated by Center for Epidemiological Studies Depression Scale (CES-D) at Wave I
Delinquency	A scale indicating level of delinquency in adolescence

Repeat arrestee	Binary indicator of whether respondent has been arrested multiple times; 1=respondent has been arrested more than once
Violent arrestee	Binary indicator of whether respondent was a violent arrestee; 1=respondent has been arrested for a violent crime

Table 2.2. Descriptive Statistics

	All Mean/prop.	Whites Mean/prop.	Blacks Mean/prop.	*p-value*
Outcomes				
C-reactive protein (log + 1)	1.30	1.29	1.40	0.016
Depressive symptoms	2.44	2.33	2.96	<0.001
Criminal justice contacts				
No contact (reference)	0.65	0.65	0.63	
Stopped by police	0.08	0.09	0.05	
Arrested	0.09	0.08	0.11	0.017
Convicted	0.05	0.05	0.05	
Incarcerated	0.13	0.13	0.16	
Incarceration history				
Formerly incarcerated (1=yes)	0.13	0.13	0.16	0.044
Age at first incarceration				
Under age 18 years	0.22	0.22	0.20	0.061
18 years or older	0.78	0.78	0.79	
Duration of incarceration				
Less than one year	0.54	0.53	0.55	0.329
One year or longer	0.46	0.47	0.45	

Notes: p-value of difference in means/proportions between Blacks and Whites; two-tailed test. The distributions for age at first incarceration and duration of incarceration are for those who were previously incarcerated only.

Table 2.3. Contacts with the Criminal Justice System and Depressive Symptoms (N=5,503)

	Model 1[a] (Coeff.)	Model 2[b] (Coeff.)	Model 3[c] (Coeff.)
Racial disparity in depressive symptoms			
Race (1=Black)	0.23***	0.21***	0.15***
Level of contact with criminal justice system[d]			
Stopped by police		0.15*	0.12†
Arrested		0.18**	0.14*
Convicted		NS	NS
Incarcerated		0.36***	0.23***

Note: Coefficient estimates presented. ***p<0.001; **p<0.01; *p<0.05; †p<0.1. NS=not significant.

a: Model 1 adjusts for age, sex, and race only.

b: Model 2 builds on Model 1 by also adjusting for level of contact with the criminal justice system.

c: Model 3 builds on Model 2 by also adjusting for family SES at Wave 1, education at Wave 4, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, and delinquency at Wave I.

d: Levels are mutually exclusive. No reported contact is the reference group.

Table 2.4. Incarceration History and Log C-Reactive Protein

	Stage 1: OLS Regression[a]			Stage 2: OLS w/Treatment-Control Groups[b]			Stage 3: IPTW with RA[c]		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Incarceration history[d]									
Formerly incarcerated	0.12*			0.20*			0.22**		
Age at first incarceration[d]									
Under age 18 years		NS			NS			NS	
18 years or older		0.19†			NS			NS	
Duration of incarceration[d]									
Less than one year			NS			NS			NS
One year or longer			0.22*			0.23*			0.20†

Notes: Coefficient estimates presented. ***p<0.001; ** p<0.01; *p<0.05; † p<0.1. NS indicates no statistically significant association.
a: Results of the OLS regression models (N=4,603). Models adjust for age, gender, race, family SES at Wave 1, education at Wave 4, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, and violent arrestee.
b: Results of the OLS regression models that include only those convicted of a crime (N=740). Models adjust for age, gender, race, family SES at Wave 1, education at Wave 4, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, and violent arrestee.

c: Results of the models utilizing inverse probability of treatment weighting with a regression adjustment estimator. Sample is restricted to only those convicted of a crime (N=740). The models predicting the "treatment" (e.g., incarceration history, age at first incarceration, and duration of incarceration) include race, gender, age, neighborhood conditions, family SES, respondent education, mental health in adolescence, delinquency, repeat arrestee, and violent arrestee. The outcome model adjusts for race, gender, age, neighborhood conditions, family SES, and respondent education.
d: The reference group is never incarcerated.

Table 2.5. Incarceration History and Depressive Symptoms

	Stage 1: Negative Binomial Reg.[a]			Stage 2: Neg. Bin. w/Treatment-Control Grps[b]			Stage 3: IPTW with RA[c]		
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9
Incarceration history[d]									
Formerly incarcerated	0.13*			0.20*			NS		
Age at first incarceration[d]									
Under age 18 years		0.25†			NS			NS	
18 years or older		NS			0.22†			NS	
Duration of incarceration[d]									
Less than one year			0.14*			NS			NS
One year or longer			0.14†			0.27**			0.20*

Notes: Coefficient estimates presented. ***p<0.001; ** p<0.01; *p<0.05; † p<0.1

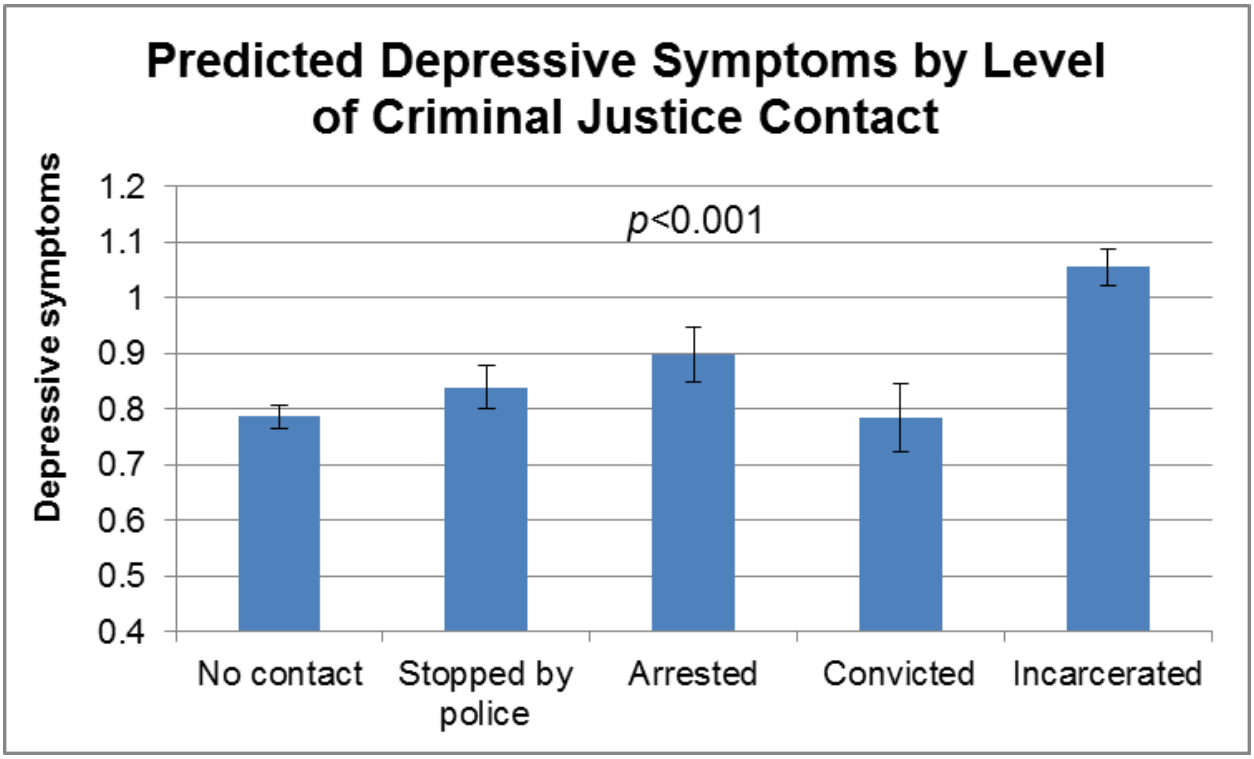
a: Results of the OLS regression models (N=5,077). Models adjust for age, gender, race, family SES at Wave 1, education at Wave 4, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, and violent arrestee.

b: Results of the negative binomial regression models that include only those convicted of a crime (N=813). Models adjust for age, gender, race, family SES at Wave 1, education at Wave 4, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, and violent arrestee.

c: Results of the models utilizing inverse probability of treatment weighting with a regression adjustment estimator. Sample is restricted to only those convicted of a crime (N=813). The models predicting the "treatment" (e.g., incarceration history, age at first incarceration, and duration of incarceration) include race, gender, age, neighborhood conditions, family SES, respondent education, mental health in adolescence, delinquency, repeat arrestee, and violent arrestee. The outcome model adjusts for race, gender, age, neighborhood conditions, family SES, and respondent education.

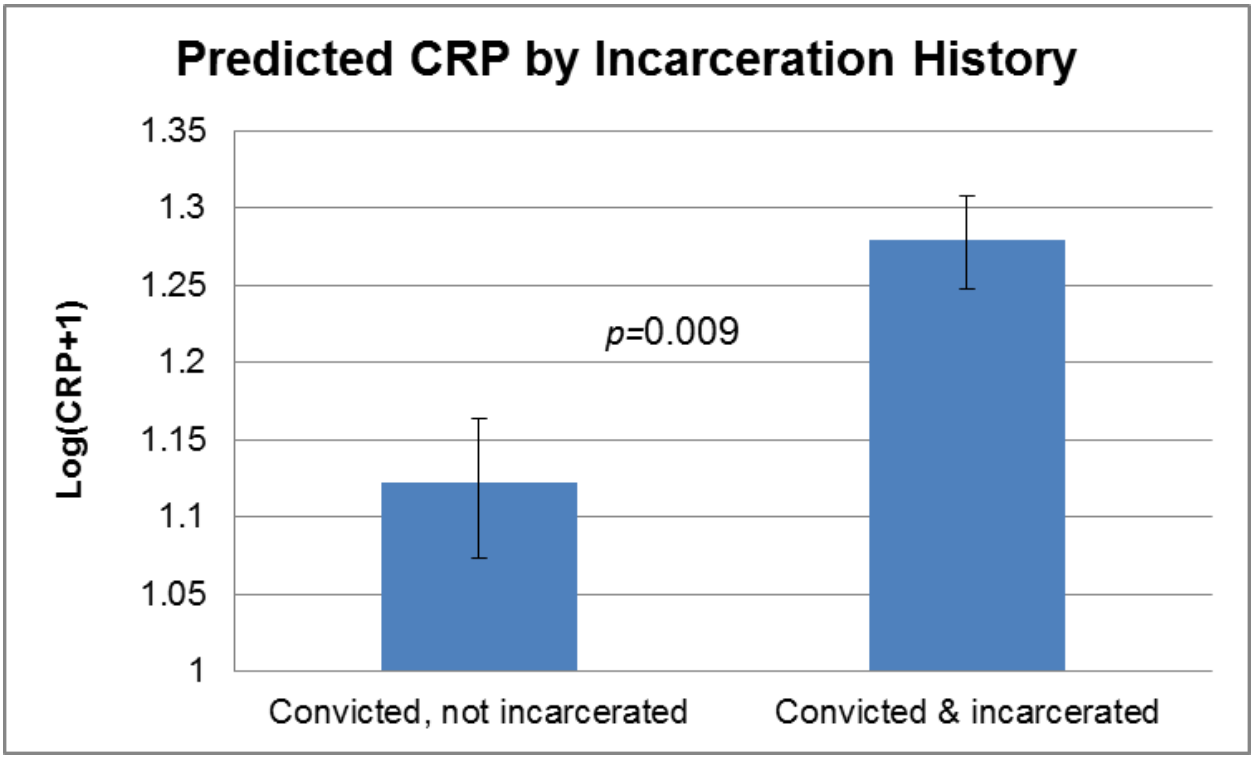
d: The reference group is never incarcerated.

Figure 2.1. Criminal Justice Contacts and Depressive Symptoms



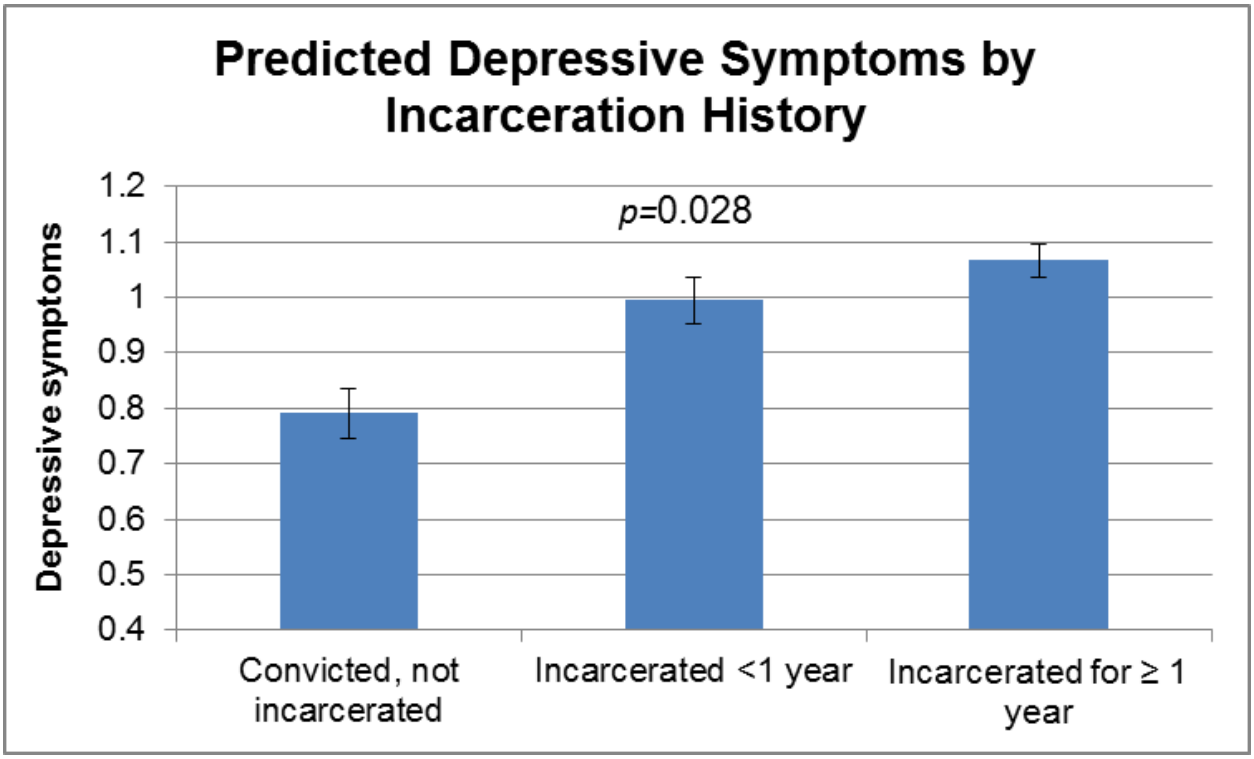
Notes: N=5,503; *p*-value of Wald test. Results based on Model 3 of Table 2.3.

Figure 2.2. Incarceration History and C-Reactive Protein



Notes: N=703; p-value of difference between not incarcerated and incarcerated. Results based on Model 7 of Table 2.4.

Figure 2.3. Duration of Incarceration and Depressive Symptoms



Notes: N=813; *p*-value of Wald test. Results based on Model 6 of Table 2.4.

CHAPTER 3: DOES INTEGRATION PRODUCE EQUITY? A LONGITUDINAL STUDY OF NEIGHBORHOOD CONDITIONS AND RACIAL HEALTH INEQUALITY

Abstract

A wide body of research across disciplines identifies racial residential segregation as a key determinant of racial health disparities(Williams and Collins 2001)(Williams and Collins 2001), with studies linking the conditions in racially segregated neighborhoods to disparities across a number of health outcomes, including low birth weight, depression, and cardiovascular disease. While research on racial residential segregation and health has boomed in recent years, critical gaps in the literature remain. In particular, it remains largely unexplored whether and how neighborhood economic conditions and neighborhood racial composition operate through similar or unique mechanisms to ultimately impact health and disease risk. Using nationally-representative, longitudinal data from the National Study of Adolescent to Adult Health (N=6,248), this study employs multilevel and neighborhood fixed effects models to examine how neighborhood economic conditions and racial composition jointly and uniquely relate to markers of metabolic function from adolescence through young adulthood. I further test the potential psychosocial mechanisms undergirding the associations between neighborhoods and biomarkers of health. Findings indicate that neighborhood economic and racial context have unique associations with metabolic dysregulation and body mass index. While neighborhood economic deprivation uniformly harms health, the associations between neighborhood racial composition and health vary by race. I find that Blacks receive some protective health benefits from living in predominately Black neighborhoods, possibly because of their higher levels of social integration and reduced exposure to discrimination in these predominately Black spaces.

By elucidating the pathways connecting neighborhoods to health, this study offers a nuanced depiction of the relationships between racism, neighborhood conditions, stress, and health.

Introduction

Understanding how where individuals live influences how they live has been the focus of social scientific inquiry for more than 70 years. Since the publication of Shaw and McKay's (1942) *Juvenile delinquency and urban areas*, sociological research on the effects of neighborhood conditions on individual well-being has surged, with a wide body of research now linking neighborhood context to a number of individual outcomes, including child development (Aneshenesel and Sucoff 1996; Brooks-Gunn et al. 1993; Ge et al. 2002; Hoffman 2002; Kowaleski-Jones 2000), educational achievement and attainment (Ainsworth 2002; Chase-Lansdale et al. 1997; Duncan 1994; Entwisle, Alexander, and Olson 1994; Halpern-Felsher et al. 1997), and social mobility (Sharkey 2012). Along this line of research, a number of studies have examined the role of neighborhood contexts in affecting health and producing racial health inequality. Given widely documented racial disparities in health, morbidity, and mortality (Williams et al. 2010) and stark levels of racial residential segregation in the US (Charles 2003; Massey and Denton 1993), scholars hypothesize that differential exposure to neighborhood contexts may be a fundamental determinant of racial health inequality (Williams and Collins 2001).

While research examining the effects of racial residential segregation on health has boomed in recent years, critical gaps in the literature remain. First, despite the widely held assertion that neighborhood racial segregation is associated with adverse health outcomes, the evidence is largely mixed. On the one hand, a body of research shows that racial segregation contributes to disease risk and thus plays a critical role in racial health inequality (e.g., Diez-Roux et al. 1997; Williams and Jackson 2005). On the other hand, other studies, particularly

those focusing on mental health, demonstrate a protective effect of residential segregation, especially for non-Whites (e.g., Pickett and Wilkinson 2008; Osypuk et al. 2009; Becares and Stafford 2009). This inconsistency in findings on the relationship between racial segregation and health has both methodological and theoretical underpinnings. For one, many studies fail to fully distinguish between the economic and racial composition of neighborhoods, resulting in a confounding of these unique characteristics of neighborhoods. Decades of racially discriminatory policies in practices in lending, real estate, educational, employment, and judiciary institutions have resulted in striking levels of racial residential segregation across the United States, as well as a concentration of poverty and economic deprivation in segregated communities of color (Charles 2003; Massey and Denton 1993). The strong association between neighborhood racial composition and neighborhood economic conditions has led some studies to conflate the effects of these neighborhood characteristics on health. However, it is possible that neighborhood economic deprivation and neighborhood racial composition operate through different mechanisms to affect physical functioning and disease risk. For one, racial segregation may contribute to disease risk by concentrating poverty, restricting access to labor market and educational opportunities, reducing socioeconomic mobility, and producing unhealthy physical environments in minority majority neighborhoods (Kramer and Hogue 2009; Williams and Collins 2001). However, racial segregation may also be protective against poor health, for people of color in particular, as individuals living in racially homogenous communities develop robust social networks and support systems that both reduce exposure to racial discrimination and buffer against stress and physiological dysregulation associated with racism (Bécares, Nazroo, and Stafford 2009). In addition to the differing mechanisms through which neighborhood economic conditions and racial composition may operate to affect health, recent research further

documents a rise in “ethnoburbs,” or middle-class or affluent suburban neighborhoods with high proportions of residents of color (Logan 2004; Wen, Lauderdale, and Kandula 2009). With the increased movement of non-Whites to economically advantaged areas, it is increasingly problematic to conflate neighborhood racial composition and neighborhood economic conditions.

Second, while research links racial residential segregation to a host of health outcomes, the biophysiological and psychosocial mechanisms underlying the associations between neighborhood conditions and health are not well specified or tested (Daniel, Moore, and Kestens 2008; Hull et al. 2008; Kramer and Hogue 2009; Wickrama and Bryant 2003; White and Borrell 2011). Most studies of neighborhood effects on health examine the associations between neighborhood conditions and measures of self-rated health (Patel et al. 2003; Subramanian et al. 2005; White and Borrell 2006), disease and morbidity (eg, Diez-Roux et al. 1997; Osypuk and Acevdo-Garcia 2008), mortality (Collins and Williams 1999; Fang et al. 1998; Jackson et al. 2000), or mental health (e.g, Halpern 1993; Pickett and Wilkinson 2008; Wickrama, Noh, and Bryant 2005), which leaves questions about how neighborhood conditions “get under the skin” to affect well-being unanswered. As a result, concerns about biological plausibility remain in much of the literature on neighborhood effects on health (Daniel, Moore, and Kestens 2008). Further, much of the research on neighborhood effects on health focuses on how structural conditions—such as the availability of healthy food sources and the presence green space for physical activity—affect individual health outcomes. However, neighborhood conditions may also affect health through psychosocial mechanisms by reducing or increasing physiological stress response (Daniel, Moore, and Kestens 2008). Because so few studies specify and test the biophysiological and psychosocial mechanisms underlying the association between neighborhood conditions and

individual outcomes, it remains to be better understood how neighborhoods contribute to disease risk through the conditioning of individual behavioral, psychosocial, and regulatory responses.

Finally, because of limited data availability, most studies of neighborhood effects on health use cross-sectional studies (Kramer and Hogue 2009; White and Borrell 2011). The lack of longitudinal research using prospective data on neighborhood conditions and markers of mental and physical well-being limits understanding of how neighborhood contexts affect health across the life course (Daniel, Moore, and Kestens 2008). It remains unknown, for example, whether neighborhood conditions experienced during critical or sensitive periods such as childhood or adolescence have stronger associations with future disease risk than neighborhood conditions experienced later in life. Further, whether and how changing neighborhood conditions impact trajectories of well-being is relatively unexplored. The use of cross-sectional data also restricts researchers' ability to draw causal inferences about the impacts of neighborhood conditions on physiological regulation and disease risk.

This study fills these gaps in the literature by using longitudinal data from the National Study of Adolescent to Adult Health (Add Health) and hierarchical and fixed effects modeling strategies to examine how neighborhood economic conditions and racial composition uniquely relate to markers of physical functioning over time. By examining the economic, psychosocial, and physiological pathways connecting neighborhoods to health, this study improves sociological understanding of the links between race, place, and health.

Theoretical Model

Neighborhoods and Health

Figure 3.1 displays the theoretical model guiding this study. This study is grounded in the ecological development perspective, which views individuals as developing and aging within a set of embedded social contexts that shape their access to resources, exposure to risks,

psychological well-being, and health (Bronfenbrenner 1986; Leventhal and Brooks-Gunn 2000; Wickrama and Bryant 2003). As indicated by Box 1 of Figure 3.1, the key social context of interest to this study is the neighborhood. While most studies in the ecological development perspective focus on the family as the central social construct shaping health, research indicates that other social contexts, such as neighborhoods, also influence individual behaviors and outcomes (Aneshenesel and Sucoff 1996; Brooks-Gunn et al. 1993; Wickrama and Bryant 2003).

Research suggests that neighborhoods may directly influence physiological functioning and health (Path 1A), as the structural characteristics of neighborhoods may function as individual risk or protective factors (Hull et al. 2008; Wickrama and Bryant 2003). This study focuses on two characteristics of neighborhood context: neighborhood economic conditions and neighborhood racial composition. Neighborhood economic conditions can affect health by either promoting or restricting access to health-promoting material resources and increasing or reducing exposure to daily stress (Aneshenesel and Sucoff 1996; Ross, Reynolds, and Karlyn 2000). Research documents that individuals living in poor neighborhoods have less access than to health-promoting resources such as grocery stores and green space, and they also experience increased exposure to hazards such as toxins and violence compared those in economically advantaged neighborhoods (Williams and Collins 2001). Further, the daily stress of living in economically deprived neighborhoods may produce feelings of hopelessness, frustration, distress, and loneliness (Aneshenesel and Sucoff 1996; Ross, Reynolds, and Karlyn 2000; Wickrama and Bryant 2003), which may further promote physiological dysregulation. Exposure to neighborhood-level stressors can serve to chronically activate the body's stress response systems, resulting in these systems becoming ineffective and inefficient over time and eventually increasing overall levels of physiological malfunctioning and allostatic load (Christou et al.

2005; Cohen et al. 2012; Hawkley et al. 2006; Kietcolt-Glaser et al. 2005; McEwen 1998; McEwen and Stellar 1993; Selye 1974). In this way, living in economically deprived neighborhoods may contribute to health inequality through stress-related physiological processes.

In addition to neighborhood economic conditions, studies across disciplines also link the racial composition of neighborhoods to a host of health outcomes, including self-rated health (Patel et al. 2003; Subramanian et al. 2005; White and Borrell 2006), disease and morbidity (eg, Diez-Roux et al. 1997; Osypuk and Acevdo-Garcia 2008), mortality (Collins and Williams 1999; Fang et al. 1998; Jackson et al. 2000) and mental health (e.g, Halpern 1993; Pickett and Wilkinson 2008; Wickrama, Noh, and Bryant 2005). The literature suggests both material and psychosocial mechanisms linking neighborhood racial composition to health. Racial residential segregation has been linked to poverty concentration, limited employment opportunities, reduced social mobility, and increased exposure to unhealthy physical environments in some segregated communities of color (Charles 2003; Massey and Denton 1993). As described above, concentrated socioeconomic deprivation can harm health through both material and psychosocial pathways. However, research also suggests that racial residential segregation may protect against poor health, particularly in segregated communities of color. First articulated by Halpern (1993), the ethnic density hypothesis suggests that racial and ethnic minorities can have better health when living in areas with high proportions of residents of the same race or ethnicity. Several studies have found evidence of the ethnic density effect (e.g., Bécares and Stafford 2009; Osypuk et al. 2009; Pickett and Wilkinson 2008; Vogt Yuan 2007). Whereas living in a racially homogenous neighborhood may promote feelings of belonging and well-being, living in racially diverse or secluded neighborhoods may induce feelings of loneliness and isolation (Mullings and

Wali 2001; Vogt Yuan 2007). In this way, the racial composition of a neighborhood may have distinct health effects from the economic conditions of a neighborhood.

Mediating Mechanisms

Social Relationships

In addition to the direct effects of neighborhoods on physiological functioning, the model presented in Figure 3.1 proposes that neighborhood conditions also exert an influence on individual health through two proximal psychosocial mechanisms. First, as indicated by Path 1B in Figure 3.1, neighborhood conditions may affect individual health by promoting or restricting the connectedness of residents (Box 2). In the literature, social integration refers to the structural dimension of social relationships and reflects the quantity and nature of ties in an individual's social network (Thoits 2011). A wide body of research links social isolation—or a lack of social integration—to a range of diseases and poor health outcomes, including cardiovascular disease (Berkman et al. 1993; Orth-Gomér et al. 1993; Eng et al. 2002), cancer (Penwell and Larkin 2010), and depression (George et al. 1989; Heikkienen and Kauppinen 2004), as well as biomarkers of physiological dysregulation such as inflammation (Kiecolt-Glaser et al. 2010; Yang et al. 2013) and infection (Cohen et al. 1997). Social isolation has also been linked to higher rates of general and cause-specific mortality (Berkman and Syme 1979; Thoits 1995; Yang at al. 2013). Studies suggest that social isolation influences physical functioning (Path 2A) through both stress inducing and behavioral mechanisms (Thoits 2011).

Neighborhood conditions may affect individual levels of social isolation (Path 1B) in two ways. First, because of social disorganization, research indicates that individuals living in economically disadvantaged communities may be less likely to form relationships with other residents (Kowaleski-Jones 2000) and less likely to participate in formal community organizations (Ross, Mirowsky, and Pribesh 2001; Sampson 2001; Shaw and McKay 1942) than

individuals living in less disadvantaged neighborhoods. Second, neighborhood racial composition may also have an effect on individual social isolation. On the one hand, neighborhood racial heterogeneity may hinder the formation of social relationships, as diversity of languages, religions, and cultural practices may restrict communication and interaction between residents (Wickrama and Bryant 2003). On the other hand, research indicates that high levels of racial segregation (e.g., neighborhood racial homogeneity) may be conducive to the creation and maintenance of social relationships. In particular, non-Whites living in highly segregated neighborhoods may have more opportunities to form and maintain social relationships than they would in racially diverse neighborhoods (Bécares, Nazroo, and Stafford 2009; Wickrama and Bryant 2003).

Racial Discrimination

Neighborhood racial composition may also affect physiological functioning and disease risk by increasing or decreasing individual exposure to racial discrimination (Box 3). A wide body of research documents a link between racial discrimination and health (Path 3A), with the stress associated with racial discrimination being linked to depression, elevated blood pressure, chronic inflammation, and increased cardiovascular disease risk (Williams and Mohammed 2009; Williams, Neighbors, and Jackson 2003). Neighborhood racial composition may affect individual exposure to racial discrimination (Path 1C) by increasing or decreasing opportunities for inter-racial interaction and discrimination. Increased levels of neighborhood racial diversity may result in increased reports of racial discrimination by non-Whites, as increased interaction between racial groups increases opportunities for racial discrimination. Similarly, in highly segregated neighborhoods, non-Whites may experience less racial discrimination than in more diverse community contexts because of decreased inter-racial contact (Halpern and Nazroo 1999; Bécares, Nazroo, and Stafford 2009).

Variation Across the Life Course

As indicated by the arrow labeled "Age" in Figure 3.1, this study is also guided by the life course perspective, which has offered considerable insights to the study of aging and health. While a wide body of literature establishes a cross-sectional link between neighborhood context at a single point in time and health, few studies have examined how neighborhood conditions experienced at different life stages may jointly or uniquely affect health (Clarke et al. 2014). For one, neighborhoods are not static, but rather they change over time (Sampson, Morenoff, and Gannon-Rowley 2002). For this reason, individuals may experience a variety of neighborhood contexts over the life course, even if they never move. Second, there is evidence that the documented association between current neighborhood characteristics and health may be subsuming the effects of earlier-life neighborhood conditions. Wheaton and Clarke (2003) find a lagged effect of childhood neighborhood conditions on later life mental health that explains the apparent bivariate effect of current neighborhood conditions on health. Life course epidemiologists suggest that events and conditions experienced during critical or sensitive experiences such as childhood or adolescence may become biologically embedded and have a greater influence on health outcomes and disease risk than conditions experienced later in the life course (Richardson et al. 2012). Given the temporal dynamics of neighborhoods and findings suggesting that the health effects of neighborhoods vary by their life course timing, further research on the temporal dynamics of neighborhoods and health is needed (Sampson, Morenoff, and Gannon-Rowley 2002).

Research Questions

Altogether, it merits further investigation whether neighborhood socioeconomic deprivation and racial composition are independent or interrelated indicators of neighborhood context that uniquely contribute to individual health and well-being. Distinguishing between the

material and psychosocial influences of neighborhood contexts on health is essential to developing policy solutions to ameliorating racial health disparities (Pickett and Wilkinson 2008).

This studies aims to address four key research questions:

1. Do neighborhood economic conditions and neighborhood racial composition have unique associations with markers of physiological well-being?
2. Are the associations between neighborhood conditions and health the same for Blacks and Whites?
3. Is there age variation in the associations between neighborhood characteristics and physiological well-being?
4. Do psychosocial factors such as social isolation and perceived discrimination mediate the associations between neighborhood characteristics and health?
5. What are the implications of these findings for research and policy efforts focused on the role of neighborhoods in Black-White health disparities?

Data and Analytic Methods

Data

Data for this study come from the National Longitudinal Study of Adolescent to Adult Health (Add Health), which is a nationally representative, longitudinal study of U.S. adolescents. Using a school-based complex cluster sampling frame, Add Health began in 1994-95 with an in-school questionnaire administered to a nationally-representative sample of students in grades 7-12. Following the in-school questionnaire, a gender- and grade-stratified random sample of 20,745 adolescents (79% response rate) was selected for in-home interviews at Wave I. The study then followed up with a series of in-home interviews conducted in 1996 (Wave 2; 88% response rate), 2001-02 (Wave 3; 77% response rate), and 2007-08 (Wave 4; 80% response rate).

In addition to respondent questionnaires and interviews, Add Health also collected questionnaires from parents, siblings, fellow students, and school administrators and interviews with romantic partners. Add Health is a particularly rich source of data for studying life course trajectories of health and well-being because the study followed young people from their teen years through their transition to adulthood, allowing researchers to gain new insights into how young people's social relationships and social contexts affect their health and well-being over time.

In addition to the interviews, Add Health also collected biological specimens from study participants at Wave IV. The collection of physical measurements, saliva samples and dried blood spots allows researchers to better understand the linkages between respondents' social lives and their cardiovascular, metabolic, and immunologic functioning. At Wave IV, Add Health interviewers collected a number of cardiovascular and anthropometric measures, including systolic blood pressure, diastolic blood pressure, height, weight, and waist circumference. The study also collected blood spots from respondents for a lipid panel and assays of glucose. For detailed information about biomarker collection procedures and protocols, see Entzel et al. (2009) and Whitsel et al. (2012).

Data for this study come from the in-home interviews at Waves I and IV and the biomarker indicators at Wave IV. I also utilize Census tract-level data linked to respondents' residences at Wave I and IV.

Measures

Dependent Variables

The outcomes of interest include two measures of physical functioning that represent important risk factors for a future morbidity and mortality: metabolic dysregulation and body

mass index (BMI). All outcomes are measured at Wave IV, when respondents were aged 24-32 years.

I construct an index of *metabolic dysregulation*, which indicates overall levels of metabolic burden using clinical markers of metabolic syndrome: waist circumference, serum triglycerides, HDL cholesterol, serum glucose, systolic blood pressure, and diastolic blood pressure. For each individual measure, I construct a dummy measure where 1 indicates high risk. Cut points for high risk were defined by clinical practice or empirically defined as the top quintile (bottom quintile was used for HDL cholesterol). I then summed the scores from each of the markers to construct the index of overall metabolic burden, which is modeled as a continuous measure ranging from 0 (low metabolic dysregulation) to 6 (high metabolic dysregulation). *Body mass index* (BMI) is included as a continuous measure. Research indicates that BMI is predictive of health and longevity, with overweight (BMI$\geq$25kg/m^2) and obese (BMI$\geq$30kg/m^2) individuals being at increased risk of cardiovascular disease, cancer, disability, and premature mortality (Kopelman 2007).

Key Explanatory Variables

Neighborhood Conditions

I measure *neighborhood economic deprivation* at Waves I and IV using a composite index of four measures of neighborhood economic conditions using Census tract-level data: proportion of residents who are unemployed, proportion of residents over the age of 25 years without a high school degree, proportion of families living in poverty, and proportion of families receiving public assistance. For each measure, I created a dummy variable indicating the top quartile of all Census tracts (e.g., the Census tracts in the top quartile for unemployment). To create the index of neighborhood economic deprivation, I summed the four measures, producing an index ranging from 0 (low deprivation) to 4 (high deprivation).

To measure neighborhood racial composition, I create a measure of *neighborhood racial density* at Waves I and IV. This measure indicates the percentage of neighborhood residents who are the same race as the respondent (White, Black or Hispanic). In order to capture non-linearities in the relationship between neighborhood racial heterogeneity, I also include a measure of *neighborhood racial density*^{2}, which captures high levels of racial segregation or homogeneity. To test whether the association between neighborhood racial density and the outcomes varies by race, I include interaction terms for *race*neighborhood racial density* and *race*neighborhood racial density*^{2}.

<u>*Psychosocial Mediators*</u>

As shown in Figure 3.1, I examine two possible mechanisms through which neighborhood conditions may get "under the skin" to affect physical well-being: social isolation (Box 2) and perceived discrimination (Box 3), both of which are measured at Wave IV using data from the in-home interviews. Measures included in the index of *social isolation* include relationship status, number of close friends, frequency of religious attendance, and frequency of volunteering. To construct the index of social isolation, I create dummy variables for each measure, with 1 indicating the top quartile of all respondents (with the exception of relationship status, where 1=married or cohabiting at Wave IV). The social integration index ranged from 0 (highest isolation) to 4 (lowest isolation). In analytic models, I include social isolation as a dummy variable where "1" indicates an isolation index score of 0 or 1.

Perceived discrimination is constructed using data from the in-home interview at Wave IV. Add Health asked respondents, "In your day-to-day life, how often do you feel you have been treated with less respect or courtesy than other people?" Perceived discrimination is included as a dummy measure, where 0=never and 1=rarely, sometimes, or often.

Other Measures

The racial disparity in the outcomes is indicated by a categorical measure of *race*, where 1=White, 2=Black, and 3=Hispanic. All analyses adjust for *gender* (1=female), *age*, and *socioeconomic status* (SES). At both Waves I and IV, I include SES as a composite measure, where I calculated SES as the mean of standardized (z-score) measures of economic well-being. At Wave I, the measures of SES included in the composite measure include parental education and household income. At Wave IV, SES reflects the respondent's level of education attainment and household income. For both measures (Waves I and IV), positive values represent higher levels of SES. Supplementary analyses also considered the role of neighborhood urbanicity in shaping these processes, though results did not offer evidence of variation in the associations between neighborhoods and health by urbanicity, so these measures were excluded from final model estimates. Further, in order to adjust for potential selection, I also included a measure indicating parents' reasons for moving to neighborhood at Wave I, but including this measure in the models had no impact on model estimates, so I did not include the measure in the final models presented here.

Analytic Sample

The analytic sample (N=6,248) includes respondents who have complete data on the variables included in the analyses as well as valid survey sampling weights. Compared to those included in the analytic samples, those who were excluded had higher levels of neighborhood economic deprivation at both waves ($p<0.05$), lower levels of neighborhood ethnic density at both waves ($p<0.001$), lower SES at Wave IV ($p=0.003$), and were more likely to be Black or Hispanic ($p<0.001$).

Analytic Methods

I use multilevel regression models to examine the influence of individual and neighborhood-level predictors on metabolic dysregulation and BMI. The use of multi-level models allows to me account for the nested nature of the data, where individuals are clustered within neighborhoods. In the multilevel models, individual outcomes are predicted by individual-level factors, neighborhood-level characteristics, and interactions between individual and neighborhood factors. The multilevel models include error terms at both the individual and neighborhood levels.

I estimate models for metabolic dysregulation and BMI separately. For each outcome, I begin with Model 1, which regresses the outcome measures on neighborhood and individual characteristics during adolescence (Wave I). Model 2 regresses the outcomes on neighborhood and individual characteristics experienced during young adulthood (Wave IV). Comparing coefficient estimates and model fit statistics across Models 1 and 2 provides insights into whether neighborhood characteristics experienced during different life course stages have differential effects on biomarkers of health during young adulthood. For each outcome, Models 3 and 4 build off of Model 2 to integrate measures of perceived discrimination and social isolation, respectively, into the multilevel models. All analyses use sample weights to ensure the representativeness of the respondents.

In addition to examining how neighborhood conditions at particular time points impact the outcomes, I also utilize multilevel neighborhood fixed effects models, where I regress the Wave IV outcomes on changes in neighborhood economic deprivation and neighborhood racial composition between Waves I and IV. These models adjust for all time-invariant neighborhood conditions and will allow me to better elucidate how changing neighborhood conditions impact the biomarker outcomes. Model 1 is the metabolic dysregulation model, and Model 2 includes

the results for the BMI outcome. For the fixed models, I run one model per outcome that includes the same covariates as those included in the previous sets of models, except that exclude the psychosocial mediators (as they were measured at Wave IV) and I model change in neighborhood economic deprivation and change in neighborhood racial density from Wave I to Wave IV, rather than including these covariates as cross-sectional measures.

Results

Descriptive Statistics

Descriptive statistics for all the variables used in the analyses are presented in Table 3.1. Table 3.2 presents the means for the outcome measures and neighborhood characteristics by race. Table 3.2 reveals racial differences in mean BMI, with Blacks and Hispanics having higher BMIs than White respondents ($p<0.001$). Further, descriptive statistics reveal stark racial disparities in neighborhood characteristics during both adolescence and early adulthood. On average, Blacks and Hispanics experience greater levels of neighborhood economic deprivation than Whites during both adolescence and the transition to adulthood ($p<0.001$). However, Whites live in neighborhoods characterized by higher levels of racial segregation than Blacks and Hispanics, as indicated by Whites' higher mean neighborhood racial density during both adolescence and young adulthood ($p<0.001$).

Multilevel Models

Metabolic Dysregulation

Table 3.3 displays the results of the multilevel regression analyses for metabolic dysregulation, where I regress metabolic dysregulation (measured during young adulthood) on neighborhood characteristics, individual characteristics, and psychosocial factors. For all models, Table 3.3 presents the coefficients estimates and standard errors.

As shown in Model 1 of Table 3.3, neighborhood context experienced during adolescence is not associated with metabolic dysregulation in young adulthood, net of individual characteristics. While neighborhood characteristics during adolescence are not associated with metabolic dysregulation, individual and family factors during adolescence predict metabolic dysregulation in young adulthood. Females have lower risk of metabolic dysregulation than males (β=-0.374, p<0.001), and family SES during adolescence is protective against metabolic dysregulation, such that increases in SES reduce metabolic risk (β=-0.139, p<0.001).
Model 2 regresses metabolic dysregulation on neighborhood and individual factors during young adulthood. Net of individual characteristics, neighborhood economic deprivation during young adulthood is not significantly associated with metabolic risk. Neighborhood racial composition is significantly associated with metabolic dysregulation, but the direction of the association varies by race. Increasing levels of neighborhood racial heterogeneity (as indicated by the coefficient for neighborhood racial density) are associated with reduced levels of metabolic dysfunction for Whites (β=-0.020, p<0.05), whereas increased levels of neighborhood racial heterogeneity are not protective against metabolic dysfunction for Blacks (β=0.020, p<0.1). However, high levels of neighborhood racial homogeneity (as indicated by the coefficient for neighborhood racial density^2) increase levels of metabolic dysregulation for Whites (β=0.017, p<0.05) but not for Blacks (β=-0.015, p<0.1). In addition to neighborhood factors, individual SES is also associated with metabolic risk, with increases in individual SES protecting against metabolic dysregulation (β=-0.136, p<0.001).

Models 3 and 4 of Table 3.3 integrate measures of perceived discrimination and social isolation, respectively. As seen in Model 3, perceived discrimination is not significantly associated with metabolic dysregulation. However, in Model 4, social isolation is predictive of

metabolic risk. Compared to more socially integrated individuals, less socially integrated individuals have higher levels of metabolic dysregulation (β=0.111, p<0.05). Further, by including the measure of social isolation in Model 4, the interaction term for Black*neighborhood racial density is fully attenuated, suggestive of potential mediation. Model 4, which includes the individual, family, and neighborhood characteristics in young adulthood as well as the measure of social isolation, provides the best model fit according to AIC and BIC statistics.

Table 3.4 presents the results of the neighborhood fixed effects models. Model 1 documents the results of the metabolic dysregulation analyses, where metabolic dysregulation at Wave IV is regressed on changes in neighborhood conditions from Wave I to Wave IV. The results in Table 3.4 largely mirror the results in Table 3.3, providing evidence of a robust link between neighborhood economic deprivation and neighborhood ethnic density with metabolic risk. While I find no association between change in neighborhood economic deprivation and metabolic dysregulation, I find that change in neighborhood racial density significantly predicts the outcome. Consistent with the previous set of models, I find that increases in neighborhood racial diversity from Wave I to Wave IV are associated with lower metabolic risk for Whites (β=-0.026, p<0.01), but higher risk for Blacks and Hispanics. Conversely, Whites who experienced increasing levels of neighborhood racial homogeneity had increased metabolic risk, whereas Blacks and Hispanics who experienced increased levels of neighborhood segregation had reduced health risk.

BMI

Table 3.5 presents the results of the multilevel regression analyses for BMI, where BMI in young adulthood is predicted by neighborhood characteristics, individual characteristics, and

psychosocial factors. Table 3.5 displays the coefficients estimates and standard errors for all models.

Model 1 of Table 3.5 indicates that neighborhood context experienced during adolescence has weak associations with BMI at young adulthood, net of individual characteristics. Increasing levels of neighborhood racial heterogeneity, as indicated by the coefficient for neighborhood racial density, are associated with higher BMI (β=0.100, p<0.1), and the association between neighborhood racial density and BMI does not vary by race. Further, high levels of neighborhood racial density are associated with lower BMI (β=-0.73, p<0.1), an association that does not vary by race. Neighborhood economic deprivation during adolescence is not associated with BMI in young adulthood. Model 1 also indicates that individual and family factors during adolescence predict BMI in young adulthood. In particular, family SES during adolescence is protective against BMI, such that increases in SES are associated with decreases in BMI (β=-0.900, p<0.001). It is worth nothing that, even after adjusting for individual, family, and neighborhood characteristics in adolescence, Blacks (β=5.551, p<0.05) and Hispanics (β=3.617, p<0.1) still have higher BMIs than Whites.

Model 2 regresses BMI on neighborhood and individual factors during young adulthood. Net of individual characteristics, including SES, neighborhood economic deprivation during young adulthood is positively associated with BMI (β=0.408, p<0.01). That is, even after controlling for individual SES, individuals living in areas of increased neighborhood economic disadvantage have higher BMIs than individuals living in less disadvantaged neighborhoods. In addition to neighborhood SES, neighborhood racial composition is also significantly associated with BMI. Similar to the model estimates for metabolic dysregulation, I find that the direction of the association between neighborhood racial composition and BMI varies by race. Increased

levels of neighborhood racial heterogeneity are associated with higher BMI for Blacks (β=0.166, $p<0.01$). Conversely, high levels of Black neighborhood segregation (as indicated by the coefficient for Black x neighborhood racial density^2) are associated with lower BMI for Blacks (β=-0.143, $p<0.01$). In addition to neighborhood factors, individual SES is also negatively associated with BMI, with increases in individual SES resulting in lower BMI (β=-0.669, $p<0.05$). After controlling for individual and neighborhood characteristics in young adulthood, the racial disparity in BMI is eliminated.

Models 3 and 4 of Table 3.5 introduce measures of perceived discrimination and social isolation, respectively. In Model 3, I find a positive association between perceived discrimination and BMI. Compared to individuals who do not report discrimination, individuals who report that they perceive discrimination have higher BMIs (β=0.623, $p<0.05$). Social isolation is not significantly associated with BMI. Model 3, which includes the individual, family, and neighborhood characteristics in young adulthood as well as the measure of perceived discrimination, provides the best model fit according to AIC and BIC statistics.

The neighborhood fixed effects models for BMI are presented in Model 2 of Table 3.4. Similar to the metabolic dysregulation analyses, results from the BMI analyses reveal remarkable consistency across cross-sectional and fixed effects modeling strategies. I find that increases in neighborhood economic deprivation from Wave I to Wave IV are associated with higher BMI. Largely consistent with the metabolic models, I find that change in neighborhood racial density significantly predicts BMI, such that increases in neighborhood diversity from Wave I to Wave IV are associated with lower BMI for Whites (β=-0.104, $p<0.05$), but higher BMI for Blacks (β=0.173, $p<0.05$). Conversely, Whites who experienced increasing levels of neighborhood

racial homogeneity had higher BMI in young adulthood, whereas Blacks who experienced increased levels of neighborhood segregation had lower BMI.

Discussion

While research widely cites racial residential segregation as fundamental determinant of racial health inequality, critical gaps in the literature remain. In particular, the evidence on the association between neighborhood racial segregation and health is mixed, with some studies finding that racial segregation harms health and others suggesting a protective effect of living in racially homogeneous communities for non-Whites, in particular. The present study aimed to improve sociological understanding of the links between race, place, and health by examining how neighborhood conditions—including neighborhood economic deprivation and racial composition—"get under the skin" to affect health and well-being as individuals age. Descriptive analyses presented in Table 3.2 reveal stark racial disparities in neighborhood context and markers of physical functioning. Compared to Whites, Blacks and Hispanics have higher BMIs and experience higher levels of neighborhood economic deprivation. On the other hand, Whites are more likely than Blacks and Hispanics to live in areas of high racial density. On average, Whites reside in overwhelmingly White spaces, with the average White adolescents living in a neighborhood where 9 in 10 residents are White. By contrast, the average Black adolescent lives in a neighborhood consisting of approximately 50 percent Black residents, and the average Hispanic adolescent lives in a neighborhood that is nearly 30 percent Hispanic.

The multilevel regression analyses in Tables 3, 4, and 5 suggest that neighborhood conditions play a fundamental role in individual disease risk, though in nuanced ways that vary by age, race, neighborhood characteristic, and outcome. In addition to cross-sectional multilevel models, I also presented results from multilevel neighborhood fixed effects models, which control for time-invariant neighborhood conditions and offer new insights into how changing

neighborhood experiences impact health and disease risk. While I found no association between neighborhood economic deprivation and metabolic dysregulation, living in an economically deprived neighborhood in young adulthood was associated with higher BMI in both the cross-sectional and fixed effects models, net of individual SES. That is, contextual disadvantages associated with neighborhood deprivation confer health risks to residents, above and beyond the health risks associated with low individual or household socioeconomic status. These findings offer further support for previous research that suggests that neighborhood economic conditions can act as protective or risk factors by either promoting or restricting access to health-promoting material resources and increasing or reducing exposure to daily stress (Aneshenesel and Sucoff 1996; Ross, Reynolds, and Karlyn 2000).

Further, results suggest that neighborhood economic conditions and neighborhood racial composition have unique associations with health. Models 2, 3, and 4 of Tables 3-5 consistently indicate that, net of neighborhood socioeconomic conditions, neighborhood racial composition is significantly related to physical functioning, but the direction of the association varies by race. Results from the metabolic dysregulation models indicate that, after adjusting for neighborhood economic conditions, Whites receive protective benefits from living in areas of increasing racial heterogeneity. For Whites, increased levels of neighborhood racial diversity are associated with lower levels of metabolic dysregulation and BMI. However, high levels of White segregation are associated with worse outcomes for Whites.

Compared with Whites, the association between neighborhood racial composition and physical function is in the opposite direction for Blacks. Whereas Whites have improved outcomes with increasing neighborhood racial diversity, Blacks have worse outcomes in areas of increased racial heterogeneity. Conversely, while high levels of White segregation prove

detrimental for Whites, high levels of Black segregation offer protective benefits for Blacks. Net of neighborhood economic deprivation, Blacks living in predominately Black neighborhoods have lower levels of metabolic dysregulation and BMI, compared to Blacks living in racially diverse neighborhoods. Further, results from the fixed effects models indicated that Blacks who experienced increasing levels of Black segregation from adolescence to young adulthood also had lower metabolic risk and BMI. Together, findings from the multilevel, cross-sectional and fixed effects models speak to the importance of considering both neighborhood economic conditions and neighborhood racial composition in studies of health inequality, as these characteristics of neighborhoods confer different health effects to residents.

Earlier research has hypothesized that high levels of segregation may be protective for people of color because of the increased opportunities for social integration and decreased exposure to racial discrimination that result from living in a segregated neighborhood (Wickrama and Bryant 2003). Results from Models 3 and 4 of Tables 3 and 5 offer some preliminary evidence that these psychosocial mechanisms may, in fact, mediate the association between neighborhood racial composition and health. I find that social isolation, in particular, is associated with increased risk of metabolic dysregulation. Further, after including the measure of social isolation in Model 4 of Table 3.3, the coefficient indicating the protective effect of living in a predominately Black neighborhood was no longer statistically significant, suggestive of potential mediation. In addition, I found that perceptions of discrimination are associated with higher BMI. Though formal Sobel-Goodman mediation tests did not provide statistical evidence that these psychosocial factors mediate the association between neighborhood racial composition and the outcomes, these findings nevertheless indicate that more research in this area is needed. Together, these findings offer preliminary evidence that the protective benefits Blacks receive

from living in hyper-segregated communities may be because of the increased opportunities for social integration and the decreased exposure to racial discrimination in these racially homogenous communities, which may buffer them against the biophysiological stress response associated with living in a racially stratified society. Conversely, in predominately White or more diverse neighborhoods, Blacks may be subject to the increased stigmatization based on their social positioning in these places (Pickett and Wilkinson 2008). Blacks living in neighborhoods where they are identified as "other" may experience higher levels of social isolation, racial discrimination, and social stigma, all of which may contribute to health decline. As visually depicted in Figure 3.2, I find that, as the percentage of White neighborhood residents increases, Blacks report higher levels of perceived discrimination. Future analyses should further explore the roles of social isolation and perceived discrimination as mechanisms linking neighborhood conditions to health risk.

Findings also provide some evidence of age variation in the associations between neighborhood characteristics and physical functioning. Previous research has documented that neighborhood conditions experienced earlier in the life course may be particularly important for later life health (Wheaton and Clarke 2003), offering support for the critical or sensitive period hypothesis. However, I do not find evidence for this hypothesis. I find no association between neighborhood characteristics experienced during adolescence and metabolic function, and I document only weak associations between adolescent neighborhood conditions and BMI. Instead, I find that neighborhood context experienced during young adulthood—as well as the changing neighborhood contexts from adolescence through young adulthood—are significant predictors of physical functioning.

This study is not without limitations. For one, as with all studies of neighborhood effects, there are concerns regarding selection and endogeneity. The use of neighborhood fixed effects models, which control for time-constant neighborhood conditions, provided confirmation of the cross-sectional, multilevel analyses, but neighborhood fixed effects models do not account for time-invariant individual characteristics. Supplementary analyses revealed that controlling for measures such as why parents decided to move to the neighborhood—which may help to indicate possible selection—did not affect model estimates. Still, the results presented here provide preliminary evidence of the unique associations of neighborhood economic and racial context with biomarkers of health, and future studies can build on the work presented here by utilizing analytic methods to account for potential selection biases. Second, the outcomes in this study were measured in Wave IV. Future analyses should integrate longitudinal biomarker outcomes to improve causal inference.

By examining the psychosocial and biophysiological mechanisms linking neighborhoods to health, this study improves sociological understanding of the contextual and structural determinants of racial health inequality. The findings presented here suggest that it is not the mere patterning of individuals of different races across space that contributes to health inequality, but it is the flow of socioeconomic resources and power that fall along racial and spatial lines that contributes to the unequal distribution of disease risk. People of color in the United States are more likely than Whites to live in economically disadvantaged households and neighborhoods, which, as shown in the present study, contributes to disparities in physiological dysregulation and disease risk. However, this study finds that, after adjusting for differences in neighborhood economic conditions, living in racially segregated spaces may confer psychosocial benefits to Blacks, in particular, that may buffer against the physiological dysregulation

associated with racism-related chronic stressors. For example, in a study of Central Harlem, Mullings and Wali (2001) found that 99% of neighborhood survey respondents indicated that living in a predominately Black community was a positive aspect of their neighborhood. The authors described that living in a "Black neighborhood" conferred to residents feelings of community, access to social and cultural resources, and less exposure to racism and discrimination. In this way, living in a segregated neighborhood may offer psychosocial benefits to Blacks that protect against the physiological dysregulation associated with the stress of living in a racially stratified society.

By no means does this study offer support for continued racial segregation, but it rather suggests that the mere mixing of individuals of different races will not eliminate racial health disparities. In fact, promoting neighborhood racial diversity—without understanding of how racism contributes to health inequality through both psychosocial and socioeconomic means—may actually exacerbate health inequality. In other words, integration does not, necessarily, result in health equity.

REFERENCES

Ainsworth, J.W.. 2002. Why Does It Take a Village? The Mediation of Neighborhood Effects on Educational Achievement. *Social Forces* 81(1):117-52.

Aneshensel, C. S. and C.A. Sucoff. 1996. The neighborhood context of adolescent mental health. *Journal of Health and Social Behavior* 37:293-310.

Berkman, L.F., and L. Syme. 1979. Social Networks, Host Resistance, and Mortality: A Nine Year Follow-up Study of Alameda County Residents. *American Journal of Epidemiology* 109(2):186-204.

Bécaresa, L., J. Nazroob, and M. Stafforda. 2009. The buffering effects of ethnic density on experienced racism and health. *Health and Place* 15(3):700-08.

Berkman, L.F., V. Vaccarino, and T. Seeman. 1993. Gender Differences in Cardiovascular Morbidity and Mortality: The Contributions of Social Networks and Support. *Annals of Behavioral Medicine* 15(2-3):112-118.

Bronfenbrenner, U. 1986. Ecology of the family as a context for human development: Research perspectives. *Developmental Psychology* 22: 723-42.

Brooks-Gunn, J., G.J. Duncan, P.K. Klebanov, and N. Sealand. 1993. Do neighborhoods influence child and adolescent development? *American Journal of Sociology* 99(2):353-95.

Brummett, B.H., J.C. Barefoot, I.C. Siegler, N.E. Clapp-Channing, B.L. Lytle, H.B. Bosworth, R.B. Williams, B. Redford, and D.B. Mark. 2001. Characteristics of socially isolated patients with coronary artery disease who are at elevated risk for mortality. *Psychosomatic Medicine, 63*(2), 267-272.

Charles, C. Z. 2003. The dynamics of racial residential segregation. *Annual Review of Sociology* 167-207.

Christou, D. D., P.P. Jones, J. Jordan, A. Diedrich, D. Robertson, and R.D. Seals. 2005. Women have lower tonic autonomic support of arterial blood pressure and less effective baroreflex buffering than men. *Circulation* 111:494– 98.

Clarke, P., J. Morenoff, M. Debbink, E. Golberstein, M.R. Elliot, and P.M. Lantz. 2004. Cumulative Exposure to Neighborhood Context: Consequences for Health Transitions Over the Adult Life Course. *Research on Aging* 36(1):115-42.

Cohen, S.D., J. William, D.P. Skoner, B.S. Rabin, and J.M. Gwaltney Jr. 1997. Social ties and susceptibility to the common cold. *JAMA* 277(24):1940-44.

Collins, Chiquita A. and David R. Williams. 1999. "Segregation and Mortality: The Deadly Effects of Racism?" 14(3):495-523.

Daniel, M., S. Moore, and Y. Kestens. 2008. Framing the biosocial pathways underlying associations between place and cardiometabolic disease. *Health and Place* 14(2):117-32.

Diez-Roux, Ana V., et al. 1997. Neighborhood environments and coronary heart disease: a multilevel analysis. *American Journal of Epidemiology* 146(1): 48-63.

Duncan, G. J. 1994. Families and neighbors as sources of disadvantage in the schooling decisions of White and Black adolescents. *American Journal of Education* 103:20-53.

Eng, P.M.., Rimm, E.B.., Fitzmaurice, G., and Kawachi, I. 2002. Social Ties and Change in Social Ties in Relation to Subsequent Total and Cause-Specific Mortality and Coronary Heart Disease in Men. *American Journal of Epidemiology* 155(8):700-09.

Entwisle, D. R., Alexander, K. L., and Olson, L. S. 1994. The gender gap in math: Its possible origins in neighborhood effects. *American Sociological Review* 59:822– 838.

Entzel, P., E. Whitsel, A. Richardson, J. Tabor, S. Hallquist, J. Hussey, C. Halpern, and K.

Mullan Harris. 2009. "Add Health Wave IV Documentation: Cardiovascular and Anthropmetric Measures." Retrieved January 2, 2014 from http://www.cpc.unc.edu/projects/addhealth/data/guides/

Fang, Jing, Shantha Madhavan, William Bosworth and Michael H. Alderman. 1998. "Residential Segregation and Mortality in New York City." *Social Science and Medicine* 47(4):469-476.

Ge, X., Gene, B., Conger, R. D., Simons, R., and Murry, V. M. 2002. Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. *Developmental Psychology* 28:42-54.

George, L.K., Blazer, D.G., Hughes, D.C., and Fowler, N. 1989. Social support and the outcome of major depression. *The British Journal of Psychiatry* 154(4): 478-85.

Glaser, R. and J. K. Kiecolt-Glaser. 2005. Stress-induced immune dysfunction: implications for health. *National Review of Immunology* 5(3): 243-251.

Halpern, David. 1993. "Minorities and Mental Health." *Social Science and Medicine* 36(5):597-607.

Halpern, D. and Nazroo, J.Y. 1999. The ethnic density effect: results from a national community survey of England and Wales. *International Journal of Social Psychiatry* 46:34–46.

Halpern-Felsher, B., Connell, J. P., Spencer, M. B., Aber, J. L., Duncan, G. J., Clifford, E., Crichlow, W., Usinger, P., and Cole, S. S. 1997. Neighborhood and family factors predicting educational risk and attainment in African American and White children and adolescents. In J.Brooks-Gunn, G. J.Duncan, and J. L.Aber (Eds.), *Neighborhood poverty: Vol. 1*. Context and consequences for children (pp. 146– 173). New York: Russell Sage Foundation.

Hawkley, L.C., Masi, C.M., Berry, J.D., and Cacioppo, J.T. 2006. Loneliness Is a Unique Predictor of Age-Related Differences in Systolic Blood Pressure. *Psychology and Aging* 21(1):152-64.

Heikkinen, R.L,, and Kauppinen, M. 2004. Depressive symptoms in late life: a 10-year follow up. *Archives of gerontology and geriatrics, 38*(3), 239-250.

Hoffman, J. P. 2002. The community context of family structure and adolescent drug use. *Journal of Marriage and Family* 64:314-330.

Hu, Y. and Goldman, N. 1990. Mortality differentials by marital status: an international comparison. *Demography*, 27(2), 233-250.

Hull, P., Kilbourne, B., Reece, M., and Husaini, B. 2008. Community involvement and adolescent mental health: Moderating effects of race/ethnicity and neighborhood disadvantage. *Journal of Community psychology* 36(4):534-551.

Jackson, S. A., R. T. Anderson, N. J. Johnson and P. D. Sorlie. 2000. "The Relation of Residential Segregation to all-Cause Mortality: A Study in Black and White." *American Journal of Public Health* 90(4):615-617

Kopelman, P. 2007. Health risks associated with overweight and obesity. *Obesity Reviews* 8(S1):13-17.

Kowaleski-Jones, L. 2000. Staying out of trouble: Community resource and problem behavior among high-risk adolescents. *Journal of Marriage and the Family* 62:449-464.
Kramer, M. R. and C. R. Hogue. 2009. "Is Segregation Bad for Your Health?" *Epidemiologic Reviews* 31:178-194.

Leventhal, T. and J. Brooks-Gunn. 2000. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin* 126:309-337.

Litwak, E., Messeri, P., Wolfe, S., Gorman, S., Silverstein, M., and, Guilarte, M. 1989. Organizational theory, social supports, and mortality rates: A theoretical convergence. *American Sociological Review 54*: 49-66.

Logan, J. 2004. “The New Ethnic Enclaves in America’s suburbs.” A Report by The Lewis Mumford Center for Comparative Urban and Regional Research. Available at: http://mumford1.dyndns.org/cen2000/suburban/SuburbanReport/page1.html.

Massey, D. S. and N.A. Denton. 1993. *American apartheid: Segregation and the making of the underclass*. Harvard University Press.

McDade, T.W., L.C. Hawkley, and J.T. Cacioppo. 2006. Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: the Chicago health, aging, and social relations study. *Psychosomatic Medicine* 68(3): 376-81.

McEwen, B.S. 1998. Stress, Adaptation, and Disease - Allostasis and Allostatic Load. *Annals of the New York Academy of Sciences* 840(1): 33-44.

Mullings, Leith and Alaka Wali. 2001. *Stress and Resilience: The Social Context of Reproduction in Central Harlem.*Springer Science and Business Media.

Orth-Gomér, K., Rosengren, A., and Wilhelmsen, L. 1993. Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. *Psychosomatic Medicine* 55(1):37-43.

Osypuk, Theresa L., Ana V. D. Roux, Craig Hadley and Namratha R. Kandula. 2009. "Are Immigrant Enclaves Healthy Places to Live? the Multi-Ethnic Study of Atherosclerosis." *Social Science and Medicine* 69(1):110-120.

Osypuk, T. L. and D. Acevedo-Garcia. 2008. "Are Racial Disparities in Preterm Birth Larger in Hypersegregated Areas?" *American Journal of Epidemiology* 167(11):1295-1304.

Patel, Kushang V., Karl Eschbach, Laura L. Rudkin, M. K. Peek and Kyriakos S. Markides. 2003. "Neighborhood Context and Self-Rated Health in Older Mexican Americans." *Annals of Epidemiology* 13(9):620-628.

Penwell, L.M., and Larkin, K.T. 2010. Social Support and Risk for Cardiovascular Disease and Cancer: A Qualitative Review Examining the Role of Inflammatory Processes. *Health Psychology Review 4*(1):42-55.

Pickett, Kate E. and Richard G. Wilkinson. 2008. "People Like Us: Ethnic Group Density Effects on Health." *Ethnicity and Health* 13(4):321-334.

Richardson, L.J., J.M. Hussey, and K.L. Strutz. 2012. A life course perspective on maternal and child health. In: Kotch, Jonathan B. *Maternal and Child Health: Programs, Problems, and Policy in Public Health, 3rd ed,* pp. 65-85. Burlington, MA: Jones and Bartlett

Ross, C.E., J. Mirowsky, and K. Goldsteen. 2001. Powerlessness and the amplification of threat: Neighborhood disadvantage, disorder, and mistrust. *American Sociological Review* 66:568-91.

Ross, C.E., J.R. Reynolds, and L.G. Karlyn. 2000. The contingent meaning of neighborhood stability for residents' psychological well-being. *American Sociological Review* 65: 581-97.

Rozanski, A., Blumenthal, J. A., and Kaplan, J. 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 99:2192-2217.

Sampson, R. 2001. How do communities undergird or undermine human development? Relevant contexts and social mechanisms. Pp. 3-30 in A. Booth and A.C. Crouter (eds.) *Does it take a village? Community effects on children, adolescents, and families*. Mahwah, NJ: Lawrence Erlbaum Associates.

Sampson, R., J. Morenoff, and DT. Gannon-Rowley. 2002. Assessing “neighborhood effects”: Social processes and new directions in research. *Annual Review of Sociology* 28:443-78.

Sesso HD, Stampfer MJ, Rosner B, Hennekens CH, Gaziano JM, Manson JE, and Glynn RJ. 2000. Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in Men. *Hypertension* 36(5):801-7.

Selye, H. 1974. *Stress Without Distress*. Philadelphia: J.B. Lippincott.

Sharkey, P. 2012. An Alternative Approach to Addressing Selection Into and Out of Social Settings: Neighborhood Change and African American Children’s Economic Outcomes. *Sociological Methods Research* 41(2):251-293.

Shaw, C.R. and H.D. McKay. 1942. *Juvenile delinquency and urban areas*. Chicago, IL: University of Chicago Press.

Subramanian, SV, Dolores Acevedo-Garcia and Theresa L. Osypuk. 2005. "Racial Residential Segregation and Geographic Heterogeneity in black/white Disparity in Poor Self-Rated Health in the US: A Multilevel Statistical Analysis." *Social Science and Medicine* 60(8):1667-1679.

Thoits, P.A. 2011. Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior, 52*(2), 145-161.

Van Uum, S.H.M., Lender, J.W.M., and Hermus, A.R.M.M. 2004. Cortisol, 11β-hydroxysteroid dehydrogenases, and hypertension. *Seminars in Vascular Medicine* 4(2):121– 28.

Vogt Yuan, Anastasia S. 2007. "Racial Composition of Neighborhood and Emotional Well Being." *Sociological Spectrum* 28(1):105-129.

Wen, M., D.S. Lauderdale, and N.R. Kandula. 2009. Ethnic Neighborhoods in Multi-Ethnic America, 1990-2000: Resurgent Ethnicity in the Ethnoburbs? *Social Forces* 88(1):425-60.

Wheaton, B. and P. Clarke. 2003. Space meets time: Integrating temporal and contextual influences on mental health in early adulthood. *American Sociological Review* 68:680-706.

White, K. and L. N. Borrell. 2006. "Racial/ethnic Neighborhood Concentration and Self Reported Health in New York City." *Ethnicity and Disease* 16(4):900-908.

Whitsel, E., C. Cuthbertson, J. Tabor, A. Potter, M. Wener, L. Killeya-Jones, and K. Mullan

Harris. 2012. “Add Health Wave IV Documentation: Measures of Inflammation and Immune Function.” Retrieved January 2, 2014 from http://www.cpc.unc.edu/projects/addhealth/data/guides/

Whitworth, J. A., Brown, M. A., Kelly, J. J., and Williamson, P. M. 1995. Mechanisms of cortisol induced hypertension in humans. *Steroids* 60:76– 80.

Wickrama, K. and C. Bryant. 2003. Community Context of Social Resources and Adolescent Mental Health. *Journal of Marriage and Family* 65(4): 850-66.

Wickrama, KAS, Samuel Noh and Chalandra M. Bryant. 2005. "Racial Differences in Adolescent Distress: Differential Effects of the Family and Community for Blacks and Whites." *Journal of Community Psychology* 33(3):261-282.

Williams, D.R., and C. Collins. 2001. Racial residential segregation: a fundamental cause of racial disparities in health. *Public health reports* 116(5): 404.

Williams, D.R. and P.B. Jackson. 2005. Social sources of racial disparities in health. *Health Affairs* 24(2):325-334.

Williams, D.R. and S.A. Mohammed. 2009. Discrimination and racial disparities in health: evidence and needed research. *Journal of Behavioral Medicine* 32(1):20-47.

Williams, D. R., Mohammed, S. A., Leavell, J., and Collins, C. 2010. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Annals of the New York Academy of Sciences*, *1186*(1), 69-101.

Williams, D.R., H.W. Neighbors, and J.S. Jackson. 2003. Racial/ethnic discrimination and health: findings from community studies. *American Journal of Public Health* 93(2):200-208.

Yang, Y.C., McClintock, M.K., Kozloski, M., and Li, T. 2013. Social isolation and adult mortality: the role of chronic inflammation and sex differences. *Journal of Health and Social Behavior* 54(2):183-203.

Yang, Y., and Kozloski, M. 2012. Change of Sex Gaps in Total and Cause-Specific Mortality Over the Life Span in the United States. *Annals of Epidemiology* 22(2):94-103.

Table 3.1. Descriptive Statistics, Weighted (N=6,248)

	Mean or proportion	SD
Outcome measures[a]		
Metabolic dysregulation	2.614	1.243
BMI	29.165	7.573
Neighborhood characteristics		
…during adolescence[b]		
Neighborhood economic deprivation	0.855	1.465
Neighborhood racial density	79.744	29.970
…during young adulthood[a]		
Neighborhood economic deprivation	0.679	1.035
Neighborhood racial density	72.422	29.234
Individual characteristics		
Race		
White	0.757	-
Black	0.147	-
Hispanic	0.096	-
Gender (1=female)	0.509	-
Age[b]	14.663	1.802
Family SES during adolescence[b]	0.032	0.761
SES during young adulthood[a]	0.029	0.808
Psychosocial factors[a]		
Perceived discrimination (1=ever experienced discrimination)	0.697	-
Social isolation (1=low integration)	0.541	-

a: Measured during young adulthood (Wave IV)

b: Measured during adolescence (Wave I)

Table 3.2. Outcome and Neighborhood Measures, by Race (N=6,248)

	Whites	Blacks	Hispanics	p-value
Outcome measures[a]				
Metabolic dysregulation	2.608	2.671	2.569	0.439
BMI	28.795	30.824	29.551	<0.001
Neighborhood characteristics				
…during adolescence[b]				
Neighborhood economic deprivation	0.552	2.136	1.288	<0.001
Neighborhood racial density	91.481	53.603	27.057	<0.001
…during young adulthood[a]				
Neighborhood economic deprivation	0.526	1.348	0.865	<0.001
Neighborhood racial density	83.169	44.512	30.226	<0.001

a: Measured during young adulthood (Wave IV)

b: Measured during adolescence (Wave I)

c: p-value of chi-square test of difference between in means between racial groups

Table 3.3. Neighborhood Characteristics and Metabolic Dysregulation: Multilevel Regression Models, 1994-2008

	Metabolic Dysregulation during Young Adulthood			
	Model 1	Model 2	Model 3	Model 4
	Coeff (SE)	Coeff (SE)	Coeff (SE)	Coeff (SE)
Neighborhood characteristics				
…during adolescence (Wave I)				
Neighborhood economic deprivation	0.027			
	(0.021)			
Neighborhood racial density	0.02			
	(0.015)			
Black x neighborhood racial density	-0.026			
	(0.017)			
Hispanic x neighborhood racial density	-0.023			
	(0.016)			
Neighborhood racial density^2	-0.012			
	(0.010)			
Black x neighborhood racial density^2	0.019			
	(0.013)			
Hispanic x neighborhood racial density^2	0.011			
	(0.012)			
…during young adulthood (Wave IV)				
Neighborhood economic deprivation		0.025	0.024	0.023
		(0.030)	(0.030)	(0.029)
Neighborhood racial density		-0.020*	-0.020*	-0.019*
		(0.009)	(0.009)	(0.009)
Black x neighborhood racial density		0.020+	0.020+	0.018+
		(0.011)	(0.011)	(0.011)
Hispanic x neighborhood racial density		0.017	0.017	0.016
		(0.013)	(0.013)	(0.014)

Neighborhood racial density^2		0.016*	0.016*	0.015*
		(0.007)	(0.007)	(0.006)
Black x neighborhood racial density^2		-0.015+	-0.015+	-0.013
		(0.009)	(0.009)	(0.009)
Hispanic x neighborhood racial density^2		-0.014	-0.013	-0.013
		(0.011)	(0.011)	(0.012)
Individual characteristics				
Race (white is reference)				
Black	0.878	-0.507	-0.506	-0.470
	(0.573)	(0.338)	(0.338)	(0.333)
Hispanic	0.833	-0.421	-0.419	-0.409
	(0.552)	(0.389)	(0.389)	(0.390)
Gender (1=female)	-0.374***	-0.358***	-0.358***	-0.358***
	(0.041)	(0.044)	(0.044)	(0.045)
Age	0.061***	0.064***	0.064***	0.065***
	(0.014)	(0.015)	(0.015)	(0.015)
Family SES during adolescence	-0.139***			
	(0.029)			
SES during young adulthood		-0.136***	-0.135***	-0.125***
		(0.031)	(0.031)	(0.033)
Psychosocial factors				
Perceived discrimination (1=rarely, sometimes, or often)			0.043	
			(0.047)	
Social isolation (1=isolated)				0.111*
				(0.045)
Intercept	1.085+	2.306***	2.270***	2.200***
	(0.592)	(0.457)	(0.450)	(0.455)
Model Fit Statisics				
AIC	3687060	3682527	3682196	3579500
BIC	3687161	3682628	3682304	3579607

N (unweighted)	6,248	6,248	6,248	6,248

***p<0.001, **p<0.01, *p<0.05, +p<0.1

Note: all models are survey design adjusted and weighted

Table 3.4. Neighborhood Fixed Effects Models, 1994-2008

	Metabolic Dysreguation	Body Mass Index
	Model 1	Model 2
	Coeff (SE)	Coeff (SE)
Neighborhood characteristics		
Δ Neighborhood economic deprivation from Wave I-IV	0.003	0.252†
	(0.026)	(0.137)
Δ Neighborhood racial density from Wave I-IV	-0.026**	-0.104*
	(0.009)	(0.049)
Black x Δ neighborhood racial density	0.029**	0.173*
	(0.010)	(0.072)
Hispanic x Δ neighborhood racial density	0.029*	0.045
	(0.013)	(0.086)
Δ Neighborhood racial density^2	0.020**	0.075*
	(0.006)	(0.038)
Black x Δ neighborhood racial density^2	-0.022**	-0.138*
	(0.007)	(0.059)
Hispanic x Δ neighborhood racial density^2	-0.021*	-0.011
	(0.010)	(0.079)
Individual characteristics		
Race (white is reference)		
Black	-0.010	0.970
	(0.086)	(0.669)
Hispanic	0.044	1.127*
	(0.100)	(0.569)
Gender (1=female)	-0.350***	0.256
	(0.046)	(0.291)
Age	0.059**	0.205*
	(0.015)	(0.101)
Family SES during adolescence	-0.077*	-0.607**
	(0.038)	(0.229)
SES during young adulthood	-0.102**	-0.270
	(0.033)	(0.264)

***p<0.001, **p<0.01, *p<0.05, †p<0.1

Note: N=6248; outcomes measured in young adulthood, at Wave IV; all models are survey design adjusted and weighted and adjust for time-invariant neighborhood characteristics.

Table 3.5. Neighborhood Characteristics and BMI: Multilevel Regression Models, 1994-2008

	BMI during Young Adulthood			
	Model 1 Coeff (SE)	Model 2 Coeff (SE)	Model 3 Coeff (SE)	Model 4 Coeff (SE)
Neighborhood characteristics				
…during adolescence (Wave I)				
Neighborhood economic deprivation	0.153			
	(0.174)			
Neighborhood racial density	0.100+			
	(0.056)			
Black x neighborhood racial density	-0.139			
	(0.092)			
Hispanic x neighborhood racial density	-0.064			
	(0.073)			
Neighborhood racial density^2	-0.073+			
	(0.040)			
Black x neighborhood racial density^2	0.095			
	(0.087)			
Hispanic x neighborhood racial density^2	-0.007			
	(0.073)			
…during young adulthood (Wave IV)				
Neighborhood economic deprivation		0.408**	0.396**	0.391**
		(0.141)	(0.138)	(0.134)
Neighborhood racial density		-0.082	-0.080	-0.070
		(0.059)	(0.059)	(0.059)
Black x neighborhood racial density		0.166*	0.165*	0.162*
		(0.080)	(0.079)	(0.080)
Hispanic x neighborhood racial density		0.063	0.060	0.045
		(0.092)	(0.091)	(0.094)

Neighborhood racial density^2		0.054	0.053	0.041
		(0.045)	(0.045)	(0.045)
Black x neighborhood racial density^2		-0.143*	-0.142*	-0.140*
		(0.067)	(0.067)	(0.066)
Hispanic x neighborhood racial density^2		-0.062	-0.058	-0.044
		(0.083)	(0.082)	(0.085)
Individual characteristics				
Race (white is reference)				
Black	5.551*	-2.775	-2.767	-2.649
	(2.280)	(2.586)	(2.562)	(2.626)
Hispanic	3.617+	-1.397	-1.375	-1.129
	(1.996)	(2.226)	(2.211)	(2.289)
Gender (1=female)	-0.139	-0.036	-0.024	-0.105
	(0.283)	(0.280)	(0.278)	(0.263)
Age	0.198*	0.223**	0.226**	0.232**
	(0.082)	(0.084)	(0.084)	(0.082)
Family SES during adolescence	-0.900***			
	(0.199)			
SES during young adulthood		-0.669*	-0.647*	-0.732**
		(0.274)	(0.273)	(0.276)
Psychosocial factors				
Perceived discrimination (1=rarely, sometimes, or often)			0.623*	
			(0.264)	
Social isolation (1=isolated)				-0.268
				(0.269)
Intercept	23.06***	28.29***	27.78***	28.12***
	(2.416)	(2.331)	(2.366)	(2.339)
Model Fit Statisics				
AIC	7897454	7892485	7655394	7894327
BIC	7897555	7892593	7655501	7894428

N (unweighted)	6,248	6,248	6,248	6,248

***p<0.001, **p<0.01, *p<0.05, †p<0.1

Note: all models are survey design adjusted and weighted

Figure 3.1. Theoretical Model

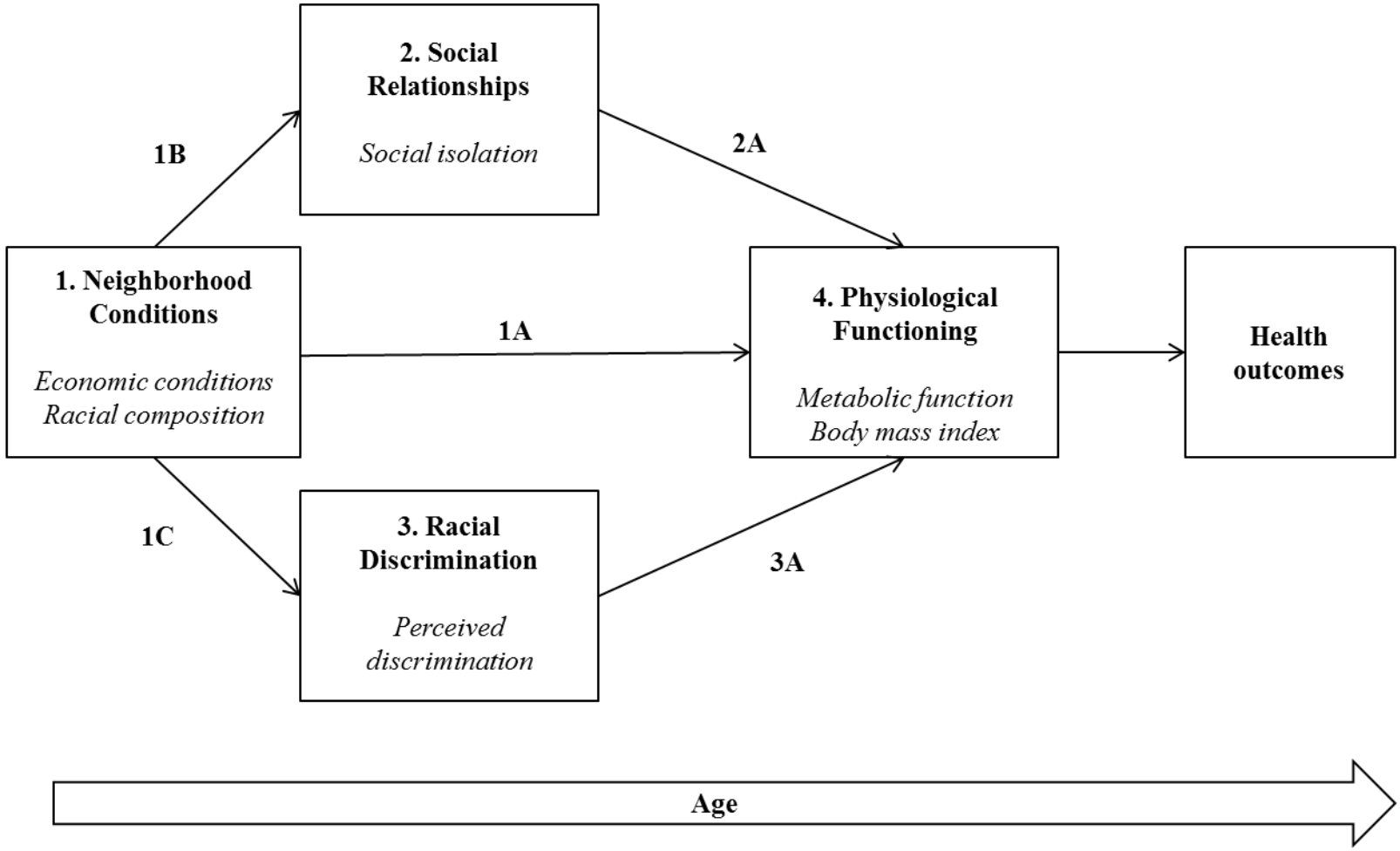
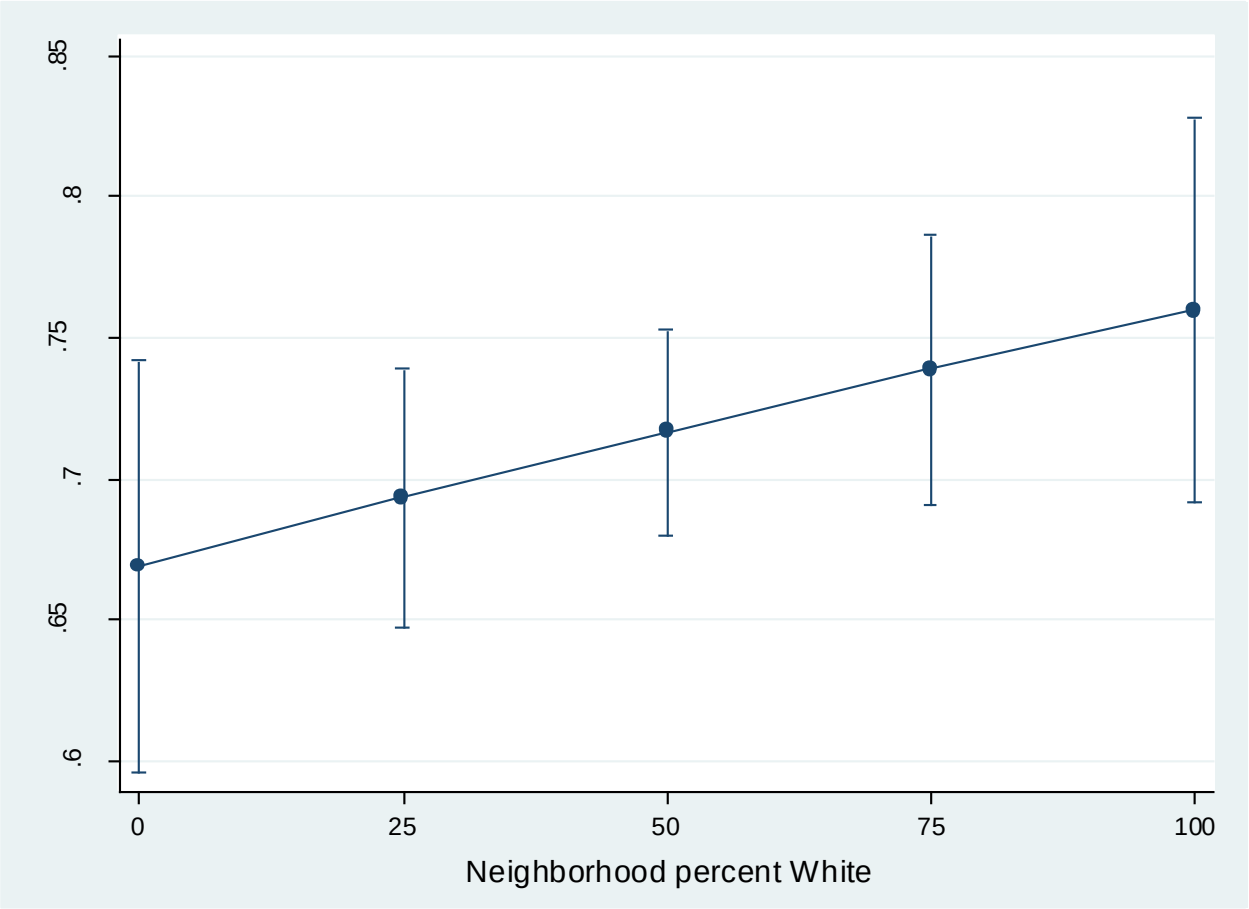


Figure 3.2. Neighborhood Racial Composition and Black Reports of Discrimination



CHAPTER 4: LIFE COURSE PSYCHOSOCIAL STRESS EXPOSURE AND BLACK-WHITE HEALTH DISPARITIES IN OLD AGE

Abstract

Research suggests that that Blacks may experience accelerated biological aging relative to Whites, due in large part to Blacks' cumulative exposure to repeated stress and high-effort coping with stressors across the life course. Despite a growing body of research linking psychosocial stress to health, critical gaps in our understanding of the role of stress exposure in racial health inequities remain. Using nationally representative, longitudinal data from the Health and Retirement Study (2004-2012), this paper documents how structural racism patterns individual psychosocial stress exposure; examines how exposure to psychosocial stressors across the life course impacts markers of physiological and psychological well-being; and assesses whether racial differences in stress exposure contribute to Black-White health disparities in late life. Findings reveal that Blacks report higher levels of multiple forms of generalized and discrimination-related stress—including more stressful life events, financial strain, ongoing chronic stress, and racial discrimination—than Whites across the life course. I also find that while acute stressors show modest associations with some of the outcomes, exposure to chronic stressors across the life course is consistently associated with the greater inflammatory, metabolic, and depressive risk. Formal mediation tests indicate that racial disparities in stress exposure contribute to Black-White disparities in markers of physical and mental health in late life, indicating that differential stress exposure by race is a critical, yet largely underestimated, source of racial health disparities at older ages. By examining the role of multiple, diverse measures of subjective and objective lifetime stress exposure in racial health inequities, findings

from this study elucidates the links between structural racial inequality, psychosocial processes, and health.

Introduction

A body of research on the age patterning of Black-White health inequality documents a divergence of health disparities with age (Geronimus et al. 2001; Shuey and Willson 2008), whereby Blacks in the U.S. experience earlier onset of disease and steeper increases in disease incidence with age than Whites. The widening racial gaps in morbidity and mortality has led some scholars to argue that Blacks may experience accelerated biological aging relative to Whites (Clarke et al. 2014; Geronimus et al. 2010; Rewak et al. 2014). A growing body of research suggests that Blacks may be biologically "older" than Whites of similar chronological age, due in large part to Blacks' cumulative exposure to repeated stress and high-effort coping with stressors (Clarke et al. 2014; Geronimus et al. 2001; Geronimus 1992). Given their relative positioning in both the social class and racial hierarchies, Blacks in the U.S. are exposed to greater levels of structural disadvantage (Pager and Shepherd 2008; Taylor et al. 2011) and report higher levels of related psychosocial stress (Adkins et al. 2009; Turner, Wheaton, and Lloyd 1995; Turner 2013) than Whites. Research suggests that these racial differences in stress exposure may contribute to Blacks' accelerated biological aging relative to Whites and play a critical, yet largely underestimated role, in racial health inequality.

Despite a large body of research linking psychosocial stress to health, critical gaps in our understanding of the role of stress exposure in the production of racial health inequality remain. In particular, few studies incorporate multiple measures of both acute and chronic and discrimination-related and generalized-social stress into their analyses or consider how stress exposure across the life course may impact trajectories of health and well-being. Further, while studies have examined how stress may impact general indicators of physical health, less is

known about how stress impacts biomarkers of physiological functioning and markers of psychological risk to ultimately impact health and disease outcomes. Using longitudinal, nationally-representative data from the Health and Retirement Study (HRS), the current study examines whether and how exposure to multiple forms of both acute and chronic, discrimination-related and generalized social stress affects markers of biological and psychological well-being and ultimately contribute to racial disparities in health and mortality. By expanding the conceptualization and operationalization of "stress" to include a variety of psychosocial exposures across the life course, this study provides new evidence of the links between macro-level racial inequality, individual-level psychosocial well-being, and micro-level physiological and psychological functioning.

Background

Stress and Health

Research on the health consequences of stress surged in the mid-twentieth century, following the groundbreaking work of Selye (1974), who is credited as being one of the first researchers to identify stress as a key contributor to health. Selye's interest in the links between stress and health arose following a number of experiments in which he subjected rats to a variety of physical and emotional stimuli, including bright lights, loud noises, extreme temperatures, and chronically frustrating situations. Selye observed that, in response to these stimuli, the rats exhibited a set of physical reactions that were non-specific in character and consisted of a number of consistent physiological changes, including enlargement of the adrenal cortex; atrophy of the thymus, spleen, lymph nodes, and all other lymphatic structures in the body; and bleeding gastric and duodenal ulcers. Following these experiments, Selye argued that these stimuli served as stressors to the rats and that exposure to the stressors initiated a set of health-related physiological changes. As a result of this work, Selye (1974) also offered the first

conceptual definitions of stress as both "the non-specific response of the body to any demand for change" and "essentially the rate of all the wear and tear caused by life." In addition to defining stress, Selye (1974) further identified three stages of physiological reaction to stressors: alarm, resistance, and eventual exhaustion. Importantly, he found that the exhaustion stage—categorized as the depletion of bodily resources following cumulative, repeated, and chronic stress exposure—to have particularly strong links to physiological dysregulation and disease risk.

Researchers examining the physiological consequences of stress in human subjects have since confirmed Selye's findings, with more than forty decades of research documenting the substantial effects of psychosocial stress on health (Cohen et al. 2012; Epel and Lithgow 2014; Thoits 2010). Biological studies have built on the groundbreaking work of Seyle (1974) by examining the physiological pathways through which stress exposure "gets under the skin" to affect health and disease risk in human populations. Research finds that, in response to stress exposure, the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) respond by secreting hormones to up-regulate physiological functioning (McEwen and Stellar 1993; McEwen 1998). Whereas up-regulation of immune, neuroendicine, metabolic, and cardiovascular changes in response to immediate threats and infections is necessary to protect health and well-being, long-term activation of these systems in response to repeated stress exposure can harm health and increase mortality risk over time. Repeated activation of the body's stress response systems results in what Seyle (1974) termed "exhaustion," where the bodily mechanisms used to defend against stressors become inefficient and ineffective, resulting in allostatic load, physiological dysregulation, and, ultimately, increased risk of disease and mortality (Cohen et al. 2012; Glaser and Kiecolt-Glaser 2005; McEwen and Stellar 1993; McEwen 1998).

Structural Inequality and Stress

Paralleling biologists' studies of the physiological pathways through which stress affects human health, sociological research has examined the social patterning of stress exposure and improved understanding of how differential exposure and differential vulnerability to stress contribute to population health inequality. While, at least superficially, stress can be seen as a highly individualized psychosocial process, stress exposure is socially patterned and linked to individuals' locations within social hierarchies. As Pearlin (1999) described, "People's standing in the stratified orders of social and economic class, gender, race, and ethnicity have the potential to pervade the structure of their daily existence . . . shaping the contexts of people's lives, the stressors to which they are exposed, and the moderating resources they possess" (398-99). Stress exposure corresponds, in large part, to social status, such that socially disadvantaged groups in society are exposed to more negative life events and traumas and greater levels of chronic strain than advantaged groups (Pearlin 1999). Further, evidence suggests that differential stress exposure partially explains disparities in morbidity and mortality by race, as well as other axes of social stratification (Turner 2013; Turner, Wheaton, and Lloyd 1995).

With regard to racial health inequities, specifically, recent research documents linkages between structural racism and individual exposure to social stressors. For one, the stress inherent in a racially stratified society can be perceived, in and of itself, as stressful, such that people of color may recognize their location within the racial hierarchy as characteristically and fundamentally challenging, disheartening, and wearing (Geronimus et al. 2006). Further, as Pearlin (1999) suggests, racism also patterns exposure to a variety of acute and chronic stressors, such that Blacks report more traumatic events (Lu and Chen 2004; Umberson et al. 2017), chronic strains (Pearlin 1999; Thoits 1995; Turner and Avison 2003), and discrimination (Clark et al. 1999; Geronimus et al. 2006) than Whites. Social scientists hypothesize that Blacks'

greater exposure to stress and stigmatization and prolonged use of high-effort coping mechanisms may contribute to disproportionate physiological deterioration and accelerated biological aging, relative to Whites (Geronimus et al. 2006; Geronimus et al. 2010).

Despite the two robust bodies of recent literature—one on the relationship between stress and health and the other on the links between social stratification and stress exposure—few studies integrate these bodies of work to examine how social disparities in psychosocial stress exposure contribute to population health inequities. Further, early research on the role of stress exposure in social disparities in health, generally, and racial health inequities, specifically, produced largely inconsistent findings (Hatch and Dohrenwend 2007). The weak findings linking stress exposure to population health disparities was due in large part to limited conceptualizations and operationalizations of stress exposure, which were generally restricted to checklists or counts of negative life events. For decades, researchers used composite scales of "traumatic life events" or "stressful events" to capture the impacts of stress on health outcomes. While some of these events have been linked to adverse outcomes, the association between the number of stressful life events an individual reports and health is still relatively weak (Thoits 1983; Thoits 2010). The weak associations between measures of stressful life events and health outcomes led many scholars to hypothesize that there were many stressful experiences and situations that are not captured by life events checklists (Turner, Wheaton, and Lloyd 1995; Wheaton 1999). In particular, persistent, ongoing, and chronic stressors and strains—such as financial difficulties, exposure to discrimination, or relationship difficulties—have long been overlooked in sociological studies of stress (Thoits 2010), despite the overwhelming body of literature suggesting that these chronic stressors may be have particularly harmful effects on health.

Unsurprisingly, as researchers expanded their examinations to chronic, ongoing, and cumulative stressors, studies began to document a stronger, more consistent social patterning of stress, where people of color and socioeconomically disadvantaged groups were exposed to higher levels of chronic stress and experienced greater cumulative stress burdens than more socially advantaged groups (Pearlin 1999; Thoits 1995; Turner and Avison 2003). Further, recent studies document that differential stress exposure partially accounts for health disparities in self-rated health, functional limitations, and several physical health conditions (Ensel and Lin 2000; House et al. 1994; Kosteniuk and Dickinson 2003; Lantz et al. 2005; Lin and Ensel 1989), as well as disparities in psychological distress, depression, and alcohol and substance abuse disorders (Avison, Ali, and Walters 2007; Seeman and Crimmins 2001; Turner, Wheaton, and Lloyd 1995; Turner and Lloyd 1999; Turner and Avison 2003).

Gaps in the Literature

Despite a burgeoning literature on the contribution of psychosocial stress exposure to racial disparities in health and aging, three critical gaps in the literature remain. First, most studies measure stress exposure using one or two indicators of stress, such as everyday racial discrimination or traumatic life events. In the face of a widespread recognition that a more comprehensive measurement of stress may aid in better explaining population disparities between socially advantaged and disadvantaged population subgroups (Pearlin 1999; Turner et al. 1995), few studies measure multiple forms of both acute and chronic stress exposure, which suggests that the role of stress in Black-White disparities in health and biological aging has been underestimated (Turner, Wheaton, and Lloyd 1995; Turner 2013). For example, studies document that, compared to checklists of stressful life events that account for 1-12% of the variance in psychological distress, cumulative measures of stress burden that include stressful life events, lifetime traumas, and chronic strains explain 25-40% of the variance in psychological

distress and depressive symptoms (Turner et al. 1995; Wheaton 1999). For this reason, more research utilizing comprehensive measures of stress exposure is needed. By including only measures of stressful events and traumas in empirical studies, both racial differences in psychosocial stress exposure and the contribution of psychosocial stress to racial disparities in health may be critically underestimated.

Second, there is limited evidence of how the accumulation of stressors across the life course, as indicated by measures such as lifetime traumas and lifetime discrimination, affect markers of health and well-being. The stress process model asserts that acute stressors—such as a job loss—may give rise to additional chronic stressors—such as ongoing financial strain or housing difficulties (Pearlin 1999). Further, stress exposure can multiply over the life course, such that stress in childhood gives rise to stressful experiences in adolescence, which generates additional stress in adulthood (Thoits 2010). Studies document, for example, that adults who reported one or more traumatic events in childhood also reported greater numbers of lifetime and recent stressful events (Horwitz et al. 2001; Turner and Avison 2003; Wheaton 1999). However, few studies examine how the accumulation of stress exposure across the life course impacts health over time. As a result, it remains unknown how the accumulation of stress exposure across the life course impacts markers of physiological functioning and psychological well-being and whether racial differences in lifetime exposure to psychosocial stress contribute to racial health disparities.

Finally, there is a lack of research on the biological mechanisms underlying the associations between racial inequality, psychosocial stress exposure, and health outcomes. While a number of studies assess the associations between stress exposure and singular physical and mental health outcomes, less is known about how exposure to stress affects bodily systems to

predispose individuals to host of poor outcomes. An analytic strategy that considers the effects of stress on multiple, pre-disease markers of physiological and psychological well-being would be consistent with the non-specificity hypothesis that guides the stress process model, whereby stress exposure elicits a range of physiological and psychological changes that, over time, serve to increase disease risk from a host of causes (Seyle 1974). In this way, the integration of biomarkers of physiological stress response will improve understanding of the biological mechanisms through which stress exposure impacts disease outcomes (Turner 2013). Further, by identifying the biological systems most affected by social stress, policy and prevention efforts may be better targeted to address the predecessors of multiple forms of disease and disability (Aneshensel 2009).

Aims of the Present Study

This paper seeks to understand the contribution of psychosocial stress exposure across the life course to disparities in markers of physiological functioning and psychological well-being in late life, with particular attention to the role of psychosocial stress in the production of Black-White health inequities. Specifically, this study examines how racial differences in both chronic and acute, discrimination-related and generalized psychosocial stress exposure across the life course contribute to Black-White disparities in three critical markers of disease risk. This study tests the following hypotheses:

Hypothesis 1: Blacks will exhibit higher levels of physiological inflammation, metabolic dysfunction, and depressive symptom than Whites.

Hypothesis 2: Blacks will report higher levels of psychosocial stress exposure than Whites.

Hypothesis 3: Exposure to psychosocial stress will be associated with increased levels of physiological inflammation, metabolic dysfunction, and depressive risk.

Hypothesis 4: Racial differences in stress exposure will partially account for racial disparities in biological aging, physiological dysregulation, and depressive risk.

Data and Methods

Data and Sample

Data for this study come from five waves of the Health and Retirement Study (HRS), a nationally representative, longitudinal study of adults aged 50 years and older in the U.S. Started in 1992, the HRS collects information about the income, wealth, physical health, cognitive functioning, and social lives of older adults, primarily through the use of in-home interviews. The HRS sample was selected using a multi-stage area probability design that included oversamples of African Americans, Hispanics, and Floridians. More information about the design of HRS can be found in Heeringa and Connor (1995). This study will utilize data from five waves of the HRS: the 2004, 2006, 2008, 2010, and 2012 waves.

As part of its data collection, the HRS collected blood-based biomarkers on a random half of the sample in 2006, and the other half of the sample provided biomarker data in 2008. These sample respondents were then re-interviewed in 2010 and 2012, respectively, when they again provided blood samples for biomarker data, creating a four year interval between biomarker collection. The analytic sample for this study includes Black and White respondents who provided biomarker data in 2010-2012 and who have complete data for the variables used in the analyses. Most excluded survey respondents had missing data on the stressor measures and/or the outcomes. Compared to those included in the analyses, those excluded from the analytic samples were more likely to be Black ($p<0.001$) and older age ($p<0.001$) and had lower levels of household wealth ($p=0.001$) and completed education ($p<0.001$). To the extent that the analytic sample is, in general, younger and more socially advantaged than those excluded from the analysis, the results presented in this study may be conservative.

Measures

Outcomes

This study will include three outcomes that represent critical markers of health and disease risk across the life course: C-reactive protein (CRP), metabolic dysregulation, and depressive symptoms. All outcomes were measured in 2010-2012. *CRP*, a marker of inflammatory response and immune function, is an acute phase protein produced by the liver. Elevated levels of circulating CRP indicate systemic inflammation (Finch 2010). Studies document a relationship between inflammation and health risk, including prospective associations of CRP with higher rates of coronary heart disease, stroke, and mortality (Emerging Risk Factors Collaboration 2010; Harris et al. 1999; Ridker et al. 2000). Because of a skewed distribution, I include CRP as a log transformed measure in all multivariate models.

Consistent with previous studies (Yang, Li, and Ji 2013), I construct a composite measure of *metabolic dysregulation*, which indicates overall levels of metabolic burden using clinical markers of metabolic syndrome: systolic and diastolic blood pressure, Hba1c, waist circumference, total cholesterol, HDL cholesterol, and body mass index. For each individual measure, I construct a dummy measure where 1 indicates high risk, with cut points for high risk defined by clinical practice. I then summed the scores from each of the markers to construct the index of overall metabolic dysregulation, which is modeled as a continuous measure ranging from 0 (low metabolic dysregulation) to 6 (high metabolic dysregulation).

Finally, I also include a count measure of *depressive symptoms* based on the Center for Epidemiological Studies Depression Scale (CES-D). While often used as a marker of mental health, depressive symptoms have also been linked to declines in physical health (Kiecolt-Glaser and Glaser 2002; Penninx et al. 1998). Research shows that depressive states can stimulate the production of physiological stress response hormones, which in turn influences a host of diseases

and conditions, including cardiovascular disease, diabetes, and cancer (Kiecolt-Glaser and Glaser 2002). In addition, depressive states can down-regulate the body's immune function, making individuals with increased depressive symptoms more prone to prolonged infection and delayed wound healing (Kiecolt-Glaser and Glaser 2002).

Key Explanatory Variables

The key explanatory variables for this study include a variety of measures indicating psychosocial stress exposure. Measures of stress exposure were collected in a leave behind questionnaire, and I utilize stress exposure data from the 2004, 2006, and 2008 waves of the HRS, though respondents did not answer questions about stress exposure at every one of these waves. This study includes two categories of stressors. First, I constructed standardized count indices of the following domain-specific stressors, corresponding to stress exposure in various domains of social life: *lifetime traumas, recent stressful life events, financial strain, ongoing chronic strains, everyday discrimination, and major lifetime discrimination*. These measures of stress include both recent and lifetime stress exposure and discrimination-related and generalized stressors. For detailed information on the construction of the scales, see Table 4.1.

Next, I created composite, latent measures of chronic and acute stress exposure using confirmatory factor analysis. Given that previous research has indicated that chronic strains may have stronger associations with health and play a more prominent role in producing racial health inequities than acute stressors, creating composite measures of overall chronic and acute stress exposure allows to me better understand how these categories of psychosocial exposure impact Black-White health disparities. To create the composite measure of chronic stress, I included the measures of financial strain, ongoing chronic strains, and everyday discrimination in the confirmatory factor analyses. The composite measure of acute stress included the measures of lifetime traumas, stressful life events, and major life discrimination. Results from the

confirmatory factor analyses indicated that the measures of chronic stress exposure comprise a single underlying factor structure (Eigenvalue=1.704). Similarly, the measures of acute stress exposure loaded on a single factor (Eigenvalue=1.457). For both composite measures, all of the individual measures comprising the respective composite measure had strong item-rest correlations, indicating good internal consistency.

Other Measures

The racial disparity in the outcomes is measured by a dummy variable, where "1" indicates Black. Other covariates include sociodemographic measures such as age (continuous) and gender (1=female) and measures of socioeconomic status, including education (1=<high school, 2=high school, 3=some college, 4=bachelor's degree or higher), total household wealth (continuous), and marital status (1=married or cohabiting).

Analytic Strategy

Analytic methods for this study include a combination of descriptive analyses, OLS (for log CRP and metabolic dysregulation outcomes) and negative binomial (for the depressive symptoms outcome) regression models, and longitudinal lagged dependent variable models. First, I assess Black-White disparities in makers of health and stress exposure using descriptive statistics, using t-tests to formally test for race differences in inflammation, metabolic dysregulation, depressive symptoms, and stress exposure.

Next, I use multivariate regression analyses to model the associations between the stressors and the outcomes. The multivariate regression models also allow me to assess how the domain-specific and composite stressors impact the markers of health and whether racial differences in stress exposure are, in fact, determinants of Black-White disparities in the three outcomes. In the OLS and negative binomial regression analyses, I exploit the temporal

sequencing of the data by modeling the outcomes as a function of stress exposure in earlier waves (i.e., outcomes measured at t_2 regressed on stress measured at t_1).

Finally, I use lagged dependent variable models, also known to as residual change models (Allison 1990), to examine how stress exposure at baseline affects *change* in the depressive risk over time. Research suggests that individuals have relatively stable levels of CRP over shorter durations of time (Macy, Hayes, and Tracy 1997), particularly later in life (Kizer et al. 2011). Similarly, many markers of metabolic risk remain relatively stable in late life (Wills et al. 2011; Wilson et al. 1994) and changes in markers of metabolic functioning having varying consequences for mortality risk at older ages (Stevens et al. 1998). Given the short amount of time between the waves of data collection—there were just 4 years between biomarker measurements—and the older age of the sample, supplementary longitudinal lagged dependent variable models for CRP and metabolic dysregulation did not produce robust results. For this reason, this study only includes lagged dependent variable modeling results for the depressive symptom outcome.

In the lagged dependent variable models for depressive risk, I regress the measure of depressive symptoms measured at a follow-up (2010-2012) on the outcome measures at baseline (2006-2008), the stressor measures at baseline, and the covariates. Lagged dependent variable models take the following general form:

$$y_{i2} = \alpha_0 + \beta_1 y_{i1} + \beta_2 Stress_{i1} + \beta_3 Z_i + \varepsilon_i \quad \text{(Equation 1)}$$

As seen in Equation 1, the outcome at follow-up (y_{i2}) is regressed on the outcome measured at baseline (y_{i1}) as well as the stress exposure also measured at baseline (represented by $\beta_2 Stress_{i1}$). β_3 represents the vector of covariates, including race. Given that the use of first difference models is not preferred given that several of my hypotheses require me to model race

differences, controlling for the baseline outcome levels allows me to better estimate how social stress exposure affects change in the outcome measures. Additionally, the lagged dependent variable models include measures of the key independent variables (psychosocial stress exposure) prior to the outcome, further enhancing my ability to draw conclusions about the potentially causal associations between psychosocial stress exposure and depressive risk using non-experimental data.

In reporting the results of the multivariate OLS, negative binomial, and lagged dependent variable analyses, I pay particular attention to whether inclusion of the stressor measures in the models reduces the racial disparity in the outcomes, which is indicated by the coefficient for the dummy variable indicating race. To formally test whether the stress measures help to "explain away" the race gaps in the outcomes, I use Sobel-Goodman mediation tests.

Results

Descriptive Statistics

Table 4.2 presents descriptive statistics for the outcome variables and the psychosocial stress exposure measures by race. As seen in Table 4.2, Blacks have higher levels of CRP ($p<0.001$), metabolic dysregulation ($p<0.001$), and depressive risk ($p<0.001$) than Whites. These results show support for hypothesis 1, which states that Blacks will exhibit higher levels of physiological inflammation, metabolic dysregulation, and depressive symptoms than Whites.

Table 4.2 also reveals that Blacks report higher levels of psychosocial stress exposure than Whites across virtually all domains of stress, with the exception of lifetime traumas. Compared to Whites, Blacks report more stressful life events ($p=0.035$), financial strain ($p<0.001$), ongoing chronic strains ($p=0.008$), everyday discrimination ($p=0.014$), and major discrimination ($p<0.001$). In addition to the domain-specific stressors, I also observe racial disparities in exposure to the latent, composite stress measures, which were obtained using

confirmatory factor analysis. Blacks report higher levels of both chronic ($p<0.001$) and acute stress exposure ($p<0.001$) than Whites. These descriptive analyses offer support for hypothesis 2, confirming that Blacks report higher levels of recent and life time psychosocial stress exposure than Whites.

Multivariate Models

Tables 3-5 present results of OLS and negative binomial regression models, where the outcomes measured in 2010-2012 were regressed on the stress exposures measured in 2004-2008. Across Models 1-11 in Tables 3-4, the "unexplained" racial disparity in the outcome is indicated by the dummy indicator for race. For each outcome, Model 1 adjusts for age, sex, and race; Model 2 builds on Model 1 by also including the socioeconomic measures; and Models 3-8 include each of the domain-specific stressors in a stepwise fashion. Model 9 includes the composite chronic stress measure, and Model 10 includes the composite acute stress measure. Model 11 includes both the composite chronic and acute stressor measures. Results of the log CRP models can be seen in Table 4.3, metabolic dysregulation in Table 4.4, and depressive symptoms in Table 4.5.

Inflammation

Results in Table 4.3 document the prospective associations between stress exposure and log CRP. In addition to displaying the model results, Table 4.3 also shows the results of the mediation analyses that reveal the proportion of the race gap in log CRP "explained away" by the stress measures included in the respective models. As indicated by the coefficient for the dummy variable for race, which indicates the racial disparity in log CRP, the Black-White disparity in inflammation is largest in Model 1, where there are no adjustments for SES or stress exposure. Including the measures of SES in Model 2 reduces the racial disparity in log CRP over Model 1. Models 3-8 introduce each of the domain-specific stressors in a stepwise fashion. In most cases,

the Black-White disparity in log CRP is attenuated by the inclusion of the stress exposure measures in the models.

Model 3 indicates that lifetime traumas are positively associated with inflammation (0.328, p=0.005), such that individuals who reported more traumas over the course of their lives—including the death of a child, physical abuse by parents, or living through a natural disaster—had higher levels of CRP. However, racial differences in lifetime traumas did not account for a significant portion of the Black-White disparity in log CRP. Model 4 reveals no significant association between stressful life events and log CRP. In Models 5 and 6, I find that financial strain (0.045, p=0.012) and recent chronic stress (0.147, p<0.001) are positively associated with inflammation, respectively. Including the measure of financial strain in Model 5 reduces the race gap in log CRP by approximately 6.4 percent, net of SES. This means that, even after adjusting for racial differences in financial resources, Black-White differences in financial stress account for a significant portion of the racial disparity in physiological inflammation. Results in Models 7 and 8 of Table 4.3 indicate that both the everyday (0.052, p=0.015) and major life discrimination (0.239, p=0.030) measures are positively associated with inflammation. Further, including the measures of everyday discrimination and major life discrimination reduced the race gap in inflammation by 7.2 and 2.8 percent, respectively.

Models 9-11 include the composite stress measures. Model 9 includes the composite measure of chronic stress exposure, Model 10 includes the acute stress measure, and Model 11 includes the measures of chronic and acute stress simultaneously. Results in Models 9 and 10 reveal that increases in both chronic (0.067, p<0.001) and acute (0.050, p=0.003) stress exposure are associated with increases in log CRP and that racial differences in these stressors help to explain significant portions of the Black-White gap in inflammation. When both composite

measures are included simultaneously in Model 11, the magnitudes and statistical significance of the coefficients of the stress exposure measures are slightly diminished, though both retain a positive association with inflammation, net of one another. By comprehensively accounting for stress exposure in Model 11, the Black-White disparity in log CRP is reduced by nearly 14 percent ($p<0.001$) over Model 2. Across Models 1-11, the race gap in log CRP is smallest in Model 11, which adjusts for SES and also includes the composite chronic and acute stress exposure measures.

Metabolic Function

Table 4.4 displays results of the multivariate metabolic dysregulation models. The racial disparity in metabolic risk is greatest in Model 1 and is substantially reduced by adjusting for the host of socioeconomic covariates in Model 2. Similar to the log CRP results, Models 3-8 in Table 4.4 reveal that nearly every one of the domain-specific stressors has a positive association with metabolic dysregulation, such that individuals reporting higher levels of stress exposure in 2004-2008 have greater metabolic risk in 2010-2012. With the exception of major life discrimination, I find that all domain-specific stressors are positively associated with metabolic dysregulation. Further, results from Model 5 indicate that racial differences in financial strain account for nearly 5 percent of the race gap in metabolic dysregulation, net of racial differences in reported socioeconomic resources such as education and wealth. The measure of everyday discrimination also partially mediates the racial disparity in metabolic risk, with approximately 3 percent of the Black-White gap in metabolic dysregulation due to racial differences in the stress associated with everyday discriminatory experiences.

Also consistent with the log CRP results, Models 9 and 10 of Table 4.4 indicate that the composite measures of both chronic (0.102, $p<0.001$) and acute (0.066, $p=0.017$) stress exposure are positively associated with metabolic risk. When both composite measures are included in

Model 11, the association between acute stress exposure and metabolic dysfunction is no longer significant, but chronic stress exposure (0.089, p=0.005) maintains its positive association with the outcome. By including both of the composite stress exposure measures in Model 11, the Black-White disparity in metabolic dysregulation declined by approximately 9.2 percent, suggesting that these measures of stress are key drivers of Black-White metabolic disparities in late life. Across all models, the race gap in metabolic risk is smallest in Model 11.

Depressive Risk

Table 4.5 presents the results of multivariate negative binomial depressive symptoms regression models. Model 1 indicates that a significant racial disparity in mental health risk, with Blacks having more depressive symptoms than Whites. However, when the socioeconomic covariates are included in Model 2, the racial disparity in the outcome is no longer significant. This finding suggests that the observed racial disparity in depressive symptoms is fully accounted for by Black-White differences in socioeconomic well-being.

Models 3-8 build on Model 2 by including the domain-specific stressors in a stepwise fashion. Largely consistent with the inflammation and metabolic risk analyses, the results in Table 4.5 indicate that every one of the domain-specific stressors is positively associated with mental health risk, such that individuals reporting more lifetime traumas (1.560, p<0.001), stressful life events (0.709, p<0.001), financial strain (0.394, p<0.001), ongoing chronic stress (0.950, p<0.001), everyday discrimination (0.305, p<0.001), and major life discrimination (0.883, p<0.001) in 2004-2008 had the greatest mental health risk in 2010-2012.

Models 9-11 include the composite stress measures. Results in Models 9 and 10 reveal that chronic (0.475, p<0.001) and acute stress exposure (0.215, p<0.001) are prospectively associated with depressive risk. When both composite measures are included in Model 11, however, only chronic stress exposure maintains an association with mental health risk (0.459,

$p<0.001$). The composite measure of acute stress exposure no longer has a statistically significant relationship with the outcome.

Results of the longitudinal lagged dependent variable models are presented in Table 4.6. For the lagged dependent variable models, I utilize the same stepwise covariate adjustment strategy employed in the previous sets of models. Similar to the results presented in Table 4.5, results from the lagged dependent variable models indicate that racial differences in socioeconomic status fully explain Black-White differences in the development of depressive symptoms from baseline to follow-up, as the racial gap in depressive risk change is no longer significant in Model 2. Also consistent with the Table 4.5 results, I find that virtually every measure of domain-specific stress exposure is significantly associated with change in depressive risk, such that individuals who reported higher levels of lifetime traumas (0.963, $p<0.001$), financial strain (0.249, $p<0.001$), ongoing chronic strain (0.623, $p<0.001$), everyday discrimination (0.175, $p<0.001$), and major discrimination (0.384, $p=0.027$) had the greatest increases in reported depressive symptoms from baseline to follow-up.

Models 9, 10, and 11 include the composite stress measures. In Models 9 and 10 I find that both the measures of chronic and acute stress exposure significantly predict change in depressive risk over the period. However, once both measures are jointly included in Model 11, only the measure of chronic stress exposure maintains its association with depressive risk change (0.311, $p<0.001$).

Discussion

The divergence of Black-White health disparities from mid- through late-life has led some scholars to argue that Blacks experience accelerated biological aging relative to Whites (Clarke et al. 2014; Geronimus et al. 2010; Rewak et al. 2014) , perhaps due in part to their repeated exposure to chronic and acute stressors and strains across the life course. While

research documents that Blacks experience greater levels of psychosocial stress exposure than Whites (Adkins et al. 2009; Turner, Wheaton, and Lloyd 1995; Turner 2013) and that repeated exposure to stress can promote physiological malfunctioning and psychological distress (Cohen et al. 2012; Epel and Lithgow 2014; Thoits 2010), these two bodies of relatively disparate literatures have not yet been fully integrated to adequately assess the role of differential stress exposure in Black-White health gaps. Utilizing nationally representative, longitudinal data and a variety of markers of biological and psychological functioning, this study offers new insights into how Black-White differences in lifetime exposure to chronic and acute stressors and strains contribute to racial disparities in health in late life.

Consistent with earlier research, descriptive analyses presented in Table 4.2 offer support for hypothesis 1 by documenting that Blacks have higher levels of inflammation, metabolic risk, and psychological distress than Whites. Further, results in Table 4.2 also indicate that Blacks experience more stressful events, financial strain, ongoing chronic strain, everyday discrimination, and major life discrimination than Whites, providing evidence for hypothesis 2. In this way, findings from descriptive analyses support the notion that racism, as a system of oppression and domination, patterns exposure to a variety of acute and chronic stressors and strains—such as familial deaths, financial difficulties, caregiving obligations, and unfair and discriminatory treatment—throughout the life course. Along virtually every dimension of stress, I find that Blacks have higher levels of exposure than their White counterparts. By expanding operationalization of "stress exposure" to include a variety of acute and chronic strains and discrimination-related and generalized social stressors across the life course, this study provides new evidence of the racial patterning of stress in the United States.

Results from the multivariate analyses presented in Tables 3-6 offer robust, consistent support for hypothesis 3, where psychosocial stress exposure is strongly associated with the markers of health. In fact, of the 36 total multivariate models examining the associations between the stressors, there were only three models were I did not find evidence for a statistically significant relationship between the stressors and the outcomes. Results in Tables 3, 4, and 5, which model the prospective associations between stress exposure at baseline and health risk at follow-up, indicate that exposure to the acute and chronic stressors and strains upregulates the body's inflammatory and metabolic responses and increases levels of psychological distress to ultimately affect health and mortality risk. Still, I find that stress exposure may also be implicated in the divergence of trajectories of well-being with age, such that virtually every single measure of stress exposure, with the exception of stressful life events, had a significant impact on individuals' change in depressive risk over time, as seen in results from the lagged dependent variable models in Table 4.6. Future research utilizing fixed or mixed effects models is needed to further establish a causal link between stress exposure and health, but the evidence provided in this study offers initial support for the idea of stress as a critical determinant of population health disparities.

While I find evidence that both acute and chronic stressors are associated with the markers of health, I find that exposure to chronic stress has particularly robust associations with the outcomes. In Models 9-11 of Tables 4-6, I include the composite measures of chronic and acute stress exposure individually and then jointly in order to determine if these measures represented unique or interrelated aspects of stress exposure. Across all three outcomes, both the acute and chronic stress measures had positive associations with the outcomes. However, in Model 11, when the two measures were included simultaneously, the relationship between acute stress

exposure and the outcomes was either mostly or entirely attenuated. This finding is critical to future studies of stress and population health for two primary reasons. First, this finding suggests that individuals may be able to recuperate following acute threats, traumas, and events in ways that differ from their physiological and psychological reaction to chronic strains. Ongoing, chronic stressors and strains may initiate what Seyle (1974) termed "exhaustion," where the repeated activation of the body's stress response system results in ineffective and inefficient physiological and psychological functioning and increased allostatic load. In this way, this study offers support for the growing consensus in the literature that, in order to fully understand how stress is implicated in population health, studies must include a variety of measures of both chronic and acute stressors and strains. Second, this finding regarding the relative importance of chronic and acute stressors to health may also suggest that acute stressors give way to chronic strains. While many of the acute stress exposure measures had significant associations with the outcomes when included in models by themselves, the associations were no longer significant, or greatly reduced in magnitude and significance, when included jointly with the chronic stress measures. An essential element of Pearlin's (1981) stress process is the interrelatedness between acute events—such as the loss of a spouse or parent—and chronic strains—such as ongoing financial difficulties. More research, particularly using structural equation modeling procedures, would help to better elucidate the interrelatedness of acute events, chronic strains, and health and disease risk.

I also find evidence that racial differences in psychosocial stress exposure accounted for significant portions of the Black-White gaps in inflammation and metabolic risk, providing partial support for hypothesis 4. For these two measures of physiological functioning, Sobel-Goodman tests confirmed that, in particular, Black-White disparities in exposure to financial

strain, everyday discrimination, and major life discrimination helped to "explain away" significant portions of the "unexplained" racial gaps in the outcomes. In this way, psychosocial stress exposure is not only a critical determinant of population health, generally, but also a key driver of racial health inequities. It is also worth noting that the contribution of psychosocial stress exposure to Black-White disparities in inflammatory and metabolic risk persisted net of racial differences in socioeconomic factors, which is a relatively conservative test for mediation. Contrary to the results from the inflammation and metabolic dysregulation models, the models of depressive symptoms indicated that the Black-White gap in mental health risk was fully explained by racial differences in socioeconomic factors. In this way, this study also provides some support for the notion of socioeconomic factors being "fundamental" causes of racial health gaps (Phelan and Link 2015).

Finally, the findings presented here suggest that studies of Black-White health disparities that exclude measures of psychosocial stress exposure, or limit their conceptualization and operationalization of stress to check lists of life events and traumas, may overestimate the magnitude of "unexplained" racial health gaps. I find that, while the measures of acute stress exposure are significantly associated with markers of health, these measures did not fully reflect the role of stress exposure in Black-White health inequities. In fact, the measures of chronic stress, which are often excluded from empirical studies, had the strongest associations with the markers of physiological and psychological well-being and mediated the greatest proportions of the Black-White disparities in the outcomes. By excluding these chronic stress measures, or failing to account for psychosocial stress exposure at all, studies of Black-White health disparities may risk overestimating the "unexplained" race residual, which is not without implications (Boen 2016; Do, Frank, and Finch 2012). When studies are left with significant,

unaccounted for racial disparities in health, authors speculate about the potential explanations for these “unexplained” health gaps. As such, there has been a resurgence of biological and genetic explanations for Black-White health differences [see (Roberts 2013) for a review], which is troubling in the face of overwhelming evidence that social explanations for racial health gaps have been underestimated (e.g., (Boen 2016; Do, Frank, and Finch 2012; Pollack et al. 2007). In this way, this study serves both as essential evidence of the critical role of psychosocial stress exposure in population health inequality and as further proof that more research on the social origins of racial health gaps is needed.

REFERENCES

Adkins, Daniel E., Victor Wang, Matthew E. Dupre, Van den Oord, Edwin JCG and Glen H. Elder. 2009. "Structure and Stress: Trajectories of Depressive Symptoms Across Adolescence and Young Adulthood." *Social Forces* 88(1):31-60.

Avison, W. R., J. Ali and D. Walters. 2007. "Family Structure, Stress, and Psychological Distress: A Demonstration of the Impact of Differential Exposure." *Journal of Health and Social Behavior* 48(3):301-317.

Boen, Courtney. 2016. "The Role of Socioeconomic Factors in Black-White Health Inequities Across the Life Course: Point-in-Time Measures, Long-Term Exposures, and Differential Health Returns." *Social Science & Medicine* 170:63-76.

Clark, Rodney, Norman B. Anderson, Vernessa R. Clark and David R. Williams. 1999. "Racism as a Stressor for African Americans: A Biopsychosocial Model." *American Psychologist* 54(10):805-816 (https://auth.lib.unc.edu/ezproxy_auth.php?url=http://search.ebscohost.com/login.aspx?direct=true&db=pdh&AN=1999-11644-001&site=ehost-live&scope=site). doi: 10.1037/0003-066X.54.10.805.

Clarke, Philippa, Jeffrey Morenoff, Michelle Debbink, Ezra Golberstein, Michael R. Elliott and Paula M. Lantz. 2014. "Cumulative Exposure to Neighborhood Context Consequences for Health Transitions Over the Adult Life Course." *Research on Aging* 36(1):115-142.

Cohen, S., D. Janicki-Deverts, W. J. Doyle, G. E. Miller, E. Frank, B. S. Rabin and R. B. Turner. 2012. "Chronic Stress, Glucocorticoid Receptor Resistance, Inflammation, and Disease Risk." *Proceedings of the National Academy of Sciences of the United States of America* 109(16):5995-5999. doi: 10.1073/pnas.1118355109 [doi].

Do, D. P., Reanne Frank and Brian K. Finch. 2012. "Does SES Explain More of the Black/White Health Gap than we Thought? Revisiting our Approach Toward Understanding Racial Disparities in Health." *Social Science & Medicine* 74(9):1385-1393.

Emerging Risk Factors Collaboration. 2010. "C-Reactive Protein Concentration and Risk of Coronary Heart Disease, Stroke, and Mortality: An Individual Participant Meta-Analysis." *The Lancet* 375(9709):132-140.

Ensel, W. M. and N. Lin. 2000. "Age, the Stress Process, and Physical Distress: The Role of Distal Stressors." *Journal of Aging and Health* 12(2):139-168.

Epel, E. S. and G. J. Lithgow. 2014. "Stress Biology and Aging Mechanisms: Toward Understanding the Deep Connection between Adaptation to Stress and Longevity." *The Journals of Gerontology.Series A, Biological Sciences and Medical Sciences* 69 Suppl 1:S10-6. doi: 10.1093/gerona/glu055 [doi].

Finch, Caleb E. 2010. *The Biology of Human Longevity:: Inflammation, Nutrition, and Aging in the Evolution of Lifespans.*Academic Press.

Geronimus, Arline T., John Bound, Timothy A. Waidmann, Cynthia G. Colen and Dianne Steffick. 2001. "Inequality in Life Expectancy, Functional Status, and Active Life Expectancy Across Selected Black and White Populations in the United States." *Demography* 38(2):227-251.

Geronimus, Arline T., Margaret T. Hicken, Jay A. Pearson, Sarah J. Seashols, Kelly L. Brown and Tracey D. Cruz. 2010. "Do US Black Women Experience Stress-Related Accelerated Biological Aging?" *Human Nature* 21(1):19-38.

Geronimus, Arline T., Margaret Hicken, Danya Keene and John Bound. 2006. "“Weathering” and Age Patterns of Allostatic Load Scores among Blacks and Whites in the United States." *Journal Information* 96(5).

Geronimus, A. T. 1992. "The Weathering Hypothesis and the Health of African-American Women and Infants: Evidence and Speculations." *Ethnicity & Disease* 2(3):207-221.

Glaser, Ronald and Janice K. Kiecolt-Glaser. 2005. "Stress-Induced Immune Dysfunction: Implications for Health." *Nature Reviews Immunology* 5(3):243-251.

Harris, Tamara B., Luigi Ferrucci, Russell P. Tracy, M. C. Corti, Sholom Wacholder, Walter H. Ettinger, Harley Heimovitz, Harvey J. Cohen and Robert Wallace. 1999. "Associations of Elevated Interleukin-6 and C-Reactive Protein Levels with Mortality in the Elderly." *The American Journal of Medicine* 106(5):506-512.

Hatch, Stephani L. and Bruce P. Dohrenwend. 2007. "Distribution of Traumatic and Other Stressful Life Events by Race/Ethnicity, Gender, SES and Age: A Review of the Research." *American Journal of Community Psychology* 40(3-4):313-332.

Heeringa, Steven G. and Judith H. Connor. 1995. "Technical Description of the Health and Retirement Survey Sample Design." *Ann Arbor: University of Michigan*.

Horwitz, Allan V., Cathy S. Widom, Julie McLaughlin and Helene R. White. 2001. "The Impact of Childhood Abuse and Neglect on Adult Mental Health: A Prospective Study." *Journal of Health and Social Behavior*:184-201.

House, James S., James M. Lepkowski, Ann M. Kinney, Richard P. Mero, Ronald C. Kessler and A. R. Herzog. 1994. "The Social Stratification of Aging and Health." *Journal of Health and Social Behavior*:213-234.

Kiecolt-Glaser, Janice K. and Ronald Glaser. 2002. "Depression and Immune Function: Central Pathways to Morbidity and Mortality." *Journal of Psychosomatic Research* 53(4):873-876.

Kizer, J. R., A. M. Arnold, N. S. Jenny, M. Cushman, E. S. Strotmeyer, D. G. Ives, J. Ding, S. B. Kritchevsky, P. H. Chaves, C. H. Hirsch and A. B. Newman. 2011. "Longitudinal Changes in Adiponectin and Inflammatory Markers and Relation to Survival in the Oldest Old: The Cardiovascular Health Study all Stars Study." *The Journals of Gerontology.Series A, Biological Sciences and Medical Sciences* 66(10):1100-1107. doi: 10.1093/gerona/glr098 [doi].

Kosteniuk, Julie G. and Harley D. Dickinson. 2003. "Tracing the Social Gradient in the Health of Canadians: Primary and Secondary Determinants." *Social Science & Medicine* 57(2):263-276.

Lantz, P. M., J. S. House, R. P. Mero and D. R. Williams. 2005. "Stress, Life Events, and Socioeconomic Disparities in Health: Results from the Americans' Changing Lives Study." *Journal of Health and Social Behavior* 46(3):274-288.

Lin, Nan and Walter M. Ensel. 1989. "Life Stress and Health: Stressors and Resources." *American Sociological Review*:382-399.

Lu, Michael C. and Belinda Chen. 2004. "Racial and Ethnic Disparities in Preterm Birth: The Role of Stressful Life Events." *American Journal of Obstetrics and Gynecology* 191(3):691-699.

Macy, E. M., T. E. Hayes and R. P. Tracy. 1997. "Variability in the Measurement of C-Reactive Protein in Healthy Subjects: Implications for Reference Intervals and Epidemiological Applications." *Clinical Chemistry* 43(1):52-58.

McEwen, Bruce S. 1998. "Stress, Adaptation, and Disease: Allostasis and Allostatic Load." *Annals of the New York Academy of Sciences* 840(1):33-44.

McEwen, Bruce S. and Eliot Stellar. 1993. "Stress and the Individual: Mechanisms Leading to Disease." *Archives of Internal Medicine* 153(18):2093-2101.

Pager, D. and H. Shepherd. 2008. "The Sociology of Discrimination: Racial Discrimination in Employment, Housing, Credit, and Consumer Markets." *Annual Review of Sociology* 34:181-209.

Pearlin, Leonard I. 1999. "The stress process revisited." Pp. 395-415 in *Handbook of the sociology of mental health.*"The stress process revisited."Springer.

Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman and Joseph T. Mullan. 1981. "The Stress Process." *Journal of Health and Social Behavior* 22(4):337-356.

Penninx, Brenda W., Jack M. Guralnik, Luigi Ferrucci, Eleanor M. Simonsick, Dorly J. Deeg and Robert B. Wallace. 1998. "Depressive Symptoms and Physical Decline in Community-Dwelling Older Persons." *Jama* 279(21):1720-1726.

Phelan, Jo C. and Bruce G. Link. 2015. "Is Racism a Fundamental Cause of Inequalities in Health?" *Annual Review of Sociology* 41:311-330.

Pollack, Craig E., Sekai Chideya, Catherine Cubbin, Brie Williams, Mercedes Dekker and Paula Braveman. 2007. "Should Health Studies Measure Wealth?: A Systematic Review." *American Journal of Preventive Medicine* 33(3):250-264. doi: http://dx.doi.org.libproxy.lib.unc.edu/10.1016/j.amepre.2007.04.033.

Rewak, Marissa, Stephen Buka, Jennifer Prescott, Immaculata De Vivo, Eric B. Loucks, Ichiro Kawachi, Amy L. Non and Laura D. Kubzansky. 2014. "Race-Related Health Disparities and Biological Aging: Does Rate of Telomere Shortening Differ Across Blacks and Whites?" *Biological Psychology* 99:92-99.

Ridker, Paul M., Charles H. Hennekens, Julie E. Buring and Nader Rifai. 2000. "C-Reactive Protein and Other Markers of Inflammation in the Prediction of Cardiovascular Disease in Women." *New England Journal of Medicine* 342(12):836-843.

Roberts, Dorothy. 2013. *Fatal Invention: How Science, Politics, and Big Business Re-Create Race in the Twenty-First Century.*The New Press.

Seeman, Teresa E. and Eileen Crimmins. 2001. "Social Environment Effects on Health and Aging." *Annals of the New York Academy of Sciences* 954(1):88-117.

Selye, Hans. 1974. *Stress Sans Détresse.*Lippincott.

Seyle, Hans. 1974. *Stress without Distress.*Lippincott Williams & Wilkins.

Shuey, Kim M. and Andrea E. Willson. 2008. "Cumulative Disadvantage and Black-White Disparities in Life-Course Health Trajectories." *Research on Aging* 30(2):200-225.

Stevens, June, Jianwen Cai, Elsie R. Pamuk, David F. Williamson, Michael J. Thun and Joy L. Wood. 1998. "The Effect of Age on the Association between Body-Mass Index and Mortality." *New England Journal of Medicine* 338(1):1-7.

Taylor, Paul, Rakesh Kochhar, Richard Fry, Gabriel Velasco and Seth Motel. 2011. "Wealth Gaps Rise to Record Highs between Whites, Blacks and Hispanics." *Pew Research Center, Social and Demographic Trends*.

Thoits, Peggy A. 1983. "Multiple Identities and Psychological Well-being: A Reformulation and Test of the Social Isolation Hypothesis." *American Sociological Review*:174-187.

------. 1995. "Stress, Coping, and Social Support Processes: Where are we? what Next?" *Journal of Health and Social Behavior*:53-79.

Thoits, P. A. 2010. "Stress and Health: Major Findings and Policy Implications." *Journal of Health and Social Behavior* 51 Suppl:S41-53. doi: 10.1177/0022146510383499 [doi].

Turner, R. J. 2013. "Understanding Health Disparities the Relevance of the Stress Process Model." *Society and Mental Health*:2156869313488121.

Turner, R. J. and William R. Avison. 2003. "Status Variations in Stress Exposure: Implications for the Interpretation of Research on Race, Socioeconomic Status, and Gender." *Journal of Health and Social Behavior*:488-505.

Turner, R. J. and Donald A. Lloyd. 1999. "The Stress Process and the Social Distribution of Depression." *Journal of Health and Social Behavior*:374-404.

Turner, R. J., Blair Wheaton and Donald A. Lloyd. 1995. "The Epidemiology of Social Stress." *American Sociological Review*:104-125.

Umberson, D., J. S. Olson, R. Crosnoe, H. Liu, T. Pudrovska and R. Donnelly. 2017. "Death of Family Members as an Overlooked Source of Racial Disadvantage in the United States." *Proceedings of the National Academy of Sciences of the United States of America* 114(5):915-920. doi: 10.1073/pnas.1605599114 [doi].

Wheaton, Blair. 1999. "Social stress." Pp. 277-300 in *Handbook of the sociology of mental health.*"Social stress."Springer.

Wills, Andrew K., Debbie A. Lawlor, Fiona E. Matthews, Avan A. Sayer, Eleni Bakra, Yoav Ben-Shlomo, Michaela Benzeval, Eric Brunner, Rachel Cooper and Mika Kivimaki. 2011. "Life Course Trajectories of Systolic Blood Pressure using Longitudinal Data from Eight UK Cohorts." *PLoS Med* 8(6):e1000440.

Wilson, P. W., K. M. Anderson, T. Harris, W. B. Kannel and W. P. Castelli. 1994. "Determinants of Change in Total Cholesterol and HDL-C with Age: The Framingham Study." *Journal of Gerontology* 49(6):M252-7.

Yang, Yang C., Ting Li and Yinchun Ji. 2013. "Impact of Social Integration on Metabolic Functions: Evidence from a Nationally Representative Longitudinal Study of US Older Adults." *BMC Public Health* 13(1):1210.

Table 4.1. Domain-Specific Stressor Measure Construction

Stressor Measure	Item	Item Coding	Scale coding
Lifetime traumas	Ever experienced death of child	1=yes; 0=no	(Sum of items)/10
	Ever been victim serious physical attack or assault	1=yes; 0=no	
	Ever had life-threatening illness	1=yes; 0=no	
	Spouse or child ever had life threatening illness	1=yes; 0=no	
	Ever been in major fire, flood, earthquake, or other disaster	1=yes; 0=no	
	Ever fired a weapon in combat or been fired upon in combat	1=yes; 0=no	
	Spouse, partner, or child ever addicted to drugs or alcohol	1=yes; 0=no	
	Ever repeat a year of school before age 18	1=yes; 0=no	
	Parents' drinking or drug use ever cause problems before age 18	1=yes; 0=no	
	Ever physically abused by either parent before age 18	1=yes; 0=no	
Stressful life events (last 5 years)	Lost job	1=yes; 0=no	(Sum of items)/5
	Looked for job longer than 3 months	1=yes; 0=no	
	Anyone else in household looked for job longer than 3 months	1=yes; 0=no	
	Moved to a worse residence or neighborhood	1=yes; 0=no	
	Robbed or burglarized	1=yes; 0=no	
Financial strain	How satisfied are you with present financial situation	1=completely; 2=very; 3=somewhat; 4=not very; 5=not at all	(Sum of items)/2
	How difficult is it to meet monthly payments	1=extremely; 2=very; 3=somewhat; 4=not very; 5=not at all	
Ongoing chronic strains (currently experiencing)	Ongoing health problem	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	(Sum of items)/8
	Ongoing physical or emotional problems in spouse or child	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	

	Ongoing problems w/alcohol or drug use in fam member	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
	Ongoing difficulties at work	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
	Ongoing financial strain	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
	Ongoing housing problems	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
	Ongoing problems in a close relationship	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
	Helping a sick, limited, or frail fam member or friend on regular basis	1=no; 2=yes, but not upsetting; 3=yes, somewhat upsetting; 4=yes, very upsetting	
Everyday discrimination	How often are you treated with less courtesy or respect	1=never; 2=less than once a year; 3=a few times a year; 4=a few times a month 5=at least once a week; 6=almost everyday	(Sum of items)/5
	How often do you receive poorer service than others at restaurants or stores	1=never; 2=less than once a year; 3=a few times a year; 4=a few times a month 5=at least once a week; 6=almost everyday	

	How often do people act as if they think you are not smart	1=never; 2=less than once a year; 3=a few times a year; 4=a few times a month 5=at least once a week; 6=almost everyday	
	How often do people act as if they are afraid of you	1=never; 2=less than once a year; 3=a few times a year; 4=a few times a month 5=at least once a week; 6=almost everyday	
	How often are you threatened or harassed	1=never; 2=less than once a year; 3=a few times a year; 4=a few times a month 5=at least once a week; 6=almost everyday	
Major lifetime discrimination	Ever unfairly dismissed from a job	1=yes; 0=no	(Sum of items)/6
	Ever not hired for a job	1=yes; 0=no	
	Ever unfairly denied a promotion	1=yes; 0=no	
	Ever prevented from moving to a neighborhood because realtor refused to sell/rent to you	1=yes; 0=no	
	Ever unfairly denied a bank loan	1=yes; 0=no	
	Ever unfairly stopped by police	1=yes, 0=no	

Table 4.2. Descriptive Statistics by Race

	White	Black	p-value
Outcomes			
Log C-reactive protein	1.137	1.364	<0.001
Metabolic dysregulation	2.633	3.190	<0.001
Depressive symptoms	1.218	1.565	<0.001
Domain-specific stressors			
Lifetime traumas	0.155	0.154	0.406
Stressful events	0.043	0.058	0.035
Financial strain	2.305	2.842	<0.001
Ongoing chronic strains	1.475	1.540	0.030
Everyday discrimination	1.628	1.764	0.014
Major discrimination	0.066	0.128	<0.001
Composite stressors			
Chronic stress exposure	-0.089	0.317	<0.001
Acute stress exposure	-0.074	0.204	<0.001

Notes: Weighted descriptive statistics. P-value of race difference (t-test).

Table 4.3. Stress Exposure and Log C-Reactive Protein (N=3,773)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)
Racial disparity											
Race (1=Black)	0.279***	0.203***	0.206***	0.201***	0.189**	0.203***	0.197**	0.191**	0.189**	0.193**	0.185**
	(0.059)	(0.060)	(0.060)	(0.060)	(0.060)	(0.060)	(0.060)	(0.060)	(0.060)	(0.059)	(0.060)
Proportion race gap mediated by stressor(s)[a]	-	-	NS	NS	0.064*	NS	0.072*	0.028*	0.070**	0.067**	0.137***
Socio-demographic characteristics											
Age	-0.001	-0.003†	-0.003	-0.002	-0.002	-0.001	-0.002	-0.002	-0.000	-0.002	-0.000
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Gender (1=female)	0.092**	0.067*	0.072*	0.068*	0.067*	0.059†	0.077*	0.074*	0.070*	0.076*	0.075*
	(0.029)	(0.030)	(0.030)	(0.031)	(0.030)	(0.030)	(0.030)	(0.031)	(0.030)	(0.031)	(0.031)
Socioecononmic factors											
Education (HS is reference)											
<HS		0.094†	0.086†	0.096†	0.091†	0.095†	0.096†	0.097†	0.094†	0.095†	0.095†
		(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)
Some college		-0.032	-0.037	-0.034	-0.030	-0.033	-0.033	-0.041	-0.031	-0.043	-0.038
		(0.039)	(0.038)	(0.038)	(0.039)	(0.038)	(0.039)	(0.039)	(0.038)	(0.038)	(0.038)
BA+		-0.200***	-0.194***	-0.202***	-0.190***	-0.196***	-0.198***	-0.205***	-0.189***	-0.203***	-0.193***
		(0.036)	(0.037)	(0.036)	(0.037)	(0.036)	(0.036)	(0.036)	(0.036)	(0.036)	(0.036)
Total household wealth		-0.000***	-0.000**	-0.000**	-0.000*	-0.000**	-0.000**	-0.000**	-0.000*	-0.000**	-0.000*
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Marital status (1=married or cohabiting)		-0.021	-0.012	-0.017	-0.008	-0.004	-0.019	-0.015	-0.002	-0.007	0.003
		(0.035)	(0.035)	(0.035)	(0.036)	(0.035)	(0.035)	(0.035)	(0.035)	(0.035)	(0.035)

Domain-specific stressors									
Lifetime traumas		0.328**							
		(0.116)							
Stressful life events			0.165						
			(0.140)						
Financial strain				0.045*					
				(0.018)					
Ongoing chronic strains					0.147***				
					(0.036)				
Everyday discrimination						0.052*			
						(0.021)			
Major discrimination							0.239*		
							(0.110)		
Composite stressors									
Chronic stress exposure								0.067***	0.056**
								(0.016)	(0.017)
Acute stress exposure								0.050**	0.029†
								(0.017)	(0.018)

*** p<0.001, ** p<0.01, * p<0.05, † p<0.1

Notes: Results based on OLS regression models. C-reactive protein was assessed in 2010-2012; all other covariates were measured in 2004-2008. Model estimates are weighted.

a: Proportion of racial disparity in log CRP observed in Model 2 "explained away" by the stress measure(s) included in the model. "NS" indicates no significant mediation.

Table 4.4. Stress Exposure and Metabolic Dysregulation (N=3,351)

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**	**Model 8**	**Model 9**	**Model 10**	**Model 11**
	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)
Racial disparity											
Race (1=Black)	0.601***	0.510***	0.516***	0.505***	0.489***	0.513***	0.496***	0.500***	0.490***	0.499***	0.487***
	(0.084)	(0.086)	(0.085)	(0.086)	(0.086)	(0.086)	(0.086)	(0.086)	(0.086)	(0.086)	(0.086)
Proportion race gap mediated by stressor(s)[a]	-	-	NS	NS	0.046**	NS	0.030*	NS	0.042**	0.017†	0.092***
Socio-demographic characteristics											
Age	-0.005*	-0.007*	-0.006*	-0.005†	-0.005†	-0.005†	-0.004†	-0.006*	-0.003	-0.005†	-0.003
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Gender (1=female)	0.090	0.057	0.062	0.062	0.057	0.047	0.077	0.063	0.060	0.070	0.066
	(0.051)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)	(0.052)
Socioecononmic factors											
Education (HS is reference)											
<HS		0.042	0.035	0.050	0.036	0.042	0.042	0.044	0.038	0.044	0.040
		(0.081)	(0.081)	(0.082)	(0.081)	(0.081)	(0.080)	(0.081)	(0.081)	(0.081)	(0.081)
Some college		-0.033	-0.039	-0.040	-0.031	-0.036	-0.035	-0.041	-0.034	-0.049	-0.043
		(0.064)	(0.064)	(0.064)	(0.064)	(0.064)	(0.064)	(0.065)	(0.064)	(0.064)	(0.064)
BA+		-0.313***	-0.307***	-0.318***	-0.299***	-0.311***	-0.311***	-0.318***	-0.301***	-0.318***	-0.305***
		(0.066)	(0.066)	(0.066)	(0.066)	(0.066)	(0.066)	(0.066)	(0.066)	(0.066)	(0.066)
Total household wealth		-0.000**	-0.000**	-0.000**	-0.000*	-0.000**	-0.000**	-0.000**	-0.000*	-0.000**	-0.000*
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Marital status (1=married or cohabiting)		0.033	0.042	0.047	0.051	0.051	0.035	0.038	0.058	0.052	0.065
		(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)	(0.059)

Domain-specific stressors									
Lifetime traumas		0.329†							
		(0.177)							
Stressful life events			0.491*						
			(0.237)						
Financial strain				0.072*					
				(0.032)					
Ongoing chronic strains					0.165**				
					(0.062)				
Everyday discrimination						0.115**			
						(0.037)			
Major discrimination							0.202		
							(0.187)		
Composite stressors									
Chronic stress exposure								0.102***	0.089**
								(0.028)	(0.032)
Acute stress exposure								0.066*	0.034
								(0.028)	(0.031)

*** p<0.001, ** p<0.01, * p<0.05, † p<0.1

Notes: Results based on OLS regression models. Metabolic dysreguation was assessed in 2010-2012; all other covariates were measured in 2004-2008. Model estimates are weighted.

a: Proportion of racial disparity in metabolic dysregulation observed in Model 2 "explained away" by the stress measure(s) included in the model. P-value of Sobel-Goodman test for mediation.

Table 4.5. Stress Exposure and Depressive Symptoms (N=4,773)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)	Coeff. (SE)
Racial disparity											
Race (1=Black)	0.316***	0.081	0.119	0.074	-0.023	0.108	0.066	0.040	0.019	0.054	0.017
	(0.083)	(0.081)	(0.081)	(0.080)	(0.081)	(0.082)	(0.082)	(0.084)	(0.083)	(0.083)	(0.083)
Socio-demographic characteristics											
Age	0.006*	0.000	0.002	0.003	0.013***	0.013***	0.006*	0.003	0.020***	0.006*	0.020***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Gender (1=female)	0.232***	0.144*	0.184***	0.150**	0.162**	0.100†	0.218***	0.179**	0.187***	0.199***	0.195***
	(0.057)	(0.057)	(0.056)	(0.057)	(0.056)	(0.055)	(0.058)	(0.057)	(0.056)	(0.057)	(0.056)
Socioecononmic factors											
Education (HS is reference)											
<HS		0.266***	0.230**	0.277***	0.261***	0.279***	0.295***	0.278***	0.292***	0.272***	0.292***
		(0.072)	(0.072)	(0.072)	(0.072)	(0.072)	(0.074)	(0.072)	(0.074)	(0.072)	(0.074)
Some college		-0.088	-0.112†	-0.101	-0.039	-0.096	-0.080	-0.118†	-0.056	-0.140*	-0.066
		(0.068)	(0.067)	(0.068)	(0.067)	(0.066)	(0.068)	(0.068)	(0.067)	(0.067)	(0.067)
BA+		-0.410***	-0.377***	-0.419***	-0.303***	-0.371***	-0.393***	-0.420***	-0.312***	-0.417***	-0.316***
		(0.077)	(0.076)	(0.077)	(0.076)	(0.076)	(0.076)	(0.078)	(0.074)	(0.077)	(0.074)
Total household wealth		-0.000*	-0.000†	-0.000*	0.000	-0.000	-0.000†	-0.000†	0.000	-0.000†	0.000
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Marital status (1=married or cohabiting)		-0.278***	-0.231***	-0.262***	-0.154**	-0.165**	-0.265***	-0.253***	-0.129*	-0.218***	-0.123*
		(0.060)	(0.059)	(0.061)	(0.060)	(0.058)	(0.061)	(0.062)	(0.059)	(0.061)	(0.059)

Domain-specific stressors									
Lifetime traumas	1.560***								
	(0.176)								
Stressful life events		0.709**							
		(0.252)							
Financial strain			0.394***						
			(0.032)						
Ongoing chronic strains				0.950***					
				(0.057)					
Everyday discrimination					0.305***				
					(0.036)				
Major discrimination						0.883***			
						(0.180)			
Composite stressors									
Chronic stress exposure							0.475***		0.459***
							(0.027)		(0.030)
Acute stress exposure								0.215***	0.038
								(0.027)	(0.029)

*** p<0.001, ** p<0.01, * p<0.05, p<0.1

Notes: Results based on negative binomial regression models. Depressive symptoms were assessed in 2010-2012; all other covariates were measured in 2004-2008. Model estimates are weighted.

Table 4.6. Stress Exposure and Change in Depressive Risk 2006-2012 (N=4,411)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11
	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.	Coeff.
	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)	(SE)
Baseline depressive risk											
Depressive symptoms at Wave I	0.308***	0.292***	0.280***	0.291***	0.266***	0.250***	0.281***	0.288***	0.243***	0.282***	0.243***
	(0.011)	(0.012)	(0.012)	(0.012)	(0.012)	(0.013)	(0.012)	(0.012)	(0.013)	(0.012)	(0.013)
Racial disparity											
Race (1=Black)	0.159*	0.046	0.070	0.045	-0.010	0.067	0.036	0.027	0.011	0.032	0.011
	(0.075)	(0.078)	(0.076)	(0.078)	(0.078)	(0.077)	(0.078)	(0.079)	(0.078)	(0.077)	(0.078)
Socio-demographic characteristics											
Age	0.009***	0.006*	0.007*	0.007*	0.014***	0.014***	0.009***	0.007*	0.018***	0.009**	0.018***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Gender (1=female)	0.169**	0.124*	0.146**	0.126*	0.135*	0.101†	0.167**	0.141**	0.157**	0.152**	0.157**
	(0.054)	(0.054)	(0.054)	(0.054)	(0.053)	(0.053)	(0.055)	(0.055)	(0.053)	(0.054)	(0.053)
Socioeconomic factors											
Education (HS is reference)											
<HS		0.126†	0.110	0.129†	0.132†	0.156*	0.147*	0.134†	0.165*	0.135*	0.165*
		(0.069)	(0.067)	(0.069)	(0.068)	(0.068)	(0.070)	(0.069)	(0.069)	(0.068)	(0.069)
Some college		-0.054	-0.070	-0.058	-0.028	-0.060	-0.055	-0.068	-0.040	-0.082	-0.040
		(0.066)	(0.065)	(0.066)	(0.065)	(0.064)	(0.065)	(0.065)	(0.064)	(0.065)	(0.064)
BA+		-0.282***	-0.270***	-0.285***	-0.226**	-0.273***	-0.279***	-0.290***	-0.239***	-0.292***	-0.239***
		(0.074)	(0.073)	(0.075)	(0.073)	(0.072)	(0.074)	(0.075)	(0.071)	(0.074)	(0.072)
Total household wealth		-0.000	-0.000	-0.000	0.000	-0.000	-0.000	-0.000	0.000	-0.000	0.000
		(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Marital status (1=married or cohabiting)		-0.114†	-0.091	-0.110†	-0.052	-0.062	-0.110†	-0.105†	-0.043	-0.088	-0.043
		(0.061)	(0.059)	(0.061)	(0.059)	(0.057)	(0.060)	(0.061)	(0.057)	(0.061)	(0.057)

Domain-specific stressors									
Lifetime traumas	0.963***								
	(0.181)								
Stressful life events		0.182							
		(0.233)							
Financial strain			0.249***						
			(0.033)						
Ongoing chronic strains				0.623***					
				(0.061)					
Everyday discrimination					0.175***				
					(0.035)				
Major discrimination						0.384*			
						(0.174)			
Composite stressors									
Chronic stress exposure							0.312***		0.311***
							(0.029)		(0.032)
Acute stress exposure								0.108***	0.001
								(0.026)	(0.028)

*** p<0.001, ** p<0.01, * p<0.05, † p<0.1

Notes: Results based on negative binomial lagged dependent variable regression models. Baseline depressive symptoms were assessed in 2006-2008 and follow-up depressive symptoms were measured 2010-2012; all other covariates were measured in 2004-2008. Model estimates are weighted.

CHAPTER 5: CONCLUSION

This dissertation research aimed to provide new insights into and understanding of the role of stress in racial health disparities. Despite a long scholarly tradition of uncovering the material roots of racial health gaps, less attention has been paid to the psychosocial processes that undergird the links between structural racial inequality, material conditions, and health. Each of the three empirical studies presented here integrates diverse and nuanced measures of stress exposure—including contacts with the criminal justice system, neighborhood-level stressors, and a variety of acute and chronic psychosocial strains—and objective markers of physiological and psychological functioning into empirical examinations of the proximate determinants of Black-White health disparities. In this way, this research improves sociological understanding of how racism patterns exposure to material conditions and psychosocial stressors to promote physiological and psychological dysregulation and ultimately affect health and disease risk on a population level.

Though distinct, the three studies presented here collectively offer a more detailed, comprehensive view of the role of stress in racial health disparities across the life course. Together, the three studies examine how exposure to various dimensions of stress contribute to racial differences in markers of physical health and pyschological functioning at different life stages to contribute to the emergence and progression of disease. The first study provided new evidence of the links between the stress of criminal justice encounters and population health by detailing how contacts along the criminal justice continuum—from police stops, arrests, convictions, and incarceration—impact stress-related makers of biological and psychological

well-being and contribute to racial disparities in health and disease risk. Findings revealed that these contacts not only served to up-regulate bodily stress response systems, but that Black-White differences in criminal justice contacts account for a sizable portion of racial health disparities, net of a host of potential confounders.

The second study served to challenge scholarly assumptions about the relationship between racial residential segregation and population health disparities by investigating and documenting how neighborhood economic conditions and neighborhood racial context have unique and, at times, surprising associations with health. While a large body of literature suggests that racial residential segregation plays a fundamental role in contributing to the Black health disadvantage, this study provides new evidence that Blacks receive some protective health benefits from living in predominately Black neighborhoods. In fact, results suggest that Blacks living in racially diverse neighborhoods may experience more stress related to social isolation and racial discrimination than Blacks living in racially segregated neighborhoods, which challenges the notion that "integration produces equity."

Finally, the third study examined how Black-White differences in psychosocial stress exposure across the life course contribute to racial disparities in stress-related markers of physiological and psychological well-being in late life. By expanding conceptualization and measurement of stress exposure to include a host of chronic, acute, discrimination-related, and generalized social stressors, findings from this study revealed that racial differences in stress account for significant portions of Black-White health gaps. Further, findings also revealed that chronic stressors and strains have stronger associations with health and play a more prominent role in the production of racial health gaps than acute stressors.

By integrating diverse measures of life course stress exposure and objective markers of bodily stress response systems; utilizing a variety of advanced analytic techniques; and offering new conceptualizations of how racism harms health, findings from these studies advance understanding of the biological and psychosocial pathways generating persistent racial disparities in disease and longevity. In addition to improving scientific knowledge of the determinants of racial health gaps, these studies also offer essential insights into potential leverage points for ameliorating Black-White health gaps. As documented throughout this research, structural racial inequality patterns individual access to opportunities and resources—such as well-paying jobs, higher education, social relationships, and feelings of belonging—and shapes exposure to risk and physiological toxins—such as police stops and searches, incarceration, and discrimination—in real and tangible ways that have profound influences on population health. As such, it is time that developing policy, programmatic, and movement-based strategies to eradicate structural racism become not only become a societal priority, but a scientific one as well.