ON THE INTERSECTION OF RACE, STRESS, AND HEALTH

by

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DEDICATION

This Dissertation is dedicated to all of the people who are desperately searching for their purpose in this big crazy world. It is dedicated to all young people who need a role model to help them realize that they can be an agent of change in our world. It is finally dedicated to everyone who has ever supported and believed in me, while simultaneously putting up with my unconventional personality.

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CHAPTER ONE

Introduction

The complex web of causation associated with racial and ethnic health disparities in the United States is well documented. This web of causation has been studied extensively over the last 50 years, and yet racial and ethnic health disparities have remained a seemingly insurmountable public health challenge. There have been significant strides in this area of research however; an appreciation of fundamental and structural causes has burgeoned (Link and Phelan 1995), a call to adopt a life course perspective and an accompanying desire to collect more longitudinal as opposed to cross-sectional data (Alwin and Wray 2005), and a growing consideration of contextual influence (Israel, Schulz et al. 2001).

Another important ever-evolving area of research concerning health is the development of the stress process as a causal pathway in which health disparities are continually maintained. The term "stress" has been used to describe the ways in which the body copes with psychosocial, environmental, and physical challenges (McEwen and Seeman 1999). While it is reasonable to assume that stress can impact the health of individuals, it is unclear as to exactly how this may happen. Stress is a concept that consists of a variety of constructs, which may be measured on an individual or group level. On an individual level, it is important to understand how

experiences with stress and stressors impact physical and mental health functioning, as well as overall daily living experiences. Likewise, it is important to consider the broad array of social and psychological conditions that combine over time to create stress (Pearlin, Menaghan et al. 1981). For these reasons, the study of stress is particularly relevant to the study of racial and ethnic health disparities. However, there remain many unanswered questions. For instance, what exactly are the sources of stress that impact the health of American adults? Do experiences of stress differ between racial groups (e.g. Black and White Americans)? Is it safe to contend that the life experiences between Black and White American adults are not comparable? How do these experiences of stress impact the observed rates of racial and ethnic health disparities? Currently in the 21st century there is a contention that racism is not a relevant or widespread problem in the United States of America. Unfortunately, this may be a premature ideal. The realities of American society certainly differ from the hopes of the utopian American members' vision. The need to continually acknowledge the role race (i.e. color of one's skin) plays in racial and ethnic health disparities is crucial in eliminating the gap in health.

In many public health reports, the role of socioeconomic status in racial and ethnic health disparities is either confounded with the race, or is deemed "significant" while the impact of race on health is diminished. This approach to studying disparities has severely limited an understanding of how Black Americans, and other minorities, are adjusting to the contextual challenges faced in America. Considering the way Black Americans are treated through interactions with the media or the court and law

enforcement arenas, it is important to acknowledge that navigating through the vicissitudes of American life can ultimately impact health.

The aim of this research is to consider stress as a potential determinant of health. The experience and impact of different forms of stress among African Americans and White Americans will be examined throughout two separate research papers. Stress is the focus of this dissertation because it has been shown to greatly impact the mental and physical health of individuals, and it simultaneously demands consideration of the social context in which individuals live (Dutton and Levine 1989; Almeida, Neupert et al. 2005; Williams and Jackson 2005).

The challenge of eliminating racial and ethnic health disparities has been a daunting task. As mentioned, these disparities are highly multi-factorial in nature, and researchers have been unable to elucidate the exact mechanisms, and equally appropriate solutions, to adequately address these problems, specifically in the United States. In 2003, African Americans had the highest age-adjusted all-causes mortality rate of all races/ethnicities (NCHS 2006). In addition, African Americans had the highest age-adjusted death rate for heart disease, cancer, diabetes, and HIV/AIDS. In 2000, the homicide rate was almost six times greater for African Americans than for whites (Williams and Jackson 2005). Compared to the year 1950, by 2000, African Americans had a 30 percent higher death rate from both cancer and heart disease, compared to their white counterparts (Williams and Jackson 2005). In 2002, blacks suffered 40.5 percent more deaths than would be expected, if they had experienced

equal mortality rates of their white counterparts(Satcher, Fryer et al. 2005). Over time, the infant mortality rate for both blacks and whites has declined; however, in 1960 a black baby born in the United States was 1.6 times as likely to die before their first birthday than his or her white counterpart, but is 2.5 times as likely in 2000 (Williams and Collins 2004).

Economically, African Americans have continued to struggle as compared to their white counterparts in this post Civil Rights era. For example, in 1978, black households had earned 59 cents for every dollar earned by whites, had an unemployment rate that was 1.9 times higher, and a poverty rate that 3.5 times higher. In 1996, there was still very little economic progress. As compared to Whites, African Americans had an unemployment rate that was twice as high, a poverty rate that was 2.5 times higher, and median household earnings that were still 59 cents for every dollar earned by Whites (Williams and Collins 2004).

The study of the stress process provides an opportunity to further examine the mechanisms that contribute to racial and ethnic health disparities. Socioeconomic disparities in health have often been explained by exposure and vulnerability to stress and stressors (House and Williams 2000). The purpose of the proposed dissertation research will be to critically investigate the possible mechanisms of the effects of stress. Previous research has examined other mechanisms such as John Henryism (James 1994), Weathering (Geronimus 1992), discrimination (Williams 1999),

psychological distress (Ensel and Lin 1991), and self-regulatory behaviors (Jackson and Knight 2006).

The theoretical basis for this dissertation is as follows: Considering the history of African Americans, rooted in slavery and postslavery segregation (Satcher, Fryer et al. 2005), and the current disadvantages African Americans face: low social status, discrimination, and residential segregation (Mechanic 2005), it is reasonable to question the impact race has on the exposure to chronic stress and stressors. Next, people of disadvantaged social status tend to report elevated levels of stress and may be more vulnerable to the effects of stress (Williams and Jackson 2005). Exposure to chronic stress is associated with altered physiological functioning, which in turn may increase risks for a wide range of mental and physical health conditions (Israel 2002; Epel, Blackburn et al. 2004; Hogue and Bremner 2005; Rich-Edwards 2005; Williams and Jackson 2005). Since it is well known that stress may greatly impact a person's mental health status, considering the physical health effects of stress allows researchers to further elucidate the indirect ways in which stress operates.

Outline of Dissertation

The overall goal of this body of research is to compare and contrast the prevalence of stress and its antecedents, specifically considering the differentials by race. Before this task is undertaken, the next chapter will offer an overview of the intersection between race, health, and stress and discuss key theories and explanations of racial and ethnic health disparities. Thus, in Chapter Two, I explore the theoretical

significance of the stress process in existing explanations for racial and ethnic health disparities. First, I describe the significance of race in America. I describe the current state of racial and ethnic health and economic disparities. In doing so, I describe the rates of disease and illness among White Americans, and female and male Black Americans. Next, I attempt to synthesize and outline the vast field of stress research. I offer definitions of various forms of stress in an attempt to show how stress pervades many facets of daily life. In a literature review I then describe the relationship stress has with health. I outline how stress is related to mental, biological and physical health. The concluding section of Chapter Two highlights the more notable explanations for racial and ethnic health disparities, while elaborating on how these explanations are all undergirded by aspects and mechanisms of the stress process. I argue that in many of the theoretical frameworks used to better understand health disparities, stress is a fundamental feature of the framework. The frameworks considered include Fundamental Causes of Disease (Link and Phelan 1995), Neighborhood Exposure and Residential Segregation (Massey, Condran et al. 1987; Diez Roux, Merkin et al. 2001; Williams and Collins 2001; Massey 2004), Income Inequality (Marmot 1999; Lynch, Smith et al. 2001; House 2002), Racism and Racial Discrimination (Krieger and Sidney 1996; Williams 1999; Williams and Williams-Morris 2000), John Henryism (James 1984; James 1987; James 1994), Allostatic Load (McEwen and Seeman 1999; McEwen, Lasley et al. 2002; McEwen 2004), and the Weathering Hypothesis (Geronimus 1992; Geronimus, Hicken et al. 2006). The goal of this paper is to propose that a closer look at the stress process might offer a better understanding of racial and ethnic health disparities.

In Chapter Three I rely on data from the Americans' Changing Lives study (House, Lantz et al. 2005) to explore the rates of reported stress between White and Black Americans over a 16 year time period. Reports of financial, parental, and marital chronic stress and negative lifetime events are compared between the two racial groups. The analyses are limited to Black and White respondents who also participated at in least two of the four waves of data collection, in order to estimate longitudinal rates of stress. I use Hierarchical Linear Modeling (HLM) to compare the levels of chronic stress over time between the two groups. Selected control variables, such as age, gender, income, employment, and education are included in the multivariate models. The goal of this paper is to get a basic idea of whether or not stress levels vary between Black and White American adults. An advantage of this research is that the data used are longitudinal in order to get a better understanding of stress levels across a significant segment of the life course.

In Chapter Four, I explore the extent to which reported stress levels influence physical health outcomes differentially between Black and White adults. That is, I investigate the moderating role of race between stress and health. The selected physical health outcomes are functional health and self-rated health. The three measures of financial, parental, and marital chronic stress and negative life events are used, as in Chapter Three. Stress has been found to be associated with various health outcomes, and this study aims to look at the health of individuals over time, simultaneously considering the impact of chronic stress and negative life events. This

study also uses data from Americans' Changing Lives and control variables are age, income, education and employment.

Americans' Changing Lives Study

Americans' Changing Lives (ACL) is a long-term four-wave cohort longitudinal study that was conducted by the University of Michigan Survey Research Center and began in 1986 (see House, Lepkowski et al. 1994). The ACL studywas designed to understand the role of a broad range ofpsychosocial and behavioral factors in health functioning in middle and later life.The second data collection time was in 1989, and then was repeated in 1994, and 2001-2003.

ACL is a stratified, multistage area probability sample of noninstitutionalized adults aged 25 and older living in the coterminous United States. Two groups were oversampled: African Americans and adults aged 60 and older. The Americans' Changing Lives Dataset has been previously used for a wide variety of research analyses in peer-reviewed journal publications.

Due to the longitudinal nature of the data, multivariate multilevel modeling will be utilized to analyze the ACL dataset. Specifically, descriptive analyses will be conducted in SPSS Version 15.0.1.1 and hierarchical linear modeling (HLM) will be conducted in HLM6. There are a variety of justifications and advantages of using a HLM approach as opposed to using general linear modeling applications such as ANOVA, ANCOVA, or MANOVA. First, for the reason that the data include

repeated observations on the same person at different time points, the data is hierarchical in structure, with measurement occasions nested within individuals (O'Connell and McCoach 2004). Given this, HLM has the ability to deal with missing or staggered occasions for some individuals. Next, time does not have to be treated as a fixed effect, and there is flexibility to treat time as a random effect, in the case that time varies across individuals. The use of HLM allows for the characterization of response patterns or the growth of individuals and groups over time and allows for the investigation of whether person- or group-level characteristics may be related to variations in growth patterns (O'Connell and McCoach 2004). There are two aspects of the sampling design that must be considered while creating the multilevel models. First, the initial 3617 observations are arranged in 45 strata, and 2 clusters within each stratum. Secondly, all analyses will be adjusted using a final composite weight that consisted of five factors: household screening factor, housing unit selection weight, respondent selection factor, nonresponse adjustment weight, and poststratification weight.

Conclusion

Racial and ethnic health disparities are maintained under a very complicated web of causation. There is an essential need to understand people's characteristics, social influences, exposures to and interpretation of stressors, and psychological and physical manifestations of stress. Thus, an enhanced review of the stress process may offer an opportunity to better understand racial and ethnic health disparities. It is quite possible that Black Americans encounter unique experiences and sources of

stress that may be more detrimental to health, than as compared to their White counterparts. This research seeks to look at the psychosocial elements in which individuals live, to determine the effects of fundamental social conditions, which may operate through the human stress process. To better understand racial and ethnic health disparities, it is necessary to embrace, rather than avoid, the nature of implicit and explicit race relations in current American society. It is hopeful that this research adds to the knowledge base, a more clear view of the effects of stress by race, and how health is impacted under stressful living conditions.

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CHAPTER TWO

Implicit and Explicit, Missing and Present Linkages: The significance of stress when considering explanations for racial and ethnic health disparities.

Introduction

A common explanation for socioeconomic disparities in health in America has been differential vulnerability and exposure to stress and stressors (Almeida, Neupert et al. 2005). It is known that individuals in lower socioeconomic strata are prone to report higher rates of stress and stressors, and when exposed, suffer more severe consequences than their higher status peers. The establishment of this association has shed light on the need of more equitable distribution of resources as well as policies to alleviate socioeconomic disparities. However, in many instances, these conclusions have also obscured the role stress and strains may play in racial and ethnic health disparities. While it is clear that socioeconomic disparities and racial and ethnic health disparities may operate in similar manners, they are not a proxy for one another. The persistence of racial differences in various aspects of health, even after controlling for socioeconomic status (SES), suggests that race and SES may still have independent effects (Kahn and Fazio 2005). It has been surmised by many researchers that race and class tend to confound one another when analyzing health outcomes and health disparities. The underestimation or "explaining away" of race in research continues to miscalculate the health effects that originate from American racism. Race and SES are two *related* but not interchangeable systems of inequality

(Williams 2005). Thus, it is important to disentangle the independent contribution of each factor.

The current paper will review the role of stress and health, including key theories and explanations of racial and ethnic health disparities. We define "health disparities" as differences in morbidity, mortality, and access to health care among population groups defined by factors such as socioeconomic status, gender, and race or ethnicity (Dressler, Oths et al. 2005). This paper will specifically examine the health disparities between Black and White Americans.

This chapter will present a literature review of racial and ethnic health disparities in America, and how they may relate to stress. The depth of research on these topics is immense, so the discussion will be at times succinct. The overall aim of this chapter is to discuss how the stress process is implicated in the understanding of racial and ethnic health disparities. People who are exposed to similar social and economic stressors, who share similar ascribed social roles and positions, who come from similar contextual environments, will experience similar types and levels of stress (Pearlin 1989). Therefore, environmental, psychological, and physiological aspects of stress must be considered in understanding health disparities between Black and White Americans (Brown 2004). As will be seen, many of these aspects of stress are implicitly and explicitly discussed in explanations of racial and ethnic health disparities.

First, a review of the historical underpinnings of race in American culture, coupled with a description of racial and ethnic economic and health disparities will ensue. Part Two will outline the vast field of stress research, while the concluding section, Part Three, will highlight the more notable explanations for racial and ethnic health disparities, while elaborating on how these explanations are all undergirded by aspects and mechanisms of the stress process.

Review of Relevant Literature

Part I. Race and Racial and Ethnic Economic and Health Disparities

Race in America

In the United States, race represents the "confluence of biological factors and geographic origins, culture, economic, political and legal factors, as well as racism" (Williams 1997). Thus, race has largely been a socially constructed concept that was created by institutional and ideological forces, with the intended and unintended consequence of defining and maintaining racial superiority (Hayward, Miles et al. 2000). In the mid-1960s, landmark civil rights legislation was passed to legally ensure that America become a more egalitarian society. While there has been progress in a lot of areas, such as workplace equality, desegregation, and voting rights, complete racial equality has not yet been achieved. The social, political, and economic meanings of race, force some members of racial groups to occupy hierarchical, and often undesired, social roles and social statuses. The resulting social stratification is under girded in American society by the legacy and current manifestations of racism. Racism has become an important societal force that shaped and reshaped many social

structures (Williams 1997). As Sherman James (1994) comments, African Americans have been engaged in an unrelenting struggle to "free themselves from pervasive and deeply entrenched systems of social and economic oppression" (p.167). This struggle persists.

Racial and Ethnic Economic Disparities

The socio-political effect of the social stratification of races in America has had wide-ranging effects and consequences in American life. One major area of impact is economic status. Since the mid-1600s, for about fifteen generations, the exploitation and oppression of African Americans has redistributed income and wealth earned by black labor to generations of white Americans, thus leaving many Blacks impoverished, (Feagin 2004). With the arrival of the Civil Rights Act of 1964, there have been fluctuations in the wage differentials and employment rates of White and Black Americans (Heckman 1989; Heckman 1989; Couch and Daly 2004). Since 1968, African American males have seen increases in levels of educational attainment and occupational distributions (Couch and Daly 2004). Despite this, African Americans have continued to experience economic struggles relative to their white counterparts. For example, in 1978, black households had earned 59 cents for every dollar earned by whites, had an unemployment rate that was 1.9 times higher, and a poverty rate that 3.5 times higher. In 1996, these differences largely persisted and by some metrics worsened. As compared to Whites, African Americans had an unemployment rate that was twice as high, a poverty rate that was 2.5 times higher, and median household earnings that were still 59 cents for every dollar earned by

Whites (Williams and Collins 2004). In 2004, the median family income of blacks ages 30 to 39 ($35,000) was only 58 percent that of white families in the same age group ($60,000) (Isaacs 2007). The median net worth of white households is about ten times that of black households (Feagin 2004). Further, in one intergenerational income research study, while only 17 percent of whites born to the bottom decile of family income remained there as adults, for blacks the figure was 42 percent (Hertz 2004). The economic status of Black Americans has been impacted by the erosion of the labor force participation of black men, high rates of unemployment and underemployment, incapacitation due to poor health, and the disproportionate incarceration of black males (Wallace 1985; Lane, Keefe et al. 2004). The lack of socioeconomic resources and dearth of accumulated wealth among African Americans is closely linked to their continuing limited access to powerful and influential organizations. In terms of absolute, relative, and integrated mobility measures, white children have substantially more upward mobility than black children of similar incomes (Isaacs 2007). The far-reaching effects of these economic adversities traverse the life course for many Black Americans and eventually impact the social and health related spheres of their lives.

Racial and Ethnic Physical Health Disparities

White and African Americans

The health of the American public has never been as favorable as it is now (Isaacs and Schroeder 2004). Yet for years, there has been a wide gap in health between White and Black populations, and it is inextricably linked to the history of race and

racism in the United States (Williams and Collins 2004)Williams & Collins, 2004). African Americans have experienced varying levels of social, economic, and political exclusion that have resulted in poorer health since their arrival as slaves in North America several hundred years ago (Fiscella and Williams 2004). Many public health practitioners, researchers, policy makers and lay advocates have developed a consensus that these health inequalities are a pressing social problem (Braun 2006). Geronimus and Thompson (2004) noted that "racial inequalities in health are the predictable manifestation of linkages among: 1) Prevailing racialized ideologies; 2) Political and economic structural inequalities that follow; 3) The personal and social coping mechanisms adopted to manage dominant ideologies and structural inequalities; and 4) The physiological effects of the coping efforts." These linkages converge to present a rather grim picture of African Americans in the 21st century.

At certain age intervals over the life course, Blacks have worse health outcomes as compared to their White counterparts. For example, in 2001, the infant mortality rate per 1000 births was 5.7 for infants of white mothers, but 13.3 for infants of black mothers; a 2.3-fold excess death rate (Hogue and Bremner 2005). Further, preterm birth is a leading cause of infant death and morbidity; compared with White infants, Black infants are 1.6 times as likely to be born preterm and 2.9 times as likely to be born very preterm (Lu and Chen 2004). Once born, the life expectancy at birth for Whites in 2000 was 77.6 years, which was about 6 years longer than the 71.9 years for Blacks (Lynch, 2008). Further, the life expectancy at birth for black males in the

U.S. (68.8 years) is lower than that for males in Iran (69.0), Colombia (69.3), and Sri Lanka (71.5) (Gadson 2006).

It has been noted that racial differences in morbidity and mortality are often more pronounced in the young and middle adult age brackets (Geronimus, Bound et al. 1999). At the age of 15 years, Blacks face 1.5 times the odds of being hypertensive than Whites, but at age 44 Blacks were more than three times as likely to be hypertensive than Whites (Geronimus, Andersen et al. 1991). Further, the probability that a 15-year old African American mother or father in a poor urban setting live to see their child's 20th birthday is about the same probability that a typical American 30-year old White mother or father does (Geronimus, Bound et al. 1999). In poverty-stricken areas such as Harlem or the south side of Chicago, many African American girls who reach their 16th birthdays do not live to see their 65th birthdays. As compared to their White peers, these same young ladies have a 60% less probability of surviving to the age of 85. In one study comparing cancer survival rates, inner city Harlem residents fared far worse as compared to the rest of the country (Freeman, 1989). The overall death rate of African Americans in the United States is currently equivalent to that of Whites in America 30 years ago. Specifically, the age-adjusted all-cause mortality for all blacks in 1998 was 6.9 per 100,000, which was equal to the white value for 1969 (a 29-year lag). The age-adjusted all-cause mortality rate for blacks in 1965 revealed a lag of 27 years compared with the white rate (Levine 2001).

The causes of premature deaths in the African American population are attributable to a wide array of disorders, illnesses, and diseases. Across various health indicators, there are large disparities that do not favor African Americans. The rates for the Black non-Hispanic population are more than 20 times the best group rate for congenital syphilis and gonorrhea. Although African Americans and Hispanics represented only 26 percent of the U.S. population in 2001, they accounted for 66 percent of adult AIDS cases and 82 percent of pediatric AIDS cases reported in the first half of that year (NCHS 2002). Among U. S. adults in 2007, the age-sex adjusted prevalence of diagnosed diabetes was highest among Blacks (11.4%) than White (6.2%) and Hispanic (11.2%) persons (NCHS 2008). In deaths due to heart disease, African Americans had a rate of 321.3 per 100,000 persons, a rate that was higher than any other racial/ethnic group. The rate for Whites was 245.6, American Indian/Alaskan Natives (137.4), and Asian /Pacific Islanders (137.4) (Mays, Cochran et al. 2007).

African American Males

Deaths due to circulatory diseases alone account for more than one-third of all deaths to young adult through middle aged Black men in poverty stricken urban areas and about one fourth of the excess deaths they experience each year relative to similar aged white men in the United States (Geronimus 1998). Black middle-aged men suffer disproportionately from hypertension, diabetes, stroke, arthritis, foot and leg problems, depression, and vision and hearing difficulties (Hayward and Heron 1999). Black males aged 51 to 61, have a higher prevalence of hypertension, stroke,

diabetes, kidney and bladder problems, and stomach ulcers (Hayward, Miles et al. 2000). While the rates of disease and mortality are striking between White and Black populations, the within-group differences are stark as well. For many African American, they are stuck in a double bind. Not only are they a disadvantaged group nationwide, but also those who live in poverty stricken areas suffer worse than their peers. For example, in two U.S. poor urban areas, African American men faced an average of over 11 years of life lost between the ages of 15 and 65, this rate was almost twice the number of lost for blacks nationwide and almost four times the number for Whites (Geronimus 1998).

African American Females

While the state of Black men is rather sobering, the status of Black women is also concerning. Major health problems for African American women include several cancers, diabetes, vision loss, tuberculosis, and sexually transmitted diseases (STDs), including HIV/AIDS (Department of Health and Human Services (DHHS) 2007). The leading causes of death for African American women are heart disease, cancer, stroke, diabetes, and kidney disease. African American women are about twice as likely to have diabetes as whites of the same age and they are more likely to have other serious health problems caused by diabetes. Among women, about two out of three new HIV cases are African American. High rates of other STDs, including chlamydia, gonorrhea, and syphilis are also prevalent in African American women (DHHS 2007). African American women are less likely to receive health care; and when they do receive care, they are more likely to receive it late. In 2004, African

American mothers were twice as likely to have late or no prenatal care compared to white mothers. Black women are also more likely to have more advanced cancer at the time of diagnosis than their White peers (Williams 2002). African American women are more than twice as likely to die of cervical cancer as are white women and are more likely to die of breast cancer than are women of any other racial or ethnic group (NCHS 2002). This seething health profile of African Americans appears to suggest that there are adverse and systematic forces that continue to undermine efforts for optimal health for all members of the American society.

Part II. Stress and Health

Definitions of Stress

As early as 1914, a physiologist by the name of Walter Cannon introduced the term stress into medical vocabulary. In the 1930s, the Hungarian physiologist Hans Selye described the connection between stress and health (McEwen 2002). Ever since, however, there has been considerable debate regarding how to exactly define the term stress (Brown 1974; Young 1980; Wheaton 1994). There is general agreement that the term stress refers to a response of the organism to conditions that, either consciously or unconsciously, are experienced as noxious (Pearlin 1989). Stress is a complex phenomenon that encompasses exposure to psychosocial, environmental, and physical changes and the body's responses to those experiences. Stress has also been conceptualized from a stress process perspective- either as a stimulus a person must confront or as the response to the stimulus. A stressor is the stimulus that causes stress, whereas, stress is what Wheaton (1994) refers to as the "processing state of the

organism", and distress is the behavioral response to the stimuli. A conceptualization that may be more relevant in current society is drawn from the pivotal work of Leonard Pearlin and colleagues (Pearlin, Aneshensel et al. 1999; Pearlin, Schieman et al. 2005). Pearlin et al. (2005) suggests that stressors may also be thought of as "…the dogged hardships, demands, conflicts, and frustrations…that may disrupt the continuities" a people's lives (p.206). Further, there is a socially patterned distribution of components or domains of the stress process: stressors, stress mediators, and stress outcomes (Pearlin 1989). These patterns provide clues that the potentially stressful experiences of individuals, and the ways in which they are affected by these experiences may originate in the social context in which they are embedded. There are also three major types of stress: life events, chronic strains, and daily hassles (Thoits 1983; Kessler, Price et al. 1985; Aneshensel 1992). Life events are described as discrete generally one-time occurrences (e.g. wedding or death of a loved one) (Turner, Wheaton et al. 1995). Chronic strains, however, are different from life events in that these occurrences are more persistent and prolonged over time, such as poverty or discrimination (Brown 1978; Pearlin, Menaghan et al. 1981; Wheaton 1990; Turner and Avison 2003). The third major type of stress, daily hassles are characterized as small bothersome nuisances of living from day to day such as traffic and commuting, everyday concerns of work, or a malfunctioning appliance (Serido, Almeida et al. 2004). The next section will discuss each type of stress in greater detail.

Stressful Life Events

The early work of Seyle (1956) and Cannon (1932) demonstrated that experimental exposure to stressful stimuli produces an adaptive physiological response in both animals and humans. The work of Seyle (1956) provided an important theoretical foundation for events research. Life events are typically defined as experiences that cause the individual to substantially readjust his or her behavioral patterns (Thoits 1982). Thus stress and change become synonymous. Stressful life events can be separated into two broad categories: recent stressors and remote stressors. Clearly, recent stressors refer to an occurrence or happening in the recent past (1-3 years) and remote stressors refer to events that have occurred over the lifetime, previous to the recent past. Dohrenwend (1973) describes two distinct conceptions of the characteristic that makes life events stressful. The first is that the events are negative or undesirable in quality, and the second is that the occurrence of these events usually evoke or is associated with some adaptive or coping behavior. When considering life events as stressors, Pearlin (1989) comments that it is important for the stress research field to establish that events are in fact events, and not simply proxy indicators of chronic stressors strains in a person's life. It is also equally important, when studying life events, to be sensitive to events whose occurrence varies with a person's life circumstances and key social and economic statuses, such as age, gender, race and ethnicity, and occupational status (Pearlin 1989).

Chronic Strains

Chronic strains, also known as chronic stress, involve the relatively enduring problems, conflicts, and threats that many people face in their daily lives (Pearlin 1989; Kessler, Mickelson et al. 1999). Chronic strains are described as life difficulties that occur over a long period of time and there are two major perspectives on this type of stress. The first, attributed by Pearlin (1981), asserts that the importance and exigencies of roles are an important source of stress. Pearlin (1989) argues that a focus on role strains, when considering chronic stress, can reinforce the links between the contexts that shape a person's well-being, activities, relationships, and experiences. According to (Pearlin 1989), role strain can be divided into five types. The first, role overload, a condition that exists when demands on energy and stamina exceeds the individual's capacities. The second, interpersonal conflicts within the role sets, assumes many forms, but they all involve problems and difficulties that arise among those who interact with each other in sets of complementary roles (i.e. parent/child, husband/wife). Next, inter-role conflict entails the incompatible demands of occupying multiple roles. Fourth, role captivity, exists when a person is fulfilling an unwanted role; whether or not the role is conflict-free or onerous. The final role strain type is role restructuring, which involves the reformation or reorganization of roles that may be long-standing or entrenched. These alterations may occur as the result of the aging process or external circumstances that force readjustments and alterations, despite the possible existence of long-standing patterns or traditions.

The second perspective views chronic strains as difficulties in life as opposed to stressful life events (Brown 1978). This conceptualization juxtaposes stressful life events that occur at a discrete point in time, from life difficulties, which are viewed as persistent, ongoing problems. Not all strains are directly linked to a role; other chronic strains may include: a spouse's infidelity, a son or daughter's drug or alcohol problem, poverty, or a chronic health condition (Avison and Gotlib 1994). Pearlin (1989) warns that chronic strain is not only role strain, but also 'ambient strain' distinct from role strain. Ambient strains cut across roles and envelop people. Pearlin (1983) suggests the convergence of stressful life events and chronic strain may have 'a synergistic effect which may change the meaning of existing strains and generate or magnify existing ones'. He notes that there are at least three ways in which events and strains interface with one another: 1) events lead to chronic strains; 2) chronic strains lead to events; and 3) strains and events provide meaning contexts for each other (Pearlin 1989). Thus, just as stressful chronic problems may be created by life events, stressful events can be triggered by persistent problems.

Daily Hassles

Daily hassles are small, minor everyday annoyances such as traffic or a flat tire that may elicit stress (Dohrenwend, Dohrenwend et al. 1984). Daily hassles can also refer to small, more unexpected events that might disrupt daily life (Serido, Almeida et al. 2004). Hassles can also be thought of as events, thoughts or situations which, when they occur produce negative feelings such as annoyance, irritation, worry, or frustration, and make a person aware that their goals will be more difficult or

impossible to achieve (O'Connor, Jones et al. 2008). Nonetheless, generally speaking, the emotional effects of daily hassles and of the hassle itself are expected to moderate, decrease, or disappear in 24 to 48 hours. The research on hassles is not as extensive as the previously discussed areas, but there has been more attention paid to the stressful nature of small events (Thoits 1995).

Socioeconomic Position and Stress

A significant body of research documents that socioeconomic status and the role of structural arrangements can influence stress (Langner 1963; Brown 1978; Kessler and Neighbors 1986; Turner and Noh 1988; Ulbrich, Warheit et al. 1989; McLeod and Kessler 1990; Krause 1997). Recent developments in sociological theory (i.e. symbolic interactionism) (Thoits 1983; Thoits 1985), network theory (Fischer 1977), and organizational theory (Litwak, Messeri et al. 1989), and labeling theory (Link 1982; Link 1987) are expanding stress theoretical frameworks that link relevant social factors and mechanisms and social stress.

Differential differences in socioeconomic position may be fundamental to stress (Link and Phelan 1995; Williams 1999; Schulz, Williams et al. 2000). Often individuals must traverse complicated social arrangements of occupation, education, health care and other formal institutions. When these experiences become threatening they may result in stress (Pearlin 1983). Role strain in family life is also linked to structure and may emerge due to financial strain or other hardship (Wheaton 1990; Krause and Borawski-Clark 1994). Research indicates that socioeconomic position has a strong

relationship with constructs such as mastery (Pearlin 1985; Thoits 1999). Gallo and Matthews (1999) found that low socioeconomic status was predictive of negative cognitive emotion and cardiovascular disease. While the exact relationship between socioeconomic position and health are still unknown, it can reveal interesting data about stress, which is often obscured by focusing only on proximal causes.

The relationship between Stress and Health

Exposures to chronic stress are considered the most toxic because they are most likely to result in long lasting or permanent in the behavioral, physiological, emotional responses that influence susceptibility to and course of disease (Cohen, Janicki-Deverts et al. 2007). There is a vast array of research that has shown that reports of stressful life events, chronic, acute, and other forms of stress are related to a wide variety of mental, biological, and physical health outcomes, and the next section discusses each in turn.

Stress and Mental/Psychological Health Outcomes

The study of stress bloomed as people thought that inquiries into individual stress levels would further develop research of mental health morbidity. Associations between psychological stress have long been established with depression. Stressful life events have been linked to major depressive disorder as well as to depressive symptoms (Hammen 2005). In the 3-6 months preceding depression, 50% to 80% of depressed persons experienced a major life event as compared to 20% to 30% of nondepressed individuals evaluated during the same time period (Monroe and Simons

1991). In a study of black and white adolescent girls, negative life events continue to predict depressive symptoms years after their occurrence, and there were some events that were more predictive than others (Franko, Striegel-Moore et al. 2004). Also, although the black adolescents reported more losses, financial hardships, and other adversities, than their white counterparts, they were no more likely to experience future depressive symptomatology.

In a review paper of predictive studies from 1980 to 2001, Tennant (2002) reported that stressors are associated with greater initial severity of depressive symptoms both in adult patients, 'non-patient' community samples, and in adolescent depression. The recent studies Tennant (2002) reviewed, also demonstrated a consistent significant relation between stressors and depression. Acute stressors were more likely to be associated with a briefer illness episode with an 'at risk period' of a month or so; chronic stressors (lasting 6 months or more) had a much longer at risk period, caused longer episodes and contributed to a greater depression relapse/recurrence rate. In university students, recent stressful life events predicted anxiety while daily hassles predicted depression. Tennant (2002) also differentiated childhood and adult stressors. In relation to earlier childhood experiences the evidence was somewhat inconsistent. Childhood sexual and physical abuse were both independently associated with adult depression in women, while childhood stressors were found to 'sensitize' women to stressor induced depression in adult life in another study. Similarly in elderly depressives, both recent events and significant events earlier in life separately predicted depression. Finally, Tennant (2002) reviewed two articles on

early childhood 'loss' experiences and concluded that parental divorce predicted adult depression but parental death in childhood did not. Thus it seems clear that significant ongoing childhood stressors are indeed predictors of much later adult depression. In suicidal adolescents, separations, poor parental supports, or disciplinary conflicts further contributed to comorbid alcohol abuse. In another study of young adults aged 18 to 23 years, high levels of lifetime exposure to adversity were found to be associated with the onset of depressive and anxiety disorders (Turner and Lloyd 2004).

Stress and Physiological/Biological Outcomes

Chronic stress has been repeatedly implicated in altering the biological functioning of humans. A stressful stimulus results in the activation of several physiological pathways including the autonomic nervous system, and two endocrine response systems: the hypothalamic-pituitary-adrenal axis (HPAA) and the sympathetic-adrenal-medullary (SAM) system (Kajantie and Phillips 2006; Cohen, Janicki-Deverts et al. 2007). It is generally accepted that stress-induced activity of the HPAA and the release of glucocorticoids (i.e. cortisol) from the adrenal cortex account at least partially for detrimental health effects of chronic stress (Pruessner and Malla 2008). In laboratory rats, chronic stress has been shown to enhance or the vulnerability of the brain to the neurotoxic effects of psychostimulants (Matuszewich and Yamamoto 2004). Kiecolt-Glaser, Preacher et al.(2003) explored increases in the proinflammatory cytokine IL-6 between caregivers of Alzheimer's patients and noncaregivers. IL-6 is a key inflammatory cytokine that appears to enhance the

morbidity and mortality among older adults. Caregivers' average rate of increase in IL-6 was about four times as large as that of noncaregivers. This suggests that a chronic stressor is capable of substantially augmenting normal age-related increases, effectively aging the immune response. In another study, researchers explored human sex differences in physiological responses to acute psychosocial stress (Kajantie and Phillips 2006). The authors concluded that the observed systematic sex differences in the autonomic nervous system arise from the need for women to protect the fetus from excessive stress hormones in utero. As seen in controlled experiments with animals, frequently repeated or chronic stress is followed by a blunted, pathological HPA axis function, and this has been seen in war veterans, in vital exhaustion, and in chronic pain (Bjorntorp and Rosmond 1999). Stress has also been implicated in the dual functioning of corticotropin-releasing factor (CRF) in the human brain. CRF is considered to mediate aversive aspects of stress, fear, and anxiety. Additionally, CRF release in the brain mediates an independent function of positive incentive motivation. Thus, Pecina, Schulkin, et al. (2006) propose that the CRF release in the brain may also explain why stress may produce cue-triggered bursts of binge eating, drug addiction relapse, or other excessive pursuits of rewards. Next, Epel and colleagues (2004) provide evidence of how psychological stress results in shortened human telomere length and activity, especially among women. Telomeres are DNA-protein complexes that cap chromosomal ends, which promote chromosomal activity. In people, telomeres shorten with age; thus, telomere length can serve as a biomarker of a cell's biological (versus chronological) "age" (Epel, Blackburn et al. 2004). The authors concluded that stress-induced premature cell senescence in people might be

influenced by chronic or perceived life stress. Particularly, women with the highest levels of perceived stress had shorter telomeres, which was about the equivalent of a decade of additional aging, compared with low stress women (Epel, Blackburn et al. 2004).

Stress and Physical Health Outcomes

There is increasing considerable evidence that stress affects physical health though various pathways. It is generally accepted that stressful events influence the pathogenesis of disease by causing negative affective states, and these in turn directly effect the biological processes or the behavioral patterns that influence disease and disease risk (Cohen, Janicki-Deverts et al. 2007). Beginning in the womb, maternal psychosocial stress has emerged as a significant risk factor for preterm delivery. Stress can act in two different matters, first it can influence the biological cascade that may lead to preterm delivery; or stress can simply induce coping styles such as high-risk behaviors that catalyze preterm delivery (Lu and Chen 2004; Hogue and Bremner 2005; Rich-Edwards and Grizzard 2005). Moreover, stressful life events have also been associated with depression during pregnancy (Seguin, Potvin et al. 1995), low birthweight, and spontaneous abortion (Collins and David 1997).

Stress has also been shown to alter eating patterns and behaviors. In one study, persons who reported experiencing one or more daily hassles were significantly more likely to consume more between-meal snacks, high fat snacks, and high sugar snacks; a perceived decrease in main meals and vegetable consumption was also reported

(O'Connor, Jones et al. 2008). Another unhealthy behavior, smoking, has also been associated with stressful life events, especially among women (McKee, Maciejewski et al. 2003). Stressful life events have been shown to be a potential trigger of multiple sclerosis disease activity (Ackerman, Stover et al. 2003); daily stress worsens the clinical symptomatology perceived by lupus erythmatosus patients (Peralta-Ramirez, Jimenez-Alonso et al. 2004); and levels of chronic stress significantly predicted poor clinical outcomes for patients with irritable bowel syndrome (Bennett 2004). Finally, stress has serious implications on cardiovascular health, HIV/AIDS disease progression, and cancer. Among initially healthy individuals, long-term cardiovascular risk is increased among those who experience traumatic events, or are exposed to emotional, sexual, or physical abuse during early life (Dong, Giles et al. 2004). Prospective research conducted among HIV-positive homosexual men evidenced that severe life event stress (e.g. death of mate, arrest, chronic financial difficulty, breakup of a love relationship) was associated with an increased rate of early HIV disease progression (Evans, Leserman et al. 1997). While research studies linking stress and cancer offer mixed results, experiments in humans have indicated that stress affects key pathogenic processes in cancer, such as antiviral defenses, DNA repair, and cellular aging (Cohen, Janicki-Deverts et al. 2007). It is important to note however, that the links between stress and health have often offered mixed results. Researchers have to be sensitive to the introduction of contaminating factors that may inflate the overall event-illness correlations, and the numerous and inconsistent operationalization of stress and stressors in research (Schroeder and Costa 1984).

Part III. The implication of the stress process in explanations of racial and ethnic health disparities

It has been proposed that the major opportunity for improving the health of all Americans may lie in improving the longevity and overall health of those of disadvantaged status, as defined by race/ethnicity or socioeconomic status (House and Williams 2000). There have been numerous interdisciplinary attempts to offer explanations and solutions to why these disparities exist and persist. As will be shown in the next section, aspects of the stress process conceptually underlie all of the reviewed explanations. As seen above, stress is a far-reaching and ubiquitous in human life. It has been shown to impact physical, mental, and biological pathways. Thus, it can be proposed that stress may be a fundamental and structural element of racial and ethnic health disparities. All of the explanations that will be discussed offer information about how a broad array of social, biological, and psychological conditions combines over time to create stress. Considering the historical and current social and economic standing of African Americans, and the observed health disparities, socially induced stress may be a precipitating factor in the appearance of chronic diseases (Rabkin 1976). The stress process unfolds interactively; responses to stress is affected by not only social statuses and circumstances of the affected person, but also the dynamic and sociocultural environment that is reflected by social roles and group memberships (Broman 1995). Therefore, the interaction between stress and race in American culture deserves a more exact exploration.

The following section aims to highlight seven of the more robust and innovative theoretical models for why racial and ethnic health disparities subsist and how stress is implicated in each one. There are three other explanations that have been present in the literature: inherited biological variance, personal health behaviors, and access to health care. While these three explanations have been interesting topics, they have proven to not significantly impact or alter health disparities, and will not be further deliberated (Williams 1995; Lantz, House et al. 1998; Krieger 2003; Kawachi and O'Neill 2005). The subsequent discussions are oriented between structural- and individual-level explanations.

Structural Explanations of Racial and Ethnic Health Disparities

Fundamental Causes of Disease

In 1995, two researchers, Bruce Link and Jo Phelan introduced a theoretically rich and persuasive approach to considerations of how disease and illness are maintained in the American social structure. Entitled 'Fundamental Causes' of disease, this new concept was a continuation of previous discussions (House, Kessler et al. 1990; House, Lepkowski et al. 1994) and attempted to turn attention away from proximate causes of disease to more distal origins. The authors note that in the late twentieth century, the rise and influence of "risk-factor epidemiology" focused attention on behavioral and biological individually based risks for poor health (Phelan and Link 2005). Over time, this epidemiologic perspective has been highly successful in providing the public with information that has helped reduce individual risk, and thereby improve population health. Unfortunately, this approach has also helped

diminish the role of social conditions as important causes of poor health. Link and Phelan (1995) describe social conditions, such as social support and socioeconomic status, as fundamental causes of disease. The hallmark feature of fundamental causes is that they are causes in which their health effects cannot be eliminated by only addressing the mechanisms that seem to connect them to disease. The four essential components of the theory of fundamental causes of morbidity and mortality are as follows: first, such causes influence multiple disease outcomes. Second, such causes operate through multiple risk factors. Third, new intervening mechanisms (i.e. mediators or moderators) reproduce the association between fundamental causes and mortality over time. Finally, the "essential feature of fundamental social causes is that they involve access to resources that can be used to avoid risks or to minimize the consequences of disease once it occurs" (Link and Phelan 1995). Examples of a fundamental cause are SES and racism (Phelan and Link 2005). By adopting a fundamental cause perspective, there is a need for policies that eliminate or reduce the ability to use socioeconomic advantage to gain a health advantage--either by reducing disparities in socioeconomic resources themselves, or by developing interventions that, by their nature, are more equally distributed across SES groups (Phelan and Link 2005). Thus, the fundamental cause perspective is highly focused on resource allocations throughout society. The theory proposes that if resources were distributed equally throughout society there would be an alteration in disease gradients. These same resources are directly related to the stressful conditions experienced by individuals with poor health outcomes. If one were to agree with this theory of explaining SES and race disparities in health, the implicit role of stress would have to

be better understood. For example, the authors suggest that stress is a current intervening mechanism in the SES-health gradient. However, the authors state that resources are key to ameliorating this SES-mortality association. For example, the resources that the authors suggest are money, knowledge and power; these are also some of the resources needed to diminish the excess levels of stress experienced by individuals who are the victims of the SES-mortality or the race-health gradients. These resources are also coping mechanisms to deal with increased levels of stress in a person's life. These increased resources the authors suggest that will diminish the strength of fundamental causes of disease also implicitly are connected to the correlates and consequences of stressful living conditions.

Neighborhood Exposure/Residential Segregation

The physical location of individuals can also impact and predict racial and ethnic health disparities. Many researchers urge future research directions to consider the context in which individuals to better interpret the risks and feasibility of interventions. Two approaches, racial residential segregation and neighborhood effects, are especially relevant when considering racial and ethnic health disparities and both represent the injection of the stress process in the lives of those who live under these conditions.

Racial Residential Segregation

Racial Residential Segregation is defined as the residential physical separation of races, and is an institutional mechanism of racism that was designed to protect whites

from social interaction with blacks (Williams and Collins 2001). Williams and Collins (2001) argue that racial residential segregation is the keystone on which U.S. black-white health disparities began and can be considered a fundamental cause of disease. Segregation has been imposed by legislation and has been supported by economic institutions, the federal government, the U.S. judicial system, and other cultural institutions (Williams and Collins 2001). In 1972, the National Academy of Sciences commissioned an investigation into residential segregation, and they concluded that there was a "web of discrimination" that involved brokers, lenders, local governmental officials, developers and school administrative staff. These perpetrators all worked to reduce the supply of housing that was available to blacks (Farley 2000). Further, they also successfully steered black consumers to certain residential segments. It has been suggested that racial residential segregation can undermine the relative social and economic position of African Americans, as compared to other groups, by restricting access to jobs, exposure to greater health risks, poorer public services and higher taxes, increasing the cost of housing, and automatic attendance in inferior schools (Massey, Condran et al. 1987). Segregation limits residents' access to full-service grocery stores, safe, walkable streets, and a healthy environment. In fact, polluting businesses and factories are located much more frequently in communities of color, which means a less healthy neighborhood with more air and soil contamination (PolicyLink 2007). In 2000, the national index of dissimilarity (a measure of segregation) was 0.66. This means that 66% of Blacks would have to relocate to eliminate segregation. An index greater than .60 represents extremely high segregation (Williams and Collins 2001). According to the 2000

Census, about 74 Metropolitan Statistical Areas were found to have dissimilarity scores greater than 0.60. Further, although there has been documented segregation among immigrant groups in the United States, no group has lived under the current extreme levels of segregation of African Americans. Thus, racial residential segregation can help explain the extreme racial and ethnic health disparities that are unfavorable for African Americans. All of these negative by-products of residential segregation ultimately result in varying degrees and forms of stress imposed upon residents. For example, the social and economic sanctions of segregation are a direct link to financial chronic strains and work stress. Racial residential segregation not only is caused by racism, but it increases it by keeping people apart and different. The value of housing in segregated neighborhoods is likely to devalue, thus reducing the equity and possible wealth of Black homeowners. Segregation can also concentrate poverty, and poverty concentrates a host of other challenges (Massey 2004). The correlates of poverty include violence and crime, social disorganization, and the drug trade and drug use. Violence and crime increase the levels of traumatic lifetime events experienced by residents. By default, residents require increased coping skills to manage the realities of the blockage of social mobility enacted by segregation. By relegating disadvantaged minorities to areas with fewer opportunities and amenities, residential segregation exacerbates their existing social status. In one prospective study among African American students, those from segregated neighborhoods experienced increased levels of family stress, as compared to whites, Latinos, and other non-segregated African Americans (Charles 2004). This family stress was largely a function of violence and disorder in the segregated neighborhoods. As can

be seen, the consequences of racial residential segregation are highly likely to impose chronic activation of the human stress response among residents, also creating an environment in which experiences of chronic stress, daily hassles, and traumatic events are likely more widespread. With theoretical elaboration, segregation can be thought of as a form of stress imposed upon residents.

Neighborhood Effects

Over the years, there have been attempts to direct researchers attention to structural and environmental influences on health and health behaviors. These include paying more attention to the upstream causes of poor health and health inequalities, rather than downstream, which is to focus on more individual level behaviors and risk factors (Macintyre 2002). An example of upstream causes of poor health is that of neighborhood or area effects. In the field of public health, neighborhood effects are thought of as certain aspects or characteristics of neighborhoods that have 'effects' on the health of its residents. The neighborhood is defined as a subsection of a larger community; a collection of people and institutions who occupy a spatially defined area. This spatially defined area is influenced by ecological, cultural, and political forces (Sampson 2002). There are three ways in which neighborhoods may impact health: social and economic, the physical environment, and services. As with segregation, neighborhood effects impact the public education choices of residents, especially considering that the tax base of neighborhoods typically finances public education. Other ways in which neighborhood effects affect residents are: social networks, role models, mutual trust, social disorganization, violence and crime, social

cohesion, and delinquency. Also, the structure of neighborhoods impact access to food, food choices, the possibility of exercise, and transportation options. All of these neighborhood effects translate into increased stressors for inhabitants. The daily hassles associated with the poor physical environment and quality of neighborhood services likely convert to chronic strains. In essence, negative neighborhood effects require residents to live under stressful living circumstances. While the study of neighborhood effects has been plagued with measurement issues, it certainly offers insight to aggregate level rationales of health differences across certain populations.

Socioeconomic Status/Poverty/Income Inequality

The relationship between income and health is well established (Rodgers 1979; Wilkinson 1992; Kennedy, Kawachi et al. 1996; Kawachi and Kennedy 1997). Rodgers (1979) was one of the first to examine the association between income and health, demonstrating that the curvilinear association between individual income and health meant that countries with greater income inequality would experience lower life expectancy. One of the most notable explorations of SES and health is the Whitehall Study of mortality (Marmot, Shipley et al. 1984). For over 10 years, this study covered 17,350 British civil servants, and concluded that the relative risk of mortality significantly increased as grade of employment decreased. For example, men in the lowest classification of employment had three times the risk of mortality from coronary disease as compared to men in the first (i.e. administration) grade of employment. Socioeconomic status represents one of the most important risk factors

for mortality, disability, and chronic disease and illness (Alley, Seeman et al. 2006). Socioeconomic inequalities in health have long been associated with racial and ethnic health disparities in American society as well. In recent years, there have been increased efforts to better understand the relationships between race, socioeconomic status, and health; however, an understanding of how these concepts interact to produce the persistent disparities is rather limited (Kahn and Fazio 2005). Socioeconomic status is defined as a "composite measure that typically incorporates economic status, measured by income; social status, measured by education; and work status, measured by occupation" (Dutton and Levine 1989). The notion that socioeconomic status explains racial and ethnic health disparities has been referred to as "The Socioeconomic Status Model" (Dressler, Oths et al. 2005). There are a couple of points to consider. First, health interviews and surveys have repeatedly shown that rates of mortality, morbidity, functional health, illness, and disease are consistently and significantly higher for members of racial and ethnic groups *and* those of low socioeconomic status. Thus, it has been relatively easy for researchers to argue that income inequalities are the driving force behind the wide and consistent health disparities seen between groups. This conclusion seems reasonable, because race inequalities in health are so closely linked to socioeconomic inequalities, and it is quite difficult to parse out the effects of each, when explaining health disparities (Kahn and Fazio 2005). However, it is important to note that there has been the persistence of racial disparities in various health outcomes, even after controlling for SES, which suggests that race and SES may still have independent effects. Even among minorities who have the same socioeconomic status of their white

counterparts, why is their health status still comparably poor and/or compromised? In other words, why does SES provide unequal returns to health for minority populations? If it is true that income inequality is the driving force behind disparities, disparities should not appear when there is income equality. Thus, researchers have to consider what are the true structural causes for the health disparities seen. Secondly, the relationships among race, SES, and health are complex, but the complexity is compounded by the fact that the interrelationships are dynamic across cohort, age, and time (Lynch 2008).

Therefore, a deeper understanding of the SES-health gradient may emerge if there is heightened consideration of how variables across multiple dimensions and levels interact and simultaneously occur (Adler, Boyce et al. 1994). SES is also inversely correlated with stress (Lantz 2005; Chen 2007; Hatch and Dohrenwend 2007). As discussed in the previous section, the effects of lower socioeconomic status or lower SES itself, directly results in higher reports of stress and stressors; while the resources that diminish lower SES status, are the same to relieve certain excess stressors. Although it must be remembered that upward mobility (e.g. higher socioeconomic status) also may result in chronic psychological stress, especially for minority populations. Many of the aspects that link lower socioeconomic status, race, and health can all be considered as excess stress and stressors on the individual.

Racism and Racial Discrimination

When considering the mechanisms in which racial and ethnic health disparities operate, there has long been interest in the roles of race, racism, and racial prejudice

and discrimination. Although these constructs can be experienced on a structural or individual level, they are constructs that institutionalized in American culture. Thus racism and racial discrimination will be characterized as a structural explanation for racial and ethnic health disparities. Historically, the meaning of race was attributed to readily observable external biological features, such as skin color. Over time, social sciences have come to reject race as a biological notion, now in favor of race as a social concept (Omi and Winant 2007). The resulting ranking of identified groups (e.g. by skin color), determined access to societal resources. The term "racism" refers to an "organized system, based on an ideology of inferiority that categorizes, ranks, and differentially allocates desirable societal resources to socially defined "races"" (Williams and Collins 2004). In the United States, the racial category of "black" evolved with the advent and success of slavery. Racism was supported by societal institutions, and once created, became a societal force that continuously altered social roles and structures (Williams 1997). Further, integral to racism is the ideal that some populations groups are regarded as being inferior to other groups. This ideal tends to lead to prejudice, defined as, the development of negative attitudes and beliefs toward certain racial groups, and discrimination, defined as the differential treatment of members of these groups by both individuals and social institutions (Williams and Collins 2004). Racism operates within objective life conditions, popular culture, and religious and educational institutions (Harrell, Hall et al. 2003).

Clearly, experiences of racism, racial prejudice, and racial discrimination are certain to elicit negative responses, which may directly or indirectly adversely effect mental

and physical health. There has been considerable interest in establishing the relationship between racial experiences and health, especially among African Americans. Existing literature suggests that there are multiple pathways in which racism adversely affects the health of non-majority racial/ethnic groups over time. Williams et al. (2003; 2004) suggests three specific pathways; first, institutional discrimination can affect health by creating racial/ethnic differences in residential environments, socioeconomic status, and access to goods and services. As outlined above, these mechanisms of institutional discrimination are also sources of excess stress among socially disadvantaged populations. Second, reports of discrimination may be a neglected psychosocial stressor that unfavorably affects health. A literature review firmly supports the assertion that the outcomes of perceived racism and discrimination are forms of stress, specifically entitled 'race-related stressors' (Williams 1997; Loo, Singh et al. 1998; Utsey, Chae et al. 2002; Danoff-Burg, Prelow et al. 2004; Franklin 2006). Finally, internalization of society's negative characterization of the nondominant group may have adverse health consequences. Research has shown that self-reported experience of interpersonal racism has been shown to be associated with increased psychological distress, stress, and depression, poorer self-rated health and raised blood pressure (Broman 1996; Krieger and Sidney 1996; Williams 2003).

Klassen et al. (2002), examined access to the cadaveric transplant waiting list among patients with end stage renal disease. Patients with previous experiences of racial discrimination were less likely to be placed on the waiting list. The authors

concluded that victims of discrimination are less likely to seek and obtain new or alternative treatment methods, as they may not expect successful outcomes. Further, discrimination measures predicted list access more strongly than the patient's race (Klassen, Lowrie et al. 2002). The phenomenon of racial discrimination has not been exclusive to only American populations. In a study conducted among minorities in England and Wales, significant associations existed between reported experiences and perceptions of racism and various mental and physical health indicators, such as self-rated health, high blood pressure, and weekly prevalence of depression (Karlsen and Nazroo 2002). Williams (1997) listed institutional racism as a basic cause in theoretical framework that outlined the relationship between health and race. This framework described racism as shaping other important social determinants of health outcomes, such as economic resources and culture. Thus, the realities of the African American experience offers clues to the ways in which racial and ethnic health disparities operate, specifically understanding how stress operates across different social groups. It has been suggested that the psychosocial effects of racism can produce and maintain levels of conscious and subconscious stress in Black Americans (Feagin 2003; Carter 2007; Franklin-Jackson 2007). Nevertheless, the notion that racism is a stressor that can harm or injure its targets is not recognized in psychological or psychiatric diagnostic systems (Carter 2007).In conclusion, scholars and researchers have posited that racism embedded in American society and enacted by individuals, institutions, and systems can act as a chronic or life event stressor for Blacks and the experience of racism may play a role in the high rate of stress-related mental and physical illnesses prevalent among Blacks (Franklin-Jackson 2007).

Individual Level Explanations of Racial and Ethnic Health Disparities

John Henryism

According to American folklore, John Henry was an African American railroad worker in the late 1800s (Williams 1983). Henry became legend as he competed with a steam-powered drill in a steel driving competition. Henry used his considerable strength to persevere during the competition, and he eventually defeated the machine. Unfortunately, as soon as he finished the competition, Henry collapsed and died from the mental and physical toll the battle exacted upon him. This folklore became the main impetus for a body of research that has developed over recent years. Sherman James proposed the John Henryism construct as a link between low socioeconomic status and marginal health outcomes of African Americans, specifically hypertension. More exactly, John Henryism can be defined as prolonged, high effort coping with difficult psychosocial environmental stressors. James further suggests that John Henryism may offer insight about the relationship of African Americans and core principles of popular American culture. The "John Henryism" hypothesis assumes that individuals of lower socioeconomic status are more often exposed to chronic psychosocial stressors, and these challenges require elevated amounts of vigor and thought to handle. The construct consists of three major themes: efficacious mental and physical vigor; a strong commitment to hard work; and single-minded determination to succeed (Bennett 2004). Thus, John Henryism as an explanation for social and racial health disparities explicitly acknowledges the role of stress in the lives of individuals. Exposure to stress is the one of the defining characteristics of this construct. Further, this theory suggests that as personality trait, John Henryism also

offers reasoning of why and how disparities persist under chronically stressful environments. James' (1994) early investigations tested John Henryism among American Southeastern African Americans and found an inverse relationship between hypertension and socioeconomic status only among those who tested high on the John Henryism scale.

Over the years there have been a wide array of methodological inquiries into the validity of the John Henryism Hypothesis, by many researchers (James 1983; James 1984; James 1987; Duijkers 1988; Francis 1991; Wiist 1992; Jackson 1994; Light, Brownley et al. 1995; Nordby 1995; Scribner 1995; Somova 1995; McKetney and Ragland 1996; Wright 1996; Dressler, Bindon et al. 1998; Markovic, Bunker et al. 1998; Adams, Aubert et al. 1999; Clark 2001; Loon 2001; Williams 2001). The John Henryism construct has considered a wide array of health status outcomes and negative health outcomes. There have been mixed results; some empirical investigations supported the John Henryism Hypothesis, suggesting that high John Henryism scores were associated with adverse cardiovascular profiles and other specific health outcomes (Dressler, Bindon et al. 1998). There were other results that failed to support the John Henryism Hypothesis (Nordby 1995), but many of these empirical inquiries were conducted in diverse populations that differed from the initial research population.

Allostatic Load

The recent advent of the terms "allostasis" and "allostatic load" are an effort to better clarify the biological and physiological stress responses. As described by McEwen and colleagues (McEwen 1998; McEwen and Seeman 1999; McEwen 2003), the concept of allostasis literally means "maintaining stability (or homeostasis through change)". For example, allostasis can describe how the cardiovascular system adjusts to active and resting states of the body. Related, the concept of allostatic load refers to the wear and tear that the body experiences due to repeated cycles of allostasis as well as the inefficient regulation of these responses. Allostatic load can be thought of as the cost to the body for being forced to adapt to various psychosocial challenges and adverse environments. More specifically, allostatic load is the physiological burden imposed by stress on the body and brain, including the cardiovascular, immune, and metabolic systems. As with John Henryism, the theory of allostatic load explicitly acknowledges the biological and physiological effects of stress. Stress is the bedrock of which this theory is built. Allostatic load develops, according to the theory, when the repeated costs of dealing with stress accumulate to cause "wear and tear" on the body (McEwen 1998).

As time passes the health consequences of allostatic load include diabetes, abdominal obesity, obesity, and cardiovascular disease (Geronimus 2001). As measured by two categories of stress hormone-related biomarkers, allostatic load scores are calculated by use of an allostatic load logarithm that may include 8, 10, 13, 14 or 16 components, dependent upon available data. This theory has been tested in various

populations and settings, each with mixed results; some attempts to measure allostatic load have failed to distinguish between measures of aging and measures of the impact of aging on metabolic processes (Hogue and Bremner 2005). Further, it is not clear to what extent allostatic load causally influences clinical endpoints such as mortality or cardiovascular disease (Loucks, Juster et al. 2008). Nonetheless, these concepts are relevant in the study of racial and ethnic health disparities. Comparing allostatic load scores offers the opportunity to gain more insight into the mechanisms of Black/White health disparities in the U.S. and Geronimus et al. (2006) did just that. Using data from the 1999-2002 National Health and Nutrition Examination Survey IV, the researchers examined gender and race differences in age-related allostatic load scores. Analyses revealed the existence of distinct racial disparities in health in clinical and subclinical conditions across a range of biological systems among young through middle-aged adults. Among both men and women, Blacks had higher mean allostatic load scores that Whites at all ages, and the differential in scores increased with age (Geronimus, Hicken et al. 2006). Even more telling, Black women bore the burden of allostatic load, as compared to Black men or White women. Among Whites, there were little gender differences until age 55. Among Blacks, however, Black women had higher allostatic load scores than their male counterparts at all ages. McEwen (2004)acknowledges that stress is a common experience and the blame for many illnesses, but research has yet to understand exactly how stressful experiences lead to disease. So, although the study of allostasis and allostatic load has been challenged, the concepts do provide an interesting impetus to further explore the biological effects and mechanisms in which disparities operate.

Weathering

In an attempt to better understand the early health deterioration of Blacks, (Geronimus 1992). Geronimus (1992) introduced the "weathering" hypothesis. Weathering describes the process of premature aging, or an accelerated aging process experienced by Blacks as a consequence of the cumulative impact of repeated experiences and exposure to social and economic adversities, coupled with political marginalization (Geronimus, Hicken et al. 2006). For example, urban African American men and women in high poverty areas have rates of health induced disabilities at ages thirty-five and fifty-five that are comparable to the national averages for fifty-five and seventy-five year olds, respectively (Geronimus 2004). Notably, the theory of weathering has been established among Black women, specifically those of child-bearing age. Geronimus (1992) has shown that weathering is associated with adverse pregnancy outcomes and hypertension among Black and poor women. Weathering attempts to describe the ways in which the daily life experiences and social status of Blacks has implications in the manifestation and timing of certain diseases and illnesses. As with John Henryism, Weathering is an explanation for the early onset of disease, especially among African Americans, as a result of cumulative stress experienced throughout the life course. Geronimus (2001) contends that from the time African Americans are in utero until their deaths, their exposure to chronic stressors, such as racism, environmental insults, and taxing social networks, places them at heightened risk for early disability and mortality. As with the other individual level explanations reviewed, the notion of stress is at the heart of the theory.

Conclusion

The goal of this chapter was to deconstruct several of the more popular explanations for racial and ethnic health disparities in America, specifically explicitly acknowledging the role of stress and stressors. This chapter began with a historical review of race and race relations in American culture. Next, a literature review revealed the current state of racial and ethnic health and economic disparities. The literature review concluded with an attempt to better define and characterize the stress process. This chapter then highlighted contemporary explanations of racial and ethnic health disparities and the implication of the stress process in each explanation. Pearlin (1989) noted that the structural contexts of people's lives are not extraneous to the stress process but are fundamental to that process. It appears that the stress process is also fundamental to many of the approaches to better understand persistent and unyielding health disparities.

Persons who do not live under chronic and unrelenting stressful living conditions are more likely to experience a wide range of resources, ranging from money, knowledge, and power to beneficial social connections. These persons tend to be individuals of a higher socioeconomic status, and a social standing that may include membership of the dominant American cultural group. The impact of stress varies depending upon the resources possessed by an individual (Lazarus 1991). The past and current realities of social or racial stratification must be acknowledged, even when race is not the significant variable in a regression analysis.

Each of the mechanisms of racial and ethnic health disparities discussed above are not relevant for every Black and all do not result in negative health and consequences for every affected individual. It is important to note that stress reactions depend on the individual's perception that the event or experience is negative and unwanted. The aim of this chapter was to theoretically elaborate on the role stress has in the current and more popular explanations of racial differences in health, and describe the impact of stress and stressors on disparities, regardless of the approach used to understand these disparities. Future research requires an honest assessment of the possibility that experiences with stress, either as a natural part of the human experience or relatively excessive, are gradated across racial and ethnic group memberships. If this is true, future research must continue to focus on and elaborate the structural mechanisms that dictate varying stressful living conditions that ultimately result if poor health outcomes. Future research should seek to better understand how to decide whether a person's living condition is excessively stressful as compared to other members of social groups. For example, there has been little research examining the daily stressors of Black Americans, and among what is available, much of the research focuses on women (Brown 2004). Reformulated objective analyses of experiences with chronic stressors, daily hassles, and traumatic life event stressors should impact public policy and help find solutions to this form of human suffering, that may be stratified by the perceived color of a person's skin (See Slavin 1991 for a discussion of the need of a multicultural approach to the stress process) or (Chun 2006 for a discussion of the role of culture in stress research). There is a distinct possibility that many minorities are living a quiet desolation that is defined and constantly influenced

by varying forms of daily and chronic stressors, which results in earlier mortality for several minority individuals across the life course.

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CHAPTER THREE

Exploratory Analysis of the prevalence of stress and negative life events among a sample of African American and White American adults, measured four times, over a sixteen-year time span.

Introduction

Racial and ethnic health disparities are prominent in the United States. Reducing or eliminating health disparities can improve the health of all Americans. A variety of factors contribute to the persistent health disparities observed between Black and White Americans (herein, "Black" refers to all nonimmigrant persons of African heritage living in the United States, and may be interchanged with "African American"). In health disparities research, it is acutely important to be sure that characteristics, such as race, class, and gender, are not only analysis control variables, but also clues to an individual's historical, social and political background (Schulz 2006). The elimination of racial and ethnic health disparities is a research interest not only among public health researchers and practitioners, but also among scholars in the social sciences, humanities, and the medical community. This interdisciplinary interest has facilitated opportunities to explore disparities from various theoretical vantage points as well. One such vantage point proposes that the stress process may play an active role in determining the nature of mental and physical health disparities. As Williams (1997) notes, stress is linked to social structure and is not randomly distributed in the population; therefore stress can affect racial differences in health.

Social status and social roles can determine both the types and quantity of stress to which an individual is exposed. Thus, the structural location of many blacks in society could lead them to have higher levels of stress than whites (Williams 1997). As a prerequisite to these discussions, it is important to first understand the prevalence and descriptions of stress among Black and White Americans. Such an understanding is essential to further elucidate the role the stress process may play in racial and ethnic health disparities.

In numerous discussions of race and ethnicity, researchers from the stress tradition have focused on discrimination and racial prejudice (Meyer, Schwartz et al. 2008). This paper will extend these discussions by focusing on other non race-related constructs of stress and life events. Thus, the goal of this research is to explore if race is associated with the level of stress which people experience. As an exploratory analysis, the primary research question is: do reports of stress and life events differ between Black and White Americans over a sixteen-year time span? This paper will first provide a brief literature review of the distribution of stress in American society focusing on four types of stressors: financial, marital, and parental chronic stress and major negative life events. Next, the paper will draw on data from the Americans' Changing Lives (ACL) study to explore racial differences in reports of stress longitudinally. This paper will only focus on the differences between Blacks and Whites.

Background

The discussion of existing literature that follows is divided into two main sections. First, there will be a description of the racial and ethnic distribution of stress. Second, there will be an in depth overview of the chronic stressors and negative life events to be analyzed in the current study, offering definitions and racial differences in exposure to each.

Review of the Literature: The Racial and Ethnic Distribution of Stress

Hatch and Dohrenwend (2007) delineated the distribution of traumatic and other stressful life events by race, gender, age, and socioeconomic status (SES) from 1967 to 1980 and 1989 to 2005. In a review of 9 separate published studies, African Americans/non-whites reported higher traumatic and stressful life events in six of the nine studies. In data from the Detroit Area Study, Williams (1997) reported that there were significant racial differences on chronic stress, with blacks having lower levels of chronic stress than whites, but with blacks reporting higher levels of financial stress. Blacks also had double the average score on the life-events scale than their white counterparts. When comparing debt between blacks and whites, Drentea and Lavrakas (2000) found that blacks experience more stress due to overall debt.

There have also been observed differences in the distribution of stress in African American subpopulations, such as gays and lesbians, young adults, college students, and women. Among minorities, sexual orientation or age are also associated with further increased stress exposure. In a comparison of lesbian, gay, and bisexual

individuals, those of minority status reported higher levels of exposure to general stressors than white heterosexual men (Meyer, Schwartz et al. 2008). Existing literature suggests that there are stress differences between Black and White younger adults as well. In a study of young adults, the typical African American indicated exposure to nine major and potentially traumatic events, as compared to their counterparts, who averaged around six (Turner 2004). Among African American college students reported significantly higher levels of family stress (e.g. death in a social network, crime in immediate family, or social problems in family) compared to their white student colleagues. Furthermore, this elevated exposure was also structured by the African American students' level of segregation at their home away from college. (Charles 2004).

Due to the social interplay of race and gender, Black women often struggle to assume power and control comparable to their male or white female peers. In a study of Detroit metropolitan area women, city-dwelling African American women reported a significantly greater number of acute life events than suburban-dwelling white women. Further, independent of city or suburban residence, African American women reported significantly more frequent exposure to everyday unfair treatment than white women who lived outside of the city (Schulz, Israel et al. 2000). Since stress is also a determinant of birth outcomes, there has been a growth of pregnancy stress research. In one such study, Black women reported the higher levels of stressful life events (emotional, financial, partner-related, and traumatic constructs) in the 12 months before delivery than any other race/ethnicity (Lu and Chen 2004). In another

study of pregnant women, black women reported significantly higher levels of individual and community level stressors (Culhane, Rauh et al. 2002).

Review of the Literature: Chronic Stressors and Life Events

The circumstances inimical to well-being are most prevalent among groups having the highest rates of morbidity and mortality (Kahn and Pearlin 2006). Further, many of these circumstances may originate far in advance of the health inequalities that become striking in middle and late life. The impact of chronic stressors may be particularly severe when they surface within major social domains and social roles (Pearlin 1983; Turner and Avison 2003). Among the stressors that have been identified as capable of impacting well-being are economic and financial strains, troubles and conflicts in marriage, the trials and tribulations of nurturing and socializing children, and major life events that affect the life course in unexpected ways (Kahn and Pearlin 2006). Each of these stressors will be discussed in turn.

Financial Chronic Stress

Among the array of chronic stressors that people may confront in their daily lives, there is probably none more pivotal than financial hardships and strains (Kahn and Pearlin 2006). Many fundamental activities of daily living and chances to succeed are closely tied to a person's financial resources (Peirce, Frone et al. 1996). Chronic financial stress can be defined as the persistent inability to afford the basic necessities of life (Pearlin and Radabaugh 1976). For example, in one telephone interview study of 366 male and female householders in the southeastern United States, 58% of

respondents reported that they did not have enough money to meet all monthly expenses and 74.5% reported that they had to cut out unnecessary expenses because of financial problems (Fox 1998).

Reports of financial stress include perceptions of one's capacity to manage economic resources, pay bills, repay debts, and provide the needs and wants of life. Thus, two individuals in comparable financial situations may have different perceptions of their personal level of financial stress. These differing perceptions are likely a reflection of diverse demands on income associated with family size, where and how people live, personal proclivities, and aspirations (Kahn and Pearlin 2006). When comparing financial stress levels between Blacks and Whites, the literature generally suggests that Blacks are more likely to have higher levels of financial stress. In one study of college students, African American students reported higher levels of credit card debt and financial stress levels (Grable 2006). In another study of retired workers, Black women were more likely to report higher levels of financial stress than White women (Logue 1991).

In terms of the prevalence of financial stress over the life course, the literature is mixed. Certain studies suggest that individuals presumably experience financial stress more frequently in young and middle adulthood, while attempting to develop and maintain a career (Wrosch, Heckhausen et al. 2000). While other studies have suggested that elderly persons have more financial difficulty because they may live on fixed incomes and have fewer opportunities to manage financial problems with

personal resources (Krause 1987). More research needs to be done in this area to understand these relationships better.

Marital Chronic Stress

Marital quality is a multidimensional concept that includes negative and positive experiences. Negative experiences include marital conflict and excessive demands from one's spouse, while positive experiences include feelings of love, satisfaction, being cared for, and a harmonious relationship (Umberson and Williams 2005). Marital stress can impact overall well-being (Trief, Wade et al. 2002). Unfortunately, the research base that explicitly focuses on chronic marital stress is limited; rather the focus has been on marital quality. Marital and family roles contribute to the quality and chronic stressors associated with marriage. For example, the responsibility of household and childcare may differ between men and women. Although different measures of marital quality have been used across different studies, women have consistently reported lower marital quality than men in national surveys (Umberson and Williams 2005).

Broman (1993; 2002; 2005) has contributed several studies in recent years describing racial differences in marital quality. Using the Americans' Changing Lives (ACL) database, Broman (2005) has reported that blacks have lower marital quality than do whites. Blacks were significantly more likely than whites to report their spouse having affairs, hitting, pushing, or slapping, and wasting money. Blacks also were less likely to report feeling loved by their spouse, than their white counterparts.

Broman (1993) has also argued that premarital factors, such as poor financial and educational status, and differing expectations of marriage may contribute to levels of marital chronic stress and well-being among Black respondents.

Parental Chronic Stress

Parental chronic stress can be seen as the result of a perceived discrepancy between demands of parenthood and personal resources (Östberg and Hagekull 2007). Abidin's (1990; 1992) conceptualization of parental stress emphasizes the strain experienced by a parent in his or her parenting role. Another conceptualization addresses the potential everyday frustrations and irritations that accompany childrearing and children's typical, but sometimes challenging behavior, as it translates into levels of parental stress (Crnic 2005). Regardless of the conceptualizations of parenting stress and outcomes of interest, higher reports of parenting stress is associated with poorer outcomes (Chang 2004).

Few studies have defined and investigated parenting stress in nationally representative samples. Most of the studies have focused on subgroup populations who face specific challenges. For example, there has been research examining parental stress among low-income single black mothers (Gyamfi, Brooks-Gunn et al. 2001), single fathers and mothers (Avison and Davies 2005), parents of children with attention deficit hyperactivity disorder (Fischer 1990; Mash 1990; Anastopoulos, Guevremont et al. 1992), and mothers of low-birth weight babies (Singer, Salvator et al. 1999). This research focus is likely because it is assumed that parents of children with chronic or acute health conditions are under higher levels of stress than other

parents. Nonetheless, it is also important to better understand the levels of chronic parental stress among the general population. In one such study, Pinderhughes et al., investigated the relationship between certain parental characteristics and disciplinary practices; African American parents reported higher levels of parenting stress than the White parents (2000). Middlemiss (2003) found that among low income African American and White mothers, both groups reported experiencing high levels of stress with African American mothers reporting more frequent stress, though differences in mothers' reported severity of stress was not significant. Further, despite long interest from the field of child development, very few studies have analyzed differences by race of parental stress.

Negative Life Events

As Dohrenwend (1973) comments, it is highly unlikely that all of the untoward events individuals experience are public disasters or national catastrophes. Most of the crises people experience in their lifetimes are private events. These events can be thought of as negative life events. The assessment of negative life events was originally captured by Holmes and Rahe (1967) in a life events checklist, the Schedule of Recent Experiences (SRE), which has been used over the past forty years. Life events are characterized as events that disrupt social relationships, personal habits, and patterns of activity (Kanner, Coyne et al. 1981). According to Pearlin et al. (1981), the convergence of life events and chronic strains may produce stress in two ways. First, the experience of stressful life events may redefine the

meanings of existing chronic strains. Second, the occurrence of life events may exacerbate or generate new chronic stress and strains.

A review of the literature reveals mixed results while trying to assess racial and ethnic differences in reports of negative life events. In one telephone interview study, while trying to assess depression medication acceptability between racial and ethnic groups, Cooper et al. (2003) found no differences in reports of life events between Black, White and Hispanic participants. However, in another study of high school students, African American students were significantly more likely to report negative life events, compared to their White peers (Weist 1995). Other empirical evidence suggests that African Americans may be at increased risk for exposure to life events (Utsey, Giesbrecht et al. 2008), however, while there is plentiful research studies of socioeconomic variations in exposure to life events, more research is needed to better understand racial variations in reports of negative life events (McLeod and Kessler 1990).

Methods

Data

Americans' Changing Lives (ACL) is a long-term four-wave cohort study that was begun by the University of Michigan Survey Research Center in 1986. The goal of ACL was to focus on socioeconomic disparities, while monitoring age-related health changes during middle and later life, especially in terms of compression of morbidity

and functional limitations (House, Lantz et al. 2005).The second data collection occurred in 1989, and then repeated in 1994, and 2001-2003.

ACL is a stratified, multistage area probability sample of noninstitutionalized adults aged 25 and older living in the coterminous United States. At Wave One, two groups were oversampled: African Americans and adults aged 60 and older. Wave One, conducted in 1986, included 3,617 face-to-face interviews, which represented 70% of sampled households and 68% of sampled individuals. In 1989, Wave Two represented 83% of Wave One survivors or 2,867 face-to-face interviews. In 1994, Wave Three interviewees represented 83% of Wave One survivors or 2,562 respondents. These interviews were conducted either by telephone or face-to face interviews, and also included proxy interviews. Wave Four interviews began in 2001 and concluded in 2003. These 1787 respondents represented 49% of baseline survivors. Much of the Wave Four nonresponse was due to mortality, with 1184 or 33% of the original respondents dead by Wave Four. Included in Appendix A is a table that describes the response rates of survivors across the four waves of data collection. As with Wave Three, the last data collection period entailed face-to-face and telephone interviews, which were supplemented with proxy interviews when appropriate. The study included continuous mortality tracking via the National Death Index and other methods yielding over 99% mortality ascertainment, with over 97% of deaths confirmed via death certificates.

Measures

Demographic and Socioeconomic Characteristics

Individual characteristics include the following variables, all measured at baseline: gender (female=1), education (number of years of schooling), continuous age, continuous income, race (Black =1; White =0), and employment status (employed=1). Table 3.1 describes the predictor variables and their level of hierarchy in the multilevel model. In the appendix, there are two tables (Appendix B and Appendix C), which further describe the unweighted characteristics of the 474 individuals who were loss to follow after Wave One.

Table 3.1. Predictor Variables tested on each level of the hierarchy

Level	Variable Code	Variable Description
Level-1 **Time-Varying (Within-Person)**	WAVE	Time of interview since baseline (0-16 years)
	EMPLOYMENT	Employment Status (0=not employed; 1 = employed
	INCOME*	Continuous Income (divided by $1000)
Level-2 **Fixed (Between-Person)**	RACE	Race of the respondent (0 = White; 1 = Black)
	EDUCATION*	Highest grade of school completed (0 = 0 years through 17 = 17 or more years)
	GENDER	Sex of the respondent (0 = male; 1 = female)
	AGE*	Continuous Age (24-96)
Level- 3 **(Between PSU)**	Primary Stage Sampling Unit (PSU)	The selection of primary stage sampling units (PSU's), which depending on the sample stratum, are either U.S. Standard Metropolitan Statistical Areas, single counties and/or groupings of small counties, is based on the county-level 1980 Census Reports of Population and Housing.
	STRATUM	There are 45 strata and 2 clusters within each stratum.

* These variables were centered at their grand mean.

Dependent Variables

There are four primary dependent variables to compare various levels of stress experienced among the sample.

Financial Chronic Stress. This scale is comprised of responses to two questions: 1- "How satisfied are you with your/your family's present financial situation?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all) and 2- "How difficult is it for you/your family to meet monthly payments on your bills?" (5-point response scale with 1 = extremely difficult and 5 = not difficult); this variable was reverse coded. High values indicate a higher level of Financial Chronic Stress for the respondent. A standardized index was constructed by taking the arithmetic mean of the two questions.

Parental Chronic Stress. This scale was created from responses to three questions: 1-"At this point in your life, how satisfied are you with being a parent?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all); 2-"How often do you feel bothered or upset as a parent?" (5-point response scale with 1 = almost always and 5 = never), this variable was reverse coded; and 3-"How happy are you with the way your child/children have turned out to this point?" (5-point response scale with 1 = very happy and 5 = not at all happy). High values indicate a higher level of Parental Chronic Stress, and responses were only obtained from respondents who had children. Responses to this scale were standardized and then averaged to create this index.

Marital Chronic Stress. This scale was created from responses to three questions: 1-"Taking all things together, how satisfied are you with your (marriage/relationship)?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all),this item was reverse coded; 2-"How often would you say the two of you typically have unpleasant disagreements or conflicts?" (7-point response scale with 1= daily or almost daily, 2 = 2 or 3 times a week, 3 = about once a week, 4 = 2 or 3 times a month, 5 = about once a month, 6 = less than once a month, and 7 = never); and 3-"Taking everything into consideration how often do you feel bothered or upset by your (marriage/relationship)?" (5-point response scale with1 = almost always and 5 = never). Cases missing on 2 or 3 of the three input variables at a given wave were imputed using a simple ordinal least squares regression prediction model, with no random residuals. Three cases were imputed for the Wave 1 index and 3 cases were

imputed for the Wave 3 index. There were no missing cases on the indices at either Wave 2 or Wave 4.

Total Life Events. A continuous scale was created to assess the number lifetime and recent life events experienced by the respondents. At baseline, respondents were asked if they had ever been widowed, divorced (or had a marriage annulled), had a child die, or been the victim of a serious physical attack or assault at any time in their life. These four events were considered events that may have happened to the respondent at any point in their life prior to baseline. At baseline, and every subsequent wave, respondents were also asked about recent negative events. The five recent negative events that could have occurred in the previous three years were death of a parent/step parent, death of a close friend/relative, involuntary loss of a job (excluding retirement), being robbed or burglarized, or having any other bad thing occur that greatly upset the respondent. At baseline, lifetime events were summed with recent negative events, and at each subsequent wave, these scores were added to that wave's reported recent negative events, thus a cumulative continuous score was created for each wave. These indexes were not standardized.

Statistical Analysis

Descriptive statistics were calculated to assess sample characteristics at each wave (Table 3.2). Also, at each wave, t-tests were performed to compare unadjusted stress and life events means between Black and White respondents (Table 3.3). Repeated measures on a group of individuals can be regarded as having a hierarchical structure in which the measurements at different points in time are "nested" within individuals.

Thus, hierarchical linear modeling (HLM) was used to model the data longitudinally, specifically to examine levels of chronic stress and negative life events, on the same subjects, over time, because the observations for an individual are not independent, and ordinary least squares regression analysis is inappropriate. Hierarchical relationships occur when variables at one level of analysis influence, or are influenced by, variables at another level of analysis (Hofmann 1997). In this case, a three-level hierarchical linear model was employed to simultaneously investigate the associations between race and stress over four waves of data spanning 15 years, while taking into account the complex sample survey design of the study. Particularly in longitudinal data, where there is a greater likelihood of attrition and nonresponse, the flexibility of HLM in its treatment of the spacing of observations and of missing data is highly beneficial. The fact that HLM permits nonequidistant times of observation is appropriate in the current analyses using the ACL database. HLM provides explicit modeling of reports of stress that can describe the pattern of both within-person change and the effects of between-person characteristics such as race or gender on the within-person change. In addition, an HLM analysis can consider the interactive effects across these levels (Bryk 1992). The multilevel analysis is also appropriate to overcome the problems of conflation of individual and group effect.

Table 3.2. Predictor Variables Descriptive Statistics (Unweighted) Waves One-Four

	Level-1 Variables				Level-2 Variables							
	Employment (Employed)		Income		Race*		Gender* (Female)		Education*		Age**	
	Black	White	Black	White	Black	White	Black	White	Black	White	Black	White
Wave One N=3497												
Mean	---	---	15663.89	25243.81	---	---	---	---	10.4	12.0	52.6	54.6
(SD)	---	---	(15513.6)	(19625.7)	---	---	---	---	(3.7)	(3.1)	(17.3)	(17.7)
%	50.9	51.7	---	---	34	66	61	66.3	---	---	---	---
Wave Two N=2780												
Mean	---	---	17914.44	33340.08	---	---	---	---	---	---	55.1	56.4
(SD)	---	---	(19629.7)	(38206.07)	---	---	---	---	---	---	(16.9)	(17.2)
%	49.4	51.5	---	---	---	---	---	---	---	---	---	---
Wave Three N=2491												
Mean	---	---	23558.91	38691.71	---	---	---	---	---	---	58.1	59.0
(SD)	---	---	(23688.29)	(43593.26)	---	---	---	---	---	---	(16.3)	(16.6)
%	46	49.2	---	---	---	---	---	---	---	---	---	---
Wave Four N=1737												
Mean	---	---	37620.08	58773.95	---	---	---	---	---	---	56.1	58.6
(SD)	---	---	(45315.78)	(102826.67)	---	---	---	---	---	---	(15.9)	(16.7)
%	44.3	49.3	---	---	---	---	---	---	---	---	---	---

* Only measured at Wave One

** In subsequent analyses, only baseline age was considered

The level-1 model captures within-person variation over time. The level-2 model describes between-person variability in the relationships of leve1-1 predictors with the outcomes of interest. Although longitudinal sampling weights are used in the analyses, the level-3 model captures variance between ACL sampling clusters to guard against variance underestimation (Skinner and Vieira 2007). The first step in the multilevel analyses was to fit an "unconditional model" to estimate the variance available to be explained at each level of the data hierarchy (Raudenbush 2002). The unconditional model contained only the dependent variable, random errors at Level 1, random person effects at Level 2, and random cluster effects at Level 3, and no predictor variables were specified at any level. The second step of the model building process was to build up the Level-1 time-varying (within-person) model. This involved adding Level-1 predictors to the model without entering predictors at the second level, in an attempt to explain variance in the outcomes within individuals with time-varying predictors (e.g., years since 1986, income, employment). The third step was to enter the predictors at the second level (e.g., individual-level, time-invariant predictors) to the model. This step allowed for a comparison of the levels of stress at baseline between blacks and whites. The final step was to create an interaction between race and time to assess the changing effect that race has on stress as time passes; or, equivalently, to determine if race explains between-person variance in changes in the outcomes over time. The final model for each of the four dependent variables had the following form:

Level-1 Model

$$Y_{ijK} = \pi_{0jK} + \pi_{1jK}(\text{EMPLOY}_{ijK}) + \pi_{2jk}(\text{WAVE}_{ijK}) + \pi_{3jk}(\text{INCOME\_T}_{ijK} - \mu\text{INCOME\_T}_{\text{ijK...}}) + e_{ijK}$$

Where Y is the dependent variable, measured at time i on person j within sampling cluster k, and $\pi_{0jK}$is the intercept and the expected mean stress level or number of negative life events for respondent j in sampling cluster k at baseline (who is unemployed, and has an average income). The term e_{ijK} is the residual or error.

Level-2 Model

$$\pi_{0jk} = \beta_{00k} + \beta_{01k}(AGE_{jk} - \mu AGE..) + \beta_{02k}(EDUC_{jk} - \mu EDUC..) + \beta_{03k}(FEMALE_{jk}) + \beta_{04k}(BLACK_{jk}) + r_{0jk}$$

$$\pi_{2jk} = \beta_{20k} + \beta_{24k}(BLACK_{jk}) + r_{2jk}$$

In the level-2 model, the level-1 intercept $\pi_{0jk}$is now a dependent variable, β_{00k} is the level-2 intercept for sampling cluster k, and r_{0jk} is a level-2 random effect associated with the j-th respondent from sampling cluster k. In this equation, β_{00k} represents the grand mean of the outcome at baseline, or the mean of the intercepts for all individuals within sampling cluster k. The random effect r_{0jk} represents the deviation of individual j's mean from the overall grand mean. When the variance of these random effects is large, there are large differences between individuals at baseline. The coefficient β_{04k} is the expected gap in chronic stress levels or number of negative life events between Blacks and Whites at baseline in sampling cluster k, adjusted for employment, income, education, age and gender. The random coefficient $\pi_{2jk}$is the expected change in chronic stress levels or number of negative life events for every additional year since 1986. The coefficient β_{24k} is the effect of interest in the final model. It is included only in Model 5, and represents the change in slope of the stress outcome over time for Black relative to White respondents.

Level-3 Model

$\beta_{00k} = \gamma_{000} + u_{00k}$

Where β_{00k} is the overall grand mean of the outcome at baseline across all sampling clusters, and u00k is the random effect associated with the *k*-thsampling cluster (capturing between-cluster variance). All other coefficients in the Level-2 model are assumed to be fixed at Level 3 (i.e., there are no additional random cluster effects).

The current study uses all four available waves of data to model trends of stress over time between racial groups. Hierarchical linear models were run separately for each chronic stress and life events outcome, using the HLM software v.6.06 (Raudenbush 2004). In addition to the attrition and nonresponse discussed above, individuals who did not respond on the outcome variable on at least two or more waves were excluded. This was to ensure that a slope would be calculated for all respondents, allowing comparisons of stress levels over time. Also, individuals who reported their race and/or ethnicity as other than Black or White were excluded. For example, the sample size was n=2982 for analyses regarding financial chronic stress (FCS) as an outcome. This number included only Black or White individuals and persons who have a value on the FCS scale at two or more time points. Thus, the sample size varied with each set of analyses: marital chronic stress (MCS) as an outcome (n= 1754); parental chronic stress (PCS) as an outcome (n=2555); and negative total life events (TOTLE) as an outcome (n=3023).

Centering was used for income (a level-1 predictor), age and education (both two level-2 predictor variables), and two of four the outcomes, Financial Chronic Stress

and Parental Chronic Stress, so that the value of zero represented the grand mean for the sample for that particular variable in the HLM analyses. All analyses incorporated sampling weights to account for the complex sample survey design. In summary, three-level hierarchical models were employed to specifically investigate two main analysis outcomes: First, a comparison of stress and life events at the beginning of the study between Black and White respondents. Second, to model the trajectory of levels of stress and life events for Black and White respondents over four waves of data spanning 15 years.

Results

Table 3.3 presents the unadjusted chronic stress and life events mean values for Black and White respondents at each wave. Independent samples t-tests revealed that, except for Marital Chronic Stress at Wave Three, for every stress and negative life events indicator at each wave, Black respondents reported significantly higher mean levels, as compared to their White counterparts. As mentioned, Financial Chronic Stress and Parental Chronic Stress variables were centered at their mean. The numerical range for each chronic stress and negative total life events variable differed: Financial Chronic Stress (-1.76-2.72); Marital Chronic Stress (1-7); Parental Chronic Stress (-1.65-4.65) and Negative Total Life Events (0-14).

Financial Chronic Stress

For the unconditional model (Model1), the results in Table 3.4 show that the estimated mean of FCS was -0.087, across all waves, for all cases, with a within-

person level-1 variance of 0.456 and a between-person level-2 intercept variance of 0.448, as shown in Table 3.5. The within-person variance describes how much variance is present around an individual's mean, while the between-person variance describes the variance between the respondents' means. In Model 2, the slope for wave is introduced and it is significantly negative. The addition of wave in the model decreased the within-person variance by 12.2%, which means that time explained about 12% of an individual's FCS mean variance. Model 2 also introduces the between-person variance in slopes. In Model 3, the remaining level-1 predictors are included, and they explain about 0.834% of the remaining within-person variance. In this model, interestingly, employment is not significant, thus, employed or not, reported levels of FCS do not differ significantly. However, an individual's FCS level significantly decreases by about 0.002172 for every one thousand-dollar increase in income. In Model 4, the level-2 predictors are included. All predictors, save gender, were significantly associated with Financial Chronic Stress. Model 4 demonstrates that Black respondents reported significantly higher levels of Financial Chronic Stress averaged across all time points, than their white counterparts. The addition of the level-2 predictors explained about 16.4% of the between-person intercept variance, as compared to Model 3, which included only level-1 predictors. Finally, a race by wave interaction term was added in Model 5 to examine whether the time trajectory of FCS differed significantly by race. While FCS decreased across time for Black and White respondents, there was not a significant difference in the rate of decline (See Figure 3.1). This reinforces what was seen in Table 3.3; in which independent samples t-test unadjusted analyses showed that Black respondent

reported higher mean levels of Financial Chronic Stress at each wave as compared to their White counterparts. In all of the financial chronic stress models, there was significant variance across level-3 primary sampling units.

Marital Chronic Stress

In the null model for Marital Chronic Stress, Table 3.6 shows that the overall mean was a predicted 2.64 for all respondents across the four waves of data. The addition of the time variable (Wave) in Model 2 demonstrates that as time passed, mean values for MCS significantly increased. The complete level-1 model (Model 3) shows that employment and increasing income were also significantly associated with Marital Chronic Stress. In Model 4, once all the level-2 variables were added to the model, education, gender, and race all proved to be significant predictors of MCS. Women and Black people were significantly more likely to report higher levels of Marital Chronic Stress across all time points. However, in the final model (Model 5), there were no race differences in the increasing slope of marital chronic stress levels over the four waves of data, as seen in Figure 3.2. Unadjusted analyses revealed that at waves 1, 2, and 4, Blacks were more likely to report higher mean levels of Marital Chronic Stress that White respondents (Table 3.3). Table 3.7 shows that once race was added to the model, 6.34% of the between-person level-2 intercept variance was explained.

Parental Chronic Stress

With Parental Chronic Stress centered at its mean, the estimated overall mean across waves for all respondents was 0.0031 (Table 3.8). In Model 2, with time (wave) added to the unconditional model, the rate of Parental Chronic Stress significantly decreased with every year from baseline, with the time variable also explaining about 15.88% of the within-person level-1 variance. Once the level-1 model was complete, only employment and wave held a significant association with Parental Chronic Stress. In Model 4, Blacks and individuals younger than the average age were significantly more likely to report higher levels of parental chronic stress across all time waves, but when considering the trajectory (Model 5), the overall increase of parental chronic stress over time was not significantly different between Blacks and Whites (Figure 3.3.). Finally, as with FCS, unadjusted means revealed that Blacks reported higher levels of Parental Chronic Stress at each wave than White study participants.

Total Life Events

The results in Table 3.10 show that the overall expected mean of Negative Total Life Events (TOTLE) was 2.67 for all cases in all measurements. Model 2 shows that with every additional year of observation, the mean of TOTLE significantly increased. The addition of a time variable, explained 86.6% of the within-person variance (Table 3.11). In the complete level-1 model (Model 3), higher income persons were significantly more likely to report lower total life events. Although in the unadjusted analyses there were significant mean differences in reports of TOTLE

at each wave between Black and White respondents (Table 3.3), there were no differences in reports of total life events at across all time waves between Black and White respondents in Model 4 of the Hierarchical Linear Models. Blacks (Model 5) were significantly more likely to have a higher increase of negative total life events over time. Thus, Blacks had reports of significantly more total life events than Whites, over time, despite no significant baseline differences. This relationship is also shown in Figure 3.4.

Discussion and Conclusions

The majority of studies on the stress process have focused exclusively on socioeconomic factors, largely ignoring potential racial differences in experiences of stress. The data from this study provide evidence that suggests this may be true. More specifically, the findings indicate that throughout the study, chronic stressors are experienced at higher rates among Black respondents compared to their white counterparts after adjustment for income and education. However, the trajectory of chronic stressors between Blacks and Whites are similar, with nonsignificant declines (FCS, PCS) and nonsignificant increases (MCS) over time. The data further reveal that, averaged across all time points, reports of negative life events are not significantly different between Black and White subjects. Blacks experienced significantly increasing levels of negative life events than their White counterparts. The findings indicate that throughout the 15-year study, Black and White respondents were not equal in terms of exposure to chronic stress. Thus, over this observed segment of the life course Black respondents were continuously suffering and enduring higher levels of chronic stress. Likewise, as time progressed, Blacks were

more likely to experience total negative lifetime events at a faster rate, despite nonsignificant differences averaged across all four data collection time points.

Considering that there is a widespread interest to resolve racial and ethnic health disparities, it is important to consider possible mechanisms by which these disparities operate. The present study shows that levels of chronic stress and negative life events are consistently higher among this nationally representative longitudinal cohort. As has been argued here, the social status enjoyed by White Americans may facilitate their advantageous health outcomes. This social status may be another explanation of why Whites reported lower levels of stress than their Black counterparts. Social status may act as a buffer to guard against the occurrence of stressors and life events across the life course. It is also important to better understand if and how these higher rates of stressors and events may also be related to higher levels of poor health outcomes. Consistent with previous stress research, it should be assumed that higher stress levels translate into an ill impact on mental health. However, research in recent years has shown that while Blacks suffer higher poor health outcomes, they often show an advantage in mental health quality. It has been suggested by some researchers (Jackson and Knight 2006) that the effects of stressful living conditions and stressful patterns of life may manifest differently in certain populations, for example, Blacks may suffer elevated levels of physical health insults, possibly a consequence for the preservation of mental health. Geronimus et al. (2006) note: "The stress inherent in living a race-conscious society that stigmatizes and disadvantages Blacks may cause disproportionate physiological deterioration, such that a Black

individual may show the morbidity and mortality typical of a White individual who is significantly older" (p.826). Thus, while there has been a justified focus to examine health behaviors or other physical health related variables in order to explain disparities, it may be important to consider alternative pathways in which disparities work, while also moving beyond socioeconomic status as the only viable explanation. As Pearlin (1989) observed, "the various structural arrangements in which individuals are embedded determine the stressors they encounter" (p.167). It is also likely that the structural arrangements in people are embedded can also determine the nature of their physical health outcomes. These structural arrangements extend beyond income and education levels. There is a need for more discussion regarding the impossibility of health equity given the structural conditions that continue to impinge on the life experiences and chances of many adults (Jackson 2005).

Limitations

Although this study used measures of stress that were not race-specific, it is possible that the stress indices may have different meaning among and between the Black and White American research participants as no particular group is unimodal in its perception. People with stronger personal and social resources such as mastery and social support, even if poor, may better withstand the influence of chronic and acute economic stressors (Ennis, Hobfoll et al. 2000). Parental stress may also differ due to single parent households; I did not control for number of children or two parent households. Additionally, people move in and out of the role of single parenthood throughout the life course (Avison and Davies 2005). In the chronic stress models, the

Level 1 within-person variance decreased over time; likely an artifact of the model, people became more similar to one another over time. In other words, for the chronic stress measures, people were most dissimilar at the beginning of the study, and began to report more comparable levels of stress as time progressed. However, it must be remembered that Blacks had higher levels of stress across time. Finally, it is possible that the rates of recall of various stressors and life events may differ between Black and White individuals; these recall differences may under or over estimate rates for one or both of the racial groups. Also, personality characteristics such as neuroticism may lead to exaggerate reports of negative life events (Harrell, Hall et al. 2003). Future research should also consider gender and age stress differences within and between Black and White populations.

Table 3.3. Unadjusted Mean Levels of Dependent Variables for Black and White respondents at each wave

	Wave One		Wave Two		Wave Three		Wave Four	
	Black	White	Black	White	Black	White	Black	White
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Financial Chronic Stress*	.3913 (1.06)	-.1255 (1.00)	.2648 (1.05)	-.2198 (0.92)	.1887 (1.06)	-.2026 (0.95)	.0879 (0.99)	-.3453 (0.88)
	$p < .001$		$p < .001$		$p < .001$		$p < .001$	
Marital Chronic Stress	2.6693 (1.15)	2.4362 (1.01)	2.8571 (1.31)	2.5246 (1.05)	2.7542 (1.22)	2.6173 (1.06)	2.8742 (1.18)	2.5613 (1.03)
	$p < .001$		$p < .001$		$p = .063$		$p < .001$	
Parental Chronic Stress*	.1129 (1.09)	-.117 (0.99)	.1763 (1.15)	-.0578 (1.04)	.1375 (1.14)	-.1143 (0.99)	.1873 (1.08)	-.1050 (1.02)
	$p < .001$		$p < .001$		$p < .001$		$p < .001$	
Total Negative Life Events	1.76 (1.29)	1.61 (1.29)	2.73 (1.66)	2.43 (1.60)	3.69 (2.01)	3.37 (1.95)	4.98 (2.39)	4.59 (2.34)
	$p = .002$		$p < .001$		$p < .001$		$p = .003$	

*** Grand Mean Centered**

Table 3.4. Weighted Estimates of Fixed Effects; HLM Output for Financial Chronic Stress

Financial Chronic Stress	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 9733 Level-2 Units- 2982 Level-3 Units-90	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**
Fixed Effects					
Intercept[1]	-0.086626 (0.022239) 0.000*	-0.015243 (0.025246) 0.547	-0.044121 (0.036475) 0.23	-0.04595 (0.033861) 0.015*	-0.086776 (0.034192) 0.013*
Wave (Level 1)	----	-0.013538 (0.001751) 0.000**	-0.008559 (0.002531) 0.001**	-0.011748 (0.002492) 0.000**	-0.011335 (0.002592) 0.000**
Employment (Level 1)	----	----	0.036003 (0.036475) 0.324	-0.116257 (0.034573) 0.001**	-0.116577 (0.034631) 0.001**
Income (Level 1)	----	----	-0.002172 (0.000737) 0.004**	-0.002261 (0.000755) 0.003**	-0.000002 (0.000001) 0.003**
Age (Level 2)	----	----	----	-0.020524 (0.001232) 0.000**	-0.020525 (0.001232) 0.000**
Education (Level 2)	----	----	----	-0.03712 (0.007475) 0.000**	-0.037046 (0.00748) 0.000**
Gender (Level 2)	----	----	----	0.033797 (0.030049) 0.261	0.033767 (0.030056) 0.262
Race (Level 2)	----	----	----	0.263382 (0.037186) 0.000**	0.284781 (0.049036) 0.000**
Race x Wave[2]	----	----	----	----	-0.003906 (0.004548) 0.391

* $p < .05$, **$p < .01$

[1] Variance of Means in 1986
[2] Cross-Level Interaction

Table 3.5. Weighted Estimates of Random Effects; Variance Components for FinancialChronic Stress

Random Effects	Model 1	Model 2	Model 3	Model 4	Model 5
Within-Person Variance--Level 1	0.45606	0.40063[1]	0.39729[2]	0.39516	0.39513
Between-Person Variance-- Level 2 (Intercept)	0.44775	0.55011	0.52808	0.44148[3]	0.44140
Between-Person Variance-- Level 2 (Slope)	----	0.00138	0.00144	0.00142	0.00142[4]
Between-PSU Variance-- Level 3 Variance	0.02058*	0.0195*	0.01714*	0.01307*	0.01307*

*$p < 0.05$

[1] Level 1 Within-Person Variance Model 2 vs. Model 1 explained: 12.2%
[2] Level 1 Within-Person Variance Model 3 vs. Model 2 explained: 0.834%
[3] Level 2 Between-Person Intercept Variance Model 4 vs. Model 3 explained: 16.40%
[4] Level 2 Between-Person Slope Variance Model 5 vs. Model 4 explained: 0.00%

Table 3.6 Weighted Estimates of Random Effects; HLM output for Marital Chronic Stress

Marital Chronic Stress	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 5448 Level-2 Units- 1754 Level-3 Units-90	β **(SE)** **p-value**	β **(SE)** **p-value**	β **(SE)** **p-value**	β **(SE)** **p-value**	β **(SE)** **p-value**
Fixed Effects					
Intercept[1]	2.63673 (0.028652) 0.000**	2.605349 (0.029543) 0.000**	2.543607 (0.037225) 0.000**	2.440615 (0.046345) 0.000**	2.44064 (0.046431) 0.000**
Wave (Level 1)	----	0.006433 (0.002126) 0.003**	0.007946 (0.002144) 0.000**	0.006429 (0.002203) 0.004**	0.006423 (0.003531) 0.005*
Employment (Level 1)	----	----	0.080474 (0.039385) 0.041*	0.001307 (0.04173) 0.975	0.001314 (0.041755) 0.975
Income (Level 1)	----	----	-0.000329 (0.000145) 0.023*	-0.000447 (0.000135) 0.001**	-0.000447 (0.000135) 0.001**
Age (Level 2)	----	----	----	-0.010804 (0.002066) 0.000**	-0.010804 (0.002066) 0.000**
Education (Level 2)	----	----	----	0.023776 (0.009233) 0.010**	0.023775 (0.009239) 0.010*
Gender (Level 2)	----	----	----	0.047557 (0.130015) 0.007**	0.130016 (0.047556) 0.007*
Race (Level 2)	----	----	----	0.318221 (0.066655) 0.000**	0.317842 (0.071422) 0.000**
Race x Wave[2]	----	----	----	----	-0.000086 (0.006161) 0.989

* p < .05, **p < .01

[1] Variance of Means in 1986
[2] Cross-Level Interaction

Table 3.7. Weighted Estimates of Random Effects; Variance Components for Marital Chronic Stress

Random Effects	Model 1	Model 2	Model 3	Model 4	Model 5
Within-Person Variance--Level 1	0.47854	0.41957[1]	0.42041[2]	0.41986	0.41986
Between-Person Variance--Level 2 (Intercept)	0.62569	0.68193	0.67565	0.63279[3]	0.63279
Between-Person Variance--Level 2 (Slope)	----	0.00176	0.00173	0.00172	0.00172[4]
Between-PSU Variance--Level 3 Variance	0.02174*	0.02028*	0.02014*	0.0183*	0.01830*

*$p < 0.05$

[1] Level 1 Within-Person Variance Model 2 vs. Model 1 explained: 12.32%
[2] Level 1 Within-Person Variance Model 3 vs. Model 2 explained: -0.200%
[3] Level 2 Between-Person Intercept Variance Model 4 vs. Model 3 explained: 6.34%
[4] Level 2 Between-Person Slope Variance Model 5 vs. Model 4 explained: 0.00%

Table 3.8. Weighted Estimates of Fixed Effects; HLM Output for Parental Chronic Stress

Parental Chronic Stress Level-1 Units- 8176 Level-2 Units- 2555 Level-3 Units-90	**Model 1** β **(SE)** **p-value**	**Model 2** β **(SE)** **p-value**	**Model 3** β **(SE)** **p-value**	**Model 4** β **(SE)** **p-value**	**Model 5** β **(SE)** **p-value**
Fixed Effects					
Intercept[1]	0.003086 (0.025375) 0.904	0.039467 (0.029819) 0.189	-0.023405 (0.038382) 0.543	-0.00363 (0.048493) 0.941	-0.001156 (0.048611) 0.981
Wave (Level 1)	----	-0.00693 (0.002413) 0.005**	0.000084 (0.000184) 0.012*	-0.008153 (0.002571) 0.002**	-0.008648 (0.002764) 0.002**
Employment (Level 1)	----	----	0.03086 (0.091582) 0.003**	0.00686 (0.0366) 0.852	0.007095 (0.036519) 0.846
Income (Level 1)	----	----	0.000184 (0.000084) 0.649	0.000014 (0.000169) 0.936	0.000017 (0.000168) 0.919
Age (Level 2)	----	----	----	-0.009449 (0.001548) 0.000**	-0.009454 (0.001549) 0.000**
Education (Level 2)	----	----	----	-0.003804 (0.0082) 0.642	-0.003864 (0.008205) 0.637
Gender (Level 2)	----	----	----	-0.070908 (0.043245) 0.101	-0.070917 (0.043243) 0.101
Race (Level 2)	----	----	----	0.202894 (0.045285) 0.000**	0.179937 (0.050376) 0.001**
Race x Wave[2]	----	----	----	----	0.004498 (0.005191) 0.387

* p < .05, **p < .01

[1] Variance of Means in 1986
[2] Cross-Level Interaction

Table 3.9. Weighted Estimates of Random Effects; Variance Components for Parental Chronic Stress

Random Effects	Model 1	Model 2	Model 3	Model 4	Model 5
Within-Person Variance--Level 1	0.52907	0.44504[1]	0.44579[2]	0.44539	0.44543
Between-Person Variance--Level 2 (Intercept)	0.50377	0.61107	0.60419	0.58908[3]	0.58902
Between-Person Variance--Level 2 (Slope)	----	0.00238	0.00236	0.00234	0.00234[4]
Between-PSU Variance--Level 3 Variance	0.02593*	0.02636*	0.02542*	0.027*	0.027*

*$p < 0.05$

[1] Level 1 Within-Person Variance Model 2 vs. Model 1 explained: 15.88%
[2] Level 1 Within-Person Variance Model 3 vs. Model 2 explained: -0.169%
[3] Level 2 Between-Person Intercept Variance Model 4 vs. Model 3 explained: 2.501%
[4] Level 2 Between-Person Slope Variance Model 5 vs. Model 4 explained: 0.00%

Table 3.10. Weighted Estimates of Fixed Effects; HLM Output for Total Negative Life Events

Total Life Events	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 10021 Level-2 Units- 3023 Level-3 Units-90	**β (SE) p-value**	**β (SE) p-value**	**β (SE) p-value**	**β (SE) p-value**	**β (SE) p-value**
Fixed Effects					
Intercept[1]	2.668529 (0.053602) 0.000**	1.595665 (0.042337) 0.000**	1.632281 (0.044499) 0.000**	1.645999 (0.070146) 0.000**	1.639892 (0.070345) 0.000**
Wave (Level 1)	----	0.004326 (0.206979) 0.000**	0.207814 (0.004309) 0.000**	0.207549 (0.004308) 0.000**	0.191686 (0.00566) 0.000**
Employment (Level 1)	----	----	-0.054577 (0.031415) 0.082	-0.019022 (0.029988) 0.526	-0.028878 (0.030539) 0.345
Income (Level 1)	----	----	-0.000842 (0.00027) 0.002**	-0.000784 (0.00026) 0.003**	-0.000805 (0.000267) 0.003**
Age (Level 2)	----	----	----	0.005197 (0.002008) 0.010**	0.00525 (0.002007) 0.009*
Education (Level 2)	----	----	----	-0.032714 (0.010532) 0.002**	-0.032598 (0.010534) 0.002**
Gender (Level 2)	----	----	----	0.034846 (0.066402) 0.599	0.035198 (0.066443) 0.596
Race (Level 2)	----	----	----	0.075781 (0.08214) 0.357	0.114145 (0.084571) 0.177
Race x Wave[2]	----	----	----	----	0.030819 (0.008118) 0.000**

* $p < .05$, **$p < .01$

[1] Variance of Means in 1986
[2] Cross-Level Interaction

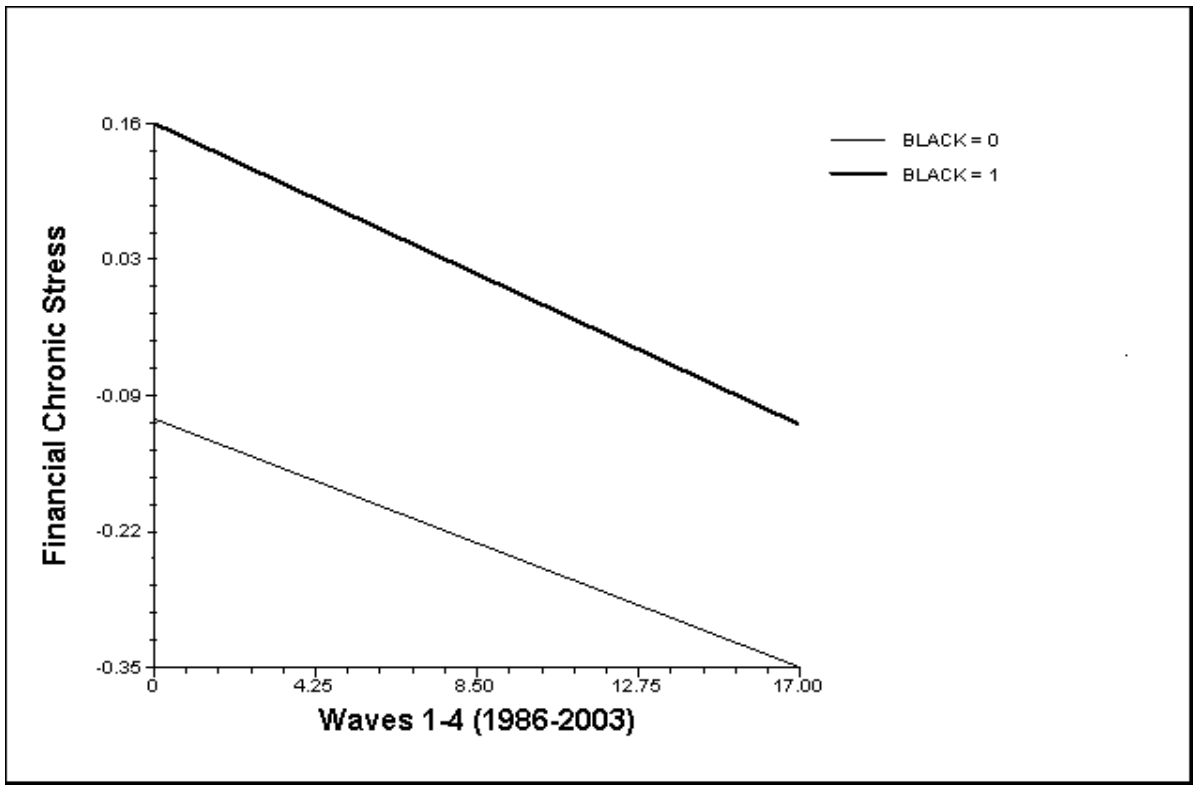
Table 3.11. Weighted Estimates of Random Effects; Variance Components for Total Negative Life Events

Random Effects	Model 1	Model 2	Model 3	Model 4	Model 5
Within-Person Variance--Level 1	2.36462	0.31663[1]	0.31741[2]	0.31702	0.31674
Between-Person Variance--Level 2 (Intercept)	1.91169	1.59415	1.58006	1.55668[3]	1.55693
Between-Person Variance--Level 2 (Slope)	----	0.01336	0.01328	0.0133	0.01323[4]
Between-PSU Variance--Level 3 Variance	0.13268*	0.06415*	0.0647*	0.07272*	0.07270*

*p <0.05

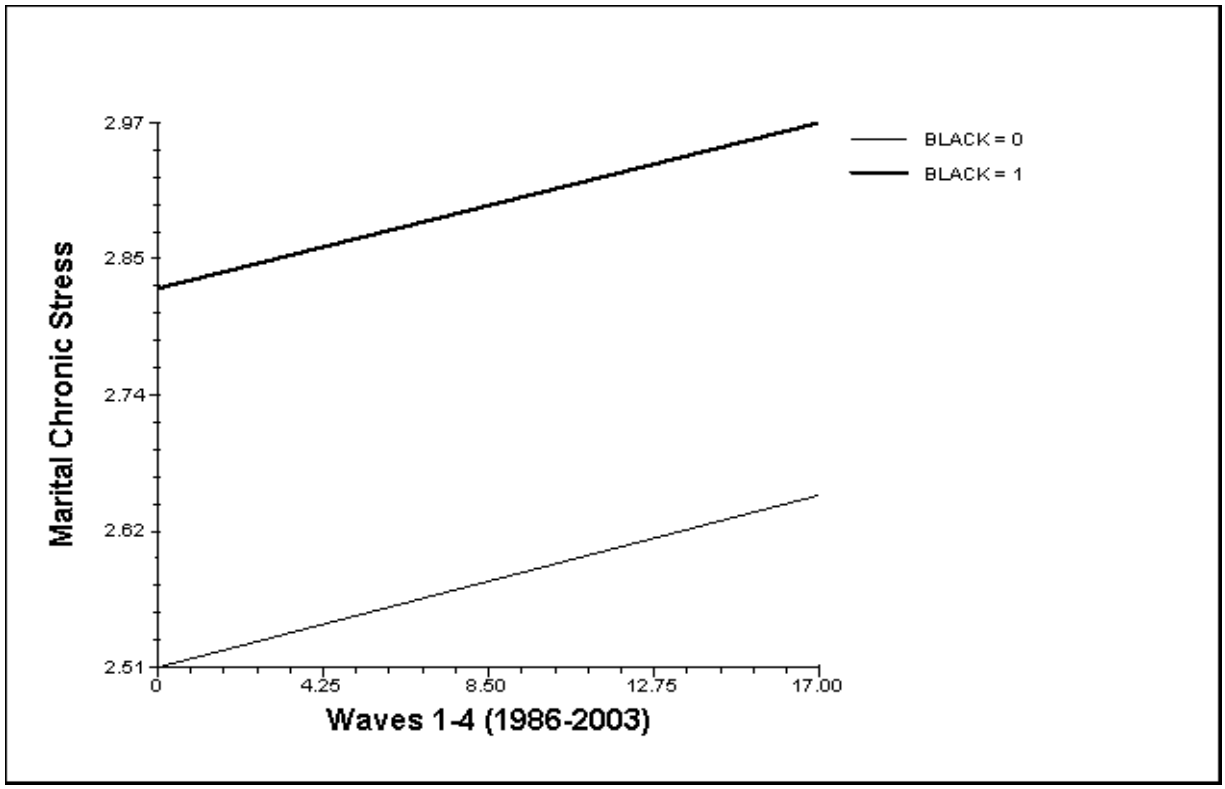
[1] Level 1 Within-Person Variance Model 2 vs. Model 1 explained: 86.6%
[2] Level 1 Within-Person Variance Model 3 vs. Model 2 explained: -0.246%
[3] Level 2 Between-Person Intercept Variance Model 4 vs. Model 3 explained: 1.480%
[4] Level 2 Between-Person Slope Variance Model 5 vs. Model 4 explained: 0.0275%

Figure 3.1. Trajectory and Levels of Financial Chronic Stress (FCS) between Black and White Respondents[1] across all waves.



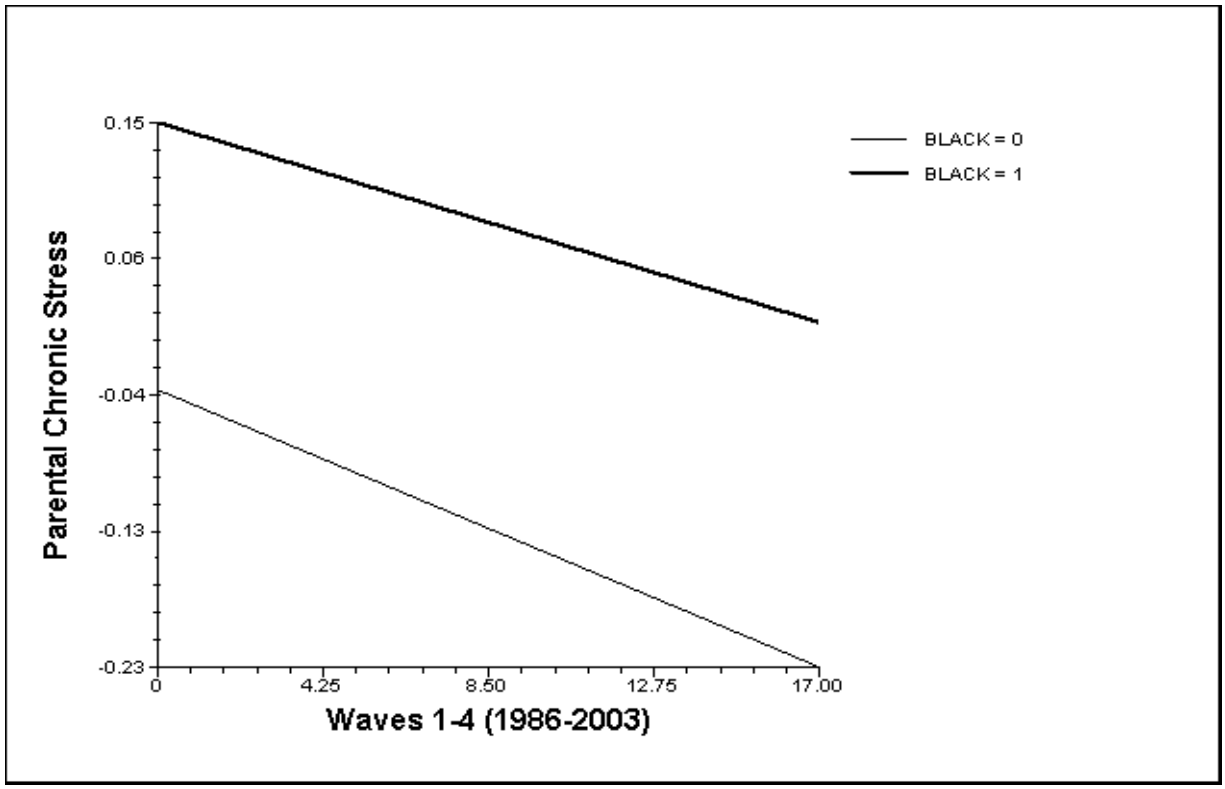
[1] Black = 1: Black Respondents; Black = 0: White Respondents

Figure 3.2. Trajectory and Levels of Marital Chronic Stress (MCS) between Black and White[1] Respondents across all waves.



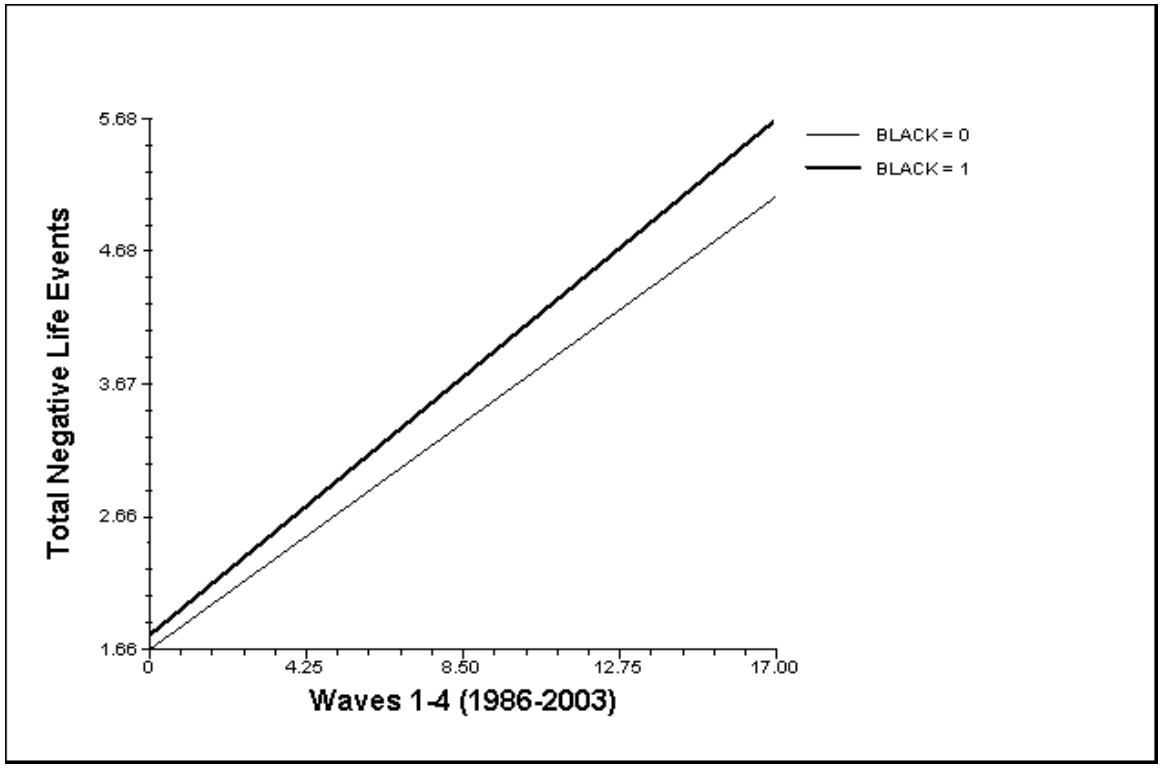
[1] Black = 1: Black Respondents; Black = 0: White Respondents

Figure 3.3. Trajectory and Levels of Parental Chronic Stress (PCS) between Black and White[1] Respondents across all waves.



[1] Black = 1: Black Respondents; Black = 0: White Respondents

Figure 3.4. Trajectory and Levels of Total Negative Life Events (TOTLE) between Black and White[1] Respondents across all waves.



[1] Black = 1: Black Respondents; Black = 0: White Respondents

Appendices

Appendix A. Americans' Changing Lives Response Rates for Survivors.

	Sample Size	Number Dead by End of Wave	Response Rate of Survivors
Wave One	3617	---	---
Wave Two	2867	178	83%
Wave Three	2562	544	83%
Wave Four	1787	1184	73%

Appendix B. Unweighted Descriptive Statistics of Level One Variables for Persons Loss to Follow-Up after Wave One

		Characteristics of Persons Loss to Follow Up after Wave One	p-value
		(%)	
Employed	**Race (n)**		
	Black (81)	(39.7)	0.063
	White (85)	(31.5)	
		Mean (SD)	
Income (Dollars)	**Race (n)**		
	Black (204)	13,901(13,896)	0.039*
	White (270)	16,651 (14,565)	
Body Mass Index			
	Black (204)	26.02 (5.1)	0.003**
	White (270)	24.7 (4.8)	
Alcoholic Drinks per Day			
	Black (79)	3.49 (3.8)	0.006**
	White (124)	2.31 (2.3)	
Cigarettes per Day			
	Black (204)	6.04 (10.1)	0.919
	White (270)	6.14 (11.3)	
Financial Chronic Stress			
	Black (204)	.473 (1.1)	0.000**
	White (270)	-.138 (1.1)	
Marital Chronic Stress			
	Black (63)	2.65 (1.1)	0.013*
	White (145)	2.2 (1.1)	
Parental Chronic Stress			
	Black (153)	-1.43 (1.2)	0.000**
	White (223)	-.261 (1.1)	
Life Events			
	Black (204)	1.02 (.86)	0.773
	White (270)	.90 (.82)	

*p < .05, **p < .01

Appendix C. Unweighted Descriptive Statistics of Level 2 Variables for Persons Loss to Follow-Up after Wave One

		Race	Gender (Female)	Education	Age
		n	%	Mean (SD)	Mean (SD)
	Black	204	59	9.99 (4.1)	55.6 (18.9)
	White	270	54	10.86 (3.2)	62.7 (18.7)
	p-value	---	---	0.000**	0.001**

*p < .05, **p < .01

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CHAPTER FOUR

The relationship between health and chronic stress and negative life events, among a sample of African American and White American adults, measured four times, over a sixteen-year time span.

Introduction

For nearly the past 50 years, the United States has made considerable gains in decreasing the black-white gap in education, income, and civil rights. However, large disparities still persist in mortality and morbidity between these two racial/ethnic groups. There has been a continued effort to explore and dissect the reasons and explanations of why these disparities still exist. A variety of hypotheses have been advanced, and many are likely to be very useful pieces to the puzzle of racial and ethnic health disparities. More often, socioeconomic factors, rather than race, have been cited as the culprit for mortality, and other health-related outcomes. Despite the preoccupation of the public health field with socioeconomic status (Kaplan and Lynch 1997, p. 206), it is important to explore the role of race and other psychosocial health determinants, when considering current American racial and ethnic health disparities.

One such psychosocial determinant of health, stress, has been very useful in better understanding the health of minority populations. As Williams et al. (1997) note, racial differences in health can be affected by stress in two distinct ways. First, stress is not randomly distributed in the population, it is linked to social structure, and these

in turn determine the types and amounts of stress to which an individual is exposed. Thus, it can be concluded that the structural location of blacks in society may lead to higher levels of stress and more negative outcomes to stress than whites. Second, the experience of specific incidences of race-related distress can lead to psychological distress, which in turn may impact physical health. This paper is concerned with the former of these two mechanisms of how stress affects health. Any attempt to examine health disparities between Whites and other minority groups must consider status placement and the resulting correlates. The purpose of this study is two-fold; first, to examine if there are racial differences in levels of self-rated health and functional health over a segment of the life course, and second, to assess if reports of chronic stressors and negative life events are associated with the physical health outcomes of Black and White respondents.

Background

The concept of stress has been studied by many disciplines, and thus has acquired numerous interpretations, and multiple layers of meaning. Despite the resulting "conceptual integration" (Pearlin 1975, p. 345), the concept of stress is very helpful when trying to better understand the context in which people live, and the many experiences that occur over the life course. Stress can expose the psychological interior of a person and reveal the social circumstances and environments of an individual. For the current study, the conceptualization of stress will be drawn from the work of Pearlin, Schieman et al. (2005). Thus, the term stress will be operationalized as the "dogged hardships, demands, conflicts, and frustrations that

may be instrumental in structuring people's experiences across time and to events that may disrupt the continuities of their lives" (Pearlin, Schieman et al. 2005, p. 206). Many researchers have considered the impact of chronic stressors that are associated with and arise out of major social roles and role sets (Pearlin 1989). Examples of these roles are wife/husband, worker/supervisor, and parent/child (Pearlin 1989). Expanded research is needed to better understand how the role set of being a Black (or White) American impacts the experience and health consequences of chronic stressors and negative life events. In a review paper describing the distribution of traumatic and other stressful life events, Hatch and Dohrenwend (2007) present ten research studies that examined race differences and were published during the years of 1967-1980 and 1989-2005. Among these ten studies, African Americans were shown to report higher levels of traumatic and other stressful life events in seven of the ten publications. The otherwise surprising absence of studies on racial differences in exposure to stress has been noted by others (Brown, Sellers et al. 1999; Turner and Avison 2003).

However, in recent years there has been an explosion of research demonstrating that various measures of stress are related to physical and psychiatric measures of health. For example, stressful life events and chronic strains are significantly associated with the incidence of depressive symptoms (Avison and Turner 1988; Moos, Schutte et al. 2005) and the progression from HIV to AIDS (Leserman, Jackson et al. 1999). Chronic exposure to stressors can lead to dysregulation across multiple physiological systems and biological cascades of the human body; such as increasing

proinflammatory cytokine IL-6 among cases infected with the Influenza A virus (Cohen, Doyle et al. 1999). The use of subjective measures of health such as, self-rated and functional health, are another pathway to study the relationship between health and stress.

Self-rated health is a subjective appraisal that is highly correlated with other health indicators and has been shown to be a powerful indicator of subsequent mortality, even after controlling for chronic illness and functional limitations (Fayers and Sprangers 2002; Kahn and Fazio 2005). Self-rated health is an individual's perception of his or her general well-being and quality of life; it is not an objective evaluation of the absence of disease (Cummings and Braboy Jackson 2008). Psychosocial and material conditions as well as lifestyle factors (i.e. smoking, drinking, obesity) are related to self-rated health. Functional health represents the presence or lack of physical health impairments that result from disease and disability. Many discussions of the association between race and functional health have also considered the effects of age. Thus, overall past research has shown that large racial differences in mean functional capacity are reduced when social class is controlled and when socioeconomic status covariates are not controlled, Black/White differences are found to be age dependent (Clark, Maddox et al. 1993). In a study of HIV infected patients, patients reporting higher levels of stress and trauma, also reported lower levels of physical health functioning (Leserman, Whetten et al. 2005). However, there is a dearth of research that describes the patterns of race and stress and associated health outcomes. Race has been a key indicator of differential access to societal resources

and rewards, and health status is no exception (Williams 2005). Further, there is a need to move empirically from collecting and analyzing primarily cross-sectional and short-term longitudinal data to developing and analyzing long-term prospective studies of multiple indicators of health in representative national samples (House, Lantz et al. 2005). The present study was conducted to provide additional data on associations between race, stress, and physical health outcomes. This study assessed these associations among a sample of Black and White Americans. Also, this study is unique because it utilizes a 15-year longitudinal sample, and fundamentally considers whether physical health outcomes are racially patterned, and assesses whether stress differentially affects these physical health outcomes.

The primary aim of the present study was therefore to assess the effect of race on two physical health outcomes, functional health and self-rated health, at baseline and over time among a cohort of White and Black study participants. A secondary aim was to evaluate whether the moderating role of certain stress and negative life events (i.e. financial, marital, and parental chronic stress and negative total life events) on the relationship between the two racial groups and functional health and self-rated health. It was expected that Black respondents would report less favorable levels of health than their White counterparts when averaged across time, and that the slope over time would differ between Black and White participants. In addition, it was hypothesized that if stress negatively impacted health outcomes, it would do so at a greater rate for Black than White respondents.

Data and Methods

Data

American's Changing Lives is a long-term four-wave cohort longitudinal study that was conducted by the University of Michigan Survey Research Center and began in 1986. The study investigators intended to construct an artificial representation of aging over the entire life course by connecting the experiences of these four cohorts over the 15 year time period of the study. The second data collection time was in 1989, and then was repeated in 1994, and 2001-2003.

Sample

ACL is a stratified, multistage area probability sample of noninstitutionalized adults aged 25 and older living in the coterminous United States. At Wave One, two groups were oversampled: African Americans and adults aged 60 and older. As mentioned, Wave One, conducted in 1986, included 3,617 face-to-face interviews, which represented 70% of sampled households and 68% of sampled individuals. In 1989, Wave Two represented 83% of Wave One survivors or 2,867 face-to-face interviews. In 1994, Wave Three interviewees represented 83% of Wave One survivors or 2562 respondents. These interviews were conducted either by telephone or face-to face interviews, and also included proxy interviews. Wave Four interviews began in 2001 and concluded in 2003. These 1787 respondents represented 49% of baseline survivors. Much of the Wave Four nonresponse was due to mortality, with 1184 or 33% of the original respondents dead by Wave Four implementation. As with Wave Three, the last data collection period entailed face-to-face and telephone interviews,

which were supplemented with proxy interviews when appropriate. The study included continuous mortality tracking via the National Death Index and other methods yielding over 99% mortality ascertainment, with over 97% of deaths confirmed via death certificates.

Measures

Demographic and Socioeconomic Characteristics

The relationships between race, physical health, and stress were evaluated after the effects of gender, education, age, employment and income had been controlled statistically. Individual characteristics measured at baseline include gender (male =1), education (number of years of schooling), race (Black =1; White =0), while continuous income, age and employment status (employed=1) were measured at each wave.

Independent Variables

Financial Chronic Stress. This scale is comprised from responses to two questions: 1- "How satisfied are you with your/your family's present financial situation?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all) and 2- "How difficult is it for you/your family to meet monthly payments on your bills?" (5-point response scale with 1 = extremely difficult and 5 = not difficult); this variable was reverse coded. High values indicate a higher level of Financial Chronic Stress for the respondent. This standardized index was constructed by taking the arithmetic mean of the two questions.

Parental Chronic Stress. This scale was created from responses to three questions: 1-"At this point in your life, how satisfied are you with being a parent?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all); 2-"How often do you feel bothered or upset as a parent?" (5-point response scale with 1 = almost always and 5 = never), this variable was reverse coded; and 3-"How happy are you with the way your child/children have turned out to this point?" (5-point response scale with 1 = very happy and 5 = not at all happy). High values indicate a higher level of Parental Chronic Stress, and obviously responses were only available from respondents who had children. Responses to this scale were standardized and then averaged to create this index.

Marital Chronic Stress. This scale was created from responses to three questions: 1-"Taking all things together, how satisfied are you with your (marriage/relationship)?" (5-point response scale with 1 = completely satisfied and 5 = not satisfied at all), this item was reverse coded; 2-"How often would you say the two of you typically have unpleasant disagreements or conflicts?" (7-point response scale with 1= daily or almost daily, 2 = 2 or 3 times a week, 3 = about once a week, 4 = 2 or 3 times a month, 5 = about once a month, 6 = less than once a month, and 7 = never); and 3-"Taking everything into consideration how often do you feel bothered or upset by your (marriage/relationship)?" (5-point response scale with 1 = almost always and 5 = never). Cases missing on 2 or 3 of the three input variables at a given wave were imputed using a simple ordinal least squares regression prediction model, with no random residuals. Three cases were imputed for the Wave 1 index and 3 cases were

imputed for the Wave 3 index. There were no missing cases on the indices at either Wave 2 or Wave 4.

Total Life Events. A continuous scale was created to assess the number lifetime and recent life events experienced by the respondents. At baseline, respondents were asked if they had ever been widowed, divorced (or had a marriage annulled), had a child die, or been the victim of a serious physical attack or assault at any time in their life. These four events were considered events that may have happened to the respondent at any point in their life prior to baseline. At baseline, and every subsequent wave, respondents were also asked about recent negative events. The five recent negative events that could have occurred in the previous three years were death of a parent/step parent, death of a close friend/relative, involuntary loss of a job (excluding retirement), being robbed or burglarized, or having any other bad thing occur that greatly upset the respondent. At baseline, lifetime events were summed with recent negative events, and at each subsequent wave, these scores were added to that wave's reported recent negative events, thus a cumulative continuous score was created for each wave.

Dependent Variables

Functional Health. Functional health was assessed by responses to six questions: "1) Are you currently in bed or in a chair most or all of the day because of your health? (Yes or No), 2) Do you currently have any difficulty bathing by yourself? (Yes or No), 3) How much difficulty do you have climbing stairs? (1= A little and 4 = Cannot do), 4) Do you currently have any difficulty walking several blocks because of your

health? (1= A little and 4 = Cannot do), 5) Would you currently have any difficulty doing heavy work around the house such as shoveling snow or washing walls, because of your health? (Yes or No), and 6) If yes, how much?" A functional health index was then formed by creating a Guttman-type scale[1](De Souza 1999) which was formed with the following levels of functional impairment: 1) Most severe level = respondents who are currently in bed or chair and/or who have a lot of difficulty bathing or cannot bathe, 2) Moderately severe = respondents who have a lot of difficulty walking or cannot do it and/or have a lot of difficulty climbing stairs or cannot do it but were not in previously defined severity level, 3) Least severe level = respondents who have a lot of difficulty doing heavy housework or cannot do it but who are not in two previously defined severity levels, and 4) No functional impairment = respondents answered no to all of the functional impairment questions. A higher score indicates better health.

Self-Rated Health. Self-rated health was assessed by a single question asked of respondents: "How would you rate your health at the present time? Would you say it is excellent (1), very good (2), good (3), fair (4), or poor (5)?"

Statistical Analysis

Descriptive statistics were calculated to assess sample characteristics at each wave (Tables 4.1 and 4.2). Hierarchical Linear Modeling (Raudenbush 2004) was used in

[1]A Guttman Scale consists of individual items ranked in order of difficulty so that all patients pass or fail the items in the same order. Thus, when an assessment of the Guttman type is used, testing can be terminated when an item has been failed. This is because the scale implies that all items above the failed one will also be failed by a statistically significant number of subjects. (De Souza, L.H. The Development of a scale of the Guttman type for the assessment of mobility disability in multiple sclerosis. Clinical Rehabilitation, Dec. 13; (6): 476-81. 1999.)

order to examine physical health outcomes over time between Black and White respondents, as well as how levels of chronic stress and negative life events influenced these outcomes. In a longitudinal HLM framework, interviews are nested within individuals. In such a model, estimates of effects are estimated for each individual from data contributed over the course of the study. Individual intercepts and slopes (changes across time) are estimated from available data. A distinct advantage of the HLM approach is that it allows for modeling within-individual variation over time. In addition, it handles missing and unbalanced data better than other statistical procedures typically used with repeated-measures data. Hierarchical relationships occur when variables at one level of analysis influence, or are influenced by, variables at another level of analysis (Hofmann 1997). In this case, a three-level hierarchical linear model was employed to simultaneously investigate the associations between physical health outcome and race over four waves of data spanning 15 years, while also acknowledging the complex sample survey design of the study. The Level 1 Model was the within-person model, Level 2, was the between-person model, while Level 3 consisted of strata and study clusters.

Table 4.1. Waves 1-4 Unweighted Descriptive Statistics of Level One Variables

		Wave 1	Wave 2	Wave 3	Wave 4
		n (%)	n (%)	n (%)	n (%)
Employed					
	Black	597 (50.9)	432 (49.4)	340 (46.0)	196 (44.3)
	White	1200 (51.7)	981 (51.5)	862 (49.2)	636 (49.3)
		n Mean (SD)	n Mean (SD)	n Mean (SD)	n Mean (SD)
Income					
	Black	1174 15663.89 (15513.61)	874 17914.44 (19629.79)	739 23558.91 (23688.29)	445 37620.08 (45315.78)
	White	2323 25243.81 (19625.68)	1906 33340.08 (38206.07)	1752 38691.71 (43593.26)	1292 58773.95 (102861.7)
Body Mass Index					
	Black	1174 27.24 (5.7)	874 27.58 (5.9)	739 28.17 (6.0)	445 29.46 (6.5)
	White	2323 25.45 (4.6)	1906 25.7 (4.7)	1752 26.1 (5.0)	1292 26.85 (5.2)
Alcoholic Drinks per Day					
	Black	1057 1.22 (2.2)	800 1.01 (2.0)	702 .69 (1.3)	412 .70 (1.6)
	White	2104 1.32 (1.8)	1704 1.12 (1.5)	1667 1.04 (1.6)	1174 .95 (1.4)
Cigarettes per Day					
	Black	1170 5.02 (9.1)	874 4.41 (8.6)	736 2.84 (7.0)	445 2.29 (6.4)
	White	2319 5.63 (10.9)	1906 4.57 (10.1)	1747 3.65 (8.9)	1292 2.35 (6.9)
Financial Chronic Stress					
	Black	1174 .398 (1.1)	874 .265 (1.1)	683 .189 (1.1)	415 .088 (.99)
	White	2323 -.128 (1.0)	1906 -.220 (.92)	1648 -.203 (.95)	1231 -.345 (.88)
Marital Chronic Stress					
	Black	497 2.70 (1.2)	377 2.87 (1.3)	305 2.76 (1.2)	167 2.87 (1.2)
	White	1493 2.4 (1.0)	1233 2.53 (1.1)	1074 2.6 (1.1)	795 2.57 (1.0)
Parental Chronic Stress					
	Black	983 .113 (1.1)	748 .176 (1.2)	592 .138 (1.1)	370 .187 (1.1)
	White	1939 -.117 (.99)	1608 -.058 (1.0)	1392 -.114 (.99)	1035 -.105 (1.0)
Life Events					
	Black	1174 1.79 (1.3)	874 2.73 (1.7)	739 3.69 (2.0)	441 4.98 (2.4)
	White	2323 1.62 (1.3)	1906 2.43 (1.6)	1752 3.37 (1.9)	1291 4.59 (2.3)

Table 4.2. Waves 1-4 Unweighted Descriptive Statistics of Level Two Variables

Baseline (Wave One)		Race	Gender (Female)	Education	Age*
		n %	n %	n Mean (SD)	n Mean (SD)
	Black	1174 33.6	716 61	1174 10.39 (3.7)	1174 52.6 (17.3)
	White	2323 66.4	1540 66.3	2323 12.04 (3.1)	2323 54.61 (17.7)

* Despite this variable being measured at each wave, age was considered a Level Two variable due to its' high correlation with the time (wave) variable, and only baseline age was considered in subsequent analyses.

The primary aim of the present study was therefore to assess the effect of race on two physical health outcomes, functional health and self-rated health, at baseline and over time among a cohort of White and Black study participants. A secondary aim was to evaluate whether race moderates the association between stress and health. The dependent variable is the score on the outcome measure (physical health and self-rated health) at each study time point. The independent variables controlled for level-1 (time-varying) predictors, such as employment, income, daily alcohol consumption, body mass index, and number of cigarettes smoked per day and level-2 (time-invariant person-specific) predictors, such as age, education, and gender. The predictors of primary interest in this analysis were measured both at level-1 (stress measures) and at level-2 (race). Cross-level interactions were included in the model to allow the effects of the stress variables to differ across racial groups, and the Level-1 interaction between stress and wave was included to allow the slope of the stress variable to differ across time. .

The first step in the multilevel analysis was to fit a 'null model' (Model 1) to estimate

the amounts of variance available to be explained at each level of the hierarchy (Raudenbush 2002). Each null model contained only the dependent variable (Functional Health or Self-Rated Health), random errors at Level 1, random person effects at Level 2, and random cluster effects at Level 3; no predictor variables were specified at any level. The second step (Models 2 and 3) was to build up the Level-1 (within-person) model. This involved adding level-1 predictors to the model, but without entering predictors at the second level. This step allowed the opportunity to examine which of the level-1 variables had a significant relationship with on Functional Health or Self-Rated Health. The next step (Model 4) was to add the Level-2 variables, including race and other control variables. This step allowed for a comparison of the levels of physical health averaged across time between blacks and whites. In Model 5 an interaction was included between race and wave to assess the differences in the trajectories of health across time for black vs. white participants; equivalently, Model 5 can be used to explore whether race explains between-person variance in changes in the outcomes over time. To explore the secondary aim: the to assess the differential impact of specific chronic stressors or negative life events on physical health between Black and White respondents, the chronic stress and negative life events variables were added to the each of the functional health and self-rated health models to specify Model 6. In the next step, Model 7 included the two-way interaction terms: race by stress (to see if there was a differential impact of stress for black vs. white participants), and time (wave) by stress (to see if the slope over time was impacted by levels of stress). In the final step, Model 8, the three-way interaction term race by stress by wave was added to see if the possibly differential effect of

stress on health over time differed by racial groups. The set of Models 1 through 8 was fitted for each of the two health outcomes (functional health and self-rated health) and was repeated for each of the stress indicators (financial, marital, and parental chronic stress and total negative lifetime events).

Level-1 Model

$$Y_{ijK} = \pi_{0jK} + \pi_{1jK}(\text{EMPLOY}_{ijK}) + \pi_{2jK}(\text{WAVE}_{ijK}) + \pi_{3jK}(\text{INCOME\_T}_{ijK} - \mu\text{INCOME\_T}_{\text{ijK...}}) + \pi_{4jK}(\text{SMOKE}_{ijK}) + \pi_{5jK}(\text{DRINK}_{ijK}) + \pi_{6jK}(\text{BMI}_{ijK} - \mu\text{BMI}_{\text{ijK...}}) + \pi_{7jK}(\text{STRESS}_{ijK}) + \pi_{8jK}(\text{WAVE}_{ijK} * \text{STRESS}_{ijK}) + e_{ijK}$$

Where Y_{ijK} is the dependent variable, measured at time *i* on person *j* within sampling cluster *k*, and π_{0jK} is the intercept (expected mean) functional health or self-rated health rating for respondent *j* in sampling cluster *k* at baseline (wave=0), who is unemployed, has an average income, smokes zero cigarettes per day, drinks zero alcoholic beverages per day, has an average body mass index, and has either an average chronic stress level or zero total negative lifetime events. Recall that both financial chronic stress and parental chronic stress are standardized measures, while marital chronic stress was centered when it was included in the model, and total negative lifetime events was not centered or standardized. The term e_{ijK} is the residual or error.

Level-2 Model

$$\pi_{0jk} = \beta_{00k} + \beta_{01k}(\text{AGE}_{jk} - \mu\text{AGE..}) + \beta_{02k}(\text{EDUC}_{jk} - \mu\text{EDUC..}) + \beta_{03k}(\text{FEMALE}_{jk}) + \beta_{04k}(\text{BLACK}_{jk}) + r_{0jk}$$

$$\pi_{2jk} = \beta_{20K} + \beta_{24K}(\text{BLACK}_{jK}) + \pi_{7jK}(\text{STRESS}_{ijK}) + r_{2jK}$$

$\pi_{7jk} = \beta_{24K}(\text{BLACK}_{jK}) + r_{2jK}$

$\pi_{8jk} = \beta_{24K}(\text{BLACK}_{jK}) + r_{2jK}$

In the level-2 model, the level-1 intercept $\pi_{0jk}$is now a dependent variable, β_{00k} is the level-2 intercept for sampling cluster *k*, and r_{0jk} is a level-2 random effect associated with the *j*-th respondent from sampling cluster *k*. In this equation, β_{00k} represents the grand mean of the outcome at baseline, or the mean of the intercepts for all individuals within sampling cluster *k*. The random effect r_{0jk} represents the deviation of each individual's mean from the grand mean. When the variance of these random effects is large, there are large differences between individuals at baseline. The coefficient $\beta_{04k}$is the expected gap in functional health or self-rated health levels between Blacks and Whites at baseline in sampling cluster *k*, adjusted for employment, income, education, age and gender. The equation beginning with the intercept π_{2jk}represents two two-way interaction terms between wave and race and wave and stress. The equation beginning with the intercept π_{7jk}represents the two-way interaction between stress and race, while the equation beginning with the intercept beginning with π_{8jk}represents the three-way interaction term between race, stress, and wave.

Level-3 Model

$\beta_{00k} = \gamma_{000} + u_{00k}$

Where β_{00k} is the overall grand mean of the outcome at baseline across all sampling clusters, and u00k is the random effect associated with the *k-th* sampling cluster

(capturing between-cluster variance). All other coefficients in the Level-2 model are assumed to be fixed at Level 3 (i.e., there are no additional random cluster effects). The current study uses all available four waves of data to model trends of physical health over time between racial groups. Hierarchical linear models were run separately for functional health and self-rated health outcome, using the HLM software v.6.06 (Raudenbush 2004). In addition to the attrition and nonresponse discussed above, individuals who did not respond on the outcome variable at two or more waves were excluded. This was to ensure that a slope would be calculated for all respondents, allowing comparisons of physical health over time. Also, individuals who reported their race and/or ethnicity as other than Black or White were excluded.

Two of the level-1 predictor variables (income and body mass index (BMI)) and four of the level-2 predictor variables, age, education, income and chronic stress were grand centered so that the intercept term would represent the average score for the respondents. All analyses incorporated sampling weights to account for the complex sample survey design. In conclusion, three-level hierarchical models were employed to specifically investigate two primary analysis outcomes: first, a baseline comparison of physical health between Black and White respondents and secondly, model the slopes of functional health and self-rated health for Black and White respondents over four waves of data spanning 15 years. The secondary analysis outcome was to assess the influence of chronic stress and negative life events on physical health between the two racial groups.

HLM also provides a chi-square test for the two residual variances. These chi-square tests indicate whether the variance components differ significantly from zero and provide a direct test of necessary conditions 2 and 3. In other words, these tests determine whether the variance in the intercepts and slopes across groups is significantly different from zero.

The remainder of this paper is organized as follows. The results section is separated into two parts for each of the two study outcomes: Self-Rated Health and Functional Health. Within these two sections, results were also divided into four different subanalyses, one for each of the chronic stress (financial, marital, parental) and negative total life events covariates. In order to better understand the exact relationship between health outcome and each of these stressors, there was a need to conduct separate analyses. These eight separate analyses were conducted to ensure optimal sample sizes, since only persons who responded on at least two or more waves on all independent and dependent variables were included in any given set of models.

Results

Self-Rated Health

Self-Rated Health and Financial Chronic Stress

The first set of results in Table 4.3, shows hierarchical models 1 through 5, and helps to answer the question: Are there racial differences in levels of Self-Rated Health (SRH) over four waves of data collection? Model 1 in Table 4.3 is the unconditional

model, providing the estimated unadjusted level of self-rated health for all respondents across all waves. Recall that SRH ranged from 1 (excellent health) to 5 (poor health). Model 2 shows how self-rated health changes as people age. Time was defined as number of years from baseline. The positive coefficient means that as people aged, or time passed, people reported higher scores on the SRH measure (corresponding to a decrease in their self-rated health over time). Model 3 includes the remaining level 1 variables: employment, income, body mass index, alcohol consumption, and cigarette smoking. Persons employed, and persons with higher incomes reported better self-rated health. As expected, those with higher body mass indexes and those who smoked reported worse self-rated health. However, alcohol consumption did not behave as expected. With an increase of alcoholic drinks per day, self-rated health significantly improved. Model 4 included the remaining level-2 covariates. Those with higher educational attainment reported better self-rated health, while as people aged, their level of self-rated health declined. Women reported worse levels of self-rated health than men, and Blacks reported worse self-rated health than Whites. Model 5 added an interaction term to identify whether the rate of change of self-rated health over time differed between Black and White respondents. The interaction term between race and wave was significant and positive, indicating that over the study period, Black respondents had a more rapid rate of declining self-rated health than their White counterparts.

Table 4.4 shows the second set of results, in which the independent variable, financial chronic stress is added to the model, to discern how financial chronic stress influences

self-rated health, and whether the effect of FCS is different for black and white respondents (Table 4.4). Model 6 included the independent variable, financial chronic stress, and the significant and positive intercept indicates that those with higher financial chronic stress reported lower levels of self-rated health. Model 7 adds two interaction terms to further explore the relationship between health, stress, race, and time. The interaction between financial chronic stress and wave indicates that the slope for financial chronic stress does not significantly differ by wave for all respondents. The interaction between financial chronic stress and race, also nonsignificant, indicates that there is no difference in the financial chronic stress slope for black and white respondents across all waves. The final model, Model 7, adds a three-way interaction term to discover if the effect of financial chronic stress on the self-rated health slope over time is different for Black and White respondents, and it was not.

Self-Rated Health and Marital Chronic Stress

Table 4.6 presents the results from the Self-Rated Health and marital chronic stress subanalyses. These analyses are unique from the previous, as they specifically include persons who were married, and responded on two or more waves on all independent and dependent variables. Model 3, the complete level-1 model, indicated that persons who were employed, persons with higher income, higher body mass, and smoked cigarettes, were more likely to report worse levels of self-rated health. For every year a person aged, their self-rated health became worse, as expected. In this subanalysis, there was no gender or racial difference in reports of self-rated health

(Model 4). Model 5 indicated that there was no significant interaction between race and wave with self-rated health. In Model 6 (Table 4.7), marital chronic stress was added. Persons experiencing higher levels of marital chronic stress reported lower levels of self-rated health. In Models 7 and 8, interaction terms were added between race, wave, and marital chronic stress, none were significant.

Self-Rated Health and Parental Chronic Stress

Table 4.9, presents the weighted estimates for the Self-Rated Health and parental chronic stress subanalysis. These analyses were significant, as they only included persons who were parents and responded to at least two or more waves of all independent and dependent variables. As seen in Model 4, Black respondents reported significantly lower levels of self-rated health. The race by wave interaction term was added in Model 5, and indicated that Black respondents had a significantly faster rate of self-rated health decline than White respondents. Model 6 shows that persons with higher reports of parental chronic stress also had worse self-rated health (Table 4.10). Model 7 added the interaction terms between race, parental chronic stress, and wave. The slope for parental chronic stress significantly differed by wave for all respondents, and there was no significant difference in parental chronic stress slope for Black and White respondents.

Self-Rated Health and Negative Total Life Events

Overall, the count of negative total life events increased over the study time period. This subanalysis included individuals who reported their experience of negative total

life events on at least two or more waves of data collection. An examination of Models 4-5 indicate that Black respondents reported significantly worse levels of self-rated health and, their self-rated health declined at a more rapid rate over the study period, as compared to their White counterparts (Tables 4.12, 4.13).

Functional Health

Functional Health and Financial Chronic Stress

The next set of analyses focus on the second health outcome, Functional Health. Table 4.15 reveals the first set of analyses, and the null model indicates that functional health levels were relatively advantageous for study participants across all waves, with an intercept of 3.72. The functional health scale ranged from 1 (severe functional impairment) to 4 (no functional impairment). Model 2 shows that as expected, functional health significantly decrease as time passes. The addition of the level-1 variables, in Model 3, show that persons who were unemployed had significantly worse functional than those employed, and those with higher income had significantly less impairment to their functional health as compared to those with lower income. Regarding the health behaviors, with every one-unit increase in body mass index, functional health significantly declines. As seen above with the Self-Rated Health analyses, persons who reported higher alcohol consumption also reported significantly less functional health impairment. Model 4 includes the remaining level-2 variables. Those with higher education levels also reported more optimal functional health. There were no significant gender differences in functional health; however, Black respondents reported significantly more functional health

impairment as compared to their White counterparts. There was no significant difference in the slope of functional health over the study period between Black and White respondents as indicated by the addition of the interaction term in Model 5. The next set of results, shown in Table 4.16 adds financial chronic stress to the existing models. Those reporting higher levels of financial chronic stress had significant worse functional health as compared to those with lower financial chronic stress. Once the two- and three-way interaction terms were added between financial chronic stress, wave, and race were added in Models 7-8, there were no significant coefficients, indicating that there was no relationship between the financial chronic stress slope by wave for all respondents, no relationship between the financial chronic stress slope by wave between Blacks and Whites.

Functional Health and Marital Chronic Stress

Table 4.18 presents the weighted estimates of functional health among respondents reporting marital chronic stress on at least two or more waves of data collection. The null model, Model 1, predicts a mean functional health value of 3.78 among these respondents. After completing the level-1 model, Model 3, indicates that those employed and those with higher incomes, reported significantly better functional health than their counterparts. Also, those with a higher body mass index and those with higher daily alcohol consumption were significantly more likely to report lower levels of functional health. Despite the absence of a significant racial difference in functional health at baseline (Model 4), Black respondents' functional health did decline at a significantly faster rate than their White counterparts (Model 5). Table

4.19 presents the results of the functional health analyses with the addition of marital chronic stress as a predictor variable. Model 6 indicates that persons reporting higher levels of marital chronic stress also reported significantly lower levels of functional health. Once the interaction terms were added in Models 6 and 7, none of the race-marital chronic stress-wave interactions were significant.

Functional Health and Parental Chronic Stress

As with the previous functional health analyses, the unconditional model indicated that most respondents reported average functional health levels. In Model 4, once all level-1 and level-2 variables were added, Black respondents reported significantly worse functional health than their White respondents, controlling for the many sociodemographic and health behavior variables. There were no differences in the slope of functional health between Black and White respondents (Model 5). In Model 6 parental chronic stress was added to the model, and there was no significant relationship between parental chronic stress levels and functional health among respondents. Furthermore, there was no significant association between any of the interaction terms added in Models 7 and 8.

Functional Health and Negative Total Life Events

The last set of analyses present the weighted estimates of functional health among Black and White study participants who responded on two or more on all covariates, including total life events. Model 2, as seen in Table 4.24, adds the wave variable to the null model, and indicates that as time passes, people report significantly more

negative total life events. Model 3, the complete level-1 model, shows that as expected, those employed and those with higher incomes, report significantly better functional health than those unemployed, and those with lower incomes. Unexpectedly, but seen in preceding analyses, those who reported more daily alcohol consumption, also reported significantly better functional health. Remaining level-2 variables are added in Model 4, and there were no significant racial or gender differences in functional health in these subanalyses. However, there were significant relationships between functional health and age and education. As expected, as a person aged their functional health declined, and those with higher education reported better functional health. There was no significant relationship between interaction of race and wave on functional health, as shown in Model 5. Once total life events was added to the model in Model 6, a significant relationship between total life events and functional health emerged, with the negative intercept indicating that the more total life events a person reported, the more likely they would also have worse functional health. Once the interaction terms were added in Model 7, it was shown that the slope for total life events significantly differed by wave for all respondents; there was no significant difference in the effect of total life events on functional health by race. However, in Model 8, a three-way interaction term of stress by race by wave was added to the model and indicated that the effect of total life events on the functional health slope over time was significantly different for Black and White respondents.

Discussion and Conclusions

Through multivariate analyses of nationally representative data for 1986 to 2001, there was an examination of racial health disparities in functional health and self-rated health over time. The goal of these analyses were twofold; first, to examine whether there were differences in functional health and self-rated health between Black and White respondents, over a segment of the life course, while also controlling for the effects of various sociodemographic variables. The second goal was to explore the moderating role of stress on the relationship between Black and White respondents and functional health and self-rated health. Eight different analyses, each containing eight different models, were conducted in an effort to better understand the unique contribution of four different types of stressors on functional health and self-rated health between the two racial groups. The results showed that in two of the four functional health analyses, and three of the four self-rated health analyses, there were significant racial differences in health, unfavorable for Black respondents. Furthermore, it was seen that Black respondents often reported faster declines of functional health and self-rated health over the study period than their White counterparts. Taken together, it was shown that there are significant and persistent health disparities between the Black and White respondents of this sample.

The next question asked what role stress and negative life events play in explaining the health status of these two racial groups. It was found that, especially for self-rated health, persons who reported lower levels of stress were also more likely to report more optimal health outcomes. Considering the fact that racial minorities have

differential access to power and other valued societal resources, it is believed that the addition of stress to the model will reveal that there are racial differences in health when considering stress levels. Thus, an interaction between stress and race was added to the analyses to examine whether experiences of stress differentially affect the health of Black respondents. It was found, however that there were no significant interactions between race and stress on health for the present adjusted models. However, there was a single significant three-way interaction between race, wave, and negative total life events on functional health, suggesting that the effect of total life events on the functional health slope over time was significantly different for Black and White respondents. It was also shown that once stress was added to the model, the health differences between Black and White respondents remained, with significant interactions indicating that despite stress, Black respondents' health still declined faster than their White counterparts. There was also the possibility that the inclusion of the sociodemographic and health risk behavior variables accounted for the effect stress may have upon health. Therefore, in analyses not shown, a very slim model was fit, which only included, race, stress, wave, and a race by stress interaction in the model. This model was fit for all of the eight health-stress analysis combinations, and of the eight, three indicated a differential effect of stress on health, not favoring Black respondents.

It is therefore concluded that there is a racial disparity in functional health and self-rated health among this nationally representative sample over a segment of the life course. As expected, this disparity did not favor Black respondents, and often

demonstrated that there were faster declines of health for the minority population. While there were was no substantial evidence that stress negatively impacted the health of the sample over time, in the current analyses, the results do raise many questions. For example, in the models unadjusted for predictor variables, the differential impact of stress between the two race groups emerged. These analyses indicated that financial chronic stress significantly impacted the self-rated and functional health unfavorably for Black Americans. The experience of total negative life events also unfavorably and significantly impacted the functional health of Black Americans. Thus, it must be considered that the sociodemographic and personal health risk behaviors may account for the lack of significant stress by race interactions.

This study underscores the need for more systematic work examining the role of stress and race in health disparities research. There is a need to better understand the way in which race and stress may converge to alter the health of disadvantaged populations. This requires that researchers acknowledge that social inequalities are at the root of health disparities. There is a need to better interpret how current American ideological perspectives do not favor many Black Americans, thus creating more stress that in turn enforces the development of disease (Geronimus 2004). Future analyses may consider stratifying racial groups based upon income, education, and gender. Having high income and education typically protects individuals from financial strain and other stressors that may have negative effects on health (Cummings and Braboy Jackson 2008). Also, the experiences of Black women and

men may differ greatly from White women and men. For the reason that socioeconomic status has a huge impact on life experiences, stratifying the racial groups by income, and examining the role of stress on health between and within these groups may offer insight into how the interplay between race, stress, and health is impacted by access or lack of societal resources.

Limitations

An examination of Gibson(1991)of the structure and measurement of three dimensions of health (disease, functional health, and subjective interpretation) using structural equations methods revealed that Blacks and Whites differ in the validity of subjective interpretations of health and in the measurement error of a chronic conditions indicator. It is possible because this analysis was not further stratified by age; functional health and self rated health differences may be more pronounced in one racial/age group versus another. For example, there is a possibility that significant racial differences in health outcomes may be more pronounced in young and middle ages, while the older Black and White respondents' health may converge to similar levels. Although the primary aim of this study was to evaluate the unique contribution of different forms of stressors considered individually, there might have been knowledge gained by combining the three chronic stressors and the total negative life events into a single composite stress index. However, I elected not to include a composite stress score. Similarly, I did not control for the other stressors in each separate model, that is, I only included one stress variable at each step. My primary aim was to explore the association of each individual source of stress and

race. Future analyses are encouraged to include an aggregate stress variable and to conduct multivariate analyses controlling for other types of stress.

Whereas the present research explored the moderating role of stress between race and health, a logical extension of the present study is the question "Does stress mediate the relationship between race and health?" While some of the models presented touched on this question, it was not the theoretical focus of work. Future research is needed to better explicate the mediating role of stress and whether race moderates this association.

Other limitations of this study acknowledge that there may be sources of error in measurement when asking participants to recall events; these include selective memory, denial of certain events or overreporting.

Table 4.3. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Financial Chronic Stress

Self Rated Health	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 7852 Level-2 Units- 2645 Level-3 Units-90	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**
Intercept[1]	2.37 (0.0226) 0.000**	2.31 (0.0242) 0.000**	2.61 (0.0372) 0.000**	2.53 (0.0456) 0.000**	2.53 (0.0457) 0.000**
Wave (Level 1)	----	0.0136 (0.0020) 0.000**	0.0102 (0.0023) 0.000**	0.0131 (0.0023) 0.000**	0.0121 (0.0024) 0.000**
Employment (Level 1)	----	----	-0.4213 (0.0300) 0.000**	-0.2656 (0.0366) 0.000**	-0.2641 (0.0368) 0.000**
Income (Level 1)	----	----	-0.0011 (0.0002) 0.000**	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.002
Body Mass Index (Level 1)	----	----	0.0302 (0.0035) 0.000**	0.0271 (0.0035) 0.000**	0.0269 (0.0035) 0.000**
Alcohol Consumption (Level 1)	----	----	-0.0245 (0.0099) 0.014**	-0.0095 (0.0097) 0.328	-0.0094 (0.0097) 0.335
Cigarette Smoking (Level 1)	----	----	0.0108 (0.0016) 0.000**	0.0113 (0.0015) 0.000**	0.01130 (0.0015) 0.000**
Age (Level 2)	----	----	----	0.0101 (0.0014) 0.000**	0.01006 (0.0014) 0.000**
Education (Level 2)	----	----	----	-0.0502 (0.0074) 0.000**	-0.0505 (0.0074) 0.000**
Gender (Level 2)	----	----	----	0.0691 (0.0381) 0.069	0.0694 (0.0381) 0.068
Race (Level 2)	----	----	----	0.1170 (0.0424) 0.006**	0.0724 (0.0499) 0.147
Race x Wave[2]	----	----	----	----	0.0108 (0.0043) 0.013*

* $p < .05$, **$p < .01$

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.4. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Financial Chronic Stress

Self Rated Health Level-1 Units- 7852 Level-2 Units- 2645 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	2.55 (0.0462) 0.000**	2.55 (0.0462) 0.000**	2.55 (0.0461) 0.000**
Wave (Level 1)	0.0131 (0.0024) 0.000**	0.01304 (0.0024) 0.000**	0.0131 (0.0024) 0.000**
Employment (Level 1)	-0.2589 (0.0374) 0.000**	-0.2575 (0.0374) 0.000**	-0.2578 (0.0374) 0.000**
Income (Level 1)	-0.0003 (0.0002) 0.131	-.00034 (0.0002) 0.112	-0.0003 (0.0002) 0.112
Body Mass Index (Level 1)	0.0261 (0.0036) 0.000**	0.0261 (0.0035) 0.000**	0.0261 (0.0035) 0.000
Alcohol Consumption (Level 1)	-0.0108 (0.0095) 0.258	-0.0107 (0.0095) 0.261	-0.0107 (0.0095) 0.259
Cigarette Smoking (Level 1)	0.0108 (0.0015) 0.000**	0.0109 (0.0015) 0.000**	0.0109 (0.0015) 0.000**
Financial Chronic Stress (Level 1)	0.1077 (0.0175) 0.000**	0.0992 (0.0227) 0.000**	0.0968 (0.0238) 0.000**
Age (Level 2)	0.0121 (0.0014) 0.000**	0.0120 (0.0014) 0.000**	0.0120 (0.0014) 0.000**
Education (Level 2)	-0.0469 (0.0074) 0.000**	-0.0468 (0.0074) 0.000**	-0.046 (0.0074) 0.000**
Gender (Level 2)	0.0604 (0.0371) 0.104	0.0605 (0.0372) 0.103	0.0603 (0.0372) 0.105
Race (Level 2)	0.0409 (0.0514) 0.427	0.0227 (0.0495) 0.646	0.0182 (0.0484) 0.706
Race x Wave[2]	0.0109 (0.0044) 0.012*	0.0118 (0.0044) 0.008*	0.0124 (0.0043) 0.004**
FCS x Wave	----	0.0001 (0.0021) 0.969	0.0006 (0.0024) 0.811
FCS x Race	----	0.0635 (0.0357) 0.075	0.0840 (0.0450) 0.062
FCS x Wave x Race	----	----	-0.0043 (0.0051) 0.401

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.5. Weighted Estimates of Random Effects; Variance Components for Self-Rated Health with Financial Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.50290	0.53231	---	0.01937
Model 2	0.48880[1]	0.53396	0.00015	0.01949
Model 3	0.50527[2]	0.37247[3]	0.00008	0.01030
Model 4	0.49473	0.33950[4]	0.00010	0.00857
Model 5	0.49438	0.34021	0.00009[5]	0.00854
Model 6	0.49641[6]	0.31904[7]	0.00009[8]	0.00872
Model 7	0.49628	0.31873	0.00008	0.00871
Model 8	0.49628	0.31862	0.00008	0.00869

[1] Level 1 Model 2 vs. 1: 2.80%
[2] Level 1 Model 3 vs. 2: -3.34%
[3] Level 2 Intercept Model 3 vs. 2: 30.24%
[4] Level 2 Intercept Model 4 vs. 3: 8.85%
[5] Level 2 Slope Model 5 vs. 4: 10.0%
[6] Level 1 Model 6 vs. 5: -0.411%
[7] Level 2 Intercept Model 6 vs. 5: 6.22%
[8] Level 2 Slope Model 6 vs. 5: 0.0%

Table 4.6. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Marital Chronic Stress

Self Rated Health	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 4482 Level-2 Units- 1554 Level-3 Units-90	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**
Intercept[1]	2.34 (0.0267) 0.000**	2.27 (0.0290) 0.000**	2.54 (0.0448) 0.000**	2.46 (0.0601) 0.000**	2.48 (0.0579) 0.000**
Wave (Level 1)	----	0.0136 (0.0025) 0.000**	0.0105 (0.0028) 0.000**	0.0132 (0.0027) 0.000**	0.0131 (0.0028) 0.000**
Employment (Level 1)	----	----	-0.3748 (0.0380) 0.000**	-0.2536 (0.0443) 0.000**	-0.2541 (0.0443) 0.000**
Income (Level 1)	----	----	-0.0011 (0.0003) 0.000**	-0.0008 (0.0002) 0.002**	-0.0008 (0.0002) 0.002
Body Mass Index (Level 1)	----	----	0.0332 (0.0048) 0.000**	0.0305 (0.0047) 0.000**	0.0302 (0.0047) 0.000**
Alcohol Consumption (Level 1)	----	----	-0.0210 (0.0112) 0.072	-0.0072 (0.0119) 0.547	-0.0070 (0.0119) 0.558
Cigarette Smoking (Level 1)	----	----	0.0104 (0.0019) 0.000**	0.0106 (0.0019) 0.000**	0.0106 (0.0019) 0.000**
Age (Level 2)	----	----	----	0.0099 (0.0017) 0.000**	0.0010 (0.0017) 0.000**
Education (Level 2)	----	----	----	-0.0425 (0.0086) 0.000**	-0.0420 (0.0086) 0.000**
Gender (Level 2)	----	----	----	0.0846 (0.0456) 0.063	0.0841 (0.0457) 0.065
Race (Level 2)	----	----	----	0.0745 (0.0629) 0.237	0.1418 (0.0824) 0.085
Race x Wave[2]	----	----	----	----	-0.0016 (0.0091) 0.86

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.7. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Marital Chronic Stress

Self Rated Health Level-1 Units- 4482 Level-2 Units- 1554 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	2.49 (0.0573) 0.000**	2.49 (0.0572) 0.000**	2.49 (0.0572) 0.000**
Wave (Level 1)	0.0125 (0.0027) 0.000**	0.0123 (0.0074) 0.097	0.0134 (0.0071) 0.059
Employment (Level 1)	-0.2556 (0.0443) 0.000**	-0.2557 (0.0443) 0.000**	-0.2560 (0.0442) 0.000**
Income (Level 1)	-0.0007 (0.0002) 0.003	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.003**
Body Mass Index (Level 1)	0.0299 (0.0046) 0.000**	0.0298 (0.0046) 0.000**	0.0299 (0.0046) 0.000**
Alcohol Consumption (Level 1)	-0.0087 (0.0116) 0.452	-0.0087 (0.0115) 0.452	-0.0086 (0.0115) 0.455
Cigarette Smoking (Level 1)	0.0103 (0.0019) 0.000**	0.0103 (0.0019) 0.000**	0.0103 (0.0019) 0.000**
Marital Chronic Stress (Level 1)	0.0821 (0.0174) 0.000**	0.0853 (0.0221) 0.000**	0.0868 (0.0220) 0.000**
Age (Level 2)	0.0107 (0.0017) 0.000**	0.0107 (0.0017) 0.000**	0.0106 (0.0017) 0.000**
Education (Level 2)	-0.0445 (0.0083) 0.000**	-0.0446 (0.0084) 0.000**	-0.0446 (0.0084) 0.000**
Gender (Level 2)	0.0703 (0.0465) 0.131	0.0690 (0.0463) 0.136	0.0687 (0.0463) 0.138
Race (Level 2)	0.1141 (0.0806) 0.157	0.1049 (0.0816) 0.199	0.1015 (0.0832) 0.223
Race x Wave[2]	-0.0017 (0.0089) 0.849	-0.0022 (0.0090) 0.809	0.0098 (0.0231) 0.673
MCS x Wave	----	0.00010 (0.0025) 0.973	-0.0003 (0.0023) 0.902
MCS x Race	----	0.0493 (0.0537) 0.359	0.0707 (0.0556) 0.204
MCS x Wave x Race	----	----	-0.0043 0.0079 0.589

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.8. Weighted Estimates of Random Effects; Variance Components for Self-Rated Health with Marital Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.47296	0.48863	---	0.02056
Model 2	0.45927[1]	0.48746	0.00014	0.02062
Model 3	0.46967[2]	0.36105[3]	0.00008	0.01456
Model 4	0.46208	0.33224[4]	0.00010	0.01503
Model 5	0.46209	0.33138	0.00010[5]	0.01432
Model 6	0.46262[6]	0.31999[7]	0.00009[8]	0.01452
Model 7	0.46254	0.31978	0.00009	0.01435
Model 8	0.46248	0.31999	0.00009	0.01433

[1] Level 1 Model 2 vs. 1: 2.89%
[2] Level 1 Model 3 vs. 2: -2.26%
[3] Level 2 Intercept Model 3 vs. 2: 25.93%
[4] Level 2 Intercept Model 4 vs. 3: 7.98%
[5] Level 2 Slope Model 5 vs. 4: 0.0%
[6] Level 1 Model 6 vs. 5: -0.115%
[7] Level 2 Intercept Model 6 vs. 5: 3.44%
[8] Level 2 Slope Model 6 vs. 5: 10.0%

Table 4.9. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Parental Chronic Stress

Self Rated Health Level-1 Units- 6578 Level-2 Units- 2256 Level-3 Units-90	**Model 1** **β** **(SE)** **p-value**	**Model 2** **β** **(SE)** **p-value**	**Model 3** **β** **(SE)** **p-value**	**Model 4** **β** **(SE)** **p-value**	**Model 5** **β** **(SE)** **p-value**
Intercept[1]	2.39 (0.0257) 0.000**	2.33 (0.0269) 0.000**	2.62 (0.0370) 0.000**	2.54 (0.0501) 0.000**	2.54 (0.0502) 0.000**
Wave (Level 1)	----	0.0123 (0.0023) 0.000**	0.0088 (0.0025) 0.001**	0.0120 (0.0025) 0.000**	0.0110 (0.0027) 0.000**
Employment (Level 1)	----	----	-0.4176 (0.0313) 0.000**	-0.2758 (0.0357) 0.000**	-0.2749 (0.0357) 0.000**
Income (Level 1)	----	----	-0.0012 (0.0003) 0.000**	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.002**
Body Mass Index (Level 1)	----	----	0.0334 (0.0039) 0.000**	0.0305 (0.0039) 0.000**	0.0304 (0.0039) 0.000**
Alcohol Consumption (Level 1)	----	----	-0.0185 (0.0109) 0.090	-0.0051 (0.0107) 0.634	-0.0049 (0.0107) 0.649
Cigarette Smoking (Level 1)	----	----	0.0104 (0.0017) 0.000**	0.0111 (0.0017) 0.000**	0.0111 (0.0017) 0.000**
Age (Level 2)	----	----	----	0.0101 (0.0015) 0.000**	0.0101 (0.0015) 0.000**
Education (Level 2)	----	----	----	-0.0497 (0.0076) 0.000**	-0.0498 (0.0076) 0.000**
Gender (Level 2)	----	----	----	0.0715 (0.0434) 0.099	0.0717 (0.0434) 0.098
Race (Level 2)	----	----	----	0.1328 (0.0469) 0.005*	0.0919 (0.0540) 0.088
Race x Wave[2]	----	----	----	----	0.0095 (0.0048) 0.048*

* $p < .05$, **$p < .01$

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.10. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Parental Chronic Stress

Self Rated Health Level-1 Units- 6578 Level-2 Units- 2256 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	2.55 (0.0491) 0.000**	2.56 (0.0492) 0.000**	2.55 (0.0492) 0.000**
Wave (Level 1)	0.0119 (0.0027) 0.000**	0.0117 (0.0026) 0.000**	0.0116 (0.0026) 0.000**
Employment (Level 1)	-0.2783 (0.0351) 0.000**	-0.2793 (0.0349) 0.000**	-0.2791 (0.0349) 0.000**
Income (Level 1)	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.002**
Body Mass Index (Level 1)	0.0292 (0.0039) 0.000**	0.0295 (0.0039) 0.000**	0.0295 (0.0039) 0.000
Alcohol Consumption (Level 1)	-0.0071 (0.0160) 0.500	-0.0077 (0.0106) 0.470	-0.0076 (0.0106) 0.472
Cigarette Smoking (Level 1)	0.0106 (0.0015) 0.000**	0.0105 (0.0016) 0.000**	0.0105 (0.0016) 0.000**
Parental Chronic Stress (Level 1)	0.0902 (0.0171) 0.000**	0.1138 (0.0233) 0.000**	0.1162 (0.0237) 0.000**
Age (Level 2)	0.0107 (0.0015) 0.000**	0.0107 (0.0015) 0.000**	0.0107 (0.0015) 0.000**
Education (Level 2)	-0.0502 (0.0074) 0.000**	-0.0505 (0.0074) 0.000**	-0.0505 (0.0074) 0.000**
Gender (Level 2)	0.0743 (0.0428) 0.082	0.0740 (0.0427) 0.083	0.0741 (0.0427) 0.082
Race (Level 2)	0.0744 (0.0538) 0.167	0.0643 (0.0518) 0.215	0.0675 (0.0519) 0.194
Race x Wave[2]	0.0093 (0.0048) 0.053	0.0105 (0.0048) 0.029*	0.0098 (0.0049) 0.044*
PCS x Wave	----	-0.0049 (0.0023) 0.030*	-0.0054 (0.0025) 0.029*
PCS x Race	----	0.0228 (0.0353) 0.518	0.0036 (0.0385) 0.927
PCS x Wave x Race	----	----	-0.0039 0.0046 0.388

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.11. Weighted Estimates of Random Effects; Variance Components for Self-Rated Health with Parental Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.51101	0.52402	---	0.02454
Model 2	0.49752[1]	0.52248	0.00016	0.02452
Model 3	0.51222[2]	0.37127[3]	0.00008	0.01383
Model 4	0.50277	0.33897[4]	0.00009	0.01107
Model 5	0.50249	0.33956	0.00009[5]	0.01103
Model 6	0.50130[6]	0.32288[7]	0.00010[8]	0.01021
Model 7	0.50134	0.32072	0.00009	0.01031
Model 8	0.50129	0.32097	0.00009	0.01030

[1] Level 1 Model 2 vs. 1: 2.64%
[2] Level 1 Model 3 vs. 2: -2.95%
[3] Level 2 Intercept Model 3 vs. 2: 28.94%
[4] Level 2 Intercept Model 4 vs. 3: 8.70%
[5] Level 2 Slope Model 5 vs. 4: 0.0%
[6] Level 1 Model 6 vs. 5: 0.237%
[7] Level 2 Intercept Model 6 vs. 5: 4.91%
[8] Level 2 Slope Model 6 vs. 5: -11.11%

Table 4.12. Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Total Life Events

Self Rated Health	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Level-1 Units- 7953 Level-2 Units- 2648 Level-3 Units-90	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**	**β** **(SE)** **p-value**
Intercept[1]	2.38 (0.0226) 0.000**	2.31 (0.0242) 0.000**	2.60 (0.0382) 0.000**	2.53 (0.0459) 0.000**	2.53 (0.0459) 0.000**
Wave (Level 1)	----	0.0138 (0.0020) 0.000**	0.0107 (0.0024) 0.000**	0.0134 (0.0023) 0.000**	0.0124 (0.0025) 0.000**
Employment (Level 1)	----	----	-0.4175 (0.0304) 0.000**	-0.2619 (0.0371) 0.000**	-0.2604 (0.0373) 0.000**
Income (Level 1)	----	----	-0.0012 (0.0003) 0.000**	-0.0007 (0.0002) 0.002**	-0.0007 (0.0002) 0.002
Body Mass Index (Level 1)	----	----	0.0230 (0.0035) 0.000**	0.0271 (0.0035) 0.000**	0.0269 (0.0035) 0.000**
Alcohol Consumption (Level 1)	----	----	-0.0242 (0.0099) 0.015*	-0.0091 (0.0097) 0.348	-0.0090 (0.0097) 0.354
Cigarette Smoking (Level 1)	----	----	0.0242 (0.0016) 0.000**	0.0112 (0.0015) 0.000**	0.0112 (0.0015) 0.000**
Age (Level 2)	----	----	----	0.0099 (0.0014) 0.000**	0.0099 (0.0014) 0.000**
Education (Level 2)	----	----	----	-0.0509 (0.0074) 0.000**	-0.0512 (0.0074) 0.000**
Gender (Level 2)	----	----	----	0.0697 (0.0380) 0.067	0.0700 (0.0380) 0.065
Race (Level 2)	----	----	----	0.1158 (0.0423) 0.007*	0.0722 (0.0498) 0.147
Race x Wave[2]	----	----	----	----	0.0102 (0.0042) 0.016*

* p < .05, **p < .01

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.13 Weighted Estimates of Fixed Effects; HLM Output for Self-Rated Health and Total Life Events

Self Rated Health Level-1 Units- 7953 Level-2 Units- 2648 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	2.49 (0.0462) 0.000**	2.40 (0.0482) 0.000**	2.40 (0.0492) 0.000**
Wave (Level 1)	0.0062 (0.0034) 0.067	0.0210 (0.0046) 0.000**	0.0211 (0.0048) 0.000**
Employment (Level 1)	-0.2572 (0.0366) 0.000**	-0.2611 (0.0367) 0.000**	-0.2610 (0.0366) 0.000**
Income (Level 1)	-0.0006 (0.0002) 0.006*	-0.0006 (0.0002) 0.002**	-0.0006 (0.0002) 0.002**
Body Mass Index (Level 1)	0.0263 (0.0035) 0.000**	0.0262 (0.0036) 0.000**	0.0262 (0.0036) 0.000
Alcohol Consumption (Level 1)	-0.0099 (0.0097) 0.305	-0.0111 (0.0093) 0.234	-0.0111 (0.0093) 0.234
Cigarette Smoking (Level 1)	0.0107 (0.0015) 0.000**	0.0101 (0.0015) 0.000**	0.0101 (0.0015) 0.000**
Total Life Events (Level 1)	0.0296 (0.0097) 0.003**	0.0723 (0.0150) 0.000**	0.0725 (0.0160) 0.000**
Age (Level 2)	0.0098 (0.0014) 0.000**	0.0095 (0.0014) 0.000**	0.0095 (0.0014) 0.000**
Education (Level 2)	-0.0506 (0.0074) 0.000**	-0.0503 (0.0074) 0.000**	-0.0502 (0.0074) 0.000**
Gender (Level 2)	0.0641 (0.0375) 0.087	0.06219 (0.0377) 0.099	0.0622 (0.0372) 0.099
Race (Level 2)	0.0680 (0.0496) 0.171	0.0439 (0.0617) 0.477	0.0472 (0.0722) 0.513
Race x Wave[2]	0.0094 (0.0043) 0.027*	0.0096 (0.0053) 0.068	0.0090 (0.0095) 0.344
TOTLE x Wave	----	-0.0052 (0.0011) 0.000**	-0.0052 (0.0012) 0.000**
TOTLE x Race	----	0.0073 (0.0193) 0.706	0.0058 (0.0269) 0.829
TOTLE x Wave x Race	----	----	0.0002 0.0024 0.937

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.14. Weighted Estimates of Random Effects; Variance Components for Self-Rated Health with Total Life Events

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.50687	0.53115	---	0.01934
Model 2	0.49308[1]	0.53444	0.00013	0.01947
Model 3	0.50968[2]	0.37384[3]	0.00007	0.01013
Model 4	0.49924	0.34054[4]	0.00008	0.00821
Model 5	0.49890	0.34122	0.00008[5]	0.00817
Model 6	0.49852[6]	0.33971[7]	0.00009[8]	0.00765
Model 7	0.49246	0.34328	0.00012	0.00779
Model 8	0.49246	0.34327	0.00012	0.00778

[1] Level 1 Model 2 vs. 1: 2.72%
[2] Level 1 Model 3 vs. 2: -3.37%
[3] Level 2 Intercept Model 3 vs. 2: 30.05%
[4] Level 2 Intercept Model 4 vs. 3: 8.91%
[5] Level 2 Slope Model 5 vs. 4: 0.0%
[6] Level 1 Model 6 vs. 5: 0.076%
[7] Level 2 Intercept Model 6 vs. 5: 0.443%
[8] Level 2 Slope Model 6 vs. 5: -12.5%

Table 4.15. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Financial Chronic Stress

Functional Health Level-1 Units- 8767 Level-2 Units- 2834 Level-3 Units-90	**Model 1** **β** **(SE)** **p-value**	**Model 2** **β** **(SE)** **p-value**	**Model 3** **β** **(SE)** **p-value**	**Model 4** **β** **(SE)** **p-value**	**Model 5** **β** **(SE)** **p-value**
Intercept[1]	3.72 (0.0146) 0.000**	3.79 (0.0145) 0.000**	3.44 (0.0275) 0.000**	3.50 (0.0328) 0.000**	3.48 (0.0343) 0.000**
Wave (Level 1)	----	-0.0149 (0.0013) 0.000**	-0.0097 (0.0014) 0.000**	-0.0116 (0.0012) 0.000**	-0.0109 (0.0013) 0.000**
Employment (Level 1)	----	----	0.3889 (0.0256) 0.000**	0.2755 (0.0295) 0.000**	0.2748 (0.0294) 0.000**
Income (Level 1)	----	----	0.0009 (0.0002) 0.000**	0.0006 (0.0002) 0.001**	0.0006 (0.0002) 0.001
Body Mass Index (Level 1)	----	----	-0.0073 (0.0025) 0.004**	-0.0057 (0.0024) 0.019*	-0.0056 (0.0024) 0.021**
Alcohol Consumption (Level 1)	----	----	0.0356 (0.0050) 0.000**	0.0250 (0.0046) 0.000**	0.0249 (0.0046) 0.000**
Cigarette Smoking (Level 1)	----	----	0.0003 (0.0010) 0.746	-0.0003 (0.0010) 0.748	-0.0003 (0.0010) 0.757
Age (Level 2)	----	----	----	-0.0082 (0.0010) 0.000**	-0.0082 (0.0009) 0.000**
Education (Level 2)	----	----	----	0.0232 (0.0040) 0.000**	0.0233 (0.0040) 0.000**
Gender (Level 2)	----	----	----	-0.0295 (0.0228) 0.196	-0.0233 (0.0228) 0.195
Race (Level 2)	----	----	----	-0.0514 (0.0243) 0.034*	-0.0296 (0.0250) 0.271
Race x Wave[2]	----	----	----	----	-0.0062 (0.0037) 0.097

* p < .05, **p < .01

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.16. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Financial Chronic Stress

Functional Health Level-1 Units- 8767 Level-2 Units- 2834 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	3.47 (0.0340) 0.000**	3.48 (0.0341) 0.000**	3.48 (0.0341) 0.000**
Wave (Level 1)	-0.0114 (0.0013) 0.000**	-0.0118 (0.0015) 0.000**	-0.0117 (0.0015) 0.000**
Employment (Level 1)	0.2700 (0.0291) 0.000**	0.2707 (0.0293) 0.000**	0.2705 (0.0293) 0.000**
Income (Level 1)	0.0004 (0.0001) 0.006**	0.0004 (0.0001) 0.007*	0.0004 (0.0001) 0.007*
Body Mass Index (Level 1)	-0.0051 (0.0024) 0.035*	-0.0050 (0.0024) 0.039*	-0.0050 (0.0024) 0.038*
Alcohol Consumption (Level 1)	0.0254 (0.0047) 0.000**	0.0254 (0.0047) 0.000**	0.0254 (0.0047) 0.000**
Cigarette Smoking (Level 1)	-0.0000 (0.0010) 0.968	-0.0001 (0.0010) 0.954	-0.0001 (0.0010) 0.955
Financial Chronic Stress (Level 1)	-0.0659 (0.0010) 0.000**	-0.0518 (0.0135) 0.000**	-0.0532 (0.0140) 0.000**
Age (Level 2)	-0.0094 (0.0009) 0.000**	-0.0094 (0.0001) 0.000**	-0.0094 (0.0009) 0.000**
Education (Level 2)	0.0213 (0.0042) 0.000**	0.0215 (0.0041) 0.000**	0.0215 (0.0041) 0.000**
Gender (Level 2)	-0.0253 (0.0224) 0.258	-0.0260 (0.0224) 0.254	-0.0257 (0.0224) 0.252
Race (Level 2)	-0.0086 (0.0254) 0.734	-0.0040 (0.0253) 0.875	-0.0065 (0.0245) 0.791
Race x Wave[2]	-0.0064 (0.0037) 0.082	-0.0061 (0.0039) 0.121	-0.0057 (0.0037) 0.127
FCS x Wave	----	-0.0022 (0.0020) 0.285	-0.0019 (0.0022) 0.395
FCS x Race	----	-0.0305 (0.0217) 0.159	-0.0057 (0.0037) 0.127
FCS x Wave x Race	----	----	-0.0025 0.0033 0.447

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.17. Weighted Estimates of Random Effects; Variance Components for Functional Health with Financial Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.28024	0.2463	---	0.00643
Model 2	0.22287[1]	0.2312	0.00139	0.00633
Model 3	0.22995[2]	0.17538[3]	0.00119	0.0017
Model 4	0.22559	0.16828[4]	0.00122	0.00052
Model 5	0.22554	0.16823	0.00121[5]	0.00051
Model 6	0.22625[6]	0.1627[7]	0.00118[8]	0.00057
Model 7	0.22639	0.16296	0.00117	0.00054
Model 8	0.22634	0.16302	0.00117	0.00055

[1] Level 1 Model 2 vs. 1: 20.47%
[2] Level 1 Model 3 vs. 2: -3.18%
[3] Level 2 Intercept Model 3 vs. 2: 24.14%
[4] Level 2 Intercept Model 4 vs. 3: 4.05%
[5] Level 2 Slope Model 5 vs. 4: 0.820%
[6] Level 1 Model 6 vs. 5: -0.315%
[7] Level 2 Intercept Model 6 vs. 5: 3.29%
[8] Level 2 Slope Model 6 vs. 5: 2.48%

Table 4.18. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Marital Chronic Stress

Self Rated Health	Model 1	Model 2	Model 3	Model 4	Model 5
Level-1 Units- 4889 Level-2 Units- 1659 Level-3 Units-90	β (SE) p-value	β (SE) p-value	β (SE) p-value	β (SE) p-value	β (SE) p-value
Intercept[1]	3.78 (0.0144) 0.000**	3.83 (0.0157) 0.000**	3.53 (0.0351) 0.000**	3.56 (0.0427) 0.000**	3.56 (0.0427) 0.000**
Wave (Level 1)	----	-0.0109 (0.0015) 0.000**	-0.0065 (0.0016) 0.000**	-0.0079 (0.0014) 0.000**	-0.0075 (0.0015) 0.000**
Employment (Level 1)	----	----	0.3417 (0.0313) 0.000**	0.2691 (0.0359) 0.000**	0.2684 (0.0358) 0.000**
Income (Level 1)	----	----	0.0006 (0.0002) 0.001**	0.0004 (0.0001) 0.005*	0.0004 (0.00014) 0.006**
Body Mass Index (Level 1)	----	----	-0.0086 (0.0031) 0.006*	-0.0071 (0.0031) 0.022*	-0.0070 (0.0031) 0.022*
Alcohol Consumption (Level 1)	----	----	0.0261 (0.0053) 0.000**	0.0195 (0.0053) 0.001**	0.0194 (0.0054) 0.001**
Cigarette Smoking (Level 1)	----	----	0.0010 (0.0012) 0.387	0.0008 (0.0012) 0.487	0.0008 (0.0012) 0.484
Age (Level 2)	----	----	----	-0.0063 (0.0011) 0.000**	-0.0063 (0.0011) 0.000**
Education (Level 2)	----	----	----	0.0160 (0.0046) 0.001**	0.0160 (0.0047) 0.001**
Gender (Level 2)	----	----	----	-0.0224 (0.0285) 0.432	-0.0225 (0.0285) 0.429
Race (Level 2)	----	----	----	0.0172 (0.0336) 0.608	0.0083 (0.0329) 0.800
Race x Wave[2]	----	----	----	----	-0.0069 (0.0047) 0.014*

* p < .05, **p < .01

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.19. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Marital Chronic Stress

Functional Health Level-1 Units- 4889 Level-2 Units- 1659 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	3.56 (0.0429) 0.000**	3.56 (0.0430) 0.000**	3.56 (0.0430) 0.000**
Wave (Level 1)	-0.0073 (0.0015) 0.000**	-0.0105 (0.0048) 0.029*	-0.0086 (0.0053) 0.109
Employment (Level 1)	0.2681 (0.0356) 0.000**	0.2683 (0.0357) 0.000**	0.2681 (0.0357) 0.000**
Income (Level 1)	-0.0004 (0.0001) 0.006*	0.0004 (0.0001) 0.006*	0.0004 (0.0001) 0.007*
Body Mass Index (Level 1)	-0.0069 (0.0030) 0.024*	-0.0070 (0.0030) 0.021*	-0.0070 (0.0030) 0.020*
Alcohol Consumption (Level 1)	0.0201 (0.0054) 0.000**	0.0202 (0.0054) 0.000**	0.0201 (0.0054) 0.000**
Cigarette Smoking (Level 1)	0.0010 (0.0012) 0.422	0.0010 (0.0012) 0.420	0.0010 (0.0012) 0.415
Marital Chronic Stress (Level 1)	-0.0259 (0.0141) 0.065	-0.0331 (0.0186) 0.074	-0.0294 (0.0191) 0.125
Age (Level 2)	-0.0065 (0.0012) 0.000**	-0.0065 (0.0012) 0.000**	-0.0065 (0.0012) 0.000**
Education (Level 2)	0.0168 (0.0048) 0.001**	0.0169 (0.0048) 0.001**	0.0168 (0.0048) 0.001**
Gender (Level 2)	-0.0181 (0.0285) 0.526	-0.0183 (0.0285) 0.521	-0.0181 (0.0285) 0.526
Race (Level 2)	0.0166 (0.0337) 0.622	0.0162 (0.0349) 0.642	0.0247 (0.0352) 0.484
Race x Wave[2]	-0.0070 (0.0047) 0.137	-0.0074 (0.0047) 0.118	-0.0292 (0.0141) 0.038*
MCS x Wave	----	0.0013 (0.0018) 0.478	0.0005 (0.0020) 0.797
MCS x Race	----	0.0097 (0.0280) 0.729	-0.0239 (0.0305) 0.433
MCS x Wave x Race	----	----	0.0076 0.0045 0.092

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.20. Weighted Estimates of Random Effects; Variance Components for Functional Health with Marital Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.22319	0.19219	---	0.00328
Model 2	0.18749[1]	0.17715	0.00091	0.00436
Model 3	0.19149[2]	0.13959[3]	0.00074	0.00081
Model 4	0.18907	0.13502[4]	0.00076	0.00026
Model 5	0.18888	0.13506	0.00076[5]	0.00027
Model 6	0.18849[6]	0.1345[7]	0.00077[8]	0.00025
Model 7	0.18851	0.13452	0.00077	0.00024
Model 8	0.18837	0.13455	0.00077	0.00023

[1] Level 1 Model 2 vs. 1: 16.00%
[2] Level 1 Model 3 vs. 2: -2.13%
[3] Level 2 Intercept Model 3 vs. 2: 19.54%
[4] Level 2 Intercept Model 4 vs. 3: 3.27%
[5] Level 2 Slope Model 5 vs. 4: 0.0%
[6] Level 1 Model 6 vs. 5: 0.206%
[7] Level 2 Intercept Model 6 vs. 5: 0.415%
[8] Level 2 Slope Model 6 vs. 5: -1.32%

Table 4.21. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Parental Chronic Stress

Functional Health Level-1 Units- 7346 Level-2 Units- 2422 Level-3 Units-90	**Model 1** **β** **(SE)** **p-value**	**Model 2** **β** **(SE)** **p-value**	**Model 3** **β** **(SE)** **p-value**	**Model 4** **β** **(SE)** **p-value**	**Model 5** **β** **(SE)** **p-value**
Intercept[1]	3.71 (0.0155) 0.000**	3.78 (0.0157) 0.000**	3.45 (0.0303) 0.000**	3.50 (0.0354) 0.000**	3.49 (0.0353) 0.000**
Wave (Level 1)	----	-0.0160 (0.0015) 0.000**	-0.0109 (0.0015) 0.000**	-0.0133 (0.0013) 0.000**	-0.0126 (0.0014) 0.000**
Employment (Level 1)	----	----	0.3973 (0.0289) 0.000**	0.2866 (0.0323) 0.000**	0.2862 (0.0323) 0.000**
Income (Level 1)	----	----	0.0009 (0.0002) 0.000**	0.0005 (0.0002) 0.001**	0.0005 (0.0002) 0.001**
Body Mass Index (Level 1)	----	----	-0.0083 (0.0026) 0.002**	-0.0065 (0.0026) 0.011**	-0.0065 (0.0026) 0.012*
Alcohol Consumption (Level 1)	----	----	0.0360 (0.0054) 0.000**	0.0260 (0.0050) 0.000**	0.0259 (0.0051) 0.000**
Cigarette Smoking (Level 1)	----	----	0.0004 (0.0012) 0.725	-0.0004 (0.0011) 0.691	-0.0004 (0.0011) 0.703
Age (Level 2)	----	----	----	-0.0086 (0.0011) 0.000**	-0.0086 (0.0012) 0.000**
Education (Level 2)	----	----	----	0.0239 (0.0052) 0.000**	0.0240 (0.0052) 0.000**
Gender (Level 2)	----	----	----	-0.0256 (0.0230) 0.265	-0.0257 (0.0230) 0.264
Race (Level 2)	----	----	----	-0.0614 (0.0291) 0.035*	-0.0385 (0.0291) 0.185
Race x Wave[2]	----	----	----	----	-0.0059 (0.0040) 0.144

* p < .05, **p < .01

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.22. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Parental Chronic Stress

Functional Health Level-1 Units- 7346 Level-2 Units- 2422 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	3.49 (0.0356) 0.000**	3.49 (0.0357) 0.000**	3.49 (0.0357) 0.000**
Wave (Level 1)	-0.0127 (0.0014) 0.000**	-0.0126 (0.0014) 0.000**	-0.0126 (0.0014) 0.000**
Employment (Level 1)	0.2864 (0.0322) 0.000**	0.2864 (0.0323) 0.000**	0.2865 (0.0323) 0.000**
Income (Level 1)	0.0005 (0.0002) 0.001**	0.0005 (0.0002) 0.001**	0.0005 (0.0002) 0.001**
Body Mass Index (Level 1)	-0.0064 (0.0026) 0.012**	-0.0065 (0.0026) 0.012*	-0.0065 (0.0026) 0.012*
Alcohol Consumption (Level 1)	0.0261 (0.0051) 0.000**	0.0262 (0.0051) 0.000**	0.0262 (0.0051) 0.000**
Cigarette Smoking (Level 1)	-0.0004 (0.0011) 0.000**	-0.0004 (0.0011) 0.725	-0.0004 (0.0011) 0.723
Parental Chronic Stress (Level 1)	-0.0056 (0.0106) 0.599	-0.0075 (0.0147) 0.608	-0.0057 (0.0153) 0.707
Age (Level 2)	-0.0086 (0.0011) 0.000**	-0.0086 (0.0011) 0.000**	-0.0086 (0.0011) 0.000**
Education (Level 2)	0.0240 (0.0052) 0.000**	0.0241 (0.0052) 0.000**	0.0241 (0.0052) 0.000**
Gender (Level 2)	-0.0257 (0.0230) 0.264	-0.0249 (0.0229) 0.277	-0.0248 (0.0229) 0.279
Race (Level 2)	-0.0374 (0.0293) 0.202	-0.0313 (0.0292) 0.285	-0.0292 (0.0290) 0.315
Race x Wave[2]	-0.0058 (0.0040) 0.145	-0.0126 (0.0014) 0.000**	-0.0067 (0.0040) 0.096
PCS x Wave	----	0.0012 (0.0019) 0.515	0.0009 (0.0021) 0.689
PCS x Race	----	-0.0295 (0.0244) 0.228	-0.0437 (0.0238) 0.065
PCS x Wave x Race	----	----	0.0031 0.0035 0.376

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.23. Weighted Estimates of Random Effects; Variance Components for Functional Health with Parental Chronic Stress

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.30149	0.23543	---	0.00604
Model 2	0.23601[1]	0.22512	0.00155	0.00562
Model 3	0.24291[2]	0.16911[3]	0.00131	0.00183
Model 4	0.23855	0.16036[4]	0.00134	0.00038
Model 5	0.23848	0.16023	0.00133[5]	0.00048
Model 6	0.23865[6]	0.15969[7]	0.00133[8]	0.00061
Model 7	0.2382	0.15971	0.00134	0.00069
Model 8	0.23829	0.15964	0.00134	0.00070

[1] Level 1 Model 2 vs. 1: 21.72%
[2] Level 1 Model 3 vs. 2: -2.92%
[3] Level 2 Intercept Model 3 vs. 2: 24.88%
[4] Level 2 Intercept Model 4 vs. 3: 5.17%
[5] Level 2 Slope Model 5 vs. 4: 0.746%
[6] Level 1 Model 6 vs. 5: -0.071%
[7] Level 2 Intercept Model 6 vs. 5: 0.337%
[8] Level 2 Slope Model 6 vs. 5: 0%

Table 4.24. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Total Life Events

Functional Health Level-1 Units- 9051 Level-2 Units- 2886 Level-3 Units-90	**Model 1** **β** **(SE)** **p-value**	**Model 2** **β** **(SE)** **p-value**	**Model 3** **β** **(SE)** **p-value**	**Model 4** **β** **(SE)** **p-value**	**Model 5** **β** **(SE)** **p-value**
Intercept[1]	3.69 (0.0148) 0.000**	3.78 (0.0143) 0.000**	3.43 (0.0274) 0.000**	3.47 (0.0329) 0.000**	3.47 (0.0328) 0.000**
Wave (Level 1)	----	-0.0202 (0.0012) 0.000**	-0.0147 (0.0013) 0.000**	-0.0163 (0.0012) 0.000**	-0.0156 (0.0012) 0.000**
Employment (Level 1)	----	----	0.4103 (0.0263) 0.000**	0.2853 (0.0293) 0.000**	0.2846 (0.0293) 0.000**
Income (Level 1)	----	----	0.0011 (0.0003) 0.000**	0.0008 (0.0002) 0.000**	-0.0008 (0.0002) 0.000**
Body Mass Index (Level 1)	----	----	-0.0036 (0.0025) 0.152	-0.0021 (0.0025) 0.389	-0.0020 (0.0024) 0.406
Alcohol Consumption (Level 1)	----	----	0.0368 (0.0052) 0.000**	0.0255 (0.0047) 0.000**	0.0254 (0.0047) 0.000**
Cigarette Smoking (Level 1)	----	----	0.0010 (0.0010) .299	0.0003 (0.0010) 0.764	0.0003 (0.0010) 0.754
Age (Level 2)	----	----	----	-0.0092 (0.0009) 0.000**	-0.0092 (0.0009) 0.000**
Education (Level 2)	----	----	----	0.0263 (0.0043) 0.000**	0.0264 (0.0043) 0.000**
Gender (Level 2)	----	----	----	-0.0168 (0.0235) 0.474	-0.0169 (0.0235) 0.472
Race (Level 2)	----	----	----	-0.0444 (0.0246) 0.070	-0.0196 (0.0247) 0.427
Race x Wave[2]	----	----	----	----	-0.0068 (0.0037) 0.067

* p < .05, **p < .01

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.25. Weighted Estimates of Fixed Effects; HLM Output for Functional Health and Total Life Events

Functional Health Level-1 Units- 9051 Level-2 Units- 2886 Level-3 Units-90	**Model 6** **β** **(SE)** **p-value**	**Model 7** **β** **(SE)** **p-value**	**Model 8** **β** **(SE)** **p-value**
Intercept[1]	3.50 (0.0330) 0.000**	3.48 (0.0337) 0.000**	3.47 (0.0340) 0.000**
Wave (Level 1)	-0.0106 (0.0015) 0.000**	-0.0060 (0.0029) 0.000**	-0.0045 (0.0031) 0.150
Employment (Level 1)	0.2827 (0.0294) 0.000**	0.2810 (0.0295) 0.000**	0.2814 (0.0296) 0.000**
Income (Level 1)	0.0007 (0.0002) 0.000**	0.0007 (0.0002) 0.000**	0.0007 (0.0002) 0.001**
Body Mass Index (Level 1)	-0.0017 (0.0025) 0.497	-0.0016 (0.0025) 0.519	-0.0015 (0.0025) 0.529
Alcohol Consumption (Level 1)	0.0264 (0.0047) 0.000**	0.0263 (0.0047) 0.000**	0.0263 (0.0047) 0.000**
Cigarette Smoking (Level 1)	0.0007 (0.0010) 0.467	0.0006 (0.0010) 0.524	0.0006 (0.0010) 0.535
Total Life Events (Level 1)	-0.0242 (0.0056) 0.000**	-0.0138 (0.0077) 0.071	-0.0105 (0.0079) 0.185
Age (Level 2)	-0.0091 (0.0009) 0.000**	-0.0092 (0.0009) 0.000**	-0.0092 (0.0009) 0.000**
Education (Level 2)	0.0262 (0.0043) 0.000**	0.0263 (0.0043) 0.000**	0.0263 (0.0043) 0.000**
Gender (Level 2)	-0.0128 (0.0234) 0.584	-0.0131 (0.0234) 0.574	-0.0131 (0.0235) 0.577
Race (Level 2)	-0.0163 (0.0243) 0.503	-0.0155 (0.0319) 0.628	0.0499 (0.0406) 0.220
Race x Wave[2]	-0.0063 (0.0038) 0.098	-0.0055 (0.0049) 0.266	-0.0196 (0.0077) 0.011*
TOTLE x Wave	----	-0.0015 (0.0008) 0.042*	-0.0020 (0.0008) 0.016*
TOTLE x Race	----	-0.0018 (0.0151) 0.907	-0.0294 (0.0176) 0.095
TOTLE x Wave x Race	----	----	0.0043 0.0016 0.010*

[1] Variance of Means in 1986

[2] Cross-Level Interaction

Table 4.26. Weighted Estimates of Random Effects; Variance Components for Functional Health with Total Life Events

Random Effects	Within-Person Variance-- Level 1	Between-Person Variance-- Level 2 (Intercept)	Between-Person Variance-- Level 2 (Slope)	Between-PSU Variance-- Level 3 Variance
Model 1	0.31666	0.26909	---	0.00638
Model 2	0.23725[1]	0.23167	0.00186	0.00599
Model 3	0.24623[2]	0.17452[3]	0.00156	0.00159
Model 4	0.24147	0.17013[4]	0.00159	0.00061
Model 5	0.24146	0.17010	0.00158[5]	0.00059
Model 6	0.24212[6]	0.16902[7]	0.00156[8]	0.00060
Model 7	0.24218	0.16868	0.00153	0.00056
Model 8	0.24196	0.16873	0.00153	0.00062

[1] Level 1 Model 2 vs. 1: 25.07%
[2] Level 1 Model 3 vs. 2: -3.79%
[3] Level 2 Intercept Model 3 vs. 2: 24.67%
[4] Level 2 Intercept Model 4 vs. 3: 2.52%
[5] Level 2 Slope Model 5 vs. 4: 0.629%
[6] Level 1 Model 6 vs. 5: -0.273%
[7] Level 2 Intercept Model 6 vs. 5: 0.635%
[8] Level 2 Slope Model 6 vs. 5: 1.266%

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CHAPTER FIVE

Conclusion

In this dissertation I have examined the conceptual and empirical intersection of race, stress, and health. Among many urban African American populations, there is an excess mortality that is coupled with persistent and long-standing physical health disparities and stress-related chronic diseases are the primary reasons for this excess mortality (Geronimus 2004). Racial inequalities in health have been an unrelenting reality of American society, yet there have been few advancements. Further there are racial inequalities in economic and social arenas as well that have long persisted. Black Americans continue to be residentially segregated and materially disadvantaged. Considering these facts, it is thus wise for pubic health professionals to consider the many psychosocial determinants of health that contribute to these social inequalities that are likely a precursor to poor health. One such determinant, stress, is the topic of this dissertation. People of disadvantaged social statuses tend to report elevated levels of stress and may be more vulnerable to the effects of stress (Williams and Jackson 2005). Exposure to chronic stress is associated with altered physiological functioning, which in turn may increase risks for a wide range of mental and physical health conditions (Israel, Farquhar et al. 2002; Epel, Blackburn et al. 2004; Hogue and Bremner 2005; Rich-Edwards and Grizzard 2005; Williams and Jackson 2005). It has also been suggested that certain negative chronic health disease risk behaviors may be used in response to exposure to chronic stressors, and these

also may impact the health of individuals. Thus the goal of this research is to explore racial patterns in the experience of stress and negative life events, and to examine if these are related to poorer health outcomes among Black respondents, as compared to their White counterparts.

Specifically, I have examined the differences in stress responses between Black and White respondents who participated in a longitudinal research study. Conceptually, I pondered the question: What is the role of the stress process in public health's explanations of racial and ethnic health disparities? My primary empirical research questions were as follows: Are there Black/White differences in reports of chronic stress and negative life events over a 15-year time period? Are there Black/White differences in functional and self-rated health, and how does stress impact these levels of health? I will use the remainder of this chapter to present an overview of my findings.

In Chapter Two, I provided an overview the state of racial and ethnic health disparities in the United States of America. I also described the economic disparities between the two racial groups, and discussed the historical underpinnings of race in America. Many contemporary theorists have offered various explanations of why and how racial and ethnic health disparities have been produced and maintained in America. In Chapter Two, I attempted to distinguish and provide an overview of the more popular and robust public health theories that address racial and ethnic health disparities. There was a discussion of individual level explanations: John Henryism,

Allostatic Load, and the Weathering Hypothesis; and structural level explanations: Fundamental Causes of Disease, Neighborhood Effects, and Income Inequality, and Racism and Racial Discrimination. While doing so, I considered the implication of the stress process. In Chapter Two, I also argued that the stress process is ubiquitous in the lives of individuals, but may be more prevalent and offer more dire consequences for members of society that endure less favorable living conditions. I reasoned that due to the historical and current social injuries against African Americans, it is reasonable to assume that this racial group exists under more stressful living conditions. The stress process is a major factor in health status and many theories and explanations directly acknowledge the role of stress, however others only indirectly infer its' role. For example, when considering income inequality and poverty as an explanation for racial and ethnic health disparities, more often than not, it is just assumed that the disparities persist due to the material deprivation and its antecedents. However, after critical review, one can understand that stress is also a consequence. As a chronic stressor, poverty contributes to distress in the form of economic hardships, poor accessibility to health care, limited resources, feelings of powerlessness, and low educational achievement (Anderson 1991).

In Chapter Three, I utilized data from the American's Changing Lives study database to discern Black/White differences in experiences of stress and negative life events. I used four measures: financial, marital and parental chronic stress and total negative life events. I reviewed the literature to describe the current racial and ethnic distribution of chronic stress, and I also defined the three chronic stressors and

negative life events. A literature review revealed that African Americans were generally more likely to report higher levels of chronic stress, and these trends extended to subpopulations within the African American community. For example, African American gays and lesbians, young adults, college students, and women were more likely to report higher stress levels than their White counterparts. For the planned multivariate analyses, I used Hierarchical Linear Modeling to examine Black/White differences in reports of chronic stress and negative life events over a longitudinal study period. Using all available four waves of data, I modeled the trends in the four different stressors, while controlling for key sociodemographic predictors, such as age, gender, education and employment status, and income. The results showed that after controlling for all of these variables African American respondents reported higher levels of each chronic stress measure (financial, marital, parental) than their White counterparts throughout the entire study period. However, there were no racial differences in the chronic stress slope over time. When considering total negative life events, the results were reversed. Black respondents did not report more total negative life events over an average of the four waves of data, but there were significant increases in the stress slope over time as compared to White respondents. In other words, Black respondents were experiencing significantly more total negative life events, at a faster rate, than their peers, over the study period. Chapter Three showed that Black respondents were experiencing unfavorable amounts of stress over time, as compared to White respondents. This chapter answered a fundamental question, Are there Black/White differences in chronic stress and total negative life events over a segment of the life course in a

general population study? The answer was yes. With this advancement, it is now possible to further explore the role of the stress process in racial and ethnic health disparities. It is important to understand if there are vastly different living conditions, patterned by race, and examine how stress operates for each group.

Chapter Three demonstrated that there are significant racial differences in the prevalence of chronic stress and total negative life events in my study population. In Chapter Four I now consider are there Black/White health differences in functional health and self-rated health. After considering this I will assess if stress moderates the association between race and health. In Chapter Four, I wanted to know the unique impact of each of my four stressors: Financial, Parental, and Marital Chronic Stress, and Total Negative Life Events on each of my two health outcomes: Functional Health and Self-Rated Health. I controlled a variety of sociodemographic variables (age, gender, education, employment, and income) and personal health behaviors that may impact the two health outcomes (daily cigarette use, daily alcohol consumption, and body mass index). I use Hierarchical Linear Modeling to longitudinally examine health trends between Black and White respondents, and secondarily examine the moderating role of stress. In Chapter Four there were eight different analyses, one for each of the health-stress combinations. When reviewing the self-rated health of the two racial groups, Black respondents suffered worse overall self-rated health and significant worse self-rated health declines over time in the three different analyses that included financial and parental chronic stress and total negative life events.

In the analyses for functional health, Black respondents reported an average of significantly worse functional health over the study period than White respondents when considering their financial and parental chronic stress in the models. When considering their marital and parental chronic stress in the analyses, Black respondents reported significantly faster health declines over time than their White counterparts. Finally, when considering the interaction between race, stress and time on health, there was a significant difference in the impact of stress on health between Black and White respondents, with Black participants reporting worse health.

In the public health arena, race and class are inextricably linked with one another. When trying to examine the social determinants of health researchers are typically examining one construct, while ignoring the other. In American society, race and socioeconomic class are also linked, but often not confused with one another. In general, research has shown that the social class position of Blacks does not afford them the degree of social acceptance by others (Cummings and Braboy Jackson 2008). The attainment of middle or higher socioeconomic position by Black Americans does not translate into the mental or physical health or personal lifestyle returns seen in other racial groups. These systems of social inequality are likely to prove different levels of, and effects of stress in the vulnerable populations. A major challenge facing public health professionals is identifying the social determinants that produce the existing and persistent health disparities. Thus, one of the most important implications of this research is that there are increased levels of stress and negative life events among Black Americans. In other words, race is a marker of

increased risk for stress exposure. There was not overwhelming evidence in this dissertation that these increased levels of stress directly exacerbates the health status of Black respondents. My research has not provided definitive answers, rather serves as an impetus to other research directions. Building upon the research agenda of this dissertation, I will suggest other research paths. First, the mechanisms in which the negative effects of stress response may be more efficiently measured other health measures. The stress response may be exhibited via biomarkers, which links chronic and traumatic stress to mental and bodily disorders (J Hellhammer 2004). So while I was unable to measure the effect of the stress response functional health and self-rated health, future research should explore the stress response on neural and neuroendocrine cascades. A stressful stimulus results in the activation of several physiological pathways, including the HPA axis and the autonomic nervous system (Kajantie and Phillips 2006). Second, there is a need for more interdisciplinary investigations that contain information about the broad array of social and psychological conditions that combine over time to create stress. Further, people typically confront stress-provoking conditions with a variety of behaviors, perceptions, and cognitions that are often capable of altering the difficult conditions, or may mediate the impact of stressful situations. These coping mechanisms may be the key to better understanding the nature of racial and ethnic health disparities. To be clear, the suggestion is that if there is a pathway between racially patterned living conditions and experiences, stress responses, and health, there needs to be political and economic structural change that equalizes the living conditions of majority and

minority American populations, in order to diminish racial and ethnic health disparities.

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