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The things they carried: The biological residue of childhood misfortune

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THE THINGS THEY CARRIED: THE BIOLOGICAL RESIDUE OF CHILDHOOD MISFORTUNE

For the degree of Doctor of Philosophy



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Date

THE THINGS THEY CARRIED:
THE BIOLOGICAL RESIDUE OF CHILDHOOD MISFORTUNE

A Dissertation

Submitted to the Faculty

of

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by

Patricia M. Morton

In Partial Fulfillment of the

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of

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To Ian who always said he would die before I finished school. He was right.
Ian Randolph Buxton
1981-2013

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ABSTRACT

Morton, Patricia M. Ph.D., Purdue University, May 2016. *The Things They Carried: The Biological Residue of Childhood Misfortune*. Major Professor: Kenneth Ferraro.

There is a well-established relationship between childhood misfortune and adult health, but how these early-life experiences “get under the skin” to later manifest as poor health is less clear. To elucidate this process, this dissertation investigates (1) how childhood conditions influence immune functioning and (2) whether these physiological consequences of early misfortune lead to poor health, indicated by ischemic heart disease (IHD) onset. Guided by cumulative inequality theory and biological embedding, this dissertation also examines adult health lifestyles and socioeconomic status (SES) as possible mechanisms linking childhood misfortune to inflammation and IHD in later life. Data come from six waves of the Health and Retirement Study between 2004 and 2014, comprising a sample of over 8,000 adults aged 51 and older. The empirical investigation is presented in two main articles. The first article presented in Chapter 2 investigates the relationship between childhood misfortune and chronic inflammation, and examines mediators of adult health lifestyles and SES. Building on the findings in Chapter 2, the second article presented in Chapter 3 investigates the relationship between childhood misfortune and IHD risk, and examines mediators of adult health lifestyles, SES, and

inflammation. In both articles, alternative specifications of childhood misfortune are tested. Findings from this dissertation reveal that childhood misfortune predicts higher levels of inflammation and IHD risk in later-life. For inflammation, additive childhood misfortune and lower childhood SES led to elevated levels of inflammation via adult health lifestyles and SES. For IHD risk, lower childhood SES raised IHD risk by directly impacting adult health lifestyles and SES, which subsequently led to higher levels of inflammation, resulting in onset of IHD. These findings clarify how childhood misfortune impacts health among older adults. Using multiple mediating domains to assess the long-term effects of early-life conditions can enhance health policy in an effort to reduce the associated disease burden of childhood misfortune.

CHAPTER 1. INTRODUCTION

1.1 Statement of the Problem

As life expectancy rises, health research in epidemiology, gerontology, psychology, and sociology has begun to emphasize healthspan in addition to lifespan (Crimmins 2015). This shift to healthspan has garnered interest in optimal aging. Consequently, scholars have identified threats to optimal aging in an effort to reduce the health burden faced by the older population. One of the liveliest areas studying optimal aging is life course research, which has revealed that threats to optimal aging can occur in the earliest stages of life. Indeed, health differentiation in adulthood can be traced back to childhood events and experiences. For instance, adults who experienced childhood misfortune are at increased risk for poor health, such as chronic morbidity (Greenfield and Marks 2009a), ischemic heart disease (Dong et al. 2004), hypertension (Felitti et al. 1998), and cancer (Morton, Schafer, and Ferraro 2012), as well as premature mortality (Brown et al. 2009). Thus, childhood misfortune, which refers to a wide-range of childhood events and experiences including—but not limited to—maltreatment, parental death, poor health, and socioeconomic disadvantage, can threaten optimal aging.

To understand these threats and how to overcome them, scholars have begun to identify mechanisms that may help explain how childhood misfortune becomes manifest as poor physical adult health. The preponderance of this burgeoning literature has

focused on behavioral, emotional, cognitive, and social pathways (Due et al. 2011; Graham and Power 2010; Kendall-Tackett 2002). With the continual focus on behavioral and psychosocial links, one fundamental question remains: How does childhood misfortune “get under the skin”? Recent findings suggest that misfortune is associated with physiological changes in immune functioning, and that these alterations persist into adulthood. Although this research has advanced our understanding of the biological mechanisms of misfortune, *the process of how* early-life misfortune influences the immune system and subsequent health is not well researched. Therefore, a more comprehensive approach to elucidate how childhood experiences manifest on a physiological level, and, in turn, impact the health and aging process is warranted.

To provide a more comprehensive approach to studying this life course process, this dissertation integrates conceptual and empirical work from multiple disciplines to explicate how early-life misfortune influences immune functioning, eventuating in disease onset. Conceptually, this research bridges together two interdisciplinary theoretical frameworks. First, this research draws from cumulative inequality (CI) theory (Ferraro and Shippee 2009). CI theory identifies childhood as a sensitive period for later-life health and expounds upon the mechanisms underlying long-term processes of health. Second, this research is also guided by biological embedding (Hertzman and Boyce 2010). Biological embedding explicates how biological development during childhood is susceptible to the environment, providing theoretical plausibility for how childhood disadvantage becomes manifest on a physiological level in later life. Together, these two frameworks create a theoretical platform for the present research.

Empirically, this research builds upon recent, multidisciplinary research, examining intermediary biological, behavioral, and psychosocial processes to explicate how health consequences of early misfortune unfold over the life course. Given the rising costs of long-term healthcare coupled with increasing life expectancy, a better understanding of the residual effects of childhood misfortune is paramount to reducing the health burden faced by older populations.

1.1.1 Specific Aims and Innovation

Using the Health and Retirement Study (HRS), this dissertation systematically examines the lasting health consequences of childhood misfortune among Black, White, and Hispanic men and women. The HRS data are ideal to understand how early misfortune influences health in later-life via chronic inflammation. These data include a wealth of childhood indicators and biological measures on a large, random sample of midlife and older adults residing in the United States. In addition, the data structure enables me to investigate the *occurrence* and *timing* of disease onset as well as conduct formal tests of mediation given the temporal ordering of key variables. This research has three specific aims:

Aim 1. To determine which specifications of childhood misfortune are most consequential to chronic inflammation among older adults.

Recent research has revealed that the effects of childhood misfortune can vary based on the operationalization of misfortune (Morton et al. 2012). Therefore, alternative specifications of misfortune are examined to determine which forms of childhood misfortune are most salient in predicting chronic inflammation. Chronic inflammation is

assessed using C-reactive protein levels (CRP). CRP is a common, reliable, and valid marker of inflammation (McDade, Burhop, and Dohnal 2004). Since childhood experiences and inflammation vary by race and gender (Geronimus et al. 2006; Herd, Karraker, and Friedman 2012; Slopen et al. 2010), the moderating effects of race and gender are also considered.

Aim 2. To examine whether adult health lifestyles or socioeconomic status explain the relationship between childhood misfortune and chronic inflammation.

Adults who experienced childhood misfortune are more likely to engage in unhealthy behaviors and lifestyles and experience socioeconomic disadvantage, all of which are associated with chronic inflammation (Alley et al. 2006; Anda et al. 1999; Case, Fertig, and Paxson 2005; Felitti et al. 1998; Haas 2006; Morey and Segerstorm 2015; Williamson et al. 2002). To elucidate the relationship between childhood misfortune and adult inflammation, this study investigates the mediating effects of adult health-related and socioeconomic factors (see Figure 1.1).

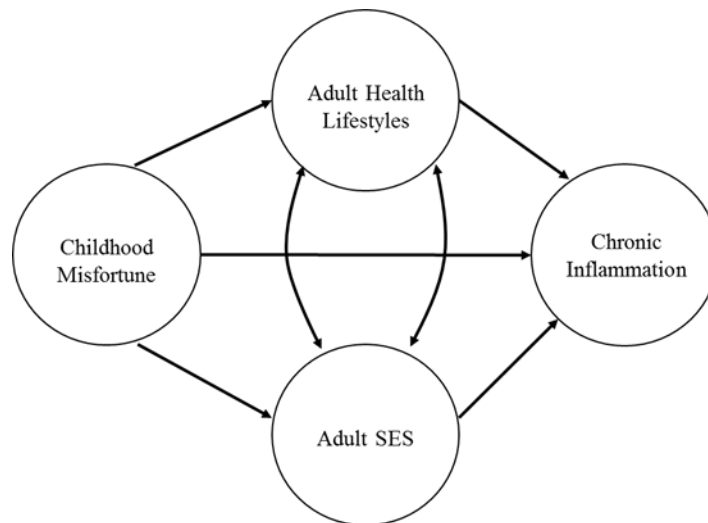


Figure 1.1 Conceptual Model of Proposed Relationships Among Childhood Misfortune, Adult Health Lifestyles, Adult SES, and Chronic Inflammation.

Aim 3: To investigate the relationships among childhood misfortune, adult health lifestyles, adult socioeconomic status, chronic inflammation, and incident ischemic heart disease.

Prior research has demonstrated that the effects of childhood misfortune on adult health and mortality are most salient for cardiovascular health and mortality (e.g., Beebe-Dimmer et al. 2004; Bowen 2010; Elo, Martikainen and Myrskylä 2010; Hamil-Luker and O’Rand 2007; Kaplan and Salonen 1990; Korkeila et al. 2010; Morton, Mustillo, and Ferraro 2014; Singer and Ryff 1999; Singh-Manoux et al. 2004). Therefore, this study examines whether childhood misfortune raises the risk of ischemic heart disease (IHD), a common type of cardiovascular disease (CVD) and cause of death in the United States (Center for Disease Control and Prevention [CDC] 2015; Gillespie, Wigington, and Hong 2013; Mensah and Brown 2007). Incident IHD is measured using data from both living and deceased respondents and includes two main types of IHD: myocardial infarction (MI) and angina (Dong et al. 2004). To elucidate the relationship between childhood misfortune and incident IHD, a pathway model investigating the mediating effects of adult health lifestyles, socioeconomic status (SES), and chronic inflammation is tested (see Figure 1.2). Alternative forms of childhood misfortune and the moderating effects of race and gender are also investigated.

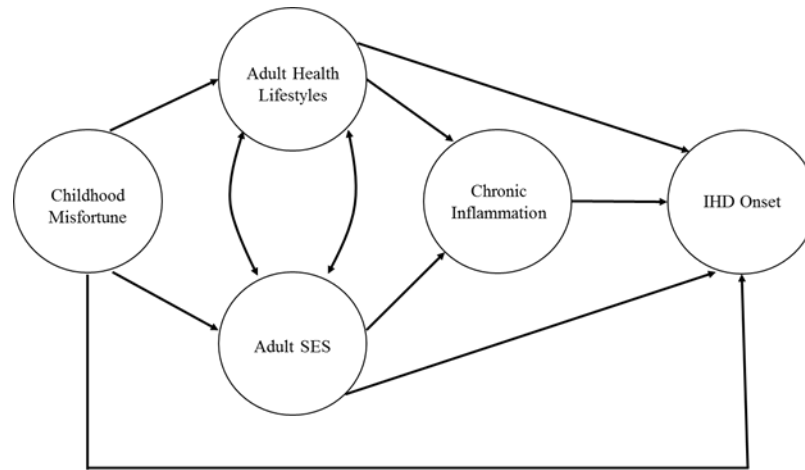


Figure 1.2 Conceptual Model of Proposed Relationships Among Childhood Misfortune, Adult Health Lifestyles, Adult SES, Chronic Inflammation, and IHD Onset.

Overall, this dissertation contributes to the emerging research investigating the biological consequences of childhood misfortune. Specifically, there are four novel aspects of this dissertation. First, this project blends together conceptual and empirical research from fields of biology, epidemiology, gerontology, psychology, and sociology to test theoretically proposed causal pathways from childhood to adulthood. This integrative approach draws fresh multidisciplinary insight to understand how *social forces can shape physiology* and, subsequently, trajectories of health.

Second, the methodological approach advances research on the early origins of adult health. Much of the childhood misfortune research, especially research on the biological consequences of misfortune, focuses on a single type of misfortune, such as childhood SES. However, the effects of childhood misfortune can vary based on the operationalization of misfortune, and this dissertation investigates alternative forms of misfortune that encompass multiple domains to determine which types and specifications are most consequential to inflammation and health in later life. In addition, the

longitudinal design of these data enables me to test theoretically driven pathways from childhood to older adulthood. Mapping how childhood misfortune unfolds over the life course can inform public health policy as to how and where to intervene. Whereas biological markers of health are often used as outcomes, this study also considers the mediating effect of markers of inflammation.

Third, this project uses biological markers of inflammation from a large, representative sample of US adults. Often, studies obtain biological markers on small, non-representative samples because collecting biological data is somewhat invasive as well as time and monetarily consuming.

Fourth, the large sample size in tandem with oversamples of Black and Hispanic Americans enables me to investigate the moderating effects of race and gender, *contextualizing* the unique experiences of childhood misfortune and its aftermath for Black, White, and Hispanic men and women.

1.1.2 The Pervasive Long-arm of Childhood Misfortune

In 1998, a pivotal life course study on health revealed that the additive effect of multiple types of childhood misfortune increased the risk of several diseases in adulthood (Felitti et al. 1998). This study, commonly known as the first Adverse Childhood Experiences or ACE study, connected a host of childhood events and experiences, such as maltreatment and household dysfunction, to many adult diseases, including cancer, chronic lung disease, ischemic heart disease, liver disease, and skeletal fractures. By demonstrating that the combined effect of disadvantageous early-life experiences could impact health later in life, this study was a catalyst for research on the early origins of

adult health. A profusion of subsequent research has since affirmed that childhood events and experiences can influence adult health (e.g., Anda et al. 2008; Blackwell, Hayward, and Crimmins 2001; Greenfield and Marks 2009a; Morton et al. 2012; O’Rand and Hamil-Luker 2005; Thomas, Hyppönen, and Power 2008). Now, there is overwhelming evidence linking childhood misfortune to adult health.

Because there is often a substantial period between childhood experiences and onset of a major illness or disease, a natural progression of this research has been to elucidate the mechanisms connecting childhood experiences to adult health. This emerging phase of life course research has been seeking to better understand what happens between early-life experiences of disadvantage and disease onset, and whether these intermediary factors can help clarify how childhood misfortune becomes manifest as poor health in adulthood. This burgeoning research has revealed that childhood misfortune influences multiple life domains which can impact health. Experiencing disadvantage early in life can influence health behaviors and lifestyles (Anda et al. 1999; Greenfield and Marks 2009b; Felitti et al. 1998), psychosocial resources (Morton et al. 2016), and economic outcomes (Palloni 2006) in adulthood. This body of work has been instrumental in advancing our understanding of how life course processes of health are initiated during childhood. But one question remains: How does childhood misfortune impact health on a physiological level? This question, rooted in classic biology (i.e., the assumption that an organism’s environment interacts with its internal physiology), has spurred the most recent phase of research on the childhood origins of adult health: examining the biological consequences of childhood misfortune.

Although fewer studies have examined biological markers, children of misfortune appear to exhibit immunological signs of dysregulation over time. Chronic inflammation, which can lead to disease, has been observed in adults who experienced childhood misfortune (Carpenter et al. 2010; Danese et al. 2007; Pearson et al. 2003; Slopen et al. 2010).¹ Taken together, these bodies of research demonstrate the “long-arm” of childhood misfortune, raising the question of whether immune dysregulation is a precursor to the health consequences of early misfortune. However, few studies examine chronic inflammation as a mediator of childhood misfortune and adult health.

In addition, inflammation may provide invaluable insight into the intermediary processes of how childhood misfortune manifests as disease and disability onset. Inflammation has been linked to many of the aforementioned mechanisms of childhood misfortune, including adult health lifestyles and SES (Alley et al. 2006; Friedman and Herd 2010; Herd et al. 2012; Koster et al. 2006; Lubbock et al. 2005). Given the research documenting the relationships among childhood misfortune, adult health lifestyles, SES, inflammation, and health, it is plausible that childhood misfortune may indirectly influence adult health through a multidimensional process of health lifestyles, SES, and chronic inflammation. Although many conceptual frameworks posit a multidimensional pathway model of health, these literatures have yet to be connected empirically through formal tests of mediation.

¹Whereas acute inflammation is a natural immune response, chronically elevated levels of inflammation can signal immune dysregulation and lead to cancer, CVD, disability, and premature mortality (Fagundes and Way 2014).

1.2 Data and Methods

This dissertation utilizes longitudinal data from the HRS to examine how childhood misfortune influences health and aging processes among Black, White, and Hispanic older adults. Sponsored by the National Institutes of Health (NIH) (NIA U01AG009740) and conducted by the University of Michigan, the HRS is a biennial survey of American adults 51 years and older (Health and Retirement Study 2015). Initiated in 1992, the HRS utilized a multistage, stratified area probability sample of American households, including oversamples of Black and Hispanic Americans and Floridians, and replenishes the sample with new cohorts every six years to maintain a representative sample.² Response rates for initial and follow-up surveys have been good—all above 70% with most exceeding 85% for each cohort (Sonnegga et al. 2014).

The HRS is the largest, nationally representative panel study of older adults in the United States and provides exceptional data to investigate the hypothesized relationships among childhood misfortune, adult health lifestyles, SES, chronic inflammation, and IHD risk. The HRS is replete with information on childhood, health behaviors and lifestyles, and socioeconomic indicators. In addition, the HRS collected information on the timing of disease onset (for living and deceased respondents) as well as biological markers for the entire sample. Thus, the HRS is among a few studies that have biological data on a large, national survey of Americans. Finally, because the HRS has several waves

²The HRS currently includes 6 cohorts: Asset and Health Dynamics Among the Oldest Old (AHEAD; born ≤ 1923); Children of the Depression (CODA; born 1924-1930); Original HRS (HRS; born 1931-1941); War Babies (WB; born 1942-1947); Early Baby Boomers (EBB; born 1948-1953); Mid Baby Boomers (MBB; born 1954-1959). Because the analytic sample utilizes survey data from 2004-2014, the MBB cohort which joined the HRS in 2010 are excluded from all analyses.

(currently 14 waves for the entire study), I am able to maintain temporal ordering among my key exogenous and endogenous variables laid out in Figures 1.1 and 1.2.

This dissertation draws from 6 waves of the HRS spanning 10 years from 2004 to 2014. I use the 2004 (wave 7) survey year as baseline because 2004 is when the majority of the childhood measures were implemented into the core surveys. In addition to the core surveys, this dissertation also incorporates biomarker data from the 2006/08 Enhanced Face-to-Face Interviews (EFTF) and mortality data from the restricted death data.³ Because the sampling frame of the HRS is comprised of midlife and older adults, information about respondents' childhood was collected retrospectively. For some, this means recalling events several decades after they were experienced. Although prior research has shown that most adults are able to recall the occurrence of past events, and particularly salient and/or noxious ones, with a relatively high degree of accuracy (Beckett et al. 2001; Krall et al. 1988; Smith 2009), these data, nonetheless, raise the issue of recall bias. To reduce issues related to recall bias, I controlled for variables that can influence recollection reliability as suggested by Vuolo et al. (2014), and set several criteria parameters for the analytic samples to increase the reliability and validity of the childhood measures. I describe these inclusion criteria in my Sample Description sections in Chapters 2 and 3.

³The EFTF was first implemented in 2006 when a random half of the sample was selected to participate. In the following survey year (2008), the EFTF was administered to the other random half of the sample. During the EFTF, inflammation levels were collected via dried blood spots (DBS). Response rate for completion of 2006 DBS was 81%. In 2008, DBS completion response rate was 87%.

Data analysis was conducted in Stata version 14 and Mplus version 7.

Preliminary and exploratory analyses as well as descriptive statistics were conducted in Stata. Final analyses were estimated in Mplus. The first study in Chapter 2 employs a series of regressions using childhood misfortune to predict inflammation levels. The second study in Chapter 3 estimates a series of Cox proportional hazards regression models using childhood misfortune to predict IHD onset. In both studies, statistical tests of mediation are conducted using Monte Carlo integration and a maximum likelihood robust estimator (MLR); the delta method is used to produce standard errors for direct and indirect effects (Muthén 2011). Because the HRS uses multistage clustered probability sampling, I use complex variance estimation in all analyses including descriptive statistics to account for stratification and clustering in the sample design. Failure to account for the complex sampling design, especially clustering, may lead to biased estimates (Korn and Gradbard 1995).

1.3 Description of Chapters

The remainder of this dissertation is divided into two main empirical articles followed by a concluding chapter. The first empirical study is presented in Chapter 2, and focuses on the process of how childhood misfortune influences immune functioning. In this study, the main outcome is chronic inflammation in adulthood and attention is given to the mechanisms connecting childhood misfortune to adult inflammation levels. I focus on mediators of adult health lifestyles and SES. Using multiple indicators of health lifestyles and SES, I expect that the effect of childhood misfortune on adult inflammation is mediated by adult health lifestyles and SES.

The second empirical study is presented in Chapter 3. Building upon the findings in Chapter 2, I propose a multidimensional pathway model of health over the life course. In this study, the main outcome is incident IHD in adulthood. I investigate how childhood misfortune influences the timing and onset of IHD over a six-year observation period. I expect that childhood misfortune raises IHD risk through multiple mediators of adult health lifestyles, SES, and chronic inflammation. Chapter 4 is the final chapter which discusses the findings of the two empirical chapters in light of practical policy implications as well as future directions of this area of research. Because this dissertation follows a two-article format, some of the materials, such as description of sample and variables, will be similar in each of the chapters.

CHAPTER 2. THE INFLAMMATORY SEQUELAE OF CHILDHOOD MISFORTUNE

2.1 Introduction

The proliferation of life course research has revealed that the social antecedents of many common ailments in adulthood can be traced back to childhood events and experiences. Misfortunate childhood experiences, such as maltreatment, poor health, and low socioeconomic status (SES), have been linked to arthritis (Blackwell et al. 2001), cancer (Blackwell et al. 2001; Felitti et al. 1998; Morton et al. 2012), cardiovascular disease (CVD) (Blackwell et al. 2001; Felitti et al. 1998; Hamil-Luker and O’Rand 2007), comorbidity (Greenfield and Marks 2009a), chronic obstructive pulmonary disease (Anda et al. 2008), and diabetes (Blackwell et al. 2001; Felitti et al. 1998) in adulthood. The CDC now classifies disadvantageous childhood experiences as risk factors for the leading causes of death in the United States, stating that “it is critical to understand how some of the worse health and social problems in our nation can arise as a consequence of adverse childhood experiences” (CDC 2014). The CDC also notes that the mechanisms connecting early-life experiences to adult health are less clear, calling for more research to address these “scientific gaps” in an effort to better understand how early-life experiences manifest as disease in later-life.

The immune system may help explain the long-term health consequences of childhood misfortune because many diseases associated with childhood misfortune have

potential inflammatory origins (Fagundes and Way 2014; Morey and Sergerstrom 2015). Whereas acute inflammation is a natural immune response to illness and injury, chronically elevated levels of inflammation can signal immune dysregulation and lead to Alzheimer's disease, cancer, CVD, disability, and premature mortality (Fagundes and Way 2014). Recent findings suggest that childhood misfortune can alter immune functioning, leading to chronic inflammation in adulthood (Fagundes and Way 2014; Morey and Sergerstrom 2015). Although this emerging research has advanced our understanding of the inflammatory sequelae of early misfortune, the majority of these studies utilizing samples of U.S. adults are theoretically limited, non-representative, comprised of a relatively small sample size, and focus on one type of misfortune—favoring childhood SES or maltreatment (e.g., Carpenter et al. 2010; Carpenter et al. 2011; Danese et al. 2007). Moreover, limited attention has been given to the *process* of how early-life experiences impact immune functioning and whether these processes vary by race or gender.

Utilizing data from a nationally representative study of American adults age 51 and older, this study seeks to elucidate the process of how childhood misfortune affects health on a physiological level by systematically investigating the long-term immunological consequences of early-life misfortune among Black, White, and Hispanic men and women. To extend prior research, this study considers alternative specifications of misfortune; examines whether health and socioeconomic factors mediate the effect of early misfortune on inflammation; and investigates if childhood experiences and their inflammatory sequelae vary by race, ethnicity and gender. Cumulative inequality (CI) theory and biological embedding serve as the theoretical frameworks guiding this study

(Ferraro and Shippee 2009; Hertzman and Boyce 2010). I begin with a summary of prior literature, followed by a discussion of CI theory and biological embedding.

2.2 Background

2.2.1 The Immunological Imprint of Childhood Misfortune

A burgeoning literature has revealed that children of misfortune exhibit biological signs of deterioration over time. This physiological wear and tear arising from early-life stressors has been documented for several biological markers of health such as cortisol, inflammation, and telomere length (e.g., Carpenter et al. 2009; Danese et al. 2007; Tyrka et al. 2010), and is reflected in many conceptual models of health, such as the allostatic load (McEwen 1998), biological embedding (Hertzman and Boyce 2010), ‘inflamm-aging’—i.e., immunosenescence—(Franceschi et al. 2000), and the weathering hypothesis (Geronimus et al. 2006). Among the many biomarkers assessed, the present study focuses on markers of inflammation—a reliable biomarker that can indicate dysregulation in multiple biological systems.⁴

⁴Much of the research on childhood misfortune and biological markers of health has used cortisol as the main indicator of biological dysregulation (e.g., Carpenter et al. 2009; Carpenter et al. 2011; Cicchetti, Rogosch, and Oshri 2011; Miller et al. 2009; Trickett et al. 2010; van der Vegt et al. 2009). Although a logical biological marker of choice given its theoretical and empirical relevance to the biological stress response system (Hertzman and Boyce 2010; McEwen 1998), the present study uses inflammation for several substantive and practical reasons. First, markers of inflammation have relatively good reliability and validity, especially since immunoassays with greater sensitivity became available (Pepys and Hirschfield 2003). In contrast, salivary swabs of cortisol have reliability issues due to sensitivity to extraneous factors, such as time of day and food intake (Schury and Kolassa 2012). Since cortisol levels fluctuate daily, dysregulation can be masked as either high or low levels of cortisol whereas immune dysregulation manifests as high levels. Second, markers of inflammation have good prognostic validity of health, making them apropos to study the early origins of adult health risks (Lloyd-Jones et al. 2006). Third, immune dysregulation often incurs a longer latency period than cortisol dysregulation. Since the analytics

The deleterious effects of childhood experiences on the immune system in adulthood have been documented using a variety of markers of inflammation (for review, see Baumeister et al. 2015). Childhood experiences of maltreatment and socioeconomic disadvantage are associated with elevated levels of C-reactive protein (CRP), fibrinogen, interleukin-6 (IL-6), white blood cells, and lymphocytes in adulthood (Baumeister et al. 2015; Carpenter et al. 2010; Carroll, Cohen, and Marsland 2011; Coelho et al. 2014; Danese et al. 2007; Miller et al. 2009; Slopen et al. 2010; Surtees et al. 2003; Zeugmann et al. 2012). These studies have also demonstrated the persistent effects of childhood misfortune on immune functioning. Chronic inflammation has been observed in young and midlife adults (Danese et al. 2007; Danese et al. 2009; Brummett et al. 2013; Carroll et al. 2011; Matthews et al. 2014; Rooks et al. 2012; Slopen et al. 2010; Taylor et al. 2006; Zeugmann et al. 2012) as well older adults (Carpenter et al. 2010; Stringhini et al. 2013; Surtees et al. 2003) who experienced misfortune during childhood. Some studies have linked childhood misfortune to chronic inflammation more than eight decades later, revealing the far-reaching grasp of early misfortune (e.g., Surtees et al. 2003).

Whereas the bulk of studies investigating the effect of childhood misfortune on adult inflammation have focused on the unique effects of childhood SES and maltreatment (Baumeister et al. 2015; Brummett et al. 2013; Carpenter et al. 2010; Carroll et al. 2011; Coelho et al. 2014; Danese et al. 2007; Danese et al. 2009; Lehto et

sample is comprised of older adults, immune functioning is a more optimal outcome of choice. Fourth, the HRS does not contain measures of cortisol. Future research may want to study cortisol, but immune functioning is more appropriate for addressing the research aims of this study.

al. 2012; Matthews et al. 2014; Stringhini et al. 2013; Taylor et al. 2006; Zeugmann et al. 2012), the broader literature on the early origins of adult health indicates that there other domains of early misfortune that can impact health in later life. Childhood health, for instance, has been associated with arthritis/rheumatism, cancer, CVD, and lung disease in adulthood (Blackwell et al. 2001). When combined, multiple domains of misfortune can exert a cumulative effect on health (e.g., Brown et al. 2010; Dong et al. 2004; Dube et al. 2009; Felitti et al. 1998; Morton et al. 2012), including inflammation (Rooks et al. 2012; Slopen et al. 2010; Surtees et al. 2003). Some scholars posit a “co-occurring risk hypothesis” (e.g., Danese et al. 2007), echoing the life course epidemiological concept of risk clustering (Ben-Shlomo and Kuh 2002), in which experiencing certain kinds of misfortune increases the likelihood of experiencing other kinds of misfortune. Indeed, many domains of misfortune are concatenated (e.g., childhood poverty and disease); analyzing multiple domains of misfortune not only informs scholars which types of misfortune are most consequential to health but can also rule out possible spurious relationships. An inventory of early-life experiences also enables scholars to investigate the additive effect of multiple domains of misfortune. Research comparing unique and additive specifications of childhood misfortune has uncovered the importance of considering alternative ways to measure misfortune, finding that the effects of childhood misfortune can vary based on how misfortune is operationalized (Morton et al. 2012). Therefore, it behooves research investigating the biological consequences of childhood misfortune to examine multiple domains of misfortune as well as alternative forms.

2.2.2 Pathways toward Immune Dysregulation

Despite the mounting evidence that childhood misfortune is a salient predictor of immune dysregulation in later life, the process of how early-life misfortune influences the immune system remains under-researched. Scholars examining childhood misfortune and adult inflammation often invoke the hypothalamic-pituitary-adrenal (HPA) axis to explain how early-life experiences interfere with internal physiology (e.g., Coelho et al. 2014; Fagundes and Way 2014; Hertzman 1999; McEwen 1998; Slopen, Koenen, and Kubzansky 2012; Surtees et al. 2003; Taylor, Way, and Seeman 2011). Indeed, the premise that the environment can impinge physiologically is fundamental to biology; stressful early-life experiences could trigger a physiological response, resulting in immune dysregulation. In response to environmental stressors, the hypothalamus releases corticotropin-releasing hormone (CRH), which signals the anterior pituitary gland to release adrenocorticotropic hormone (ACTH), which, in turn, signals the adrenal gland to release glucocorticoids (Schury and Kolassa 2012). These glucocorticoids released by the HPA axis modulate proinflammatory immune signaling (Webster, Tonelli, and Sternberg 2002). Whereas elevated levels of glucocorticoids suppress immune functioning, suppressed levels of glucocorticoids can lead to increased inflammation (Marketon and Glaser 2008; Webster et al. 2002). Initially, the HPA axis may flood the system with glucocorticoids, but later, as the HPA axis becomes blunted due to negative feedback at the hypothalamic level and decreased levels of glucocorticoids are released, inflammation becomes more prevalent. Through the modulatory effects of persistently elevated glucocorticoids, chronic exposure to stress can result in increased levels of inflammation (Marketon and Glaser 2008; Webster et al.

2002). Thus, higher levels of inflammation observed in adults who experienced childhood misfortune may be attributed to a dysregulated HPA axis.

Although these biological responses may give way to more permanent physiological alterations, there are additional paths through which early misfortune could affect adult immune functioning. The adverse childhood experiences study that pioneered much of the childhood misfortune and adult health research have applied their findings to develop a conceptual model that explains how childhood experiences can result in poor adult health and premature mortality (Felitti et al. 1998). This conceptual model includes health risk behaviors and psychosocial factors as potential pathways. Indeed, numerous studies have demonstrated the relationship between childhood misfortune and these non-physiological pathways. Childhood misfortune has been associated with unhealthy lifestyle factors such as smoking (Anda et al. 1999; Ford et al. 2011), high BMI/obesity (Greenfield and Marks 2009b; Power, Pinto Pereira, and Li 2015; Williamson et al. 2002), abnormal sleep patterns (Chapman et al. 2011), substance abuse (Dembo et al. 1992), and physical inactivity (Felitti et al. 1998). Childhood misfortune has also been associated with socioeconomic disadvantage, including low educational attainment (Case et al. 2005; Haas 2006; Palloni 2006), lower occupational status (Case et al. 2005; Palloni 2006), and decreased wealth accumulation (Haas 2006).

Many of the health-related and socioeconomic pathways of early misfortune are also related to inflammation. Health lifestyle factors of obesity, smoking, and exercise, for instance, are associated with chronic inflammation (Alley et al. 2006; Dandona, Alijada, and Bandyopandhyay 2004; Herd et al., 2012). Similarly, socioeconomic factors are also associated with inflammation: poverty, income, education, and wealth have been

linked to inflammation levels (Alley et al. 2006; Friedman and Herd 2010; Koster et al. 2006; Lubbock et al. 2005). Therefore, it may be that inflammatory responses are not a result of the HPA axis *per se*, but that other mechanisms are also at play. The handful of studies (only three to my knowledge) examining non-physiological mechanisms of childhood misfortune and adult inflammation report that BMI and smoking mediate the effect of early misfortune on adult inflammation levels (Brummett et al. 2013; Matthews et al. 2014; Surtees et al. 2003) whereas adult SES does not (Surtees et al. 2003). Building upon this work, the present study investigates both health-related and socioeconomic pathways through which childhood experiences influence immune functioning.

2.2.3 Race, Ethnicity, and Gender

Sociology has a rich history of considering how social structures can impact an individual's life course. The importance of social structures is rooted in classic sociological theory, with Durkheim, Marx, and Weber all privileging the role of social structures. Turning attention to more contemporary applications of the concept, the present study investigates how social structures of race, ethnicity, and gender may influence the relationship between childhood misfortune and adult inflammation.⁵

⁵Although race, ethnicity, and gender are often considered individual ascribed statuses, I draw from theoretical developments in sociology which also conceptualize these as social structures (Bonilla-Silva 1997; Risman 2004). Structures are, by definition, embedded within multiple societal dimensions—individual, organizational, institutional, and international. The pervasiveness of race, ethnicity, and gender throughout social processes and organizations as well as their ability to provide opportunities or constraints for agency qualifies them as social structures (Bonilla-Silva 1997; Risman 2004). Conceiving of race, ethnicity, and gender as social

Although limited attention has been given to racial, ethnic, and gender differences in the effects of childhood misfortune on adult inflammation, patterns of childhood misfortune and inflammation appear to vary by race, ethnicity, and gender. A study by Slopen et al. (2010) found that experiencing multiple types of misfortune early in life predicted higher levels of inflammation in Black but not White midlife adults. These racial differences may be explained by intermediary processes. Brummett et al. (2013) found that the effect of childhood SES on adult inflammation was mediated by BMI and smoking for White respondents only, not Black respondents. Thus, childhood misfortune may operate differently by race; early-life misfortune may indirectly affect inflammation for White adults whereas early-life misfortune may exert a direct effect on inflammation for Black adults.

Whereas fewer studies highlight the role of gender, mediational effects of the childhood misfortune-adult inflammation relationship also appear to vary by gender. Smoking has been shown to mediate the effect of childhood SES on adult inflammation for men, but not women (Brummett et al. 2013). Prior childhood misfortune research suggests that gender differences are most salient for childhood socioeconomic disadvantage (Hamil-Luker and O’Rand 2007). A possible explanation is that gender socially patterns how individuals respond to misfortune: women have reported similar events as more upsetting and requiring a longer recovery period than men (Surtees and Wainwright 2007). Given the emerging evidence indicating that race, ethnicity, and gender may influence the relationship between childhood misfortune and adult

structures (in addition to social statuses), I examine whether these social structures will pervade the childhood misfortune-adult inflammation relationship.

inflammation, the present study investigates the moderating effects of these social structures.

2.3 Theoretical Framework

2.3.1 Cumulative Inequality Theory

This study primarily draws from cumulative inequality (CI) theory. CI theory is a middle-range theory that elucidates how social inequality takes shape over the life course (Ferraro and Shippee 2009; Ferraro, Shippee, and Schafer 2009). Specified in five axioms and 19 propositions, the theory holds that “social systems generate inequality, which is manifested over the life course via demographic and developmental processes, and that personal trajectories are shaped by the accumulation of risk, available resources, perceived trajectories, and human agency” (Ferraro and Shippee 2009:334). CI theory is particularly useful to the current line of research because it explicates how health outcomes observed in later life have often been structured throughout the life course. Thus, it brings the past into a relational context with the present, piecing together life histories to explain how health develops and differentiates over time. Indeed, the concept of connecting early life experiences to distal outcomes in later life has not emerged solely from CI theory.

Sociology has a history of considering the life course and accumulation processes, as noted in Elder’s (1998) work on the Great Depression, Pearlin’s stress process (1981, 1989) as well as Merton’s (1968) work on scientific trajectories. CI theory is, indeed, a derivative of this prior work. However, CI theory has advanced this area of research by expounding upon the mechanisms underlying the process of cumulative inequality;

privileging critical and sensitive periods; differentiating processes of advantage from those of disadvantage; and highlighting key concerns in the study of health and aging. CI theory is, therefore, most relevant to the proposed research. I expound upon three elements of CI theory that are germane to the present study.

First, CI theory contends that early life stages, gestation through childhood, are sensitive periods during which events, experiences, and exposures have a greater influential potential on later-life outcomes.⁶ Childhood conditions have the ability to exert a lasting impact because these experiences provide different opportunities and constraints, differentiating individuals early in life. Over time, these initial differences can compound, revealing life course patterns of cumulative inequality (e.g., O’Rand and Hamil-Luker 2005). CI theory also acknowledges that childhood experiences do not solely entail individual traits and experiences. Although childhood misfortune consists of individual factors, such as health conditions, it also includes the broader family context, such as parent’s SES and maltreatment—all of which can shape trajectories of health. Guided by CI theory, the present study proposes that the cumulative health risk incurred from early misfortune is manifest on a physiological level. This study also considers multiple domains of childhood misfortune—on the individual and family level.

Second, CI theory asserts that disadvantage increases risk of further disadvantage. This process, commonly known as a chain of risks in life course epidemiology (Ben-

⁶Ferraro and Shippee (2009) use the term critical period (see p. 338), but their following discussion appears to denote a sensitive period in that experiences and events are not deterministic. Following the work of life course epidemiologists (Ben-Shlomo and Kuh 2002), I use sensitive period to describe periods of heightened susceptibility, and reserve the term critical period for which effects of exposures are definite and irreversible.

Shlomo and Kuh 2002), describes how health disparities become manifest over the life course. Essentially, experiencing disadvantage increases the likelihood of subsequent disadvantage, especially when disadvantage occurs within a sensitive period, such as childhood. If uninterrupted, this cycle of disadvantage can continue, leading to observable differences in later life. CI theory also acknowledges that this process of inequality diffuses across multiple life domains, as evidenced by the well-documented relationship between wealth and health (e.g., Shippee, Wilkinson, and Ferraro 2012). Translating this into the current study, misfortune may initiate a trajectory of disadvantage which diffuses across health behavioral and socioeconomic life domains, culminating in chronic inflammation.

Third, CI theory notes the importance of using cohorts to study how health is indexed in historical time; a cohort specification helps one to connect shared historical experiences to health risks. Indeed, sociology of age has chronicled how cohorts traverse different age strata together, sharing historical and structural influences that create unique conditions for each cohort (Riley 1973, 1987). Thus, conceptualizing age in terms of birth cohorts can reveal how cohorts structure risks over the life course. Another advantage of using cohorts is greater attention to selection processes, especially in samples of older adults (Ferraro and Shippee 2009). Health selection is important in longitudinal studies because declining health and mortality may inadvertently lead to underestimating variance in the outcome because the persons with the most health problems are omitted from the analyses. Therefore, the present study uses cohorts as a tool to better explicate these processes.

2.3.2 Biological Embedding

Although CI theory provides a comprehensive explanation for how childhood misfortune can create later-life health gradients, there is no explicit attention given to how childhood misfortune impacts health on a physiological level. To supplement CI theory, this study also draws from Hertzman and Boyce's (2010) biological embedding, a cogent framework that links psychosocial processes to physiological processes. Biological embedding posits that certain early-life circumstances, events, and experiences can cause permanent, long-term damage via their physiological effects (Hertzman 1999, 2012; Hertzman and Boyce 2010). Two components of biological embedding are essential to understanding the long-arm of childhood misfortune.

First, childhood is seen as a sensitive period, echoing one of CI theory's propositions. Childhood comprises a substantial period of growth and development during which biological systems are calibrating and susceptible to external influences. During this time, environmental stressors have the potential to interfere these developing systems. For instance, the HPA axis is particularly malleable during the early years of life as regulatory stress response systems are being set (Tarullo and Gunnar 2006). As demonstrated by allostatic load research, chronic or severe exposure to stress during childhood can alter the HPA axis; the HPA axis responds to this stress exposure, physiologically adapting to the current situation (McEwen 1998). Whereas this physiological response is adaptive in the short-term, it may become maladaptive in the long-term, resulting in physiological dysregulation throughout the life course. By framing childhood as a *biologically* sensitive period, biological embedding strengthens

the case for the association between childhood misfortune and biological markers of immune functioning in later life.

Second, biological embedding posits that these biological disturbances alter biological processes in *stable ways that endure throughout life*. As revealed above by the prior literature, childhood misfortune is associated with physiological alterations in adulthood. Biological embedding states that these environmental insults can incur damage via latency, accumulation, or pathways over time. Thus, biological embedding entails a *process* which can begin in childhood, but the physiological effects may not become manifest—i.e., remain latent—until later in life. Regardless of when the biological disruption occurred, this physiological dysregulation persists into adulthood. Guided by this tenet, it is theoretically plausible to assert that the effect of childhood misfortune on inflammation endures throughout the life course, enabling researchers to observe effects in midlife and beyond. Using a sample of older adults, the present study proposes a pathway model, in which childhood misfortune impairs immune functioning via health-related and socioeconomic factors.

2.4 Sample Description

This study draws from waves 7-9 of the Health and Retirement Study (HRS), spanning from 2004 to 2008 (Health and Retirement Study 2015). The HRS is a nationally representative, biennial panel study comprised of over 20,000 American adults aged 51 years and older. The HRS employed a multistage, stratified area probability sample of American households, with an oversample of African Americans, Hispanics, and Floridians, from which respondents over the age of 50 were selected. Overall,

baseline response rates were very good (> 80%), except for the oversample of Hispanic Americans which was, nonetheless, good (>70%) (Sonnegga et al. 2014). By 2004, the original HRS sample merged with several other samples to replenish younger cohorts, all of which have had high response rates.⁷ The present study analyzes a sample of 8,947 adults who participated in the Enhanced Face-to-Face Interview (EFTF) as well as the core surveys beginning in 2004 (see Appendix A for inclusion criteria of analytic sample).⁸ The EFTF was first implemented in 2006 when a random half of the sample was selected to participate.⁹ During the EFTF, physical measures including markers of inflammation were collected. In the following survey year (2008), the EFTF was administered to the other random half of the sample to collect the physical measures from these respondents. The present study uses data from both survey years to include data on inflammation for the entire sample. Unless stated otherwise, all variables were drawn from the raw HRS data files.

⁷The additional cohorts include the Asset and Health Dynamics Among the Oldest Old (AHEAD), Children of the Depression Age (CODA), the War Babies (WB), and the Early Baby Boomer (EBB) samples. 2004 response rates were as follows: original HRS 86.4%; AHEAD 89.4%; CODA 90.1%; WB 87.9%; and EBB 75.3%. Response rates have been greater than 70% at each subsequent wave.

⁸Selection criteria for the analytic sample were based on eligibility as well as reliability and validity of key variables. Respondents were excluded if they were not age eligible at baseline (2004); had childhood information collected from a proxy response; scored 2 SD below the mean cognition score at baseline; did not have inflammation collected; or had CRP levels >8.6 ug/mL. See Appendix A for more information.

⁹The HRS Biomarker data collected during the EFTF was sponsored by the NIH (NIA U01AG009740) and conducted by the University of Michigan (Health and Retirement Study 2006).

2.5 Measurement

2.5.1 Chronic Inflammation

Adult chronic inflammation was assessed using C-reactive protein (CRP), a reliable and valid marker of inflammation (McDade et al. 2004) and a robust inflammatory outcome of childhood misfortune (for reviews, see Baumeister et al. 2015; Coelho et al. 2014).¹⁰ Dried blood spots (DBS) were collected from respondents by pricking their cleansed finger with a sterile lancet. Blood droplets were placed on filter paper, wrapped in foil envelopes with a desiccant packet, and placed in mailing containers. This process does not require temperature control to preserve the specimens. The DBS were immediately sent to Biosafe and/or Flexsite laboratories where they were frozen upon receipt. DBS were then sent to the University of Vermont for assay of CRP. CRP is a continuous variable measured in micrograms per milliliter (ug/mL). To assess chronic inflammation, respondents with extremely high CRP levels (>8.6 ug/mL) were removed from analyses as these cases likely indicate acute inflammatory disease or injurious stimuli (Herd et al. 2012). Approximately 13% of cases were dropped due to acute inflammation. Following prior literature, CRP was log-transformed to adjust for skewness (e.g., Herd et al. 2012). To verify the robustness of the results presented herein, sensitivity analyses using an ordinal measure of CRP were also conducted in which raw CRP levels were separated into three clinical thresholds: <1.0, 1-2.99, and \geq 3.0 ug/mL (Sesso et al. 2003).

¹⁰Following the recommendation of the 2006 and 2008 HRS biomarker documentation report, the NHANES equivalent CRP values were used for analysis (Crimmins et al. 2013).

2.5.2 Childhood misfortune

The main predictors of childhood misfortune were created using 31 self-reported, retrospective indicators of childhood events and experiences. Based on prior literature, correlation matrices, and polychoric factor analysis, these 31 indicators were separated into six domains of childhood misfortune: childhood SES, parental behaviors, infectious disease, chronic disease, impairments, and adolescent behaviors (Felitti et al. 1998; Kemp, Morton, and Ferraro 2015; Morton et al. 2012; Turner, Wheaton, and Lloyd 1995). Childhood SES was measured using mother's education, father's education, father's occupation, perceived financial situation, and relocation due to financial strain. Measures of risky parental behaviors included whether a parent physically abused the respondent, had alcohol or substance abuse issues, or smoked. Childhood infectious disease included measles, mumps, and chicken pox. Childhood chronic disease included asthma, diabetes, respiratory disorder, allergies, heart disease, ear problems, seizures, migraines, stomach problems, high blood pressure, and poor self-rated health. Childhood impairment was measured using learning problems, speech impairment, vision impairment, disability (≥ 6 months), and head injury or trauma requiring medical attention. Indicators of risky adolescent behaviors and mental health (simply referred to as adolescent behaviors hereafter) included substance abuse, police troubles, and psychological problem (depression or otherwise). All measures of misfortune occurred before the age of 16 except for measures of parents' behaviors, childhood learning problems, and police troubles which could have occurred any time prior to age 18.

Because the majority of indicators (26/31) have binary response categories, all 31 measures were initially dichotomized with "1" indicating experiencing the misfortune

and “0” as otherwise.¹¹ The first specification of childhood misfortune utilized the six domains of misfortune as separate predictors to investigate the unique effects of different kinds of misfortune. After dichotomizing each indicator of misfortune, the indicators were summed within each domain to create the six domains of misfortune: childhood SES, parental behaviors, infectious disease, chronic disease, impairment, and adolescent behaviors. The measurement is summarized in Figure 2.1. Each of the domains ranges from 0-2, with 2 indicating experiencing 2 or more indicators within that domain. Each domain was top-coded at 2 because few respondents experienced more than 2 indicators. I also wanted to maintain consistency across the domains since each domain varied in total number of indicators.

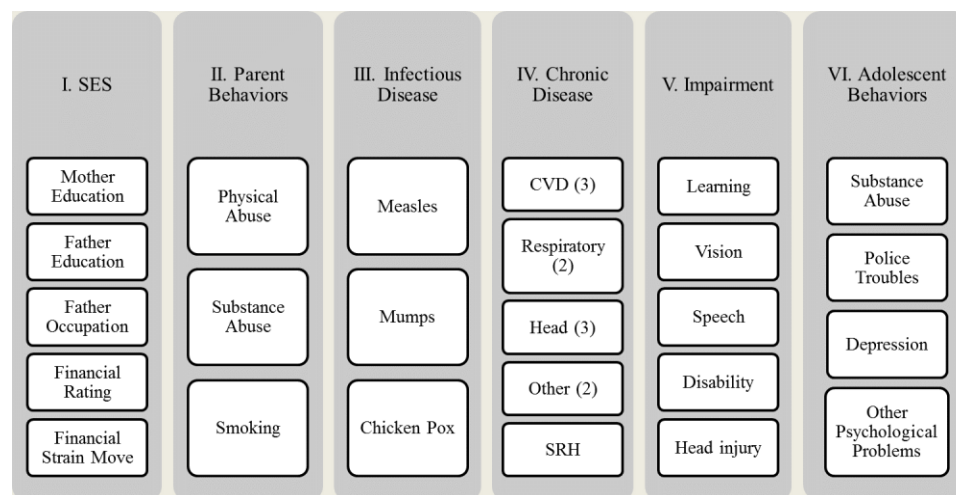


Figure 2.1 Childhood Misfortune Indicators by Domain.

¹¹Poor self-rated health was coded as “1” for respondents who rated their childhood health as either poor or fair (Haas 2007). To assess socioeconomic disadvantage during childhood, mother’s and father’s education were dichotomized so that 1 = <12 years of education (i.e., less than a high school education). Father’s occupation was coded 1 for non-skilled manual occupations, following U.S. Labor Statistics occupational categories (U.S. Bureau of Labor Statistics 2010). Perceived financial situation was coded “1” for respondents who rated their family financial situation as “poor.”

The next specification of childhood misfortune used a single count variable of childhood misfortune to investigate the additive effect of early misfortune. To create the additive childhood misfortune (ACM) variable, I created a dummy variable for each of the domains, with “1” indicating that the respondent reported experiencing at least one of the indicators in that domain. Next, I summed across the domains to create the ACM variable. This variable ranges from 0-6, with 0 indicating that the respondent did not experience any indicator of misfortune in any of the domains and 6 indicating that the respondent reported experiencing at least one indicator of misfortune in each domain.

2.5.3 Health Lifestyle Mediators

Health lifestyle factors included variables of smoking, BMI, and exercise assessed in 2004. Smoking is a continuous variable measured as pack-years. Among smokers (i.e., respondents who smoked >100 cigarettes in their lifetime), respondents reported when they began smoking and when they quit (unless they were current smokers in 2004) as well as how much they smoked daily, on average. From this information, I calculated the total years each respondent smoked (year stopped minus year started for former smokers; 2004 minus year started for current smokers). Next, I multiplied the total years smoked by the average number of cigarettes smoked daily and divided by 20 (amount of cigarettes in a pack) to calculate pack-years smoked. Pack-years is top-coded at 280. Respondents who never smoked were coded as “0”.

BMI is a continuous variable from the RAND dataset, which was calculated from respondents’ self-reports of weight and height. BMI is measured in kg/m^2 and top coded at $61.3 \text{ kg}/\text{m}^2$. The variable for exercise assesses respondent’s frequency of vigorous

exercise and was drawn from the RAND dataset. Respondents were asked how often they took part in vigorous exercise or activities such as running/jogging, swimming, cycling, aerobics/gym workouts, tennis, or digging with spade/shovel. Response categories were daily, >1 time per week, 1 time per week, 1-3 times per month, and never. The variable was reverse coded so that higher values indicate more frequent exercise.

2.5.4 Socioeconomic Mediators

Adult SES variables were drawn from the RAND dataset and include 2004 measures of education and wealth. Education is a continuous measure of years of education assessed from respondent's highest level of education completed. Wealth is a continuous variable measured in dollars at the household level. Wealth was calculated as total wealth (all assets including secondary residence) minus all debt.¹² To adjust for skewness, a cubic root transformation of wealth was used. Wealth is a more reliable indicator of finances than income for older adults as many are retired and, therefore, are not currently receiving an annual salary. Wealth taps into the accumulated assets and debts these older individuals have incurred over their lifetime.

¹²RAND used 15 indicators of wealth from the HRS dataset. If an indicator was missing, RAND imputed the values utilizing several methods of imputations. Hurd et al. (2014) provide a detailed description of this process.

2.5.5 Covariates

Models were adjusted for several adult risk factors related to either childhood misfortune or inflammation levels. These include cohort, gender, race, marital status, prescription medications, chronic conditions, and depressive symptoms (Herd et al. 2012; Jain and Ridker 2005; Kenis and Maes 2002; Schafer and Ferraro 2011). Respondents were divided into 5 birth cohorts: (1) 1908-1919; (2) 1920-1929; (3) 1930-1939; (4) 1940-1949; (5) 1950-1953. Because only four respondents were born before 1910, those born in 1908 and 1909 were collapsed into the 1910s cohort. The 1950s cohort was used as the reference group. Race was coded as four separate binary variables: non-Hispanic White, non-Hispanic Black, Hispanic, and non-Hispanic Other Race (Heisler et al. 2007). The HRS combined responses of American Indian, Alaskan Native, Asian, Pacific Islander, and other into a single category of “other” due to the low number of responses for each of these categories. Non-Hispanic White is the reference group.

Gender, marital status, prescription medication, and chronic conditions were coded as dummy variables (respectively, 1=female; 1=married; 1= taking hypertensive/psychiatric medication; 1= at least one chronic condition). Chronic conditions include physician diagnosis of hypertension, diabetes, cancer, lung disease, heart problems, stroke, and arthritis/rheumatism, all of which can affect inflammation levels. Chronic conditions were included as a single measure to reduce multicollinearity issues related to covariates of prescription medication and depressive symptoms. Depressive symptoms is a composite score of 8 items and follows the Center for Epidemiologic Studies Depression (CES-D) scale. The CES-D index assessed how often respondents felt depressed, like everything was an effort, sleep was restless, alone, sad,

couldn't get going, happy (reverse-coded) or enjoyed life (reverse-coded) in the past week. Response categories for each indicator ranged from none or almost none to all or most of the time. The depressive symptoms variable was coded so that higher values indicate more depressive symptoms. All covariates were assessed at baseline with the exception of prescription medications. Since hypertensive and psychiatric medications can directly affect inflammation levels, this variable was assessed when CRP was collected.

Models also included a flag indicating when CRP was measured. The dummy variable for measurement year is coded for 1 for respondents whose CRP was collected in 2006 and 0 for those assessed in 2008.

2.6 Analysis

Preliminary analyses were conducted in Stata version 14. For final analyses, a series of regression models were estimated in Mplus version 7 using alternative specifications of childhood misfortune as key predictors (Muthén and Muthén 2007). First, models were estimated without any mediators to establish a baseline relationship between childhood misfortune and adult inflammation. Next, health and socioeconomic mediators were simultaneously introduced into the models. Statistical tests of mediation were conducted using Monte Carlo integration and a maximum likelihood robust estimator (MLR) to produce standard errors for direct and indirect effects. For these mediations tests, paths were created to establish a direct relationship between (a) childhood misfortune and each mediator, and (b) each mediator and CRP; indirect effects were calculated as the product of the two paths ($a*b$). Standard errors were calculated

using the delta method. Within the structural equation modeling framework, Monte Carlo integration in Mplus is recommended when using mediators with missing data (Muthén 2011).

Preliminary analyses were conducted to determine whether the form and measurement of the overall models varied by race and gender. Overall model fit did not substantially improve when models were stratified by race and gender (statistical evidence for measurement invariance). Therefore, final models are not stratified by race or gender. However, a series of race and gender interactions for each of the main effects was examined, and significant findings are presented below. Given the age of the sample, additional supplementary analyses investigated the effect of mortality bias as well as a squared term of BMI. To account for selection issues due to mortality, I conducted supplementary analyses using Heckman (1979) selection bias models to determine whether parameter estimates were biased. The selection lambda was not significant and is, therefore, not included in the final models presented. In addition, the squared term of BMI was non-significant, and final models only present a single term of BMI. Sensitivity analyses using an ordinal measure of inflammation were conducted. A series of ordered regression models were conducted, following the same steps outlined above. Results (not presented) yielded similar conclusions to those presented herein and are available upon request.

All statistical analyses were weighted and adjusted for the complex survey design (i.e., stratification and clustering). In addition, item-missing data for the independent variables were dealt with using full-information maximum likelihood (FIML) (Muthén and Muthén 2007).

2.7 Results

Table 2.1 displays descriptive statistics for all variables. Since models were weighted and adjusted for clustering, weights and survey design adjustments are also applied to univariate statistics. The mean of logged CRP is 0.469. The most commonly experienced domain of childhood misfortune is infectious disease (mean=1.735) whereas the least common domain of misfortune is adolescent behaviors (mean=0.102). When combined, the average number of childhood misfortunes experienced is 3.218 (ACM mean). The mean number of pack-years smoked is 15.062 whereas the mean BMI is 27.538 kg/m². On average, these respondents do not engage in vigorous exercise often (1-3 times per month was the mean response). The mean of education (13.141 years) indicates that the average respondent has some post-high school education. The mean of the cubic root of wealth is 60.867, revealing a somewhat wealthy sample on average (\$221,225.60). Overall, the sample is predominantly White, married, born in the 1940s, has at least one chronic condition, and scored low on the CES-D scale. Approximately half of the sample is female and taking a hypertensive or psychiatric prescription medication.

Table 2.1 Descriptive Statistics of Variables, Health and Retirement Study (N=8,947)

Variable	Range	Mean	SD ^a
<i>Chronic Inflammation</i>			
CRP, logged ^b	-3.912-2.149	0.469	0.981
<i>Childhood Misfortune</i>			
ACM	0-6	3.218	0.969
SES	0-2	1.288	0.838
Parent Behaviors	0-2	0.875	0.652
Infectious Disease	0-2	1.735	0.572
Chronic Disease	0-2	0.455	0.698
Impairment	0-2	0.228	0.487
Adolescent Behaviors	0-2	0.102	0.340
<i>Adult Health Lifestyles</i>			
Pack-years Smoked	0-280	15.062	24.996
BMI	9.6-61.3	27.538	5.253
Exercise	1-5	2.168	1.357
<i>Adult SES</i>			
Education	0-17	13.141	2.891
Wealth, cubic root	-130.950-315.755	60.867	36.228
<i>Covariates</i>			
1910 Cohort	0,1	0.027	
1920 Cohort	0,1	0.131	
1930 Cohort	0,1	0.222	
1940 Cohort	0,1	0.401	
1950 Cohort ^c	0,1	0.219	
Female	0,1	0.547	
White ^c	0,1	0.836	
Black	0,1	0.073	
Hispanic	0,1	0.068	
Other Race	0,1	0.023	
Married	0,1	0.701	
Prescription Medications	0,1	0.535	
Chronic Conditions	0,1	0.767	
Depressive Symptoms	0-8	1.291	1.890
Measurement Year	0,1	0.491	

Notes: Descriptive statistics are weighted.

^aThe SD of dichotomous variables is omitted because it is a function of the mean.

^bAssessed during EFTF measurement year.

^cReference group.

Table 2.2 displays the results of regressing CRP on the independent variables. The first two models investigated the additive effect of childhood misfortune and, therefore, use ACM as the main independent variable. To establish a baseline relationship between ACM and adult inflammation, Model 1 examined the effect of ACM on adult CRP without including any of the mediators. ACM has a positive effect on CRP, net of all covariates ($b=0.034$, $p<0.05$). For each additional misfortune experienced during childhood, adult CRP levels are expected to increase by 0.034 units ($p<0.01$). Birth cohort, gender, race, medications, chronic conditions, depressive symptoms, and measurement year were also significant. Compared to those born in the 1950s, people born before 1920 have lower CRP levels, on average ($b=-0.163$, $p<0.05$). Women had higher levels of CRP than men ($b=0.158$, $p<0.001$). Compared to White respondents, Black and Hispanic respondents had higher levels of CRP (respectively, $b=0.092$, $p<0.05$; $b=0.98$, $p<0.05$) and Other racial groups had lower levels of CRP ($b=-0.216$, $p<0.05$). Respondents who were taking prescription medication, reported at least one chronic condition, and had higher levels of depressive symptoms had higher levels of CRP (respectively, $b=0.167$, $p<0.001$; $b=0.151$, $p<0.001$; $b=0.039$, $p<0.001$). Respondents whose CRP was collected in 2006 had lower levels of CRP than respondents whose CRP was collected in 2008 ($b=-0.099$, $p<0.01$).

Model 2 introduced mediators of adult health lifestyle and SES. When all five mediators were introduced, the effect of ACM was attenuated and became non-significant ($b=-0.005$, $p=0.320$). All of the five health-related and socioeconomic variables had a significant effect on CRP, net of adult risk factors. For each additional pack-year smoked, CRP increased by 0.003 ($b=0.003$, $p<0.001$). A one-unit increase in BMI was

associated with an increase of 0.045 units of CRP ($b=0.045, p<0.001$). Respondents who exercised more frequently had lower levels of CRP ($b=-0.025, p<0.05$). For each additional year of education, CRP levels decreased by 0.017 ($b=-0.017, p<0.001$). Similarly, increases in wealth predicted lower levels of CRP ($b=-0.002, p<0.001$). Similar to Model 1, female, medication, and depressive symptoms predicted higher levels of CRP whereas Other Race and 2006 measurement year predicted lower levels of CRP. However, the significant effects of the 1910 cohort, Black, Hispanic, and chronic conditions became non-significant when the mediators were introduced into the model. Whereas levels of CRP were no longer significantly different between the 1910 and 1950 cohorts, the 1940 cohort had higher levels of CRP than the 1950 cohort ($b=0.073, p<0.05$).

Models 3 and 4 examined the unique effects of each domain of misfortune to determine whether certain types of misfortune were more consequential to chronic inflammation. Parallel to Model 1, Model 3 examined the relationship between the six domains of misfortune and adult CRP without adjusting for the mediators. Among the six domains of misfortune, childhood SES predicted higher CRP levels, net of all covariates. Each additional experience of socioeconomic disadvantage during childhood increased CRP levels by 0.052 units ($b=0.052, p<0.01$). Similar to Model 1, birth cohort, gender, medications, chronic conditions, depressive symptoms, and measurement year were significant predictors of adult CRP. Although effect sizes varied between Models 1 and 3, the direction of these relationship between the significant covariates and CRP remained the same. Unlike Model 1, Black and Hispanic were not significant, and the 1920 cohort had lower levels of CRP than the 1950 cohort ($b=-0.116, p<0.05$).

Table 2.2 Regression of Independent Variables on Chronic Inflammation (HRS: 2004-2008)

Independent Variables	Model 1 <i>b</i> (SE) ^a	Model 2 <i>b</i> (SE)	Model 3 <i>b</i> (SE)	Model 4 <i>b</i> (SE)
<i>Childhood Misfortune</i>				
ACM	0.034 (0.013)*	-0.005 (0.013)		
SES			0.052 (0.018)**	-0.002 (0.018)
Parent Behaviors			0.022 (0.018)	0.013 (0.018)
Infectious Disease			-0.031 (0.022)	-0.024 (0.020)
Chronic Disease			-0.025 (0.017)	-0.017 (0.016)
Impairment			0.024 (0.027)	-0.004 (0.025)
Adolescent Behaviors			0.036 (0.035)	0.005 (0.031)
<i>Covariates</i>				
1910 Cohort ^b	-0.163 (0.069)*	-0.004 (0.074)	-0.226 (0.076)**	-0.005 (0.079)
1920 Cohort ^b	-0.066 (0.041)	0.019 (0.042)	-0.116 (0.045)*	0.021 (0.046)
1930 Cohort ^b	-0.049 (0.045)	-0.006 (0.043)	-0.087 (0.047)	-0.005 (0.045)
1940 Cohort ^b	0.055 (0.039)	0.073 (0.035)*	0.035 (0.040)	0.074 (0.036)*
Female	0.158 (0.022)***	0.200 (0.022)***	0.158 (0.020)***	0.205 (0.021)***
Black ^c	0.092 (0.046)*	-0.040 (0.043)	0.073 (0.047)	-0.042 (0.043)
Hispanic ^c	0.098 (0.039)*	-0.051 (0.043)	0.061 (0.041)	-0.056 (0.045)
Other Race	-0.216 (0.100)*	-0.224 (0.098)*	-0.235 (0.101)*	-0.231 (0.099)*
Married	-0.011 (0.030)	0.023 (0.032)	-0.011 (0.031)	0.024 (0.032)
Prescription Medications	0.167 (0.028)***	0.068 (0.024)**	0.164 (0.028)***	0.069 (0.024)**
Chronic Conditions	0.151 (0.033)***	0.049 (0.032)	0.150 (0.032)***	0.050 (0.032)
Depressive Symptoms	0.039 (0.007)***	0.019 (0.007)**	0.039 (0.007)***	0.019 (0.007)**
Measurement Year	-0.099 (0.026)**	-0.102 (0.026)**	-0.093 (0.025)***	-0.102 (0.026)***
<i>Adult Health Lifestyles</i>				
Pack-years Smoked		0.003 (0.001)***		0.003 (0.001)***

BMI		0.045 (0.003)***		0.045 (0.003)***
Exercise		-0.025 (0.010)*		-0.025 (0.010)*
<i>Adult SES</i>				
Education		-0.017 (0.005)***		-0.017 (0.005)**
Wealth, cubic root		-0.002 (0.000)***		-0.002 (0.000)***
<i>R</i> ²	0.042***	0.095***	0.045***	0.095***
AIC	24677.251	322803.705	24662.481	321288.016
N	8947	8947	8947	8947

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed tests).

^aUnstandardized coefficient (standard error).

^bReference group: 1950 Cohort.

^cReference group: White respondents.

Model 4 introduces the hypothesized health-related and socioeconomic mediators. Similar to the effect of ACM in Model 2, the effect of childhood SES became non-significant when the mediators entered the model ($b=-0.002$, $p=0.918$). Each of the health-related and socioeconomic variables was associated with adult CRP while controlling for all domains of childhood misfortune and adult risk factors. Each additional pack-year smoked increased CRP by 0.003 units ($b=0.003$, $p<0.001$). A one-unit increase in BMI was associated with an increase of 0.045 units of CRP ($b=0.045$, $p<0.001$). Respondents who exercised more frequently had lower levels of CRP ($b=-0.025$, $p<0.05$). Each additional year of education decreased CRP levels by 0.017 units ($b=-0.017$, $p<0.001$) whereas higher levels of wealth predicted lower levels of CRP ($b=-0.002$, $p<0.001$). The effects of female, race, medication, depressive symptoms, and measurement year remained significant when the mediators were introduced into the model. However, the effects of birth cohort changed. Whereas levels of CRP for the 1910 and 1920 cohorts no longer differed from the 1950 cohort, the 1940 cohort had higher levels of CRP than the 1950 cohort ($b=0.074$, $p<0.05$). The effect of chronic conditions observed in Model 3 were no longer significant in Model 4.

Table 2.3 Mediation Results: Additional Direct and Indirect Effects, HRS (2004-2008)

Childhood Misfortune	<u>Health Lifestyle Mediators</u>			<u>SES Mediators</u>	
	Pack-years Smoked <i>b</i> (SE) ^a	BMI <i>b</i> (SE)	Exercise <i>b</i> (SE)	Education <i>b</i> (SE)	Wealth <i>b</i> (SE)
ACM ^b	1.964 (0.325)***	0.446 (0.078)***	-0.019 (0.021)	-0.107 (0.034)**	-3.784 (0.577)***
Indirect Effect ^b	0.006 (0.001)***	0.020 (0.003)***	0.001 (0.001)	0.002 (0.001)*	0.008 (0.002)***
SES ^c	3.859 (0.563)***	0.409 (0.085)***	-0.222 (0.024)***	-1.104 (0.047)***	-6.578 (0.637)***
Indirect Effect ^c	0.011 (0.003)***	0.018 (0.004)***	0.006 (0.002)**	0.019 (0.006)***	0.013 (0.002)***
Female ^c	-5.143 (0.965)***				
SES x Female ^c	-3.113 (0.678)***				

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed tests).

^aUnstandardized coefficient (standard error).

^bAdditional results from Table 2.2, Model 2.

^cAdditional results from Table 2.2, Model 4.

As described above in the analysis section, I conducted statistical tests of mediation when the mediators were introduced in Table 2.2, Models 2 and 4. Table 2.3 displays the additional direct and indirect effects produced by the statistical tests of mediation for each mediator. As shown, there is statistical evidence that the effect of childhood misfortune on adult inflammation is transmitted via health lifestyles and SES. ACM had a significant direct effect on health-related mediators of smoking ($b=1.964$, $p<0.001$) and BMI ($b=0.446$, $p<0.001$). ACM also had a significant direct effect on socioeconomic mediators of education ($b=-0.107$, $p<0.01$) and wealth ($b=-3.784$, $p<0.001$). In addition, the indirect effects associated with each of these mediators were also significant, revealing that the effect of ACM on adult CRP was mediated by smoking ($b=0.006$, $p<0.001$), BMI ($b=0.020$, $p<0.001$), education ($b=0.002$, $p<0.05$), and wealth ($b=0.008$, $p<0.001$). Although exercise had a direct effect on CRP, there was no statistical evidence indicating that exercise mediated the effect of ACM on CRP.

Formal tests of mediation elucidating the path from childhood SES to adult inflammation revealed that adult health lifestyles and SES mediated the effect of childhood SES on adult inflammation. Childhood SES had a significant direct effect on smoking ($b=3.859$, $p<0.001$), BMI ($b=0.409$, $p<0.001$), exercise ($b=-0.222$, $p<0.001$), education ($b=-1.104$, $p<0.001$), and wealth ($b=-6.578$, $p<0.001$). The direct effect of childhood SES on smoking was moderated by gender; the effect of childhood SES on smoking was stronger for men than women. Indirect effects revealed that the relationship between childhood SES and adult CRP was mediated by smoking ($b=0.011$, $p<0.001$), BMI ($b=0.018$, $p<0.001$), exercise ($b=0.006$, $p<0.01$), education ($b=0.019$, $p<0.001$), and wealth ($b=0.013$, $p<0.001$).

To demonstrate how these mediation results map out on to the proposed pathway model in Figure 1.1, Figure 2.2 illustrates the relationships among childhood misfortune, adult health lifestyles, SES, and chronic inflammation.

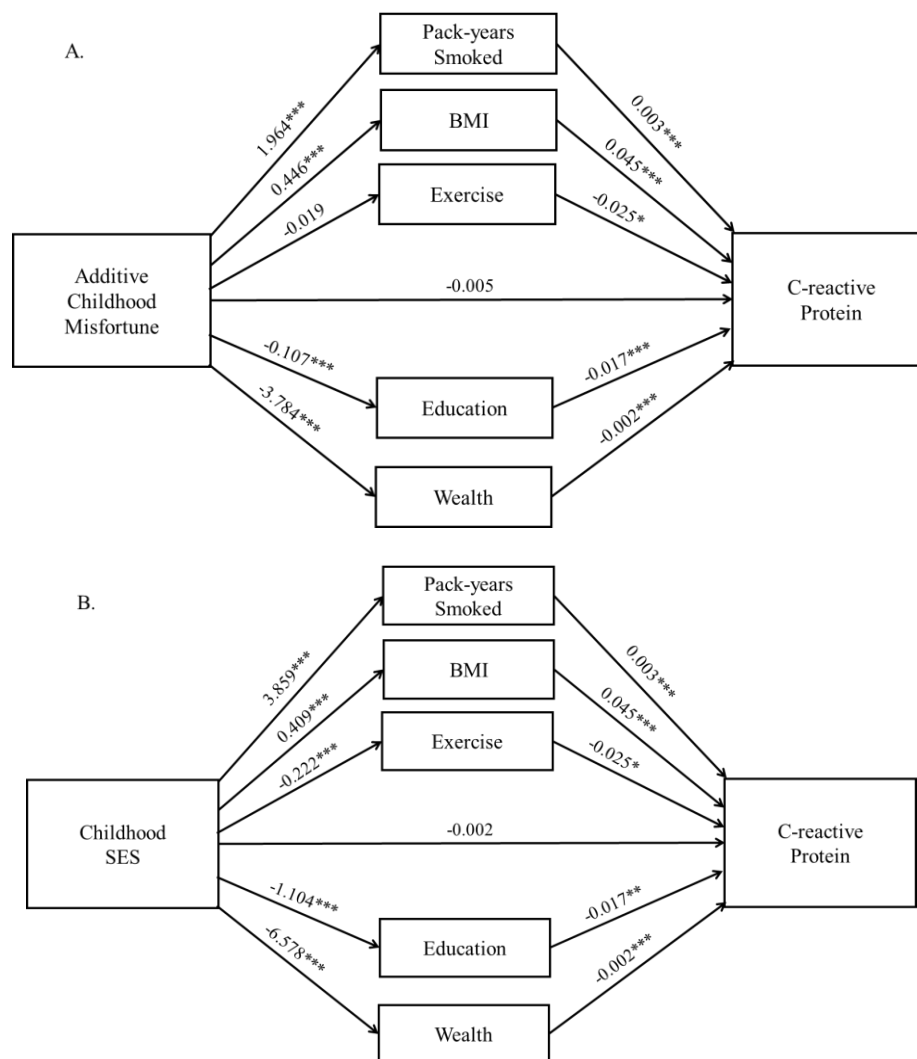


Figure 2.2 Relationships Among Childhood Misfortune, Mediators, and Chronic Inflammation. A. Pathway Model from ACM to CRP. B. Pathway Model from Childhood SES to CRP. Notes: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$. Coefficients are unstandardized.

2.8 Discussion

The purpose of this study was to systematically investigate how childhood misfortune influences immune functioning—a key pathogenic process of health for older adults (Krabbe, Pedersen, and Bruunsgaard 2004). Linking childhood experiences to chronic inflammation, albeit indirectly, in a sample of adults age 51 and older exposed the far-reaching grasp of childhood misfortune. Childhood misfortune influenced immune functioning more than four decades later, attesting to CI theory and biological embedding’s assertion that childhood is a sensitive period for health in later life (Ferraro and Shippee 2009; Hertzman and Boyce 2010). Moreover, this study revealed that childhood misfortune becomes manifest on a physiological level via health lifestyles and SES. I elaborate on three ways in which the findings of the present study contribute to the research on childhood experiences and adult chronic inflammation.

First, this study compared alternative specifications of childhood misfortune to determine which kinds and forms of misfortune were most consequential to the immune system in later-life. Findings indicated that childhood misfortune exerted both additive and unique effects on inflammation in later-life. An additive measure of misfortune revealed that multiple domains of misfortune exerted a combined effect on chronic inflammation. The notion that the quantity of misfortune experienced—regardless of type—can heighten health risks resonates with much of the childhood misfortune literature (e.g., Brown et al. 2010; Dong et al. 2004; Dube et al. 2009; Felitti et al. 1998; Morton et al. 2014). Experiencing multiple kinds of disadvantage can take a toll later in life, and these finding demonstrated that the toll can be physiological.

Disaggregating the additive measure into six different domains revealed that only one domain of misfortune was consequential to inflammation in later-life: childhood SES. Linking childhood SES to adult chronic inflammation supports prior research which has connected socioeconomic disadvantage in childhood to elevated levels of inflammation in adulthood (e.g., Brummett et al. 2013; Danese et al. 2009; Carroll et al. 2011; Stringhini et al. 2013; Taylor et al. 2006). The HRS data provide a detailed history of early-life SES and, therefore, it is not surprising that the HRS data would replicate these earlier findings. The salient nature of childhood SES lends credence to Link and Phelan's (1995) fundamental cause theory which posits that SES is a social determinant of health.

Second, this study elucidated the multidimensional pathways connecting childhood experiences to adult chronic inflammation among Black, White, and Hispanic men and women. Guided by cumulative inequality theory and biological embedding, I expected that childhood misfortune would lead to elevated levels of CRP in later life via health-related and socioeconomic mechanisms. Consistent with prior research, results indicated that children of misfortune are likely to adopt harmful coping mechanisms that lead to poor health (e.g., Brummett et al. 2013; Matthews et al. 2014; Surtees et al. 2003). This study also found that lower adult SES linked early-life experiences to chronic inflammation in adulthood. Although a prior study by Surtees and colleagues (2003) found that SES did not mediate the effect of childhood misfortune, cultural and/or measurement differences may explain these discrepant findings. Surtees et al. (2003) analyzed a sample of British adults and used different indicators of childhood misfortune and adult SES.

Among the mediators examined, exercise was the only one for which there was no consistent statistical evidence of mediation. Exercise mediated the effect of childhood SES only. Prior research suggests that exercise is often unable to explain how early-life experiences impact later-life health (for meta-analyses see Danese and Tan 2014). Some studies investigating the mediating effect of exercise on early-life experiences and health have found no mediating effect (e.g., Chartier, Walker, and Naimark 2009) whereas others found that the effect of exercise is explained by BMI (Danese and Tan 2014). Different domains of misfortune (maltreatment: Chartier et al. 2009), the operationalization of exercise (Danese and Tan 2014), or the health outcome (mental health: Chartier et al. 2009) may account for the null findings in prior research. Unlike maltreatment, childhood SES is related to many health behaviors, including physical activity (Danese and Tan 2014). In addition, there is a well-established relationship between physical activity and chronic inflammation.

Third, this study investigated whether childhood experiences and their inflammatory sequelae vary by race, ethnicity and gender. The present study did not find any evidence of moderated mediation by race or ethnicity. Moreover, several initial racial differences in inflammation were attenuated when the models adjusted for adult health lifestyles and SES. Although inflammation levels were expected to vary by race, some evidence suggests that racial health disparities decrease in older age (Herd et al. 2012), revealing an age-as-a-leveler process (Lynch 2003). Unlike race, gender moderated some of the main effects. Whereas the effects of adult health lifestyle and SES on inflammation did not vary by gender, the effects of childhood SES on smoking did. In this instance, the effect of childhood misfortune was stronger for men than

women. These gender differences reveal that chances of experiencing misfortune and its aftermath are not random. Rather, they are socially patterned. Consistent with prior research, inflammation levels also differed by gender (Herd et al. 2012).

This study is not, however, without limitations. First, the indicators of childhood misfortune are retrospective and, therefore, subject to recall bias. Since the sampling frame of the HRS is age 51 and older, some of these respondents are being asked about their childhood experiences 30, 40, 50, or more years after they occurred. However, I took precautions to increase the validity and reliability of these retrospective measures (e.g., excluding respondents with low cognition scores) and adjusted the model for variables that can bias retrospective recollection (Vuolo et al. 2014).

Second, another data limitation is that the HRS does not assess use of statins or hormonal drug therapy. Whereas medications used to lower cholesterol can reduce CRP levels, hormonal therapies can raise CRP levels. Considering the age of the sample, it is likely that a significant proportion of respondents are taking some type of cholesterol medication and/or hormonal therapy (e.g., women experiencing menopause). Because this study does not account for these medications, chronic inflammation may be masked (due to statins) or heightened (due to hormonal prescription drugs) for some individuals in this study.

Third, although the HRS included oversamples of Black and Hispanic adults, the analytics sample is, nonetheless, predominantly comprised of White adults. In addition, respondents with acute inflammation were removed from the analytic sample, effectively reducing the number of minority respondents. Moreover, social gradients in health and mortality suggest that racial disparities among older adult may be masked due to

selection processes. Therefore, initial multiple-group analyses indicating that there were no observable racial differences require careful interpretation; there may not have been sufficient statistical power to detect such differences.

Fourth, given the age of the sample, results presented herein may be underestimating the true effects of childhood misfortune. Children who experienced the most misfortune are more likely to experience premature mortality or incarceration and, therefore, less likely to be included in these analyses. The age of this sample could also mask important direct effects of misfortune on adult inflammation that have been found in other studies. It is plausible that childhood misfortune has direct effects on the immune system earlier in life. However, these initial effects could be exacerbated by subsequent health behaviors and social structures, leading to the pathway model supported by the findings of the present study. Indeed, future research should investigate this possibility in samples of young and midlife adults. However, assessing CRP in middle-aged and older adults provided a sufficient lag time for immune dysregulation to become manifest.

Fifth, the health-related and socioeconomic pathways likely tell only part of the childhood misfortune-adult inflammation story; these mechanisms are in no way exhaustive and likely paint a limited picture of the underlying mechanisms. Future studies should investigate alternative avenues through which childhood experiences can affect immune functioning. Nonetheless, this study adds to the growing literature illuminating the biological shackles of childhood misfortune.

CHAPTER 3. CHILDHOOD MISFORTUNE AND ADULT ISCHEMIC HEART DISEASE: A MULTIDIMENSIONAL LIFE COURSE PATHWAY MODEL OF HEALTH

3.1 Introduction

The last two decades have witnessed a profound growth in research on the childhood origins of adult health. Disadvantageous childhood events and experiences, broadly referred to as childhood misfortune, have been linked to many later-life ailments, such as cancer (de Kok et al. 2008; Morton et al. 2012), comorbidity (Draper et al. 2008; Non et al. 2014), diabetes (Thomas et al. 2008), myocardial infarction (MI) (Hamil-Luker and O’Rand 2007; Morton et al. 2014), lung disease (Springer 2009), and stroke (Felitti et al. 1998). Whereas the relationship between childhood misfortune and adult health is well-established, less established is an understanding of the many mechanisms connecting early experiences to adult health risks. Thus, a common goal of the many disciplines studying the childhood origins of adult health is to specify the life course processes linking childhood misfortune to adult health.

Compelling evidence suggests that childhood misfortune acts as anchor for adult health because it stratifies individuals on multiple life domains that affect health. For instance, early-life misfortune can influence health behaviors and lifestyles, psychosocial resources, and biological systems, all of which can impact health (Anda et al. 1999; Fagundes and Way 2014; Greenfield and Marks 2009b; Felitti et al. 1998; Morton et al. 2016; Palloni 2006; Taylor et al. 2006). Indeed, the mechanisms of childhood misfortune

are multidimensional; there is no single avenue through which childhood events and experiences impact health. Although many scholars studying the long-term effects of childhood experiences tend to focus on certain mechanisms favored by their respective disciplines, the present study draws from recent strides made in this multidisciplinary area of research, examining health lifestyle, socioeconomic, and biological factors as potential mechanisms of childhood misfortune. To test this multidimensional dimensional model of life course health, I use ischemic heart disease (IHD)—a common disease and leading cause of death in United States—as the health outcome for this study.

The present study weaves together two theoretical frameworks—cumulative inequality theory (Ferraro and Shippee 2009) and biological embedding (Hertzman and Boyce 2010)—to guide this integrative approach to uncovering the mechanisms of early misfortune. Guided by cumulative inequality theory and biological embedding, I raise two research questions. First, I ask whether childhood misfortune raises the risk of IHD onset among midlife and older adults. Second, I ask whether the relationship between childhood misfortune and IHD risk is explained (i.e., mediated) by adult health lifestyles, socioeconomic status (SES), and chronic inflammation. Based on CI theory and biological embedding, I expect that childhood misfortune will lead to a host of individual (health lifestyles) and structural (SES) consequences that biologically embed, resulting in heightened risk of IHD. To test these hypothesized relationships, I utilize a national, panel survey of Black, White, and Hispanic American adults aged 51 and older. I begin by outlining the theoretical background which frames this study, then move to a discussion of the prior empirical literature.

3.2 Theoretical Framework

3.2.1 Cumulative Inequality Theory

Cumulative inequality (CI) theory is the main theoretical framework of this study (Ferraro and Shippee 2009). Derived from elements of the life course perspective (Elder 1998; Ben-Shlomo and Kuh 2002), the Matthew effect (Merton 1968), the stress process (Pearlin 1989; Pearlin et al. 1981), and cumulative advantage/disadvantage (Dannefer 2003; O’Rand 1996), CI theory provides a comprehensive approach for researching multidimensional life course processes of health and aging (Ferraro and Shippee 2009). CI theory is germane to the present study because it privileges childhood as a sensitive period for life course health and expounds upon the multiple mechanisms underlying long-term processes of health inequality. Three elements of CI theory inform the present study.

First, CI theory holds that childhood is a sensitive period for later-life health. Privileging the timing of events—particularly events experienced earlier in the life course—is not a novel concept within sociology (e.g., the life course perspective; the Matthew effect). However, CI theory draws explicit attention to the life course stage of childhood, noting that childhood conditions can serve as a catalyst for cumulative inequality because they privilege some and penalize others early in life. Over time, these initial differences in advantage and disadvantage can compound, leading to observable differences in life course trajectories, including trajectories of health. Thus, CI theory views childhood misfortune as a social antecedent to adult health risks, and there is considerable evidence substantiating this proposition.

An extensive literature on childhood misfortune has documented the salience of early-life experiences for later-life health. This research has revealed that poor health may result, in part, due to events and experiences during childhood, such as socioeconomic disadvantage (Bowen 2010; de Kok et al. 2008; Hamil-Luker and O’Rand 2007; Luo and Waite 2005), maltreatment (Draper et al. 2008; Elo 2009; Fuller-Thomson and Brennenstuhl 2009; Greenfield and Marks 2009a; Springer 2009), and poor health (Blackwell et al. 2001; Bowen 2010; Luo and Waite 2005). When combined, multiple experiences of misfortune can exert an additive effect on adult health risks (e.g., Brown et al. 2010; Dong et al. 2004; Felitti et al. 1998; Morton et al. 2012; Morton et al. 2014; Non et al. 2014; Thomas et al. 2008). Whereas this body of research demonstrates the “long-arm” of childhood misfortune, CI theory provides theoretical plausibility for why childhood misfortune could raise risk of IHD several decades later.

Second, CI theory elucidates the intricate process of how health inequalities are generated over the life course, noting that cumulative processes of health entail multiple levels and domains (Ferraro and Shippee 2009). Many disciplines researching health over the life course agree that poor health often reflects a lifetime of amassed insults (Ferraro and Morton 2016). This health process can be explained by Pearlin’s (2010) stress proliferation and epidemiology’s chain of risks concepts: the occurrence of one adverse event likely leads to a subsequent adverse event (Ben-Shlomo and Kuh 2002). CI theory provides a more contingent model of health by elaborating on the multidimensional mechanisms through which early disadvantage begets subsequent disadvantage as well as instances when early disadvantage does not lead to further disadvantage.

More generally, CI theory highlights the role of both agentic and structural mechanisms of health inequality, contending that both processes should be considered simultaneously. Beyond individual markers of health and health behaviors lies social structures, which can provide unequal (1) exposure to health risks and (2) access to health protection. CI theory encourages scholars to move beyond individual risk factors of disease (e.g., smoking and obesity) and evaluate the socially patterned distributions of life course inequality by analyzing the broader social contexts wherein the individual is situated structurally and historically. Related to the present study, childhood experiences can incur subsequent disadvantage on multiple levels and diffuse across several life domains, eventuating in poor health (e.g., Felitti et al. 1998; Morton et al. 2016; Palloni 2006; Taylor et al. 2006). Because childhood misfortune can inhibit healthy aging via agentic and structural factors, the present study incorporates multiple mediators of health lifestyles, SES, and inflammation to elucidate the path from childhood misfortune to adult IHD onset.

Third, CI theory maintains that cumulative inequality can lead to premature mortality. This fatal process of cumulative inequality can be initiated in childhood. Experiences of childhood misfortune can place an individual on a life course trajectory that culminates in premature mortality (Beebe-Dimmer et al. 2004; Brown et al. 2009; Galobardes, Lynch, and Smith 2004; Hayward and Gorman 2004; Kelly-Irving et al. 2013; Lee and White 2012). Because children of misfortune may experience shorter life expectancies, it is important to study fatalities related to disease onset, not only the onset of a disease itself. This issue is of notable concern when analyzing longitudinal studies of older adults; investigating disease onset among survivors only may lead to

underestimating incidence rates as well as the true effects of childhood misfortune. Since early mortality is a risk of accumulated disadvantage, the present study investigates incident IHD using physician diagnosis and mortality data.

3.2.2 Biological Embedding

Despite the breadth of CI theory's interdisciplinary insight into health and aging over the life course, absent from CI theory is how early-life events and experiences can impinge physiologically. Therefore, this study supplements CI theory with biological embedding (Hertzman and Boyce 2010). Rather than a competing model of life course health, biological embedding reinforces CI theory by explicating how biological development during childhood can facilitate the process of childhood misfortune “getting under the skin” and altering later-life health.

Parallel to CI theory, biological embedding also contends that childhood is a sensitive period for later-life health. Unique to biological embedding, however, is the assertion that childhood is a *biologically* sensitive period for later-life health. Hertzman and Boyce (2010) note that biological systems, including the stress response system, are highly susceptible to external influences during childhood because they are developing and calibrating during this time. Disadvantageous childhood events and experiences can disrupt biological development, leading to physiological dysregulation that endures throughout life. Indeed, there is evidence to suggest that conditioning of the hypothalamic-pituitary-adrenal (HPA) axis—a fundamental part of the stress response—occurs early in life (Hertzman and Boyce 2010; Meaney, Szyf, and Seckl 2007). In addition, the HPA axis (via cortisol secretion) has system wide effects on numerous

organ systems including the immune system (Hertzman and Boyce 2010; Miller, Chen, and Zhou 2007). Therefore, childhood misfortune could negatively impact immune functioning via a strained HPA axis. The emerging research linking childhood experiences to chronic inflammation in adulthood demonstrates this lifelong biological imprint of childhood misfortune (Baumeister et al. 2015; Carpenter et al. 2010; Carroll et al. 2011; Danese et al. 2007; Miller et al. 2009; Slopen et al. 2010; Stringhini et al. 2013; Surtees et al. 2003; Taylor et al. 2006). Although childhood experiences can exert direct effects on biomarkers of health, biological embedding recognizes that intermediary pathways can accentuate these early-life tendencies of physiological dysregulation.

3.3 Background

3.3.1 Ischemic Heart Disease

As state above, there is a well-documented relationship between childhood misfortune and a variety of health outcomes, from physical functioning (Greenfield and Marks 2009a; Singh-Manoux et al. 2004) to the leading causes of death (Felitti et al. 1998). Among the many diseases studied, early-life experiences appear to have particularly salient repercussions for cardiovascular diseases (Blackwell et al. 2001; Bowen 2010; Dong et al. 2004; Felitti et al. 1998; Hallqvist et al. 2004; Hamil-Luker and O’Rand 2007; Kaplan and Salonen 1990; Korkeila et al. 2010; Morton et al. 2014; O’Rand and Hamil-Luker 2005; Singer and Ryff 1999; Singh-Manoux et al. 2004; Wannamethee et al. 1996) and mortality (Beebe-Dimmer et al. 2004; Elo et al. 2010; Hayward and Gorman 2004; Kamphuis et al. 2013; Naess et al. 2004; Power, Hyppönen, and Smith 2005). Childhood misfortune has also been linked to several risk factors of

cardiovascular disease (CVD) (Melchior et al. 2007), such as diabetes (Felitti et al. 1998; Thomas et al. 2008), hypertension (Riley et al. 2010; Stein et al. 2010), and obesity (Thomas et al. 2008; Williamson et al. 2002). Furthermore, understanding the life course process of CVD risk is of high importance for health policy in the United States where CVD remains the leading cause of death and major cause of disability (Gillespie et al. 2013). Because the majority of deaths from CVD in the United States are caused by ischemic or coronary artery diseases, which are also the most common types of heart disease, the present study focuses on IHD (CDC 2015; Gillespie et al. 2013; Mensah and Brown 2007). Prior childhood misfortune research has utilized adult IHD, including specific diseases of MI and angina, as an outcome (Bowen 2010; Dong et al. 2004; Felitti et al. 1998; Kaplan and Salonen 1990; Hallqvist et al. 2004; Hamil-Luker and O’Rand 2007; Morton et al. 2014; Morton et al. 2016; O’Rand and Hamil-Luker 2005; Wannamethee et al. 1996). Building upon this research, I elaborate on the ways in which early-life experiences can influence IHD risk.

3.3.2 Mechanisms of Childhood Misfortune

Childhood misfortune has been linked to many risk factors of and antecedents to CVD. Drawing from fields of epidemiology, gerontology, sociology, and psychobiology, the present study focuses on three key mechanisms to explain how early-life experiences raise the risk of IHD in later life: health lifestyles, SES, and inflammation.

3.3.2.1 Health Lifestyles

Not surprisingly, the majority of research linking early-life experiences to adult health, including IHD, has focused on health-related factors of smoking (Brown et al.

2010; Dong et al. 2004; Ferraro, Schafer, and Wilkinson 2016; Kaplan and Salonen 1990; Morton et al. 2014; Springer 2009; Sumanen et al. 2005; van de Mheen et al. 1998), substance abuse (drugs and alcohol) (Dong et al. 2003; Kaplan and Salonen 1990; van de Mheen et al. 1998), obesity/BMI (Dong et al. 2004; Springer 2009; Sumanen et al. 2005; Thomas et al. 2008; van de Mheen et al. 1998), and physical inactivity (Dong et al. 2004; van de Mheen et al. 1998) as explanatory mechanisms. These risk factors, mostly modifiable, are stressed in the health literature and studied by scholars from all fields because they are well-known risk factors for many diseases, including IHD, and involve agency on the part of the actor. In addition, health-related behaviors and lifestyles often become ingrained early in life and can persist into adulthood. Thus, childhood experiences have the ability to shape health lifestyles. Childhood misfortune, particularly childhood SES, has been linked to smoking (Anda et al. 1999; Ford et al. 2011; Jefferis et al. 2004; Lawlor et al. 2005), obesity/BMI (Lawlor et al. 2005; Thomas et al. 2008; Williamson et al. 2002), substance abuse (Dube et al. 2002; Dube et al. 2003; Lawlor et al. 2005), and physical inactivity (Felitti et al. 1998), all of which can accelerate the health consequences of early-life experiences.

Whereas these health-related risk factors have mediated the effects of childhood misfortune on adult health outcomes, risk factors beyond individual health lifestyles, such as psychosocial insults and resources, may have a stronger mediational effect on IHD (Dong et al. 2004). Therefore, the present study also incorporates research delineating the health consequences of exposure to social structures and resources over the life course as additional pathways through which childhood misfortune becomes manifest as adult IHD.

3.3.2.2 Socioeconomic Status

Sociology has a history of identifying the underlying social-structural determinants of health, with a special emphasis on socioeconomic disparities (e.g., Marx 1867; Preston and Taubman 1994). Thus, understanding how structural inequalities produce health disparities is central to medical sociology. Influenced heavily by the work of medical sociologists, life course research on health and aging often stresses the role of social structures conceptually and empirically (e.g., conceptual work: Dannefer 2003; Elder 1998; Ferraro and Morton 2016; Link and Phelan 1995; e.g., empirical work: O'Brien 2012; Goldman and Smith 2011; Montez et al. 2011; Sewell, Haller, and Portes 1969). Of the many social structures, SES is a robust predictor of health (Braveman, Egerter, and Williams 2011; Robert 1999). Considered a fundamental cause of disease (Link and Phelan 1995)¹³, adult SES has been linked consistently to health (Alder et al. 1994; Robert 1999) and mortality (Pappas et al. 1993). Among the many health and mortality outcomes studied, SES appears to play a particularly salient role for CVD outcomes in developed countries like the United States (Albert et al. 2006; Clark et al. 2009; Kaplan and Keil 1993; Steptoe and Marmot 2002).¹⁴

¹³SES is considered a fundamental cause of disease because it has systemic effects on many spheres of health. One way in which SES produces health disparities is by assisting or restricting access to healthcare. Other ways that SES shapes health gradients are through unequal distributions of economic resources like income, working conditions, and education as well as affecting psycho-social resources and health behaviors (O'Brien 2012; Ross and Wu 1995; Williams and Sternthal 2010). The power of SES to transform health outcomes is evident by how it pervades other social-structural disparities in health, such as racial health disparities (Hayward et al. 2000).

¹⁴One reason for why socioeconomic disparities are particularly salient for CVD outcomes is because socioeconomic gradients in some of the most common risk factors for CVD have widened over time in the United States (Kanjilal et al. 2006).

To understand how adult SES influences CVD risk, it is helpful to consider more distal factors that antecede adult SES. Research suggests that adult SES is often a function of early-life events and experiences. Childhood conditions, notably SES, can impact adult socioeconomic attainment (Sewell et al. 1969) including educational attainment (Case et al. 2005; Haas 2006; Palloni 2006), occupational status (Case et al. 2005; Palloni 2006), and wealth (Haas 2006). Moreover, adult SES has also been shown to account for part of the relationship between childhood SES and adult health (O’Rand, Hamil-Luker, and Elman 2009), including IHD (Kaplan and Salonen 1990). As such, SES is another plausible mediating factor between childhood misfortune and adult IHD.

3.3.2.3 Chronic Inflammation

Understanding how the social environment interacts with biological systems is fundamental to the field of psychobiology, which has produced much of the literature on the biological consequences of early misfortune. This burgeoning area of research has revealed that childhood experiences can embed on a physiological level, interfering with immune functioning and producing pro-inflammatory tendencies. Childhood socioeconomic disadvantage, for instance, has been linked to inflammatory signaling, indicating biological programming for proinflammatory phenotypes (Miller and Chen 2007; Miller et al. 2009). Although scholars rarely investigate the biological consequences of childhood misfortune on an epigenetic level, many studies linking childhood misfortune to chronic inflammation in adulthood corroborate the notion that childhood experiences can biologically embed into the immune system (Baumeister et al. 2015; Brummett et al. 2013; Carpenter et al. 2010; Carroll et al. 2011; Danese et al. 2007;

Danese et al. 2009; Rooks et al. 2012; Slopen et al. 2010; Stringhini et al. 2013; Surtees et al. 2003; Taylor et al. 2006). Thus, chronic inflammation may help explain how childhood misfortune disrupts health on a biological level.

Chronic inflammation is a logical next step to elucidate the relationship from childhood misfortune to adult health and mortality. Markers of inflammation have gained prominence in health research, in part, due to their remarkable predictive ability in assessing disease risk. Chronic inflammation can predict incident CVD as well as cardiac mortality risk (Cesari et al. 2003; Pearson et al. 2003; Ridker et al. 2000a; Ridker et al. 2000b; Volpato et al. 2001). Crimmins and associates (2010) put forth a conceptual model of the morbidity process in which biological risk factors, including chronic inflammation, precede disease and frailty. Moreover, chronic inflammation can arise from the aforementioned adult health lifestyles and SES mechanisms (Alley et al. 2006; Friedman and Herd 2010; Herd et al. 2012; Koster et al. 2006; Lubbock et al. 2005). Therefore, chronic inflammation may tie together the multidimensional pathways to elucidate how childhood misfortune becomes manifest as poor adult health. Drawing from these three bodies of literature, I hypothesize that childhood misfortune will lead to unhealthy lifestyles and socioeconomic disadvantage, which, in turn, lead to chronic inflammation, culminating in onset of IHD (see Figure 3.1).

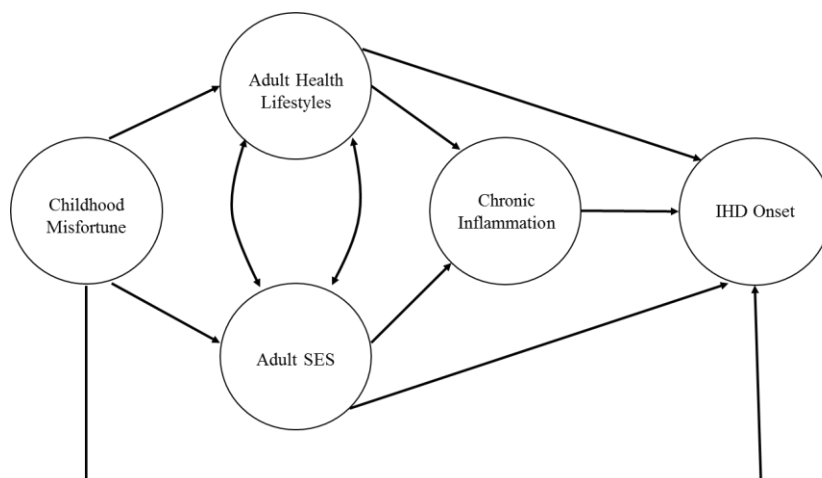


Figure 3.1 Hypothesized Relationships Among Childhood Misfortune, Adult Health Lifestyles, Adult SES, Chronic Inflammation, and IHD onset.

3.4 Sample Description

This study used 6 waves of the Health and Retirement Study (HRS) from 2004 to 2014. Initiated in 1992, the HRS is a longitudinal, biennial survey of American adults 51 years and older (Health and Retirement Study 2015). The HRS utilized a multistage, stratified area probability sample of American households, including oversamples of Black and Hispanic Americans and Floridians, and replenishes the sample with new cohorts every six years.¹⁵ Response rates for initial and each follow-up survey have been good—all above 70% (Sonnegga et al. 2014). The present study analyzes a sample of 11,697 adults (see Appendix B for inclusion criteria of analytic sample) who participated in the core surveys as well as the Enhanced Face-to-Face Interview (EFTF) during which

¹⁵The HRS currently includes 6 birth cohorts: Asset and Health Dynamics Among the Oldest Old; Children of the Depression; Original HRS; War Babies; Early Baby Boomers; Mid Baby Boomers. Because the analytic sample utilizes survey data from 2004-2014, the Mid Baby Boomers who joined the HRS in 2010 are excluded from all analyses.

the biomarker data were collected.¹⁶ Mortality data from the restricted data files was also analyzed. Unless stated otherwise, all variables were drawn from the raw HRS data files.

3.5 Measurement

3.5.1 Incident Ischemic Heart Disease

The outcome variable for this study is incident ischemic heart disease from 2008-2014. Incident IHD was observed over a six-year period from 2008 to 2014 in order to maintain temporal ordering among IHD, childhood misfortune, and all mediating variables (i.e., the observation period began after all mediating variables were assessed; final assessment of inflammation was in 2008). Following prior research, IHD was measured using MI and angina (Dong et al. 2004).¹⁷ For both diseases, respondents were asked separately if a doctor ever told them that they had a heart attack or angina. If they responded yes to having either disease, they were then asked for the year and month of their heart attack or year and month angina was first diagnosed. Using these questions, two variables for incident IHD were created for the event history analyses.

The censoring variable was created by coding respondents who reported having a heart attack or angina by 2014 as “1”, “0” otherwise. The duration variable was

¹⁶The HRS Biomarker data collected during the EFTF was sponsored by the NIH (NIA U01AG009740) and was conducted by the University of Michigan (Health and Retirement Study 2006).

¹⁷In addition to following prior research using MI and angina as indicators of IHD (Dong et al. 2004), I focused on MI and angina for substantive reasons as well. First, MI and angina differ from other types of CVD in risks and diagnoses; an individual is often aware of experiencing an MI or angina whereas certain types of CVD, such as hypertension, may go undetected, particularly for those without access to healthcare. Second, MI and angina are two of three cardiovascular diseases that include timing data which enable event history analysis.

measured in months and coded by calculating number of months until first IHD during the six-year observation period. If month was not reported, those responses were set to midway (i.e., June) of the respective year. For respondents who had not experienced an MI or angina (i.e., censored observations), year and month of latest survey or death were used for the duration variable (i.e., duration coded as months since 2008 interview).

Given the age of the sample, it is reasonable to expect that some respondents may experience IHD and die between observation periods. Therefore, I also included data from the National Death Index (NDI) which includes cause and date of death.¹⁸

Respondents who died from IHD, but had not experienced IHD prior to death were coded as “1” for the censoring variable. Information on month and year of death were also included in the duration variable, which ranges from 1-84 months for respondents who experienced IHD during the observation period. Descriptive statistics are presented in Table 3.1.

¹⁸NDI data on cause of death has only been released through 2012 by the HRS.

Table 3.1 Descriptive Statistics of Variables, Health and Retirement Study (N=11,697)

Variable	Range	Mean	SD ^a
2008-2014 Incident IHD	0,1	0.056	
Months until IHD onset ^b	1-84	19.603	14.820
<i>Childhood Misfortune</i>			
SES	0-2	1.274	0.840
Parent Behaviors	0-2	0.847	0.645
Infectious Disease	0-2	1.712	0.597
Chronic Disease	0-2	0.440	0.689
Impairments	0-2	0.211	0.469
Adolescent Behaviors	0-2	0.090	0.318
<i>Adult Health Lifestyle</i>			
Pack-years Smoked	0-287	14.586	0.311
BMI	11.7-66.1	27.720	5.559
Exercise	1-5	2.151	1.355
<i>Adult SES</i>			
Education	0-17	13.143	2.903
Wealth, cubic root	-130.950-333.672	61.000	36.479
<i>Adult Chronic Inflammation</i>			
CRP ^c	0.02-8.58	2.383	2.020
<i>Demographics</i>			
Age	50-97	62.569	9.498
Female	0,1	0.585	
White ^d	0,1	0.818	
Black	0,1	0.087	
Hispanic	0,1	0.068	
Other Race	0,1	0.027	
Married	0,1	0.684	
<i>Covariates</i>			
Prescription Medications	0,1	0.506	
Chronic Conditions	0-3	1.091	0.888
Depressive Symptoms	0-8	1.256	0.030
EFTF Measurement Year	0,1	0.534	

Notes: Descriptive statistics are weighted.

^aThe SD of dichotomous variables is omitted because it is a function of the mean.

^bAmong respondents who experienced IHD between 2008 and 2014.

^cAssessed during EFTF measurement year.

^dReference group.

3.5.2 Childhood Misfortune

Childhood misfortune was measured using 31 indicators of retrospective self-reports of childhood events and experiences. These 31 indicators comprise six domains of childhood misfortune based on prior literature, correlation matrices, and polychoric factor analysis (Felitti et al. 1998; Kemp et al. 2015; Morton et al. 2012; Turner et al. 1995). The first domain is childhood SES and included five measures: mother's education, father's education, father's occupation, perceived financial situation, and relocation due to financial strain. The second domain is risky parental behaviors and included three measures: physical abuse by parent, parental alcohol or substance abuse, and parent/guardian smoked. The third domain is childhood infectious disease and included three measures: measles, mumps, and chicken pox. The fourth domain is childhood chronic disease and included eleven measures: asthma, diabetes, respiratory disorder, allergies, heart disease, ear problems, seizures, migraines, stomach problems, high blood pressure, and poor self-rated health. The fifth domain is childhood impairment and included five measures: learning problems, speech impairment, vision impairment, disability (≥ 6 months), and head injury or trauma requiring medical attention. The sixth and final domain of risky adolescent behaviors and mental health (hereafter, adolescent behaviors) included four measures: substance abuse, police troubles, and psychological problem (depression or otherwise). All measures of childhood misfortune assessed experiences before age 16 except for items of parental behaviors, childhood learning problems, and police troubles which could have occurred any time prior to age 18.

Since the majority of indicators (26/31) have binary response categories, all 31 measures were initially dichotomized with “1” indicating experiencing the misfortune and “0” as otherwise.¹⁹ To assess the unique effects of different domains of misfortune, each of the six domains were used as a separate predictor of IHD risk. After dichotomizing each item of misfortune, the items were summed within each domain. Each domain ranges from 0-2, with 2 indicating experiencing 2 or more items within that domain. The domains were top-coded at 2 because few respondents experienced more than 2 indicators. In addition, I wanted to maintain consistency across the domains since each domain varied in number of indicators.

To assess the additive effect of childhood misfortune, a single count variable of childhood misfortune was created. First, I created a dummy variable for each of the domains, with “1” indicating that the respondent reported experiencing at least one of the indicators in that domain. Next, I summed across the domains to create the additive childhood misfortune (ACM) variable. ACM ranges from 0 (did not experience any indicator in any domain of misfortune) to 6 (experienced at least one indicator in each domain of misfortune).

¹⁹Poor self-rated health was coded as “1” for respondents who rated their childhood health as either poor or fair (Haas 2007). Mother’s and father’s education were dichotomized so that “1” = <12 years of education (i.e., less than a high school education). Father’s occupation was coded “1” for non-skilled manual occupations, following U.S. Labor Statistics occupational categories (U.S. Bureau of Labor Statistics 2010). Perceived financial situation was coded “1” for respondents who rated their family financial situation as poor.

3.5.3 Health Lifestyles

Three variables were used to assess health lifestyles in 2004: smoking, BMI, and exercise. Smoking is measured as pack-years, which combines information on years smoked and average amount smoked daily to capture cumulative exposure to smoking tobacco. Among respondents who were ever smokers (i.e., respondents who smoked >100 cigarettes in their lifetime), respondents reported when they began smoking and when they quit (unless they were current smokers in 2004) as well as how much they smoked daily, on average. From this information, I calculated the total years each respondent smoked (year stopped minus year started for former smokers; 2004 minus year started for current smokers). Next, I multiplied the total years smoked by the average number of cigarettes smoked daily and divided by 20 (amount of cigarettes in a pack) to calculate pack-years smoked. Pack-years is top-coded at 287. Respondents who never smoked were coded as “0”.

BMI was drawn from the RAND dataset, which was calculated from respondents’ self-reports of weight and height. BMI is measured in kg/m^2 and top coded at $66.1 \text{ kg}/\text{m}^2$. The exercise variable was also drawn from the RAND dataset and assesses respondent’s frequency of vigorous exercise. Respondents were asked how often they took part in vigorous exercise or activities such as running/jogging, swimming, cycling, aerobics/gym workouts, tennis, or digging with spade/shovel. Response categories were daily, >1 time per week, 1 time per week, 1-3 times per month, and never. The variable was reverse coded so that higher values indicate more frequent exercise.

3.5.4 Socioeconomic Status

Two variables from the RAND dataset were used to assess SES in 2004: education and wealth. Education was measured in years of education, indicating respondent's highest level of education completed. Wealth was measured in dollars at the household level and calculated as total wealth (all assets including secondary residence) minus all debt.²⁰ To adjust for skewness, a cubic root transformation of wealth was used. Wealth is a more reliable indicator of financial stability (or strain) than income for older adults as many are retired and, therefore, do not receive an annual income. Wealth taps into the accumulated assets and debts incurred over the life course by these older adults. In addition, wealth is an important component of SES when assessing the relationship between SES and health (Pollack et al. 2007).

3.5.5 Chronic Inflammation

Chronic inflammation was assessed using dried blood spots of C-reactive protein (CRP), a reliable and valid marker of inflammation (McDade et al. 2004) and a stronger predictor of CVD onset than other measures of inflammation (Ridker et al. 2000a). Collection of CRP occurred during the EFTF. Since the EFTF was administered to a random half of the sample in 2006 and the remaining half in 2008, this study combines the EFTF CRP data from 2006 and 2008 to include information for the entire analytic sample. CRP is a continuous variable measured in ug/mL and capped at 8.6 ug/mL.²¹

²⁰RAND used 15 indicators of wealth from the HRS dataset. If an indicator was missing, RAND imputed the values utilizing several methods of imputations. Hurd et al. (2014) provide a detailed description.

²¹Since the aim of this study is to assess the role of chronic inflammation, respondents with extremely high CRP levels (>8.6 ug/mL) were removed from analyses as these cases likely

3.5.6 Demographics

Models included several demographic variables and covariates to adjust for variables related to childhood misfortune, IHD, or any of the explanatory mechanisms. Demographic variables assessed at baseline included age, gender, race/ethnicity, and marital status. Age was a continuous variable measured in years. Gender was coded as a binary variable of female, with “1” indicating female and “0”, male. For race/ethnicity, four dummy variables of Non-Hispanic White (referred to as White), Non-Hispanic Black (referred to as Black), Hispanic, and Non-Hispanic Other (referred to as Other Race) were created following prior research investigating the HRS (Heisler et al. 2007; Kemp et al. 2015). For Other Race, the HRS combined responses of American Indian, Alaskan Native, Asian, Pacific Islander, and other due to the low number of responses for each of these categories. Marital status was represented using a dummy variable for married (1=married, 0=otherwise).

3.5.7 Covariates

Covariates included prescription medications, chronic conditions, depressive symptoms, and EFTF measurement year. Prescription medications was represented by a binary variable, coded “1” for respondents who indicated taking hypertensive and/or psychiatric medications and “0”, otherwise. I focused on hypertensive and psychiatric medications because these medications relate to childhood misfortune and key mediators (e.g., CRP), and are used to treat diseases that are well-known risk factors for CVD.

indicate acute inflammatory disease or injurious stimuli (Herd et al. 2012). In addition, the NHANES equivalent CRP values were used based on the recommendation of the 2006 and 2008 HRS biomarker documentation report (Crimmins et al. 2013).

Chronic conditions was measured as a count of conditions that are risk factors for CVD (Gabriel and Crowson 2012). Respondents were asked whether they had a physician diagnosis of hypertension, diabetes, or arthritis/rheumatism. Using this information, I created a count variable ranging from 0 to 3 based on the number of CVD risk factor conditions each respondent had. Chronic conditions was measured as a single variable to reduce multicollinearity issues related to covariates of prescription medication and depressive symptoms. Depressive symptoms were measured as a composite score of eight separate items, following the Center for Epidemiologic Studies Depression (CES-D) scale. Per the CES-D index, respondents were asked how often they felt depressed, like everything was an effort, sleep was restless, alone, sad, couldn't get going, happy (reverse-coded) or enjoyed life (reverse-coded) in the past week. Response categories for each indicator ranged from none or almost none to all or most of the time. The depressive symptoms variable was coded so that higher values indicate more depressive symptoms.

A dummy variable indicating which year respondents participated in the EFTF survey was included to adjust for possible selection bias. The variable for EFTF measurement year was coded "1" for respondents who participated in 2006 and "0" for those who participated in 2008. All covariates were assessed at baseline with the exception of the EFTF measurement year and prescription medications. Since hypertensive and psychiatric medications can directly affect inflammation levels—a key variable of the present study—this variable was assessed when CRP was collected.

3.6 Analysis

Preliminary analyses and descriptive statistics were estimated in Stata version 14. Cox proportional hazards regression models and tests of mediation were conducted in Mplus version 7 (Muthén and Muthén 2007). Cox proportional hazards models are a useful method for estimating event histories. Unlike logistic regression, Cox models utilize information on timing of IHD onset and incorporate data from both censored and uncensored cases to estimate parameters. Thus, Cox models with robust variance provide more reliable estimates than logistic regression (Barros and Hirakata 2003). As a semiparametric model, Cox models also impose fewer assumptions than standard parametric models (e.g., no assumption about baseline hazard shape). Although Cox models entail some assumptions, there was no evidence that any of them were violated in the present study.²²

My analytic strategy consisted of two stages. The first stage of analyses consisted of a series of preliminary analyses to examine who was removed from the analytic sample due experiencing IHD prior to 2008 and investigate alternative measures of key variables. Respondents who experienced an IHD before 2008 were more likely to be older, male, heavy smokers (i.e., more pack-years smoked), and taking a prescription medication, and less likely to be Hispanic or Black. In addition, respondents who had more chronic conditions, higher BMI, and less wealth; engaged in more risky adolescent behaviors; and experienced childhood socioeconomic disadvantage, infectious or chronic disease and impairment were also more likely to have experienced IHD before 2008.

²²The proportional hazards assumption was tested using Schoenfeld residuals; the assumption was not violated ($\chi^2=25.04$, $df=21$, $p=0.2455$).

Because these analyses are not a focus of the present study, tabular results are not presented nor discussed in the results section. Second, I conducted additional sensitivity analyses using alternative coding schemas for BMI and chronic conditions as well as health insurance as a possible covariate. Because this sample is comprised of older adults, preliminary analyses included a squared term of BMI. However, BMI-squared was non-significant, and main conclusions were not altered. Therefore, those results are not presented, and a single measure of BMI was used in the present study. I also investigated alternative forms of chronic conditions, which included using each condition as a separate predictor and a dummy variable of chronic conditions. Because using separate variables for each condition increased multicollinearity issues (assessed using Variance Inflation Factors) and a dummy variable of chronic conditions worsened overall model fit (assessed using Log likelihoods and AIC), final models used a single count variable for chronic conditions. Finally, I investigated the impact of health insurance because having insurance could affect diagnosis of IHD as well as influence other variables such as race. Having health insurance was not a significant predictor of IHD nor did it alter the conclusions presented below. Thus, health insurance was not included in the final models.

The second stage of analyses tested the proposed casual pathways from childhood misfortune to IHD. This second stage consisted of two steps. First, I estimated a series of Cox models to study IHD onset using childhood misfortune, demographics, and covariates as the independent variables. In order to establish a baseline relationship between childhood misfortune and IHD risk, I did not include any of the mediators in the first sets of models. During this first step, I also examined the alternative specifications

of childhood misfortune discussed above because recent research has revealed that the effects of childhood misfortune can vary based on the operationalization of misfortune (Morton et al. 2012). Testing the additive and unique effects of childhood misfortune in this first step revealed that ACM was not a significant predictor of incident IHD.

Therefore, the results using ACM as a main predictor are not presented. Second, I simultaneously introduced mediators of adult health lifestyles, SES, and CRP into the Cox models, conducting direct and indirect tests of mediation using a maximum likelihood robust estimator (MLR) and Monte Carlo integration (Muthén 2011). For these statistical tests of mediation, paths were created to establish direct relationships between (a) childhood misfortune and adult health lifestyles; (b) childhood misfortune and adult SES; (c) adult health lifestyles and CRP; (d) adult SES and CRP; and (e) CRP and IHD onset. Indirect effects were calculated as the product of the three paths from childhood misfortune to IHD onset (health behavior pathway: $a*c*e$; SES pathway: $b*d*e$). The delta method was used to calculate standard errors for the direct and indirect effects.

Because childhood experiences, adult health, and their mechanisms can vary by gender (see chapter 2 findings), a series of interactions between the key paths and gender were examined. Significant interactions are presented below. Item-missing data for the independent variables were replaced using full information maximum likelihood (FIML), and ties were adjusted using the Breslow method (Muthén and Muthén 2007). All models and univariate descriptive statistics were weighted and adjusted for the complex survey design of the HRS (i.e., stratification and clustering).

3.7 Results

As displayed in Table 3.1, approximately 6% of the sample experienced their first IHD between 2008 and 2014. Among those who experienced IHD, the average number of months until IHD onset was 19.6. The most commonly experienced domain of misfortune was infectious disease ($\bar{x}=1.712$) whereas adolescent behaviors was the least ($\bar{x}=0.090$). The mean age of the sample is 62.6 years. The majority of the sample is female, white, married, and has one chronic condition. On average, the sample is overweight, somewhat wealthy, has obtained some post-high school education, and exercises 1-3 times per month. The average number of pack-years smoked is 14.6 and mean CRP indicates that this sample has a moderate level of inflammation.

Table 3.2 displays the results of the Cox proportional hazards regression models predicting incident IHD risk from 2008-2014. Model 1 examined the relationship between domains of misfortune and IHD risk without including any of the mediating pathways to establish a baseline relationship between domains of misfortune and IHD risk. Among the six domains of misfortune, only childhood SES predicted IHD risk. Each additional experience of socioeconomic disadvantage during childhood raised the hazard of IHD by 16.5%, net of all covariates and additional domains (HR=1.165, $p<0.05$). Demographic variables of age and gender were also significant. For each additional year of age, IHD risk increased by 2.9% (HR=1.029, $p<0.001$). Women had a 38.1% lower risk of IHD than men (HR=0.619, $p<0.001$). Additional variables that were significant included chronic conditions, depressive symptoms, and EFTF measurement year. Chronic conditions (HR=1.082, $p<0.001$), depressive symptoms (HR=1.533,

$p < 0.001$), and EFTF measurement year ($HR = 1.342$, $p < 0.01$) were all associated with increased risk of IHD.

Model 2 introduced the mediating variables of adult health lifestyles, SES, and CRP. When these mediating variables were introduced, the effect of childhood SES on IHD was attenuated and became non-significant. The only mediating variables to have a direct effect on IHD risk were exercise, wealth, and CRP. Increases in exercise frequency and wealth had a protective effect, decreasing risk of IHD (respectively, $HR = 0.908$, $p < 0.05$; $HR = 0.994$, $p < 0.001$) whereas higher levels of CRP raised IHD risk ($HR = 1.079$, $p < 0.01$). As observed in Model 1, age, gender, chronic conditions, depressive symptoms, and EFTE measurement year predicted IHD risk. Whereas women had a lower risk of IHD than men ($HR = 0.609$, $p < 0.001$), age ($HR = 1.034$, $p < 0.001$), chronic conditions ($HR = 1.458$, $p < 0.001$), depressive symptoms ($HR = 1.045$, $p < 0.05$), and EFTF measurement year ($HR = 1.353$, $p < 0.001$) were all associated with increased risk of IHD. Unlike Model 1, there were significant racial differences in IHD risk. Black respondents had a decreased risk of IHD compared to White respondents ($HR = 0.629$, $p < 0.01$).

Table 3.2 Cox Proportional Hazards Regression Models for
2008-2014 Incident Ischemic Heart Disease (HRS: 2004-2014)

Independent Variables	Model 1	Model 2
	HR (CI) ^a	HR (CI)
<i>Childhood Misfortune</i>		
SES	1.165 (1.030, 1.318)*	1.084 (0.960, 1.225)
Parent Behaviors	1.005 (0.875, 1.154)	0.993 (0.864, 1.141)
Infectious Disease	0.987 (0.844, 1.156)	1.012 (0.865, 1.184)
Chronic Disease	1.091 (0.959, 1.241)	1.096 (0.965, 1.246)
Impairment	0.913 (0.746, 1.117)	0.898 (0.740, 1.090)
Adolescent Behaviors	1.151 (0.882, 1.504)	1.129 (0.858, 1.484)
<i>Demographics</i>		
Age	1.029 (1.018, 1.042)***	1.034 (1.021, 1.047)***
Female	0.619 (0.515, 0.745)***	0.609 (0.498, 0.744)***
Black ^c	0.742 (0.527, 1.042)	0.629 (0.453, 0.875)**
Hispanic ^c	0.951 (0.700, 1.292)	0.776 (0.540, 1.114)
Other Race ^c	0.962 (0.542, 1.706)	0.929 (0.520, 1.660)
Married	0.864 (0.700, 1.068)	0.996 (0.799, 1.241)
<i>Covariates</i>		
Prescription Medications	0.965 (0.776, 1.200)	0.943 (0.760, 1.169)
Chronic Conditions	1.082 (1.037, 1.129)***	1.458 (1.267, 1.677)***
Depressive Symptoms	1.533 (1.361, 1.726)***	1.045 (1.000, 1.092)*
EFTF Measurement Year	1.342 (1.133, 1.589)**	1.353 (1.149, 1.606)***
<i>Adult Health Lifestyles</i>		
Pack-years Smoked		1.002 (1.000, 1.005)
BMI		1.000 (0.975, 1.024)
Exercise		0.908 (0.832, 0.991)*
<i>Adult SES</i>		
Education		0.978 (0.951, 1.006)
Wealth, cubic root		0.994 (0.991, 0.996)***
<i>Adult Chronic Inflammation</i>		
CRP		1.079 (1.021, 1.140)**
Log Likelihood	-170483.182	-380951.623
AIC	341394.365	762391.247
N	11,697	11,697

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed tests).

^aHazard Ratio (95% confidence interval).

^cReference group: White respondents.

As described above in the Analysis section, I conducted statistical tests of mediation when the mediators were introduced into the Cox model in Model 2, Table 3.2. Table 3.3 displays the parameters for the direct and indirect effects produced by these statistical tests of mediation for the paths from childhood SES to adult IHD (Table 3.2, Model 2). As shown, there is statistical evidence that the effect of childhood SES on IHD risk was transmitted via health lifestyles and CRP as well as via SES and CRP. All three adult health lifestyles in tandem with CRP mediated the effect of childhood SES on IHD risk. For smoking, low childhood SES was associated with more pack-years smoked ($b=3.235, p<0.001$), which led to higher CRP levels ($b=0.004, p<0.01$), raising the risk of IHD ($b=0.076, p<0.01$). The indirect effect of childhood SES to IHD via CRP and smoking was significant at the 0.05 level, revealing that the effect of childhood SES on IHD was transmitted through the paths of CRP and smoking. In addition, gender moderated the effect of childhood SES on smoking. For BMI, low childhood SES was associated with higher levels of BMI ($b=0.385, p<0.001$), which led to higher CRP levels ($b=0.092, p<0.001$), culminating in increased risk of IHD ($b=0.076, p<0.01$). The indirect effect of childhood SES to IHD via CRP and BMI was significant at the 0.05 level. For exercise, low childhood SES was associated with less frequent exercise ($b=-0.195, p<0.001$); less frequent exercise was associated with higher CRP levels ($b=-0.074, p<0.001$), which raised the risk of IHD ($b=0.076, p<0.01$). The indirect effect of childhood SES to IHD via CRP and exercise was significant at the 0.05 level.

Table 3.3 Mediation Results for Childhood SES

Parameter	Model 2 <i>b</i> (SE) ^a
SES—IHD	0.081 (0.062)
SES—Pack-years	3.235 (0.539)***
Female—Pack-years	-5.461 (0.809)***
SESxFemale—Pack-years	-3.021 (0.663)***
Pack-years—CRP	0.004 (0.001)**
CRP—IHD	0.076 (0.028)**
Indirect Effect	0.001 (0.000)*
SES—BMI	0.385 (0.078)***
BMI—CRP	0.092 (0.005)***
CRP—IHD	0.076 (0.028)**
Indirect Effect	0.003 (0.001)*
SES—Exercise	-0.195 (0.020)***
Exercise—CRP	-0.074 (0.020)***
CRP—IHD	0.076 (0.028)**
Indirect Effect	0.001 (0.000)*
SES—Education	-1.144 (0.044)***
Education—CRP	-0.047 (0.010)***
CRP—IHD	0.076 (0.028)**
Indirect Effect	0.004 (0.002)*
SES—Wealth	-6.775 (0.505)***
Wealth—CRP	-0.004 (0.001)***
CRP—IHD	0.076 (0.028)**
Indirect Effect	0.002 (0.001)*

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed tests).

^aUnstandardized coefficient (standard error).

For adult SES, education and wealth in tandem with CRP significantly mediated the effect of childhood SES on IHD risk. Low childhood SES was associated with fewer years of education ($b = -1.144$, $p < 0.001$), which led to higher CRP levels ($b = -0.047$, $p < 0.001$), raising the risk of IHD ($b = 0.076$, $p < 0.01$). The indirect effect of childhood

SES to IHD via CRP and education was significant at the 0.05 level. Wealth followed a similar pattern to education: low childhood SES was associated with less wealth ($b=-6.775, p<0.001$), which led to higher CRP levels ($b=-0.004, p<0.001$), leading to an increased risk of IHD ($b=0.076, p<0.01$). The indirect effect of childhood SES to IHD via CRP and wealth was significant at the 0.05 level. These pathways fully mediated the effect of childhood SES on IHD risk ($p=0.192$). To demonstrate how these mediation results map out on to the proposed pathway model in Figure 3.1, Figure 3.2 illustrates the relationships among childhood SES, adult health lifestyles, SES, inflammation, and IHD onset.

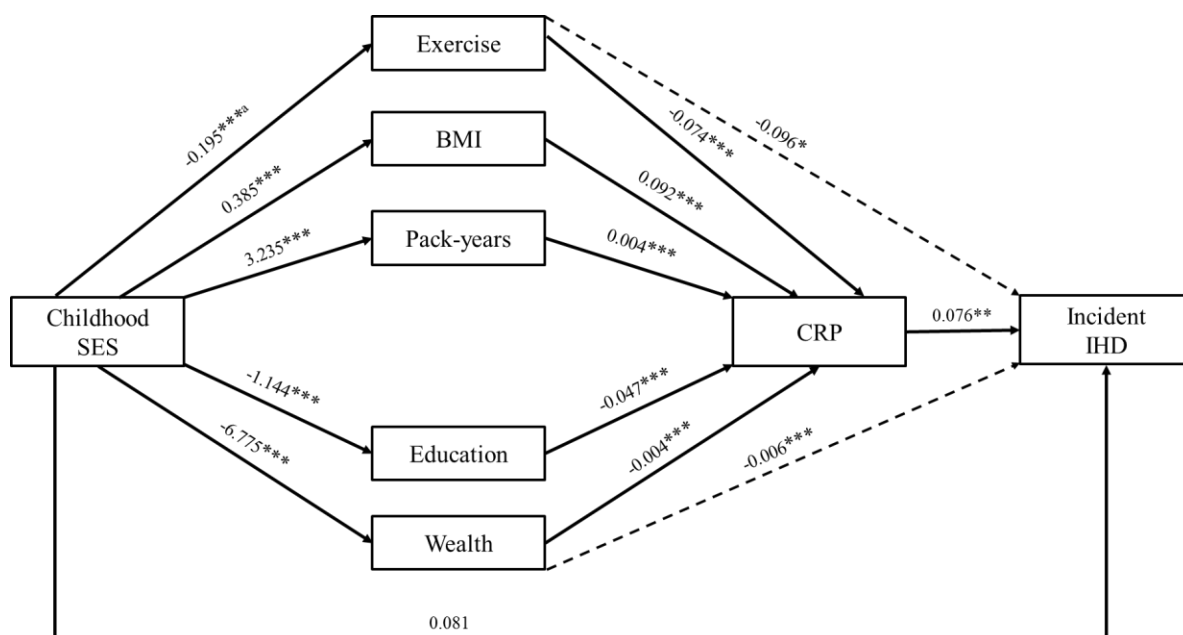


Figure 3.2 Path from Childhood SES to Incident IHD via Adult Health Lifestyles, SES, and Chronic Inflammation.

Notes: ^aUnstandardized coefficient. * $p<0.05$; ** $p<0.01$; *** $p<0.001$. All indirect effects significant at 0.05 level. Dotted lines indicate additional significant direct effects on IHD that are not part of the mediational pathway model. Model adjusts for all independent variables listed in Table 3.2, Model 2.

3.8 Discussion

The purpose of the present study was to elucidate how early-life experiences are embedded into trajectories of health over the life course. Many studies have demonstrated that childhood experiences can influence health in later-life, but they often implicitly depict a long latency period between exposure to childhood misfortune and disease onset. The present study sought to extend this literature by clarifying the multidimensional process of how early-life experiences become manifest as health risks in adulthood. In doing so, my goal was to bridge together research from multiple disciplines studying the early origins of adult health in an effort to contextualize health processes along the lines of childhood conditions, individual behaviors, social structures, and biological systems.

Health is a multidimensional process that unfolds over the life course; many conceptual models of health and aging propose this, but few surveys possess the data to empirically test this. To my knowledge, this is the first paper to empirically test a multidimensional pathway model from childhood to adult health that includes adult health behaviors, SES, and biomarkers. In particular, biomarkers are more often outcomes, but rarely tested as mediators in the childhood misfortune-adult health literature. However, biological markers of inflammation are becoming an increasingly reliable method of forecasting an individual's health and mortality risk—particularly cardiovascular health and mortality (Cesari et al. 2003; Ridker et al. 2000a; Ridker et al. 2000b; Volpato et al. 2001). Using inflammation as a mediator between childhood misfortune and adult IHD was a novel, albeit logical, next step.

Guided by CI theory and biological embedding, I expected that childhood misfortune would raise IHD risk in adulthood via health lifestyles, SES, and inflammation. Specifically, I expected that childhood misfortune would directly influence adult health lifestyles and SES which, in turn, would affect inflammation levels, leading to IHD onset. Investigating alternative types and specifications of childhood misfortune, I found evidence for this proposed pathway model of health. Although a count of misfortune did not predict IHD risk, examining the unique effects of the domains of misfortune revealed that childhood SES initiated the proposed chain of risks toward poor health. Higher levels of childhood socioeconomic disadvantage led to unhealthy lifestyles and lower SES in adulthood, both of which led to higher levels of CRP, raising risk of IHD.

The findings of this study reveal three important conclusions. First, childhood misfortune—specifically childhood SES—adversely impacts an individual’s health lifestyles *and* SES, suggesting that childhood conditions influence both agentic *and* structural processes of health. Moreover, these multidimensional processes act back on the individual and impact intra-individual health mechanisms (i.e., CRP levels), culminating in disease onset (Ferraro and Morton 2016).

Second, these findings highlight the salience of childhood SES for later-life health (Link and Phelan 1995). Consistent with prior research, socioeconomic disadvantage during childhood predicted risk of IHD in adulthood (Hamil-Luker and O’Rand 2007; Kaplan and Salonen 1990). Extending prior research, this study demonstrated that childhood SES raises adult IHD risk even when adjusting for other domains of misfortune and adult risk factors. This study also revealed that there are multiple ways in

which childhood SES can impact later-life health outcomes. Although this study did not find a relationship between ACM and IHD risk, it is important to note that prior research connecting ACM to IHD risk utilized other indicators of misfortune which measured more adverse experiences, such as household dysfunction and abuse (Dong et al. 2004; Morton et al. 2014).

Third, findings point to the value of exercise to reduce health risks in later-life. Beyond its indirect effect via chronic inflammation, exercise also exerted an independent, direct effect on IHD risk. The more frequently respondents engaged in vigorous exercise or physical activity, the lower the risk of IHD. Despite the many contributors to disease onset, it is important to remember that individuals can take control of their own health to a certain degree. One simple way is to increase the frequency of physical activity—a low cost, drug-free, and minimally invasive intervention. The benefits of exercise among older adults have been well-documented (Elward and Larson 1992; Smith and Iliffe 1997), and this study adds to the overwhelming evidence of how exercise can improve health outcomes in later-life.

One interesting finding was that education, unlike wealth, did *not* have a direct impact on IHD. Some studies suggest that wealth is more consequential to older adult's health than other aspects of SES (e.g., Pollack et al. 2007), with some scholars proposing an age-as-a-leveler hypothesis for education (e.g., Beckett 2000; Herd 2006; Preston and Taubman 1994). However, this study revealed the lingering contributions of education to health, albeit indirectly. Several theoretical frameworks note the importance of multiple indicators of SES (e.g., Link and Phelan 1995), and this study's methodological approach was able to uncover the indirect role of education in health processes for older adults. It

is important to remember that health processes are multifaceted and can take shape through many experiences and conditions, directly and indirectly.

One unexpected finding was that Black respondents had lower risk of incident IHD than White respondents. Although unexpected, there is some evidence that racial disparities in health crossover in later life. Several studies have observed a Black/White crossover effect for disease (Johnson 2000), disability (Clark, Maddox, and Steinhauser 1993; Johnson 2000; Kim and Miech 2009), and mortality (Eberstein, Nam, and Heyman 2008; Johnson 2000; Laditka and Wolf 1998; Land, Guralnik, and Blazer 1994) among older adults, and this effect appears particularly salient for cardiovascular mortality (Eberstein et al. 2008; Manhapra et al. 2004). Although this sample, on average, is relatively young compared to prior studies that have observed a crossover effect, research suggests that disease crossovers precede and, in part, explain mortality crossovers (Johnson 2000) which can begin at age 75 (Guralnik et al. 1993). In addition, some studies have found that certain types of IHD, such as MI (Alderman, Cohen, and Madhavan 2000) and transient ischemic attack (Zweifler et al. 1995), are less prevalent in midlife for Black adults compared to White adults. Given the average age of the sample (mean age during incident IHD observation period was between 66.5 and 72.5 years) and this study's outcome, these finding could be indicative of a crossover effect.

A crossover effect can arise from multiple selection processes, ranging from survivorship bias (Manhapra et al. 2004; Manton, Patrick, and Johnson 1987) to poor data (Mutchler and Burr 2011). Survivorship bias could be influenced by premature mortality as well as access to and utilization of health care, which can also reduce diagnosis of IHD

(assessment of IHD was based on physician diagnosis).²³ At the same time, the sample is disproportionately White, suggesting sampling selection bias. To what extent this apparent crossover effect is due to selective survival or data limitations is unclear. Since this race finding emerged only after the mediators were introduced into the models, the addition of the mediators may have produced a suppression effect (Guralnik et al. 1993).²⁴

The findings presented herein should be considered in light of this study's limitations. First, the childhood data were collected retrospectively. Although I attempted to reduce potential validity and reliability issues by excluding proxy responses and individuals who had a baseline cognitive score less than two standard deviations below the sample mean while adjusting for adult variables that influence retrospective recollection (e.g., depressive symptoms), recall bias may still exist (Vuolo et al. 2014).

Second, although the HRS data include a wealth of information on childhood conditions, there are some important measures of childhood misfortune that are limited in the HRS. Specifically, the HRS does not include sufficient information on child abuse and/or neglect to create a domain of child maltreatment, which some scholars consider “a life course social determinant of adult health” (Greenfield 2010:53). Although childhood

²³Although adjusting for having health insurance did not alter the race findings, I was unable to assess healthcare utilization.

²⁴Additional analyses investigating interactions between race and each mediator were not able to shed any additional insight. Thus, there may have been a compounded statistical suppression effect by two or more of the mediators. One way to detect suppression is by changes in R²; however, Mplus does not produce pseudo-R² for Cox models. Supplementary analyses investigating the interaction of race and gender did, however, reveal that White men in particular had higher risk of IHD than their Black counterparts, consistent with prior research on crossover effects among older adults (Laditka and Wolf 1998).

SES is the only domain that was related to IHD risk, future research should utilize other data sets to investigate whether the pathway model proposed in this study holds for other samples and different domains of misfortune, including maltreatment.

Third, the possibility of selection bias is another limitation. Because this sample is comprised of older adults (minimum age of the sampling frame is 51 years), there could be an issue of left-censoring. Individuals who experienced the most misfortune early in life may not be represented due to increased risk of premature mortality, institutionalization, or early onset of major illness, each of which would either disqualify individuals from the initial sampling frame or decrease the likelihood of participation. In addition, restricting the sample to incident IHD beginning in 2008 also influenced selection into the analytic sample. Preliminary analyses revealed that older, White male adults and those who experienced health and socioeconomic disadvantage were more likely to be removed from the analytic sample because they had experienced IHD prior to 2008. Although there was a strong methodological rationale for the analytic sample (e.g., temporal ordering), it is important to note that the effects of key variables on IHD risk may be underestimated for those who were excluded from the observational period (e.g., older, White men) as well as for those who are not represented in the survey (i.e., individuals with the most early-life misfortune).

Despite these limitations, this study contributes to research seeking to better understand how health risks become manifest over the life course. Mapping out the path from childhood to IHD onset demonstrates that health is not simply a result of a single, proximal event or experience. Rather, health risks can emerge from a chain of multiple events initiated in some of the earliest stages of the life course (Ben-Shlomo and Kuh

2002). In this study, childhood misfortune triggered a chain of disadvantage that led to IHD. Elucidating this chain of risks reveals that childhood misfortune may not yield permanent damage to health in later-life, especially if disadvantage is experienced within a single domain. Instead, there are ways in which health policy can intervene, and these findings can direct policymakers as to where and how to intervene. At the individual level, for instance, interventions that promote health behaviors can target children of misfortune, preferably at early ages. At the macro-level, policies can be implemented that make higher education more accessible and wealth more evenly distributed.

CHAPTER 4. CONCLUSIONS

It is easier to build strong children than to repair broken men.

Frederick Douglass (1818–1895)

4.1 Summary of Findings

Overall, the objective of this dissertation was to elucidate how childhood events and experiences “get under the skin” and threaten optimal aging. To understand how childhood misfortune can alter the aging process on a biological level, I focused on the physiological mechanism of inflammation. Using a sample of older adults, I found that childhood misfortune led to unhealthy lifestyles and lower SES, which were embedded physiologically, as observed by elevated levels of inflammation. This biological imprint of early life subsequently led to disease, threatening optimal aging. I am unaware of any study on childhood misfortune that has utilized a similar interdisciplinary, multidimensional approach to investigate the mechanisms of childhood misfortune. In addition, highlighting the role of inflammation in life course processes of health warrants special consideration for the many fields studying aging.

Studying inflammatory processes of health is pertinent to aging research as chronic inflammation is a key pathogenic process of health among older adults (Krabbe et al. 2004). As research continues to identify key mechanisms of aging, it is important to remember that many of these health and aging mechanisms often entail relatively distal

origins (Barker 2004). These distal origins of health and aging often occur during sensitive periods when a window of opportunity exists for experiences to embed themselves—physiologically or otherwise—into trajectories of health and aging (Ferraro and Shippee 2009; Hertzman and Boyce 2010). Childhood is considered a sensitive period for life course health and, therefore, research clarifying the process of how childhood misfortune becomes manifest as poor health over time is essential, especially as life expectancy continues to rise. People are living longer, and living disease-free for more years is a goal shared by many Americans (Hart Research Associates 2016). This dissertation sheds light on ways in which healthspan can be increased, even when misfortune is experienced early in life.

Using CI theory and biological embedding as theoretical templates for studying life course health, I conducted two studies, comprised of three aims, to specify how health unfolds over the life course as a multidimensional process. The first study in Chapter 2 consisted of two aims to elucidate the relationship between childhood misfortune and adult chronic inflammation. To better understand how chronic inflammation links childhood misfortune to adult health, I wanted to first understand the process of how childhood misfortune impacts inflammation levels. For aim 1, I systematically investigated *the relationship between childhood misfortune and adult chronic inflammation*. Whereas most studies using inflammation as an outcome have focused on a single domain of misfortune, such as childhood SES (e.g., Brummett et al. 2013; Carroll et al. 2011; Stringhini et al. 2013) or maltreatment (e.g., Carpenter et al. 2010; Danese et al. 2007; Lehto et al. 2012; Matthews et al. 2014), I considered multiple domains and alternative specifications of misfortune.

This study revealed that unique experiences of childhood socioeconomic disadvantage contributed to higher levels of inflammation in adulthood. In addition, the combined effect of multiple domains of misfortune also raised levels of inflammation in adulthood. These findings add to the growing evidence documenting an effect of early-life misfortune on adult chronic inflammation, and strengthen the previous research demonstrating a relationship between childhood SES and adult inflammation (Brummett et al. 2013; Danese et al. 2009; Carroll et al. 2011; Stringhini et al. 2013; Taylor et al. 2006). I found a relationship between childhood SES and inflammation among midlife and older adults even after controlling for several additional childhood experiences and adult risk factors.

Aim 2 builds upon the findings in Aim 1 and examined *whether adult health lifestyles and SES mediated the effect of childhood misfortune on adult chronic inflammation*. For this aim, I investigated both individual and structural factors as potential intermediary mechanisms to determine whether agentic or institutionalized forces contributed to the effect of childhood misfortune on adult inflammation, as posited by CI theory. This study revealed that both individual and structural factors mediated the effect of childhood misfortune on adult chronic inflammation, but specific mechanisms varied by misfortune. Whereas exercise mediated the effect of childhood SES, exercise did not mediate the effect of ACM. These findings also revealed that the effect of childhood SES on smoking varied by gender. However, the effect of smoking on chronic inflammation did not vary by gender; pack-years smoked had the same impact on inflammation for men and women.

The third aim, tested in the second study, made use of the exceptional data structure in the HRS to examine a multidimensional pathway model of life course health. For this aim, I investigated *the relationships among childhood misfortune, adult health lifestyles, SES, chronic inflammation, and ischemic heart disease onset*. Based on my findings in the previous study, I asked whether the inflammatory sequelae of childhood misfortune would lead to risk of IHD, a common disease in the United States. I found that childhood SES raised the risk of IHD via adult health lifestyles, SES, and chronic inflammation. Childhood SES led to unhealthy lifestyles and lower SES, which increased levels of inflammation, culminating in IHD onset. As observed in Study 2, gender moderated the relationship between childhood SES and smoking.

Taken together, the findings presented in this dissertation point to childhood as a sensitive period in life course health, giving credence to CI theory and biological embedding. In support of CI theory and biological embedding, I demonstrated that childhood experiences can have a *lasting impact on health and aging even if indirect*. Per CI theory, the avenues through which childhood misfortune affected health and aging were plenty; early-life experiences can affect health behaviors and lifestyles, SES, and physiological systems throughout the life course. (Ferraro and Shippee 2009). Although these multifarious consequences of childhood misfortune may seem rather daunting, this also means that health policy can intervene in multiple places and take a multi-pronged attack to combat the consequences of childhood misfortune.

Based on the findings presented in this dissertation, there are several ways in which public health policy can intervene to alleviate the consequences of childhood misfortune. The findings of health lifestyles and SES suggest that policymakers should

enact health policies that encompass multiple levels; public health should not target only individual health behaviors (e.g., smoking reduction and increased physical activity), but also broader institutions which impact health as well (e.g., making higher education more accessible and paying a living wage). In addition, using markers of inflammation to assess the long-term effects of childhood misfortune can enhance assessment of individuals' health risks, and health interventions, from exercise to mimetics, can target these high-risk individuals before onset of disease. Indeed, there are many ways in which the health burden of childhood misfortune can be reduced—and taking a multipronged approach to mitigate the consequences of childhood misfortune is warranted. Because the effect of childhood SES on IHD risk was fully mediated, interventions might be more effective when misfortune occurs within a single domain. Nonetheless, reducing exposure to misfortune should also be of chief concern to health policymakers.

In sum, this dissertation clarifies the intermediating processes of the “long-arm” of childhood misfortune and identifies *modifiable* health and mortality risks. Building upon prior contributions from several disciplines studying life course health and aging, I demonstrated how health outcomes are shaped by childhood conditions through health-related, socioeconomic, and biological factors. Indeed, the grasp of childhood misfortune is far-reaching and diverse; developing effective strategies that mitigate the health consequences of childhood misfortune requires an interdisciplinary perspective. By utilizing an interdisciplinary approach to identify the intermediary pathways between childhood misfortune and adult health, this dissertation can direct healthcare policymakers as to where and how to intervene in an effort to reduce the burden of childhood misfortune.

4.1.1 Data Limitations

Although I outlined several limitations specific to each study, there are three overarching limitations of the dataset used in this dissertation. One important limitation to consider in light of the findings presented herein is that although the HRS is replete with information about childhood, especially SES, there are other potential childhood experiences and events not captured (adequately or otherwise) by these data. Thus, other experiences of childhood misfortune should not be discounted, and future research should continue to investigate multiple domains of misfortune as unique and additive predictors of later-life health. Moreover, the effects of childhood misfortune may vary based on the outcome, as indicated by the distinct findings for ACM in Studies 1 and 2. Several types of childhood experiences, such as maltreatment and loss of a parent, are not adequately measured in the core surveys of the HRS but can be consequential to later-life health (Greenfield 2010; Luecken and Roubinov 2012). Attention to other domains is not only important for examination of unique effects of misfortune, but is also relevant to the additive effects of misfortune as other domains can be included in a count of misfortune and as controls.

A second notable limitation of this dissertation is the limited proportion of non-White respondents in the HRS. Although the HRS attempted to get an accurate representation of adults above the age of 50, descriptive statistics indicate that the sample is predominantly White. Indeed, there are several reasons for the disproportionate representation of White Americans (from non-response bias to later-life crossover and salmon effects), and the HRS took several steps to increase participation of non-White respondents, such as oversampling Black and Hispanic Americans as well as Floridians.

Nonetheless, these groups along with several other racial and ethnic minorities are underrepresented (e.g., Native and Asian Americans). Although the HRS provides weights to account for non-response bias, weighted analyses are not equivalent to the actual information missing from these non-represented individuals. The United States is a diverse country, and future surveys should take steps to ensure that samples are more representative of the U.S. population. Doing so would enable researchers to better understand how health disparities develop over the life course and can improve healthcare policy and prevention for all Americans. In light of this limitation, racial findings presented in this dissertation should be interpreted with caution.

Third, the HRS does not include medication information for several types of medications that affect inflammation levels. Many prescription drugs can influence inflammation levels but there are two notable medications missing from the core surveys: statins and hormone replacements. These medications can artificially raise (hormonal replacement) and lower (statins) inflammation levels and point to a significant limitation of this dissertation. Inflammation plays a major role in this dissertation, which sought to explore the biological residue of childhood misfortune. Chronic inflammation was the outcome of interest in Study 1 and a key mediator in Study 2. Moreover, the HRS is comprised of older adults, and it is, therefore, likely that a substantial proportion of HRS respondents are taking these medications due to high cholesterol and, for women, menopause. Adjusting for these kinds of medications would be ideal and paint a more accurate picture of the proposed relationships. Unfortunately, the dataset used in the present dissertation was unable to account for these potential confounders, and future research should do so if able.

Despite these limitations, the HRS is quite a remarkable dataset to study health and aging processes among older adults in America. Although there appears to be some bias in baseline responses, follow-up response rates are relatively high—most cohorts had an 85% or higher response rate at each wave, including supplemental surveys (Ofstedal and Weir 2011; Sonnega et al. 2014). The response rate is rather impressive given the frequency at which the HRS conducts its surveys (every two years) and the overall number of waves (14). In addition, the HRS has higher proportions of non-white respondents as well as biomarker data than many other health surveys (e.g., Midlife Development in the United States [MIDUS]).

4.2 Discussion of Findings

The objective of this dissertation was to understand why children of misfortune grow up to have worse health than others. Guided by theory, I posited that there was a ‘behind the scenes’ chain of risks that involved unhealthy lifestyles, relatively lower SES, and immune dysregulation, all of which contributed to poorer health. My theoretical frameworks of CI theory and biological embedding both asserted a pathway model of health, suggesting that childhood events and experiences could initiate a path toward poor health. Although theory can provide a plausible explanation for the links among childhood misfortune, adult health lifestyles, SES, chronic inflammation, and health, what is it about these early-life events and experiences that place children on these paths

toward poor health so early in the life course? To answer this question, it may be best to begin looking at the first two mediators: adult health lifestyles and SES.²⁵

First, why are children of misfortune more likely to engage in unhealthy lifestyles? For childhood socioeconomic disadvantage, health behaviors might be linked to access and knowledge. Considering the relationship between childhood SES and access, children growing up in disadvantaged neighborhoods may not have access to playgrounds and/or usable sidewalks to encourage physical activity (Robert 1999). Likewise, low childhood SES may also restrict access to healthy food options. For these children, socioeconomic disadvantage may mean their parents are not able to afford healthy food choices or monitor their children's eating habits if their work schedules do not permit them to be home during dining hours. Alternatively, these children may live in areas that are considered food deserts which limit their access to healthy food (for review, see Walker, Keane, and Burke 2010). These early-life health-related food behaviors may establish lifelong dietary habits that influence BMI over the life course. In addition, low childhood SES can translate into less access to and utilization of healthcare (e.g., lack of [adequate] medical insurance; healthcare may be cost or time prohibitive for parents). If so, knowledge and support for healthy lifestyles may not be given through traditional avenues like a general practitioner, leading to less physical activity, higher BMI, and smoking. Moreover, if children from economically disadvantaged backgrounds remain socioeconomically disadvantaged in adulthood, as

²⁵Although health lifestyles and SES were assessed at midlife and beyond, many of the indicators are rooted in much earlier stages of the life course, such as smoking and education.

demonstrated by the findings of this dissertation, these health lifestyles related to SES can persist.

For more adverse experiences, there might be another explanation. Unhealthy lifestyles may serve as coping mechanisms for children of misfortune. To these individuals, these health behaviors and lifestyles might not only be responses to insults, but could also be solutions to problems. As concluded by Felitti (2002) from his findings in the ACE studies, “[t]he counterintuitive aspect was that, for many people, obesity was not their problem; it was their protective *solution* to problems that previously had never been discussed with anyone” (p. 44). Thus, perhaps children who experience misfortune, particularly more adverse forms, may self-medicate through substance abuse, whether that be smoking, drugs, or overeating. Moreover, these coping mechanisms learned early-on could become standard responses to stress throughout the life course. Behaviors like smoking or overeating may be used as coping mechanisms later in life when faced with disadvantage again, such as divorce, death, and/or financial strain.

The next question is why are children of misfortune more likely to experience decreased gains in SES? For sociologists, it is not surprising that adults who experienced socioeconomic disadvantage during childhood are more likely to have lower SES than those who did not. Sociologists are aware of the strong structural hold that social institutions like SES have (Blau and Duncan 1967). Sociology recognizes that SES is often transmitted between generations, and social mobility in later-life, which is difficult but possible, does not necessarily free an individual from his or her parents’ SES. Parent’s SES can affect their children’s SES through mechanisms like capital (e.g., human: Mirowsky and Ross 1998; social: Coleman 1998; cultural: Walpole 2003) and

material assistance (e.g., Spilerman 2000; Steelman and Powell 1991).²⁶ Sociological theorists like Elder (1998) and C. Wright Mills (1959) have highlighted how lives are linked through familial generations and social structures; individuals are shaped by their historical context, and prior social forces can shape the present and future. Thus, SES is transmitted intergenerationally through micro- and macro-processes (Robert 1999).

Beyond childhood SES, other domains of childhood misfortune also shape adult SES. Childhood health as well as other domains of misfortune that impact childhood health (e.g., maltreatment) can affect adult SES. Poor health early in life can interfere with education and lead to lower income, savings, and wealth as illness can interfere with work and deplete finances due to health treatment and maintenance. Another explanation for lower educational gains is that childhood misfortune could negatively impact an individual's sense of agency and competency (i.e., self-efficacy). Many types of childhood misfortune are out of the child's control (e.g., parental death, parent's income, illness, disability, etc.). This lack of control over one's life early on may create a limited view of agency and competency. When these children become adults and reach normative life course transition periods related to SES (e.g., decide to start, continue, and graduate college), this lack of agency and competency may inhibit these adults from pursuing these economic options as they may appear futile given their past experiences.

In addition, Rosenstock, Stretcher, and Becker (1988) proposed a revised Health Belief Model that incorporated self-efficacy, asserting that belief in one's self to succeed or accomplish tasks influences health behaviors and lifestyles. Thus, this diminished

²⁶Many of these mechanisms that facilitate the intergenerational transmission of SES are also linked to health.

agency, competency, and self-efficacy may also contribute to the unhealthy lifestyles observed in these children of misfortune. Beyond perceptions, childhood misfortune can also affect other aspects of mental health (e.g., Carr et al. 2013; Kessler et al. 2010; Schilling, Aseltine, and Gore 2008; Trotta, Murray, and Fisher 2015), which can also influence health lifestyles and SES (Miech et al. 1999).

Explaining why childhood misfortune influences adult health lifestyles and SES may lead to the same answers, as noted above regarding agency and self-efficacy, and there are conceptual frameworks that could answer both questions. One conceptual framework that could potentially explain why children of misfortune are prone to engage in unhealthy lifestyles and experience lower SES is Carstensen's (1999, 2006) socioemotional selectivity theory. According to socioemotional selectivity theory, open-ended, or limitless, perceptions of time make an individual prioritize knowledge-related goals whereas perceiving of time as constrained, or limited, will make emotional goals priority. Although chronological age is a main predictor of how one senses time, Carstensen et al. (1999) argue that perceptions of time are malleable. Life experiences, like poor health, can alter an individual's sense of time, and younger adults' perception of time can mirror that of older adults. For these young adults, less importance is placed on investments for the future and knowledge-related goals are no longer a priority. Related to this dissertation, if an individual has survived a substantial amount of disadvantage early in life, then his or her future perception of time may be more limited compared to those who have not experienced such disadvantage.²⁷ If so, this would lead children of

²⁷Childhood misfortune may limit perceptions of time and lead to prioritizing socioemotional needs through various ways. For example, serious health conditions during childhood may lead to a more limited view of time, especially if the health condition is life threatening. Poor

misfortune to focus on emotional gains in the present rather than investing in future gains, as observed by decreased investments in education, savings/assets, and long-term health (being less invested in the future could lead to more risky and unhealthy lifestyles).

These explanations are beyond the scope of this dissertation, but warrant special consideration for future research on the childhood origins of adult health. Although my explanations are currently speculative and extrapolate the findings of this dissertation, understanding the link between childhood misfortune and adult health lifestyles and SES is one area for future research on the long-term effects of childhood misfortune. I highlight several additional future directions for this body of research below.

4.3 Future Directions

There are an infinite number of future directions for studying health over the life course, but I focus on five areas. First, research on health and aging over the life course should give more attention to the underutilized concept of timing (Ferraro and Morton

childhood health can easily shift the narrative of an endless sense of time to a more finite view of time, as commonly witnessed among adults who are suddenly faced with a serious health condition. Regarding childhood SES, children who experience a substantial amount of economic disadvantage may have lived on a day-to-day basis. These daily struggles may limit how time is perceived. Rather than viewing prospective time in the long-term, their early-life experiences may have taught them to view prospective time in a more short-term capacity, especially if forecasting the future is somewhat unpredictable. Likewise, childhood maltreatment and household dysfunction could also create an unstable view of the future, limiting perceptions of time as well. In addition, childhood misfortune could also affect when the shift from informational needs to socioemotional needs occurs (Carstensen 1993). Although this shift usually happens later in the life course due to age, the shift to prioritize socioemotional needs may occur earlier in life for children of misfortune, especially if their socioemotional needs are not met during early childhood (particularly in instances of abuse and neglect).

2016). Many theoretical frameworks emphasize timing, but relatively few empirical studies examine the many aspects of timing. However, timing is an important concept in life course health, especially for studying sensitive and critical periods. The timing of childhood misfortune may play a crucial role in predicting later-life consequences. The few studies investigating timing suggest that childhood misfortune can have differential effects based on when (in terms of child's age) the misfortune occurred (e.g., Cohen et al. 2004; Fisher et al. 2010; Friedman et al. 2015; Slopen et al. 2013; Thornberry, Ireland, and Smith 2001; Ziol-Guest, Duncan, and Kalil 2009). In addition, research focused on the timing of misfortune could provide a more precise definition of what period during childhood constitutes a sensitive or critical period.

The timing of the mechanisms of childhood misfortune studied in this dissertation should be examined as well. For instance, might differences in timing of higher education, obesity onset, physical activity, or debt have differential effects? Likewise, the relationship between timing and chronic inflammation should be explored: When does immune dysregulation due to early-life misfortune become measurable? When does chronic inflammation become manifest? If immune dysregulation due to childhood misfortune is experienced earlier in the life course, is it more salient and/or less malleable? Future research should make use of data sets that collect information at earlier time points in the life course because more attention to timing could greatly advance the field of life course health and aging.

Second, to facilitate research on timing, future research on health and aging in the United States should utilize surveys that observe individuals during childhood and emphasize replication studies using these prospective study designs. Replication is at the

heart of the science and future research requires more replication studies (Maxwell, Lau, and Howard 2015; Simons 2014).²⁸ However, most journals desire to publish cutting edge research and require innovation. Thus, a novel way to conduct replication studies in this area of research would be to test the pathways proposed in this dissertation using childhood data *that are not retrospective*. Hypotheses that are supported (or refuted) utilizing one dataset should be tested using different data sets to determine whether conclusions hold when respondents are not required to recall childhood conditions several decades later.

Third, future research should focus on the concept of resilience. Although children of misfortune are at higher risk for many diseases, some individuals may escape childhood misfortune relatively unscathed leaving scholars to wonder how this happens. Perhaps a link in the chain of disadvantage was broken at some point. But how was this chain of disadvantage broken? Or perhaps the event(s) or experience(s) never initiated a chain of disadvantage. To understand why responses to early-life insults might vary, future research could investigate how individuals perceive these events and experiences. What may be a misfortune or disadvantage to one may be seen as a fortune or advantage, or even neutral, to another (e.g., divorce). Prior research has shown that perception of childhood misfortune can influence how misfortune impacts an individual (Surtees and Wainwright 2007). Resilience is a somewhat fuzzy concept, but future research identifying the common threads stitching together adult who have managed to overcome early-life disadvantage may help clarify this term.

²⁸This topic of reproducibility has been recently revived in psychology and it would behoove sociologists and social gerontologists to underscore the importance of replication studies in their own field.

Fourth, future research will likely entail more multidisciplinary collaborations, particularly for hybrids fields like gerontology that bring together many disciplines. Recently, there has been an increasing awareness for the benefits of a multidisciplinary perspective of health. For example, the National Academies of Sciences, Engineering, and Medicine (2016) released a report this year calling for health professionals to address social determinants of health, including “conditions in which people are born, grow, [...] and live of health” (p. 1). As evidenced, social science’s role in the field of medicine has been increasing in tandem with multidisciplinary research on health. Additional evidence of disciplinary crossovers includes epidemiologists’ recent attention to race/ethnicity and sociological attention to biological processes.

Interdisciplinary research not only generates innovative research, but it can also have policy implications, affecting health on an institutional level. For instance, social science research on health disparities might enlighten the World Heart Federation’s (2016) view on non-modifiable risk factors, which now include race and gender. Perhaps medical sociologists’ structural approach to identifying the underlying mechanisms (e.g., discrimination) of these seemingly immutable health risks could create interventions targeting the mechanisms. Doing so could modify the risks of these “non-modifiable” risk factors. In sum, research is becomingly increasingly collaborative, and disciplines are intermixing more often. The findings of this dissertation are a testament to the interdisciplinary insight that can be gleaned when scholars consider multiple perspectives for disciplines outside their own, and future research should continue to incorporate knowledge from multiple disciplines.

Finally, another area where marked advances might be seen is in the “science of interventions” (Williams 2012) or, as referred to by Brown (2013), “advocacy social science”. Intervention research on health, aging, and the life course will likely advance, partly as a way to deal with the aging population and partly due to heightened awareness of the need for this research (Brown 2013; Williams 2012). Indeed, health studies emphasize the importance of policy-based research while highlighting the limited contributions thus far (e.g., Brown 2013; Haug 1994; McKinlay 1996; Williams 2012). Developing the “science of interventions” would give way to more translational research, and research like this dissertation can provide a starting point for intervention studies on the childhood origins of adult health.

For example, the findings of this dissertation demonstrated that childhood experiences do not always have direct effects on adult health. Rather, there is a chain of disadvantage, one that can be interrupted. But interrupting this chain requires intervention research that proves what is most effective for mitigating the unhealthy lifestyles and socioeconomic disadvantage experienced by this vulnerable population. However, intervention research should not focus solely on how to deal with the aftermath of early-life disadvantage. Because childhood misfortune permeates so many dimensions of health—and sometimes the damage is irreversible—social sciences should also promote advocacy research that focuses on ways to reduce exposure to childhood misfortune. After all, “[i]t is easier to build strong children than to repair broken men” (Douglass, public speech, 1855).

REFERENCES

REFERENCES

- Albert, Michelle A., Robert J. Glynn, Julie Buring, and Paul M. Ridker. 2006. "Impact of Traditional and Novel Risk Factors on the Relationship Between Socioeconomic Status and Incident Cardiovascular Events." *Circulation* 114:2619–26.
- Alder, Nancy E., Thomas Boyce, Margaret A. Chesney, Sheldon Cohen, Susan Folkman, Robert L. Kahn, and S. Leonard Syme. 1994. "Socioeconomic Status and Health: The Challenge of the Gradient." *American Psychologist* 49:15-24.
- Alley, Dawn. E., Teresa E. Seeman, Jung Ki Kim, Arun Karlamangla, Peifung Hu, and Eileen M. Crimmins. 2006. "Socioeconomic Status and C-Reactive Protein Levels in the US Population: NHANES IV." *Brain, Behavior, and Immunity* 20:498-504.
- Anda, Robert F., David W. Brown, Shanta R. Dube, J. Douglas Bremner, Vincent J. Felitti, and Wayne H. Giles. 2008. "Adverse Childhood Experiences and Chronic Obstructive Pulmonary Disease in Adults." *American Journal of Preventive Medicine* 34:396-403.
- Anda, Robert F., Janet B. Croft, Vincent J. Felitti, Dale Nordenberg, Wayne H. Giles, David F. Williamson, and Gary Giovino. 1999. "Adverse Childhood Experiences and Smoking During Adolescence and Adulthood." *The Journal of the American Medical Association* 282:1652-1658.

- Barker, D. J. P. 2004. "The Developmental Origins of Adult Disease." *Journal of the American College of Nutrition* 23:588S-595S.
- Barros, Aluísio J. and Vânia N. Hiraçata. 2003. "Alternatives for Logistic Regression in Cross-Sectional Studies: An Empirical Comparison of Models that Directly Estimate the Prevalence Ratio." *BMC Medical Research Methodology* 3:21.
- Baumeister, D, R Akhtar, S Ciufolini, CM Pariante, and V Mondelli. 2015. "Childhood Trauma and Adulthood Inflammation: A Meta-Analysis of Peripheral C-Reactive Protein, Interleukin-7, and Tumor Necrosis Factor- α ." *Molecular Psychiatry* doi: 10.1038/mp.2015.67
- Beckett, Megan. 2000. "Converging Health Inequalities in Later Life—An Artifact of Mortality Selection." *Journal of Health of Social Behavior* 41:106-19.
- Beckett, Megan, Julie Da Vanzo, Narayan Sastry, Constantijn Panis, and Christine Peterson. 2001. "The Quality of Retrospective Data: An Examination of Long-Term Recall in a Developing Country." *Journal of Human Resources* 36:593–625.
- Beebe-Dimmer, Jennifer, John W. Lynch, Gavin Turrell, Stephanie Lustgarten, Trivellore Raghunathan, and George A. Kaplan. 2004. "Childhood and Adult Socioeconomic Conditions and 31-Year Mortality Risk in Women." *American Journal of Epidemiology* 159:481-490.
- Ben-Shlomo, Yoav and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges, and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31:285-293.

- Blackwell, Debra L., Mark D. Hayward, and Eileen M. Crimmins. 2001. "Does Childhood Health Affect Chronic Morbidity in Later Life?" *Social Science & Medicine* 52:1269-1284.
- Blau, Peter M., and Otis Dudley Duncan. 1967. *The American Occupational Structure*. New York: John Wiley.
- Bonilla-Silva, Eduardo. 1997. "Rethinking Racism: Toward a Structural Interpretation." *American Sociological Review* 62:465-480.
- Bowen, Mary Elizabeth. 2010. "Coronary Heart Disease from a Life-Course Approach: Findings from the Health and Retirement Study, 1998-2004." *Journal of Aging and Health* 22:219-241.
- Braveman, Paula, Susan Egerter, and David R. Williams. 2011. "The Social Determinants of Health: Coming of Age." *Annual Review of Public Health* 32:381-398.
- Brown, David W., Robert F. Anda, Vincent J. Felitti, Valerie J. Edwards, Anne Marie Malarcher, Janet B. Croft, and Wayne H. Giles. 2010. "Adverse Childhood Experiences are Associated with the Risk of Lung Cancer: A Prospective Cohort Study." *BMC Public Health* 10:20.
- Brown, David W., Robert F. Anda, Henning Tiemeier, Vincent J. Felitti, Valerie J. Edwards, Janet B. Croft, and Wayne H. Giles. 2009. "Adverse Childhood Experiences and the Risk of Premature Mortality." *American Journal of Preventive Medicine* 37:389-396.

- Brown, Phil. 2013. "Integrating Medical and Environmental Sociology with Environmental Health: Crossing Boundaries and Building Connection through Advocacy." *Journal of Health and Social Behavior* 54:145-164.
- Brummett, Beverly H., Michael A. Babyak, Abanish Singh, Rong Jiang, Redford B. Williams, Kathleen Mullan Harris, and Ilene C. Siegler. 2013. "Socioeconomic Indices as Independent Correlates of C-Reactive Protein in the National Longitudinal Study of Adolescent Health." *Psychosomatic Medicine* 75:882-893.
- Carpenter, Linda L., Audrey R. Tyrka, Nicole S. Ross, Lamy Khoury, George M. Anderson, and Lawrence H. Price. 2009. "Effect of Childhood Emotional Abuse and Age on Cortisol Responsivity in Adulthood." *Biological Psychiatry* 66:69-75.
- Carpenter, Linda L., Cyrena E. Gawuga, Audrey R. Tyrka, Janet K. Lee, George M. Anderson, and Lawrence H. Price. 2010. "Association between Plasma IL-6 Response to Acute Stress and Early-Life Adversity in Healthy Adults." *Neuropsychopharmacology* 35:2617-2623.
- Carpenter, Linda L., Thaddeus T. Shattuck, Audrey R. Tyrka, Thomas D. Geraciotti, and Lawrence H. Price. 2011. "Effect of Childhood Physical Abuse on Cortisol Stress Response." *Psychopharmacology* 214:367-375.
- Carr, Clara Passmann, Camilla Maria Severi Martins, Ana Maria Stingel, Vera Braga Lemgruber, and Mario Francisco Juruena. 2013. "The Role of Early Life Stress in Adult Psychiatric Disorders: A Systematic Review According to Childhood Trauma Subtypes." *The Journal of Nervous and Mental Disease* 201:1007-1020.

- Carroll, Judith E., Sheldon Cohen, and Anna L. Marsland. 2011. "Early Childhood Socioeconomic Status is Associated with Circulating Interleukin-6 among Midlife Adults." *Brain, Behavior, and Immunity* 25:1468-1474.
- Carstensen, Laura L. 2006. "The Influence of a Sense of Time on Human Development." *Science* 312:1913-1915.
- Carstensen, Laura L., Derek M. Isaacowitz, and Susan Charles. 1999. "Taking Time Seriously: A Theory of Socioemotional Selectivity." *American Psychologist* 54:165-181.
- Case, Anne, Angela Fertig, and Christina Paxson. 2005. "The Lasting Impact of Childhood Health and Circumstance." *Journal of Health Economics* 24:365-389.
- Center for Disease Control and Prevention (CDC). 2015. "Heart Disease." Retrieved February 17, 2016 (http://www.cdc.gov/heartdisease/heart_attack.htm).
- Center for Disease Control and Prevention (CDC). 2014. "Injury Prevention & Control: Division of Violence Prevention." Updated May 13, 2014. Retrieved January 5, 2016. (<http://www.cdc.gov/violenceprevention/acestudy/>).
- Cesari, Matteo, Brenda W. J. H. Penninx, Anne B. Newman, Stephen B. Kritchevsky, Barbara J. Nicklas, Kim Sutton-Tyrrell, Russell P. Tracy, Susan M. Rubin, Tamara B. Harris, and Marco Pahor. 2003. "Inflammatory Markers and Cardiovascular Disease (The Health, Aging and Body Composition [Health ABC] Study)." *The American Journal of Cardiology* 92:522-528.
- Chapman, Daniel P., Anne G. Wheaton, Robert F. Anda, Janet B Croft, Valerie J Edwards, Yong Liu, Stephanie L. Strugis, and Geraldine S. Perry. 2011. "Adverse Childhood Experiences and Sleep Disturbances." *Sleep Medicine* 12:773-779.

- Chartier, Mariette J., John R. Walker, and Barbara Naimark. 2009. "Health Risk Behaviors and Mental Health Problems as Mediators of the Relationship Between Childhood Abuse and Adult Health." *American Journal of Public Health* 99:847-854.
- Cicchetti, Dante, Fred A. Rogosch, and Assaf Oshri. 2011. "Interactive Effects of Corticotropin Releasing Hormone Receptor 1, Serotonin Transporter Linked Polymorphic Region, and Child Maltreatment in Diurnal Regulation and Internalizing Symptomatology." *Development and Psychopathology* 23:1125-1138.
- Clark, Alexander M., Marie DesMeules, Wei Luo, Amanda S. Duncan, and Andy Wielgosz. 2009. "Socioeconomic Status and Cardiovascular Disease: Risks and Implications for Care." *Nature Reviews Cardiology* 6:712-722.
- Clark, Daniel O., George L. Maddox, and Karen Steinhauser 1993. "Race, Aging, and Functional Health." *Journal of Aging and Health* 5:536-553.
- Coelho, R. T.W. Viola, C. Walss-Bass, E Brietzke, R. Grassi-Oliveira. 2014. "Childhood Maltreatment and Inflammatory Markers: A Systematic Review." *Acta Psychiatrica Scandinavica* 129:180-192.
- Cohen, Sheldon, William J. Doyle, Ronald B. Turner, Cuneyt M. Alper, and David P. Skoner. 2004. "Childhood Socioeconomic Status and Host Resistance to Infectious Illness in Adulthood." *Psychosomatic Medicine* 66:553-558.
- Coleman, James S. 1988. "Social Capital in the Creation of Human Capital." *American Journal of Sociology* 94:S95-S120.

- Crimmins, Eileen M. 2015. "Lifespan and Healthspan: Past, Present, and Promise." *The Gerontologist* 55:901-911.
- Crimmins, Eileen, Jessica Faul, Jung Ki Kim, Heidi Guyer, Kenneth Langa, Mary Beth Ofstedal, Amanda Sonnega, Robert Wallace, and David Weir. 2013. "Documentation of Biomarkers in the 2006 and 2008 Health and Retirement Study." *HRS Documentation Report* Survey Research Center University of Michigan, Ann Arbor, MI.
- Crimmins, Eileen, Jung Ki Kim, and Sarinnapha Vasunilashorn. 2010. "Biodemography: New Approaches to Understanding Trends and Differences in Population Health and Mortality." *Demography* 47:S41-S64.
- Dandona, Paresh, Ahmad Alijada, and Arindam Bandyopandhyay. 2004. "Inflammation: The Link between Insulin Resistance, Obesity, and Diabetes." *Trends in Immunology* 24:4-7.
- Danese, Andrea, Terrie E. Moffitt, HonaLee Harrington, Barry J. Milne, Guilherme Polanczyk, Carmine M. Pariante, Richie Poulton, and Avshalom Caspi. 2009. "Adverse Childhood Experiences and Adult Risk Factors for Age-Related Disease: Depression, Inflammation, and Clustering of Metabolic Risk Markers." *Archives of Pediatrics & Adolescent Medicine* 163:1135-1143.
- Danese, Andrea, Carmine M. Pariante, Avshalom Caspi, Alan Taylor, and Richie Poulton. 2007. "Childhood Maltreatment Predicts Adult Inflammation in a Life-Course Study." *Proceedings of the National Academy of Sciences* 104:1319-1324.

- Danese, A. and M Tan. 2014. "Childhood Maltreatment and Obesity: Systematic Review and Meta-Analysis." *Molecular Psychiatry*. 19:544-554.
- Dannefer, Dale. 2003. "Cumulative Advantage/Disadvantage and the Life Course: Cross-Fertilizing Age and Social Science Theory." *The Journals of Gerontology Series B: Psychological and Social Sciences* 58B:S327–S337.
- de Kok, Inge M., Frank J. van Lenthe, Mauricio Avendano, Marieke Louwman, Jane-Willem W. Coebergh, and Johan P. Mackenbach. 2008. "Childhood Social Class and Cancer Incidence: Results of the GLOBE Study." *Social Science & Medicine* 66:1131-1139.
- Dembo, Richard, Linda Williams, Werner Wothke, James Schmeidler, and C. Hendricks Brown. 1992. "The Role of Family Factors, Physical Abuse, and Sexual Victimization Experiences in High-Risk Youths' Alcohol and Other Drug Use and Delinquency: A Longitudinal Model." *Violence and Victim* 7:245-266.
- Dong, Maxia, Shanta R. Dube, Vincent J. Felitti, Wayne H. Giles, and Robert F. Anda. 2003. "Adverse Childhood Experiences and Self-reported Liver Disease: New Insights into the Casual Pathway." *Archives of Internal Medicine* 163:1949-1956.
- Dong, Maxia, Wayne H. Giles, Vincent J. Felitti, Shanta R. Dube, Janice E. Williams, Daniel P. Chapman, and Robert F. Anda. 2004. "Insights into Causal Pathways for Ischemic Heart Disease: Adverse Childhood Experience." *Circulation* 110:1761-1766.

- Draper, Brian, Jon J. Pfaff, Jane Pirkis, John Snowdon, Nicola T. Lautenschlager, Ian Wilson, Osvaldo P. Almeida. 2008. "Long-Term Effects of Childhood Abuse on the Quality of Life and Health of Older People: Results from the Depression and Early Prevention of Suicide in General Practice Project." *Journal of the American Geriatrics Society* 56:262-271.
- Dube, Shanta R., Robert F. Anda, Vincent J. Felitti, Valerie J. Edwards, and Janet B. Croft. 2002. "Adverse Childhood Experiences and Personal Alcohol Abuse as an Adult." *Addictive Behaviors* 27:713-725.
- Dube, Shanta R., DeLisa Fairweather, William S. Pearson, Vincent J. Felitti, Robert F. Anda, and Janet B. Croft. 2009. "Cumulative Childhood Stress and Autoimmune Diseases in Adults." *Psychosomatic Medicine* 71:243-250.
- Dube, Shanta R., Vincent J. Felitti, Maxia Dong, Daniel P. Chapman, Wayne H. Giles, and Robert F. Anda. 2003. "Childhood Abuse, Neglect and Household Dysfunction and the Risk of Illicit Drug Use: The Adverse Childhood Experience Study." *Pediatrics* 111:564-572.
- Due, Pernille, Rikke Krølner, Mette Rasmussen, Anette Andersen, Mogens Trab Damsgaard, Hilary Graham, and Bjørn E. Holstein. 2011. "Pathways and Mechanisms in Adolescence Contribute to Adult Health Inequalities." *Scandinavian Journal of Public Health* 39:62-78.
- Eberstein, Isaac. W., Charles B. Nam, and Kathleen M. Heyman. 2008. "Causes of Death and Mortality Crossovers by Race [dagger]." *Biodemography and Social Biology* 54:214-228.

- Elder, Glen H. 1998. "The Life Course as Developmental Theory." *Child Development* 69:1-12.
- Elo, Irma. T. 2009. "Social Class Differentials in Health and Mortality: Patterns and Explanations in Comparative Perspective." *Annual Review of Sociology* 35:553-572.
- Elo, Irma T., Pekka Martikainen, and Mikko Myrskylä. 2010. "Early Life Conditions and Cause-Specific Mortality in Finland." *PARC Working Paper Series, WPS 10-04*.
- Elward, K., and E. B. Larson. 1992. "Benefits of Exercise for Older Adults. A Review of Existing Evidence and Current Recommendations for the General Population." *Clinics in Geriatric Medicine* 8:35-50.
- Fagundes Christopher P. and Baldwin Way. 2014. "Early-Life Stress and Adult Inflammation." *Current Directions in Psychological Science* 23:277-83.
- Felitti, Vincent J. 2002. "The Relation Between Adverse Childhood Experiences and Adult Health: Turning Gold into Lead." *The Permanente Journal* 6:44-47.
- Felitti, Vincent. J., Robert F. Anda, Dale Nordenburg, David F. Williamson, Alison M. Spitz, Valerie J. Edwards, Mary P. Koss, and James S. Marks. 1998. "Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults." *American Journal of Preventive Medicine* 14:245-258.
- Ferraro, Kenneth F. and Patricia M. Morton. 2016. "What Do We mean by Accumulation? Advancing Conceptual Precision for a Core Idea in Gerontology." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* doi: 10.1093/geronb/gbv094.

- Ferraro, Kenneth F., Markus H. Schafer, and Lindsay R. Wilkinson. 2016. "Childhood Disadvantage and Health Problems in Middle and Later Life: Early Imprints on Physical Health?" *American Sociological Review* 81:107-133.
- Ferraro, Kenneth F. and Tetyana P. Shippee. 2009. "Aging and Cumulative Inequality: How Does Inequality Get Under the Skin?" *The Gerontologist* 49:333-343.
- Ferraro, Kenneth F., Tetyana P. Shippee, and Markus H. Schafer. 2009. "Cumulative Inequality Theory for Research on Aging and the Life Course." Pp. 413-433 in *Handbook of Theories of Aging*, edited by V. L. Bengtson, M. Silverstein, N. M. Putney, and D. Gans. New York, NY: Springer Publishing Company, LLC.
- Fisher, Helen L., Peter B. Jones, Paul Fearon, Thomas K. Craig, Paola Dazzan, Kevin Morgan, Gerard Hutchinson, Gillian A. Doody, Peter McGuffin, Julian Leff, Robin M. Murray, and Craig Morgan. 2010. "The Varying Impact of Type, Timing and Frequency of Exposure to Childhood Adversity on its Association with Adult Psychotic Disorder." *Psychological Medicine* 40:1967-1978.
- Ford, Earl S., Robert F. Anda, Valerie J. Edwards, Geraldine S. Perry, Guixiang Zhao, Chaoyang Li, and Janet B. Croft. 2011. "Adverse Childhood Experiences and Smoking Status in Five States." *Preventive Medicine* 53:188-193.
- Franceschi, Claudio, Massimiliano Bonafè, Silvana Valensin, Fabiola Olivieri, Maria de Luca, Enzo Ottaviani, and Giovanna de Benedictis. 2000. "Inflamm-aging: An Evolutionary Perspective on Immunosenescence." *Annals of the New York Academy of Sciences* 908:244-254.

- Friedman, Elliot M. and Pamela Herd. 2010. "Income, Education, and Inflammation: Differential Associations in a National Probability Sample (the MIDUS study)." *Psychosomatic Medicine* 72:290-300.
- Friedman, Esther M., Jennifer Karas Montez, Connor McDevitt Sheehan, Tara L. Guenewald, and Teresa E. Seeman. 2015. "Childhood Adversities and Adult Cardiometabolic Health: Does the Quantity, Timing, and Type of Adversity Matter?" *Journal of Aging and Health* 27:1311-1338.
- Fuller-Thomson, Esme and Sarah Brennenstuhl. 2009. "Making a Link between Childhood Physical Abuse and Cancer." *Cancer* 115:3341-3350.
- Gabriel, Sherine E., and Cynthia S. Crowson. 2012. "Risk Factors for Cardiovascular Disease in Rheumatoid Arthritis." *Current Opinion in Rheumatology* 24:171-176.
- Galobardes, Bruna, John W. Lynch, and George Davey Smith. 2004. "Childhood Socioeconomic Circumstances and Cause-Specific Mortality in Adulthood: Systematic Review and Interpretation." *Epidemiologic Review* 26:7-21.
- 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." *American Journal of Public Health* 96:826-832.
- Gillespie, Cathleen D., Charles Wigington, and Yuling Hong. 2013. "Coronary Heart Disease and Stroke Deaths-United States, 2009." *MMWR Surveillance Summary* 62:157-160.
- Goldman, Dana and James P. Smith. 2011. "The Increasing Value of Education to Health." *Social Science & Medicine* 72:1728-1737.

- Graham, Hilary, and Chris Power. 2010. "Equity, Risk, and the Life-Course: A Framework for Understanding and Tackling Health Inequalities." Pp. 63-78 in *Evidence-based Public Health: Effectiveness and Efficiency*, edited by A. Killoran and M. P. Kelly. Oxford University Press.
- Greenfield, Emily A. 2010. "Child Abuse as a Life-Course Social Determinant of Adult Health." *Maturitas* 66:51-55.
- Greenfield, Emily A. and Nadine Marks. 2009a. "Profiles of Physical and Psychological Violence in Childhood as a Risk Factor for Poorer Adult Health: Evidence from the 1995-2005 National Survey of Midlife in the United States." *Journal of Aging and Health* 21:943-966.
- Greenfield, Emily A. and Nadine Marks. 2009b. "Violence from Parents in Childhood and Obesity in Adulthood: Using Food in Response to Stress as a Mediator of Risk." *Social Science & Medicine* 68:791-798
- Guralnik, Jack M., Kenneth C. Land, Dan Blazer, Gerda G. Fillenbaum, and Laurence G. Branch. 1993. "Educational Status and Active Life Expectancy Among Older Blacks and Whites." *The New England Journal of Medicine* 329:110-116.
- Haas, Steven A. 2006. "Health Selection and the Process of Social Stratification: The Effect of Childhood Health on Socioeconomic Attainment." *Journal of Health and Social Behavior* 47:339-354.
- Haas, Steven A. 2007. "The Long-Term Effects of Poor Childhood Health: An Assessment and Application of Retrospective Reports." *Demography* 44:113-135.

- Hallqvist, Johan, John Lynch, Mel Bartley, Thierry Lang, and David Blane. 2004. "Can We Disentangle Life Course Processes of Accumulation, Critical Period and Social Mobility? An Analysis of Disadvantaged Socio-Economic Positions and Myocardial Infarction in the Stockholm Heart Epidemiology Program." *Social Science & Medicine* 58:1555-1562.
- Hamil-Luker, Jenifer and Angela M. O'Rand. 2007. "Gender Differences in the Link between Childhood Socioeconomic Status Conditions and Heart Attack Risk in Adulthood." *Demography* 44:137-158.
- Hart Research Associates. 2016. *Seeing Our Way to Living Long, Living Well in 21st Century America*. The Sightlines Project. Stanford, C.A.: Stanford Center on Longevity and TIME Magazine.
- Haug, Marie R. 1994. "Elderly Patients, Caregivers, and Physicians: Theory and Research on Health Care Triads." *Journal of Health and Social Behavior* 35:1-12.
- Hayward, Mark D. and Bridget K. Gorman. 2004. "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality." *Demography* 41:87-107.
- 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* 65:910-930.
- Health and Retirement Study (HRS). 2006. 2006 Biomarker Data. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI.

- Health and Retirement Study (HRS). 2015. Public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI.
- Heckman, James. 1979. "Sample Selection Bias as a Specification Error." *Econometrica* 47:153–161.
- Heisler, Michael, Jessica D. Faul, Rodney A. Hayward, Kenneth M. Langa, Caroline Blaum, and David Weir. 2007. "Mechanisms for Racial and Ethnic Disparities in Glycemic Control in Middle-Aged and Older Americans in the Health and Retirement Study." *Archives of Internal Medicine* 167:1853-1860.
- Herd, Pamela. 2006. "Do Functional Health Inequalities Decrease in Old Age? Educational Status and Functional Decline Among the 1931-1941 Birth Cohort." *Research on Aging* 28:375-392.
- Herd, Pamela, Amelia Karraker, and Elliot Friedman. 2012. "The Social Patterns of a Biological Risk Factor for Disease: Race, Gender, Socioeconomic Position, and C-Reactive Protein." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 67:503-513.
- Hertzman, Clyde. 1999. "The Biological Embedding of Early Experience and Its Effect on Health in Adulthood." *Annals of the New York Academy of Sciences* 896:85-95.
- Hertzman, Clyde. 2012. "Putting the Concept of Biological Embedding in Historical Perspective." *Proceedings of the National Academy of Science* 109:17160-17167.

- Hertzman, Clyde and Tom Boyce. 2010. "How Experience Gets Under the Skin to Create Gradients in Developmental Health." *Annual Review of Public Health* 31:329-347.
- Hurd, Michael D., Erik Meijer, Michael Moldoff, and Susann Rohwedder. 2014. "Improved Wealth Measures in the Health and Retirement Study: Asset Reconciliation and Cross-Wave Imputation." Santa Monica, CA: RAND Corporation, Center for the Study of Aging. (Forthcoming).
- Jain, Mukesh K. and Paul M. Ridker. 2005. "Anti-inflammatory Effects of Statins: Clinical Evidence and Basic Mechanisms." *Nature Reviews Drug Discovery* 4:977-987.
- Jefferis, Barbara J. M. H., Chris Power, Hilary Graham, and Orly Manor. 2004. "Effects of Childhood Socioeconomic Circumstances on Persistent Smoking." *American Journal of Public Health* 94:279-285.
- Johnson, Nan E. 2000. "The Racial Crossover in Comorbidity, Disability, and Mortality." *Demography* 37:267-283.
- Kamphuis, Carlijn B., Gavin Turrell, Katrina Giskes, Johan P. Mackenbach, and Frank J. van Lenthe. 2013. "Life Course Socioeconomic Conditions, Adulthood Risk Factors and Cardiovascular Mortality among Men and Women: A 17-year Follow up of the GLOBE Study." *International Journal of Cardiology* 168:2207-2213.
- Kanjilal, Sanjat, Edward W. Gregg, Yiling J. Cheng, Ping Zhang, David E. Nelson, George Mensah, and Gloria L. A. Beckles. 2006. "Socioeconomic Status and Trends in Disparities in 4 Major Risk Factors for Cardiovascular Disease Among US Adults, 1971-2002." *Archives of Internal Medicine* 166:2348-2355.

- Kaplan, George A. and Julian E. Keil. 1993. "Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature." *Circulation* 88:1973-1998.
- Kaplan, George A. and Jukka T. Salonen. 1990. "Socioeconomic Conditions in Childhood and Ischaemic Heart Disease during Middle Age." *BMJ* 301:1121-1123.
- Kelly-Irving, Michelle, Benoit Lepage, Dominique Dedieu, Mel Bartley, David Blane, Pascale Grosclaude, Thierry Lang, and Cyrille Delpierre. 2013. "Adverse Childhood Experiences and Premature All-Cause Mortality." *European Journal of Epidemiology* 28:721-734.
- Kemp, Blakelee R., Patricia M. Morton, and Kenneth F. Ferraro. 2015 "Does Childhood Misfortune Raise the Risk of Cancer Among Black, White, and Hispanic Adults?" Presented at the annual meeting of the Gerontological Society of America, November 19, Orlando, Florida.
- Kendall-Tackett, Kathleen. 2002. "The Health Effects of Childhood Abuse: Four Pathways by which Abuse can Influence Health." *Child Abuse & Neglect* 26:715-729.
- Kenis, Gunter, and Michael Maes. 2002. "Effects of Antidepressants on the Production of Cytokines." *The International Journal of Neuropsychopharmacology* 5:401-412.

- Kessler, Ronald C., Katie A. McLaughlin, Jennifer Grief Green, Michael J. Gruber, Nancy A. Sampson, Alan A. Zaslavsky, Sergio Aguilar-Gaxiola, Ali Obaid Alhamzawi, Jordi Alonso, Matthias Angermeyer, Corina Benjet, Evelyn Bromet, Somnath Chatterji, Giovanni de Girolamo, Koen Demyttenaere, John Fayyad, Silvia Florescu, Gilad Fal, Oye Gureje, Josep Maria Haro, Chi-yi Hu, Elie G. Karam, Norito Kawakami, Sing Lee, Jean-Pierre Lepine, Johan Ormel, José Posada-Villa, Rajesh Sagar, Adley Tsang T. Bedirhan Üstün, Svetlozar Vassilve, Maria Carmen Viana, and David R. Williams. 2010. "Childhood Adversities and Adult Psychopathology in the WHO World Mental Health Surveys." *The British Journal of Psychiatry* 197:78-385.
- Kim, Jinyoung, and Richard Miech. 2009. "The Black–White Difference in Age Trajectories of Functional Health over the Life Course." *Social Science & Medicine* 68:717-725.
- Korkeila, Jyrki, Jussi Vahtera, Katariina Korkeila, Mika Kivimäki, Markku Sumanen, Karoliina Koskenvuo, and Markku Koskenvuo. 2010. "Childhood Adversities as Predictors of Incident Coronary Heart Disease and Cerebrovascular Disease." *Heart* 96:298-303.
- Korn, Edward L. and Barry I. Graubard. 1995. "Analysis of Large Health Surveys: Accounting for the Sampling Design." *Journal of the Royal Statistical Society. Series A (Statistics in Society)* 158:263-295.

- Koster, Annemarie, Hans Bosma, Brenda W. J. H. Penninx, Anne B. Newman, Tamara B. Harris, Jacques Th. M. van Eijk, Gertrudis I. J. M. Kempen, Eleanor M. Simonsick, Karen C. Johnson, Ronica N. Rooks, Hilsa N. Ayonayon, Susan M. Rubin, and Stephen B. Kritchevsky. 2006. "Association of Inflammatory Markers with Socioeconomic Status." *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 61:284-290.
- Krabbe, Karen Suárez, Maria Pedersen, and Helle Bruunsgaard. 2004. "Inflammatory Mediators in the Elderly." *Experimental Gerontology* 39:687-699.
- Krall, Elizabeth A., Isabelle Valadian, Johanna T. Dwyer, and Jane Gardener. 1988. "Recall of Childhood Illness." *Journal of Clinical Epidemiology* 41:1059-1064.
- Laditka, Sarah B. and Douglas A. Wolf. 1998. "New Methods for Analyzing Active Life Expectancy." *Journal of Aging and Health* 10:214-241.
- Land, Kenneth C., Jack M. Guralnik, and Dan G. Blazer 1994. "Estimating Increment-Decrement Life Tables with Multiple Covariates from Panel Data: The Case of Active Life Expectancy." *Demography* 31:297-319.
- Lawlor, Debbie A., David Barry, Susan M. B. Morton, Heather Clark, Sally Macintyre, and David Leon. 2005. "Childhood Socioeconomic Position, Educational Attainment, and Adult Cardiovascular Risk Factors: The Aberdeen Children of the 1950s Cohort Study." *American Journal of Public Health* 95:1245-1251.
- Lee, Chioun and Helene R. White. 2012. "Effects of Childhood Maltreatment on Violent Injuries and Premature Death during Young Adulthood among Urban High-Risk Men." *Archives of Pediatrics & Adolescent Medicine* 166:814-820.

- Lehto, Soili M., Antti-Pekka Elomaa, Leo Niskanen, Karl-Heinz Herzig, Tommi Tolmunen, Heimo Viinamaki, Heli Koivumaa-Honkanen, Anne Huotari, Kiris Honkalampi, Minna Valknonen-Korhonen, Sanna Sinikallio, Heli Ruotsalainen, and Jukka Hintikka. 2012. "Serum Adipokine Levels in Adults with a History of Childhood Maltreatment." *Progress in Neuro-Psychopharmacology & Biological Psychiatry* 37:217-221.
- Link, Bruce G. and Jo Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior* 35:80-94.
- Lloyd-Jones, Donald M., Kiang Liu, Lu Tian, and Philip Greenland. 2006. "Narrative Review: Assessment of C-Reactive Protein in Risk Prediction for Cardiovascular Disease." *Annals of Internal Medicine* 145:35-42.
- Lubbock, Lindsey A., Anne Goh, Sadia Ali, James Ritchie, and Mary A. Whooley. 2005. "Relation of Low Socioeconomic Status to C-Reactive Protein in Patients with Coronary Heart Disease (from The Heart and Soul Study)." *The American Journal of Cardiology* 96:1506-1511.
- Luecken, Linda J. and Danielle S. Roubinov 2012. "Pathways to Lifespan Health Following Childhood Parental Death." *Social and Personality Psychology Compass* 6:243-257.
- Luo, Ye, and Linda J. Waite. 2005. "The Impact of Childhood and Adult SES on Physical, Mental, and Cognitive Well-Being in Later Life." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 60:S93-S101.
- Lynch, Scott M. 2003. "Cohort and Life Course Patterns in the Relationship Between Education and Health: A Hierarchical Approach." *Demography* 40:309-331.

- Manhapra, Ajay, John G. Canto, Viola Vaccarino, Lori Parsons, Catarina I. Kiefe, Hal V. Barron, William J. Rogers, W. Douglas Weaver, and Steven Borzak. 2004. "Relation of Age and Race with Hospital Death after Acute Myocardial Infarction." *American Heart Journal* 148:92-98.
- Manton, Kenneth G., Clifford H. Patrick, and Katrina W. Johnson. 1987. "Health Differentials between Blacks and Whites: Recent Trends in Mortality and Morbidity." *The Milbank Quarterly* 65:129-199.
- Marx, Karl. 1867. *Capital: Critique of Political Economy*. Germany: Verlag von Otto Meisner.
- Marketon, Jeanette I. Webster and Ronald Glaser. 2008. "Stress Hormones and Immune Function." *Cellular Immunology* 252:16-26.
- Matthews, Karen A., Yue-Fang Chang, Rebecca C. Thurston, and Joyce T. Bromberger. 2014. "Child Abuse is Related to Inflammation in Midlife Women: Role of Obesity." *Brain, Behavior, and Immunity* 36:29-34.
- Maxwell, Scott E., Michael Y. Lau, and George S. Howard. 2015. "Is Psychology Suffering from a Replication Crisis? What Does "Failure to Replicate" really Mean?" *American Psychologist* 70:487-498.
- McDade, Thomas W., James Burhop, and James Dohnal. 2004. "High-Sensitivity Enzyme Immunoassay for C-Reactive Protein in Dried Blood Spots." *Clinical Chemistry* 50:652-654.
- McEwen, Bruce S. 1998. "Protective and Damaging Effects of Stress Mediators." *The New England Journal of Medicine* 338:171-179.

- McKinlay, John B. 1996. "Some Contributions from the Social System to Gender Inequalities in Heart Disease." *Journal of Health and Social Behavior* 37:1-26.
- Meany, Michael J., Moshe Szyf, Jonathan R. and Seckl. 2007. "Epigenetic Mechanisms of Perinatal Programming of Hypothalamic-Pituitary-Adrenal Function and Health." *TRENDS in Molecular Medicine* 13:269-277.
- Melchior, Maria, Terrie E. Moffitt, Barry J. Milne, Richie Poulton, and Avshalom Caspi. 2007. "Why Do Children from Socioeconomically Disadvantaged Families Suffer from Poor Health When They Reach Adulthood? A Life-Course Study." *American Journal of Epidemiology* 166: 966-974.
- Mensah, George A. and David W. Brown. 2007. "An Overview of Cardiovascular Disease Burden in the United States." *Health Affairs* 26:38-48.
- Merton, Robert K. 1968. "The Matthew Effect in Science: The Reward and Communication Systems of Science are considered." *Science* 159:56-63.
- Miech, Richard A., Avshalom Caspi, Terrie E. Moffitt, Bradley R. Enter Wright, and Phil A. Silva. 1999. "Low Socioeconomic Status and Mental Disorders: A Longitudinal Study of Selection and Causation During Young Adulthood." *American Journal of Sociology* 104:1096-113.
- Miller, Gregory and Edith Chen. 2007. "Unfavorable Socioeconomic Conditions in Early Life Presage Expression of Proinflammatory Phenotype in Adolescence." *Psychosomatic Medicine* 69:402-409.

- Miller, Gregory E., Edith Chen, Alexandra K. Fok, Hope Walker, Alvin Lim, Erin Nicholls, Steve Cole, and Michael S. Kobo. 2009. "Low Early-life Social Class Leaves a Biological Residue Manifested by Decreased Glucocorticoid and Increased Proinflammatory Signaling." *Proceedings of the National Academy of Science* 106:14716-14721.
- Miller, Gregory E., Edith Chen, and Eric S. Zhou. 2007. "If it Goes Up, Must it Come Down? Chronic Stress and the Hypothalamic-Pituitary-Adrenal Axis in Humans." *Psychological Bulletin* 133:25-45.
- Mills, C. Wright. 1959. *The Sociological Imagination*. Oxford University Press.
- Mirowsky, John and Catherin E. Ross. 1998. "Education, Personal Control, Lifestyle and Health: A Human Capital Hypothesis." *Research on Aging* 20:415-449
- Montez, Jennifer Karas, Robert A. Hummer, Mark D. Hayward, Hyeyoung Woo, and Richard G. Rogers. 2011. "Trends in the Educational Gradient of U.S. Adult Mortality From 1986 Through 2006 by Race, Gender, and Age Group." *Research on Aging* 33:145-171.
- Morey, Jennifer N. and Suzanne C. Segerstrom. 2015. "Physiological Consequences: Early Hardship and Health Across the Lifespan." Pp. 151-176 in *Traumatic Stress and Long-Term Recovery*, edited by K. E. Cherry. Switzerland: Springer International Publishing.
- Morton, Patricia M., Sarah A. Mustillo, and Kenneth F. Ferraro. 2014. "Does Childhood Misfortune Increase Acute Myocardial Infarction Risk?" *Social Science & Medicine* 104:133-41.

- Morton, Patricia M., Markus H. Schafer, and Kenneth F. Ferraro. 2012. "Does Childhood Misfortune Increase Cancer Risk in Adulthood?" *Journal of Aging and Health* 24:948-984.
- Morton, Patricia M., Nicholas A. Turiano, Daniel K. Mroczek, and Kenneth F. Ferraro. 2016. "Childhood Misfortune, Personality, and Heart Attack: Does Personality Mediate Risk of Myocardial Infarction?" *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* doi: 10.1093/geronb/gbw021.
- Mutchler, Jan E. and Jeffrey A. Burr. 2011. "Race, Ethnicity, and Aging." In Pp. 83-101 in *Handbook of Sociology of Aging*, edited by R. A. Settersten and J. L. Angel. New York: Springer.
- Muthén, Bengt. 2011. "Applications of Causally Defined Direct and Indirect Effects in Mediation Analysis using SEM in Mplus." *Manuscript submitted for publication.*
- Muthén, Linda K. and Bengt O. Muthén. 2007. *Mplus User's Guide*, 6th Ed. Los Angeles, CA: Muthén & Muthén.
- Naess, Øyvind, Bjørgulf Claussen, Dag S. Thelle, and George Davey Smith. 2004. "Cumulative Deprivation and Cause Specific Mortality: A Census Based Study of Life Course Influences Over Three Decades." *Journal of Epidemiology and Community Health* 58:599–603.
- National Academies of Sciences, Engineering, and Medicine. 2016. *A Framework for Educating Health Professional to Address the Social Determinants of Health*. Washington, DC: The National Academies Press.

- Non, Amy L., Marissa Rewak, Ichiro Kawachi, Stephen E. Gilman, Eric B. Loucks, Allison A. Appleton, Jorge C. Roman, Stephen L. Buka, and Laura D. Kubzansky. 2014. "Childhood Social Disadvantage, Cardiometabolic Risk, and Chronic Disease in Adulthood." *American Journal of Epidemiology* 180:263-271.
- O'Brien, Kymberlee M. 2012. "Healthy, Wealthy, Wise? Psychosocial Factors Influencing the Socioeconomic Status–Health Gradient." *Journal of Health Psychology* 17:1142-1151.
- Ofstedal, Mary B., and David R. Weir. 2011. "Recruitment and Retention of Minority Participants in the Health and Retirement Study." *The Gerontologist* 51:S8-S20.
- O'Rand, Angela M. 1996. "The Precious and the Precocious: Understanding Cumulative Disadvantage and Cumulative Advantage over the Life Course." *The Gerontologist* 36:230-238.
- O'Rand, Angela and Jenifer Hamil-Luker. 2005. "Processes of Cumulative Adversity: Childhood Disadvantage and Increased Risk of Heart Attack Across the Life Course." *The Journals of Gerontology Series B: Psychological and Sciences and Social Sciences* 60:S117-S124.
- O'Rand, Angela, Jenifer Hamil-Luker, and Cheryl Elman. 2009. "Childhood Adversity, Educational Trajectories, and Self-Reported Health in Later Life Among US Women and Men at the Turn of the Century." *Zeitschrift für Erziehungswissenschaft* 12:409-436.
- Palloni, Alberto. 2006. "Reproducing Inequalities: Luck, Wallets, and the Enduring Effects of Childhood Health." *Demography* 43:587-615.

- Pappas, Gregory, Susan Queen, Wilbur Hadden, and Gail Fisher. 1993. "The Increasing Disparity in Mortality between Socioeconomic Groups in the United States, 1960 and 1986." *New England Journal of Medicine* 329:103-109.
- Pearlin, Leonard I. 1989. "The Sociological Study of Stress." *Journal of Health and Social Behavior* 30:241-256.
- Pearlin, Leonard I. 2010. "The Life Course and the Stress Process: Some Conceptual Comparison." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 65:S207-S215.
- Pearlin, Leonard I., Elizabeth G. Menaghan, Morton A. Lieberman, and Joseph T. Mullen. 1981. "The Stress Process." *Journal of Health and Social Behavior* 22:337-356.
- Pearson, Thomas A., George A. Mensah, R. Wayne Alexander, Jeffrey L. Anderson, Richard O. Cannon III, Michael Criqui, Yazid Y. Faddl, Stephen P. Fortmann, Yuling Hong, Gary L. Myers, Nader Rifai, Sidney C. Smith, Jr, Kathryn Taubert, Russell P. Tracy and Frank Vinicor. 2003. "Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: A Statement for Healthcare Professionals from the Centers for Disease Control and Prevention and the American Heart Association." *Circulation* 107:499-511.
- Pepys, Mark B. and Gideon M. Hirschfield. 2003. "C-Reactive Protein: A Critical Update." *Journal of Clinical Investigation* 111:1805-1812.
- Pollack, Craig Evan, Sekai Chideya, Catherine Cubbin, Brie Williams, Mercedes Dekker, and Paul Braveman. 2007. "Should Health Studies Measure Wealth? A Systematic Review." *American Journal of Preventive Medicine* 33:250-264.

- Power, Chris, Elina Hyppönen, and George Davey Smith. 2005. "Socioeconomic Position in Childhood and Early Adult Life and Risk of Mortality: A Prospective Study of the Mothers of the 1958 British Cohort." *American Journal of Public Health* 95:1396-1402.
- Power Chris, Snehal M. Pinto Pereira, and Leah Li. 2015. "Childhood Maltreatment and BMI Trajectories to Mid-Adult Life: Follow-Up to Age 50y in a British Birth Cohort." *PLoS ONE* 10:1-16.
- Preston, Samuel H. and Paul Taubman. 1994. "Socioeconomic Differences in Adult Mortality and Health Status." Pp. 279-318 in *Demography of Aging*, edited by L. G. Martin and S. H. Preston. Washington, DC: National Academy Press.
- Ridker, Paul M, Charles H. Hennekens, Julie E. Buring, and Nader Rifai. 2000a. "C-Reactive Protein and other Markers of Inflammation in the Prediction of Cardiovascular Disease in Women." *New England Journal of Medicine* 342:836-843.
- Ridker, Paul M, Nader Rifai, Meir J. Stampfer, and Charles H. Hennekens. 2000b. "Plasma Concentration of Interleukin-6 and the Risk of Future Myocardial Infarction among Apparently Healthy Men." *Circulation* 101:1767-1772.
- Riley, Matilda White. 1973. "Aging and Cohort succession: Interpretations and Misinterpretations." *Public Opinion Quarterly* 37:35-49.
- Riley, Matilda White. 1987. "On the Significance of Age in Sociology." *American Sociological Review* 52:1-14.

- Riley, E H, R J Wright, H J Jun, E N Hibert, J W Rich-Edwards. 2010. "Hypertension in Adult Survivors of Child Abuse: Observations from the Nurses' Health Study II." *Journal of Epidemiology and Community Health* 64:413-418.
- Risman, Barbara J. 2004. "Gender as a Social Structure: Theory Wrestling with Activism." *Gender & Society* 18:429-450.
- Robert, Stephanie A. 1999. "Socioeconomic Position and Health: The Independent Contribution of Community Socioeconomic Context." *Annual Review of Sociology* 25:489-516.
- Rooks, Cherie, Emir Veledar, Jack Goldberg, Douglas Bremner, and Viola Vaccarino. 2012. "Early Trauma and Inflammation: Role of Familial Factors in a Study of Twins." *Psychosomatic Medicine* 74:146-152.
- Rosenstock, Irwin M., Victor J. Strecher, and Marshall H. Becker. 1988. "Social Learning Theory and the Health Belief Model." *Health Education & Behavior* 15:175-183.
- Ross, Catherine E and Chia-ling Wu. 1995. "The Links Between Education and Health." *American Sociological Review* 60:719-745.
- Schilling, Elizabeth A., Robert H. Aseltine, and Susan Gore. 2008. "The Impact of Cumulative Adversity on Young Adult Mental Health: Measures, Models, and Interpretations." *Social Science & Medicine* 66:1140-1151.
- Schafer, Markus H., and Kenneth F. Ferraro. 2011. "Distal and Variably Proximal Causes: Education, Obesity, and Health." *Social Science and Medicine* 73:1340-48.

- Schury, Katharina and Iris-Tatjana Kolassa. 2012. "Biological Memory of Childhood Maltreatment: Current Knowledge and Recommendations for Future Research." *Annals of the New York Academy of Sciences* 1262:93-100.
- Sesso, Howard., Julie E. Buring, Nader Rifai, Gavin Blake, Michael Gaziano, and Paul Ridker. 2003. "C-Reactive Protein and the Risk of Developing Hypertension." *Journal of American Medical Association* 290:2945–51.
- Sewell, William H., Archibald O. Haller and Alejandro Portes. 1969. "The Educational and Early Occupational Attainment Process." *American Sociological Review* 34:82-92.
- Shippee Tetyana Pylypiv, Lindsay R. Wilkinson, and Kenneth F. Ferraro. 2012. "Accumulated Financial Strain and Women's Health over Three Decades." *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 67:585-594.
- Simons, Daniel J. 2014. "The Value of Direct Replication." *Perspectives on Psychological Science* 9:76-80.
- Singer, Burton and Carol D. Ryff. 1999. "Hierarchies of Life Histories and Associated Health Risks." *Annals of the New York Academy of Sciences* 896:96–115.
- Singh-Manoux, Archana, Jane E. Ferrie, Tarani Chandola, and Michael Marmot. 2004. "Socioeconomic Trajectories across the Life Course and Health Outcomes in Midlife: Evidence for the accumulation hypothesis?" *International Journal of Epidemiology* 33:1072-1079.

- Slopen, Natalie, Karestan C. Koenen, and Laura D. Kubzansky. 2012. "Childhood Adversity and Immune and Inflammatory Biomarkers associated with Cardiovascular Risk in Youth: A Systematic Review." *Brain, Behavior, and Immunity* 26:239-250.
- Slopen, Natalie, Tene T. Lewis, Tara L. Gruenewald, Mahasin S. Mujahid, Carol D. Ryff, Michelle A. Albert, and David R. Williams. 2010. "Early Life Adversity and Inflammation in African Americans and Whites in the Midlife in the United States Survey." *Psychosomatic Medicine* 72:694-701.
- Slopen, Natalie, Katie A. McLaughlin, Erin C. Dunn, and Karestan Koenen. 2013. "Childhood Adversity and Cell-mediated Immunity in Young Adulthood: Does Type and Timing Matter?" *Brain, Behavior, and Immunity* 28:63-71.
- Smith, James. P. 2009. "Reconstructing Childhood Health Histories." *Demography* 46:387-403.
- Smith, Frank and Steve Iliffe. 1997. "Exercise Prescription in Primary Care." *British Journal of General Practice* 47 272-273.
- Sonnega, Amanda, Jessica D. Faul, Mary Beth Ofstedal, Kenneth M. Langa, John W. R. Phillips, and David R. Weir. 2014. "Cohort Profile: The Health and Retirement Study (HRS)." *International Journal of Epidemiology* 43:576-585.
- Spilerman, Seymour. 2000. "Wealth and Stratification Processes." *Annual Review of Sociology* 26:497-524.
- Springer, Kristen W. 2009. "Childhood Physical Abuse and Midlife Physical Health: Testing Multi-Pathway Life Course Model." *Social Science & Medicine* 69:138-146.

- Steelman, Lala Carr and Brian Powell. 1991. "Sponsoring the Next Generation: Parental Willingness to Pay for Higher Education." *American Journal of Sociology* 96:1505-1529.
- Stein, David J., Kate Scott, Josep M. Haro Abad, Sergio Aguilar-Gaxiola, Jordi Alonso, Matthias Angermeyer, Koen Demeyttere, Giovanni de Girolamo, Noburo Iwata, José Posada-Villa, Viviane Kovess, Carmen Lara, Johan Ormel, Ronald C. Kessler, and Michael Von Koff. 2010. "Early Childhood Adversity and Later Hypertension: Data from the World Mental Health Survey." *Annals of Clinical Psychiatry* 22:19-28.
- Steptoe, A. and M. Marmot. 2002. "The Role of Psychobiological Pathways in Socio-economic Inequalities in Cardiovascular Disease Risk." *European Heart Journal* 23:13-25.
- Stringhini, Silvia, G. David Batty, Pascal Bovet, Martin J. Shipley, Michael G. Marmot, Meena Kumari, Adam G. Tabak, and Mika Kivimäki. 2013. "Association of Lifecourse Socioeconomic Status with Chronic Inflammation and Type 2 Diabetes Risk: The Whitehall II Prospective Cohort study." *PLoS Medicine* 10:e1001479.
- Sumanen, Markku, Markku Koskenvuo, Lauri Sillanmäki, and Kari Mattila. 2005. "Childhood Adversities Experienced by Working-aged Coronary Heart Disease Patients." *Journal of Psychosomatic Research* 59:331-335.
- Surtees, Paul G. and Nicholas W. J. Wainwright. 2007. "The Shackles of Misfortune: Social Adversity Assessment and Representation in a Chronic-Disease Epidemiological Setting." *Social Science & Medicine* 64:95-111.

- Surtees, Paul, Nicholas Wainwright, Nicholas Day, Carol Brayne, Robert Luben, and Kay-Tee Khaw. 2003. "Adverse Experience in Childhood as a Developmental Risk Factor for Altered Immune Status in Adulthood." *International Journal of Behavioral Medicine* 3:251-268.
- Tarullo, Amanda R., and Megan R. Gunnar. 2006. "Child Maltreatment and the Developing HPS Axis." *Hormones and Behavior* 50:632-639.
- Taylor, Shelley E., Barbara J. Lehman, Catarina I. Kiefe, and Teresa E. Seeman. 2006. "Relationship of Early Life Stress and Psychological Functioning to Adult C-Reactive Protein in the Coronary Artery Risk Development in Young Adults Study." *Biological Psychiatry* 60:819-824.
- Taylor, Shelley E., Baldwin M. Way, and Teresa E. Seeman. 2011. "Early Adversity and Adult Health Outcomes." *Development and Psychopathology* 23:939-954.
- Thomas, Claudia, Elina Hyppönen, and Chris Power. 2008. "Obesity and Type 2 Diabetes Risk in Midadult life: The Role of Childhood Adversity." *Pediatrics* 121:e1240-e1249.
- Thornberry, Terence P., Timothy O. Ireland, and Carolyn A. Smith. 2001. "The Importance of Timing: The Varying Impact of Childhood and Adolescent Maltreatment on Multiple Problem Outcomes." *Development and Psychopathology* 13:957-979.
- Trickett, Penelope K., Jennie G. Noll, Elizabeth J. Susman, Chad E. Shenk, and Frank W. Putnam. 2010. "Attenuation of Cortisol Across Development for Victims of Sexual Abuse." *Development and Psychopathology* 22:165-167.

- Trotta, A., R. M. Murray, and H. L. Fisher. 2015. "The Impact of Childhood Adversity on the Persistence of Psychotic Symptoms: A Systematic Review and Meta-Analysis." *Psychological Medicine* 45:2481-2498.
- Turner, R. Jay, Blair Wheaton, and Donald A. Lloyd. 1995. "The Epidemiology of Social Stress." *American Sociological Review* 60:104-125.
- Tyrka, Audrey R., Lawrence H. Price, Hung-The Kao, Barbara Porton, Sarah A. Marsella, and Linda L. Carpenter. 2010. "Childhood Maltreatment and Telomere Shortening: Preliminary Support for an Effect of Early Stress on Cellular Aging." *Biological Psychiatry* 67:531-534.
- U.S. Bureau of Labor Statistics. 2010. "2010 Standard Occupational Classification User Guide." Retrieved November 11, 2014.
(http://www.bls.gov/soc/soc_2010_class_and_coding_structure.pdf).
- van de Mheen, H, K Stronks, CWN Looman, and JP Mackenbach. 1998. "Does Childhood Socioeconomic Status Influence Adult Health through Behavioural Factors?" *International Journal of Epidemiology* 27:431-437.
- van der Vegt, Esther J. M., Jan van der Ende, Clemens Kirschbaum, Frank C. Verhulst, and Henning Tiemeier. 2009. "Early Neglect and Abuse Predict Diurnal Cortisol Patterns in Adults: A Study of International Adoptees." *Psychoneuroendocrinology* 34:660-669.
- Volpato, Stefano, Jack M. Guralnik, Luigi Ferrucci, Jennifer Balfour, Paulo Chaves, Linda P. Fried, and Tamara B. Harris. 2001. "Cardiovascular Disease, Interleukin-6, and Risk of Mortality in Older Women: The Women's Health and Aging Study." *Circulation* 103:947-953.

Vuolo, Michael, Kenneth F. Ferraro, Patricia M. Morton, and Ting-Ying Yang. 2014.

“Why Do Older People Change Their Ratings of Childhood Health?”

Demography 51:1999-2023.

Walker, Renee E., Christopher R. Keane, and Jessica G. Burke. 2010. “Disparities and

Access to Healthy Food in the United States: A Review of Food Deserts

Literature.” *Health & Place* 16:876-884.

Walpole, MaryBeth. 2003. “Socioeconomic Status and College: How SES affects

College Experiences and Outcomes.” *The Review of Higher Education* 27:45-73.

Wannamethee, Goya S., Peter H. Whincup, Gerald Shaper, and Mary Walker. 1996.

“Influence of Fathers' Social Class on Cardiovascular Disease in Middle-Aged

Men.” *The Lancet* 348:1259-1263.

Webster, Jeanette I., Leonardo Tonelli, and Esther M. Sternberg. 2002. “Neuroendocrine

Regulation of Immunity.” *Annual Review of Immunology* 20:125–63.

Williams, David R. 2012. “Miles to Go before We Sleep: Racial Inequities in Health.”

Journal of Health and Social Behavior 53:279-295.

Williams, David R. and Michelle Sternthal. 2010. "Understanding Racial-Ethnic

Disparities in Health Sociological Contributions." *Journal of Health and Social*

Behavior 51:S15-S27.

Williamson, DF, TJ Thompson, RF Anda, WH Dietz, and V Felitti. 2002. “Body Weight

and Obesity in Adults and Self-Reported Abuse in Childhood.” *International*

Journal of Obesity 26:1075-1082.

World Heart Federation. 2016. "Cardiovascular Disease Risk Factors." Retrieved March 26, 2016. (<http://www.world-heart-federation.org/cardiovascular-health/cardiovascular-disease-risk-factors/>).

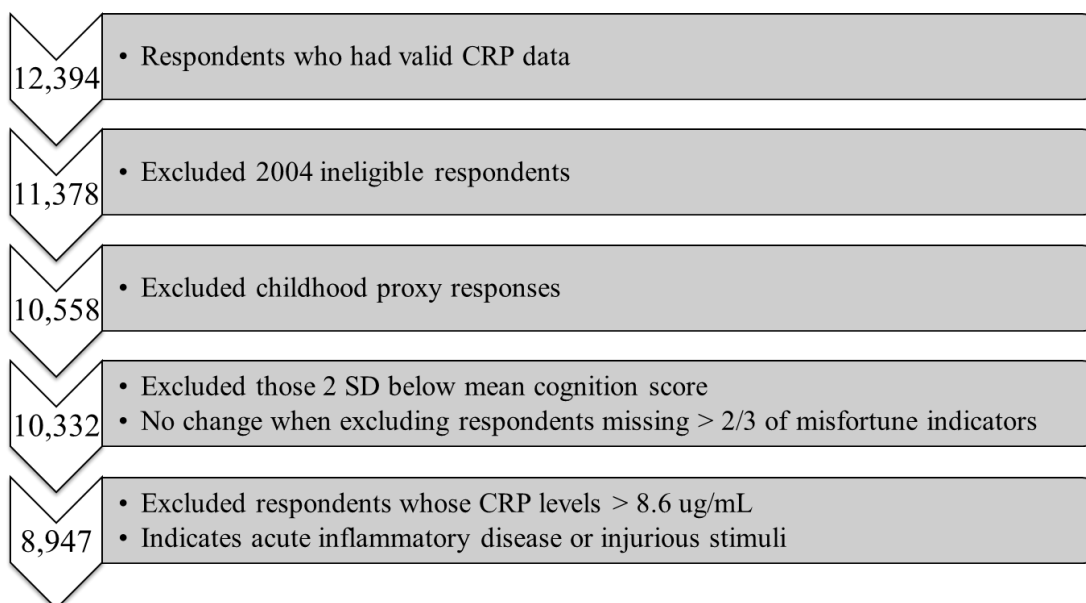
Ziol-Guest, Kathleen M., Greg J. Duncan, and Ariel Kalil. 2009. "Early Childhood Poverty and Adult Body Mass Index." *American Journal of Public Health* 99:527-532.

Zeugmann, Sue, Arnim Quante, Louchka Popova- Zeugmann, Wolfgang Kossler, Isabella Heuser, and Ion Anghelescu 2012. "Pathways Linking Early Life Stress, Metabolic Syndrome, and the Inflammatory Marker Fibrinogen in Depressed Inpatients." *Psychistria Danubina* 24:57-65.

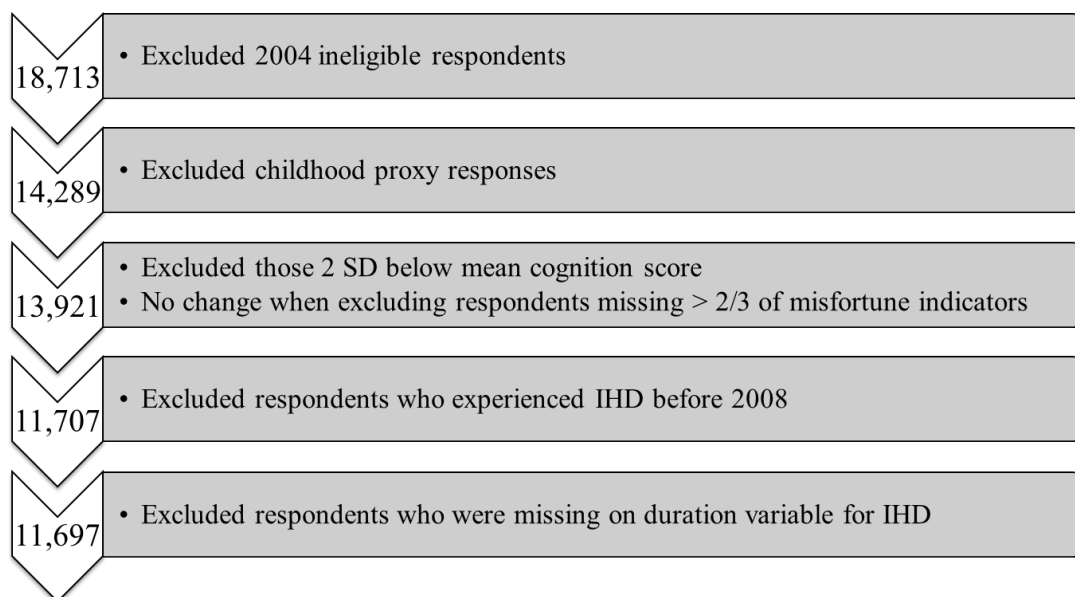
Zweifler, Richard M., Patrick D. Lyden, Barbara Taft, Nancy Kelly, and John F. Rothrock. 1995. "Impact of Race and Ethnicity on Ischemic Stroke: The University of California at San Diego Stroke Data Bank." *Stroke* 26:245-248.

APPENDICES

Appendix A Inclusion Criteria for Ch. 2 Analytic Sample



Appendix B Inclusion Criteria for Ch. 3 Analytic Sample



VITA

VITA

Patricia M. Morton

Department of Sociology
 Purdue University
 Stone Hall
 700 W. State Street
 West Lafayette, IN 47907-2059

Center on Aging and the Life Course
 Purdue University
 Hanley Hall
 1202 W. State Street
 West Lafayette, IN 47907-2055

Position

Current Ph.D. Candidate, Sociology and Gerontology, Purdue University

September, 2016 CEHI Postdoctoral Research Fellow, Statistics and Sociology, Rice University

September, 2016 Affiliate, Rice University Academy of Fellows, Rice University

Education

2016 Dual-Title Ph.D. in Sociology and Gerontology
 Purdue University, West Lafayette, Indiana
Dissertation: "The Things They Carried: The Biological Residue of Childhood Misfortune"
Committee: Kenneth F. Ferraro (chair), Elliot M. Friedman, Sarah A. Mustillo, J. Jill Suitor

2012 M.S. in Sociology
 Purdue University, West Lafayette, Indiana

2009 B.S. in Applied Sociology, Minor in Forensic Psychology
Summa Cum Laude
 Texas State University-San Marcos, San Marcos, Texas

Areas of Specialization

Aging and the Life Course, Health Disparities, Medical Sociology, Quantitative Methodology

Publications

Morton, Patricia M., Nicholas A. Turiano, Daniel K. Mroczek, and Kenneth F. Ferraro. 2016. "Childhood Misfortune, Personality, and Heart Attack: Does Personality Mediate Risk of Myocardial Infarction?" *The Journals of Gerontology Series B: Psychological Sciences* Advance online publication. doi: 10.1093/geronb/gbw021

Ferraro, Kenneth F. and **Patricia M. Morton**. 2016. "What Do We mean by Accumulation? Advancing Conceptual Precision for a Core Idea in Gerontology." *The Journals of Gerontology Series B: Social Sciences* Advance online publication. doi: 10.1093/geronb/gbv094

*2014 Best Paper Award, Theoretical Developments in Social Gerontology, Gerontological Society of America

Morton, Patricia M., Sarah A. Mustillo, and Kenneth F. Ferraro. 2014. "Does Childhood Misfortune Increase Acute Myocardial Infarction Risk?" *Social Science & Medicine* 104:133-41.

Schafer, Markus H., **Patricia M. Morton**, and Kenneth F. Ferraro. 2014. "Child Maltreatment and Adult Health in a National Sample: Heterogeneous Relational Contexts, Divergent Effects?" *Child Abuse & Neglect* 38:395-406.

Vuolo, Michael, Kenneth F. Ferraro, **Patricia M. Morton**, and Ting-Ying Yang. 2014. "Why Do Older People Change Their Ratings of Childhood Health?" *Demography* 51:1999-2023.

Morton, Patricia M. and Kenneth F. Ferraro. 2012. "Race and Health Disparities." *Encyclopedia of Health, Illness, Behavior, and Society*, edited by William C. Cockerham, Robert Dingwall, and Stella Quah. Hoboken, NJ: Wiley-Blackwell.

Morton, Patricia M., Markus H. Schafer, and Kenneth F. Ferraro. 2012. "Does Childhood Misfortune Increase Cancer Risk in Adulthood?" *Journal of Aging and Health* 24:948-84.

*Article featured in national and international news outlets.

Conference Presentations

Kemp, Blakelee R., **Patricia M. Morton**, and Kenneth F. Ferraro. 2015 "Does Childhood Misfortune Raise the Risk of Cancer Among Black, White, and Hispanic Adults?" Presented at the Annual Meeting of the Gerontological Society of America, Orlando, Florida.

Morton, Patricia M. 2015. "Race, Gender, and Class: Structural Explanations for Differences in Functional Disability Risk." Presented at the Annual Meeting of the American Sociological Association, Chicago, Illinois.

Ferraro, Kenneth F. and **Patricia M. Morton**. 2014. "Antecedents of Life Course Resilience After Major Childhood Maltreatment." Presented at Annual Meeting of the Gerontological Society of America, Washington, D.C.

Morton, Patricia M., and Kenneth F. Ferraro. 2014. "What Do We mean by Accumulation? Advancing Conceptual Precision in Defining Accumulation." Presented at Annual Meeting of the Gerontological Society of America, Washington, D.C.

*Awarded Best Paper for Theoretical Developments in Social Gerontology by the Gerontological Society of America

Morton, Patricia M., and Kenneth F. Ferraro. 2013. "Does Childhood Misfortune Increase the Likelihood of Psychotropic Drug Use in Adulthood?" Presented at the Annual Meeting of the American Sociological Association, New York City, New York.

Morton, Patricia M., Nicholas A. Turiano, Daniel K. Mroczek, and Kenneth F. Ferraro. 2013 "Childhood Misfortune, Personality, and Acute Myocardial Infarction: Does Personality Mediate Risk of Acute Myocardial Infarction?" Presented at the 20th International Association of Gerontology and Geriatrics World Congress of Gerontology and Geriatrics. Seoul, S. Korea.

Morton, Patricia M., and Kenneth F. Ferraro. 2012 "Does Childhood Misfortune Increase Acute Myocardial Infarction Risk?" Presented at the Annual Meeting of the American Sociological Association, Denver, Colorado.

Schafer, Markus H., **Patricia M. Morton**, and Kenneth F. Ferraro. 2012. "Child Maltreatment and Adult Health in a National Sample: Heterogeneous Relational Contexts, Divergent Effects?" Presented at Annual Meeting of the Gerontological Society of America, San Diego, California.

Morton, Patricia M., Markus H. Schafer, and Kenneth F. Ferraro. 2011. "Does Childhood Misfortune Increase Cancer Risk in Adulthood?" Presented at the Annual Meeting of the American Sociological Association, Las Vegas, Nevada.

Morton, Patricia M., Markus H. Schafer, and Kenneth F. Ferraro. 2011. "Long-term Effects of Childhood Misfortune on Adult Cancer Risk? Examining Socioeconomic Disadvantage, Life Events, and Adverse Experiences." Invited plenary speaker for International Conference on Successful Aging, Yonsei University, Seoul, S. Korea.

Research Experience

- 2014-present Research Assistant, "Childhood Misfortune and Adult Health among Black, White, and Hispanic Americans" Kenneth F. Ferraro, Principal Investigator, Purdue University, Funded by the National Institute on Aging, (R01 AG043544).
Participated in data analysis and manuscript preparation.
- 2010-2013 Research Assistant, "Enduring Effects of Early Adversity on Adult Health?" Kenneth F. Ferraro, Principal Investigator, Purdue University, Funded by the National Institute on Aging, (R01 AG033541).
Participated in data analysis and manuscript preparation.
- 2009-2010 Research Project Coordinator, "Parents Learning to be Decision Makers for Children with Profound Neurodevelopmental Disabilities", Nhung T, Tran, Principal Investigator, University of Texas Health Science Center at San Antonio.
Participated in data collection and management, data analysis, and Manuscript preparation.

Experience with the following statistical software packages: MPLUS, SAS, SPSS, STATA, R.

Teaching Experience

- 2013-2015 Teaching Assistant, Graduate Statistics for the Social Sciences, Department of Sociology, Purdue University
Graduate statistics courses included Longitudinal Data Analysis, Regression Analysis, and Quantitative Methods
- 2013-2014 Instructor, Introduction to Statistics for the Social Sciences, Department of Sociology, Purdue University
- 2013-2013 Instructor, Social Problems, Department of Sociology, Purdue University
- 2011-2012 Laboratory Instructor, Introduction to Statistics for the Social Sciences, Department of Sociology, Purdue University
- 2008-2010 Laboratory Instructor, Statistics for the Social Sciences, Department of

Sociology, Texas State University-San Marcos

2008-2010 Statistics Tutor, Statistics for the Social Sciences, Applied Data Analysis, Department of Sociology, Texas State University-San Marcos

2005-2006 Math Tutor, College Mathematics, Northwest Vista College, San Antonio, Texas

Guest Lectures

2015 “Applying Principles of Effective Writing.” Writing Seminar, Department of Veterinary Clinical Sciences, Purdue University, February.

2015 “Preparing for Preliminary Dissertation Exams.” Professional Development Seminar, Department of Sociology, Purdue University, February.

2014 “Early Origins of Adult Health.” Medical Sociology, Department of Sociology, Purdue University, February.

2013 “Hypothesis Testing.” Graduate Statistics for the Social Sciences, Department of Sociology, Purdue University, April.

2012 “Bootstrapping Basics.” Statistics for the Social Sciences, Department of Sociology, Purdue University, October.

Honors and Grants Awarded

2015-2016 Bilsland Dissertation Fellowship, Purdue University, \$18,000

2015 Funding awarded to attend RAND Summer Institute, RAND Corporation

2014-2015 Robert L. Eichhorn Fellowship, Purdue University, \$5,000

2014 Best Paper Award, Theoretical Developments in Social Gerontology, Gerontological Society of America, \$1,000

2014 Center on Aging and the Life Course Summer Research Award, Purdue University, \$200

2010-2011 Lynn Fellowship, Purdue University, \$15,750

2009 Clarence Schultz Scholarship, Texas State University-San Marcos, \$500

- 2008-2009 Liberal Arts Academic Excellence Award, Texas State University-San Marcos
- 2003 United Way Military Volunteer Family of the Year

Additional Training

- 2015 Mini-Medical School for Social Scientists and the Demography, Economics, Psychology, and Epidemiology of Aging Workshop. 22nd RAND Summer Institute, RAND Corporation.
- 2014 Examining the Health and Retirement Study Workshop. Summer Institute in Survey Research Techniques, University of Michigan.
- 2013 Research Integrity Workshop. Robert E. Pruitt. Graduate School, Purdue University.
- 2012 Techniques for Giving a Great Seminar. J. Paul Robinson. Center for Instructional Excellence, Purdue University.
- 2010 Seeing the Forest through the Trees: Dealing with Missing Data in Quantitative Analyses. Sarah A. Mustillo. Department of Human Development and Family Studies, Purdue University.

Professional Service

Mentor, Midwest Crossroads Alliance for Graduate Education and the Professoriate (AGEP), 2011, Kadari Taylor-Watson

Reviewed manuscripts for *American Journal of Preventive Medicine*, *American Sociological Review*, *Journal of Aging and Health*, *Journal of Health and Social Behavior*, *Nicotine & Tobacco Research*, and *Social Science & Medicine*

Reviewed abstracts for the *Gerontological Society of America Annual Meeting*

Secretary, Sociology Graduate Organization, Purdue University, 2013-2014.

Professional Affiliations

Alpha Kappa Delta Sociology Honor Society
2008-2009 *Chapter President*, XI Chapter of Texas

American Sociological Association
Section Membership:
Aging and the Life Course
Medical Sociology

Gerontological Society of America

Sociologists for Women in Society

Volunteer Experience

2005	Relief aid for Hurricane Rita evacuees, Red Cross, San Antonio, Texas
2000-2005	Summer counselor for children with disabilities, Camp C.A.M.P., Center Point, Texas
2003	Aid for orphans with disabilities, Hermano Pedro, Antigua, Guatemala