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Mechanisms of Health Disparities in Inflammation: A Test of the Differential Stress Exposure and Differential Stress Vulnerability Hypotheses

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A dissertation submitted in partial satisfaction of the

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by

Uchechi Acholonu Mitchell

2014

ABSTRACT OF THE DISSERTATION

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by

Uchechi Acholonu Mitchell

Doctor of Philosophy in Public Health

University of California, Los Angeles, 2014

Professor Carol S. Aneshensel, Chair

Despite decades of research and public health intervention, disparities in cardiovascular diseases among social groups persist and are only partially explained by parallel differences in socioeconomic status or conventional risk factors, such as smoking. For example, relative to non-Hispanic whites, racial and ethnic minorities are particularly at risk of developing and dying from cardiovascular diseases (Rogers et al., 2011) and, in terms of gender, women experience higher mortality rates than men despite their lower prevalence rate. Understanding these systematic differences in health necessitates acknowledging that the social patterning of cardiovascular diseases is not a chance occurrence; racial disparities in health are rooted in a system of social stratification that consistently and systematically disadvantages certain subgroups of the population (Adler & Rehkopf, 2008) through the unequal distribution of fiscal

and material resources, on the one hand (Williams & Mohammed, 2009), and through the unequal distribution of stressors and coping resources, on the other hand (Pearlin, 1989; Williams & Mohammed, 2009).

Discrimination is a chronic stressor that disproportionately affects racial minorities (Kessler, Mickelson & Williams, 1999). It manifests as discrete life events (i.e., major lifetime discrimination) that significantly hinder opportunities for status attainment and as day-to-day experiences of unfair treatment (i.e., everyday discrimination), such as being treated with less respect than others or being threatened or harassed (Williams, Yu, Jackson, & Anderson, 1997). Prior research has linked exposure to discrimination to multiple adverse health outcomes including low infant birth weight, depressive symptoms, all-cause mortality, and risk factors for cardiovascular diseases, such as high blood pressure (for a review of this literature see: Mays, Cochran & Barnes, 2007; Pascoe & Richman, 2009; Williams & Mohammed, 2009). Therefore, racial disparities in cardiovascular diseases may be attributed to differences in exposure to discrimination.

The current study sheds light on the mechanisms through which group differences in exposure to and the impact of discrimination generate disparities in cardiovascular diseases. The study specifically looks at its effects on C-reactive protein (i.e., CRP), a protein produced in response to exposure to stressors. It is a marker of systemic inflammation that is positively associated with cardiovascular diseases such as stroke, atherosclerosis, and myocardial infarction (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997; Ridker, Hennekens, Buring, & Riafai, 2000). The current study goes beyond a basic description of the population-level distribution of CRP by focusing on psychosocial factors that mediate and/or moderate these relationships. The specific aims of the dissertation are to investigate: (1) racial/ethnic and gender differences in

CRP levels; (2) differential exposure to discrimination as an explanation for the social patterning of CRP; (3) differential vulnerability to discrimination; and (4) the extent to which coping resources dampens these effects.

The dissertation uses data from the Health and Retirement Study (HRS), a multi-cohort longitudinal survey of a large and nationally representative sample of adults age 51 years and older. The HRS aims to identify and better understand the social, economic, psychosocial, and physical factors that influence and result from retirement. Since its inception in 1992, data have been collected every two years on the original HRS cohort and on subsequent cohorts added to the study. The "core" HRS interview is conducted face-to-face at baseline and collects data on demographic characteristics; housing and family structure; employment, income, assets, and insurance; health care, health status, cognition, and disability; and life expectations. Beginning in 2004, HRS respondents were asked to complete a psychosocial questionnaire that included measures of everyday and lifetime discrimination, among other factors, and in 2006 biomarkers—including CRP—were collected from one half of the study sample; biomarkers were collected from the other half in 2008. To maintain sufficient statistical power to detect differences in CRP, multivariate analyses are conducted with data from a hybrid 2006/2008 sample. The analysis involves statistical methods needed to appropriately adjust for the complex sampling design of HRS and to test the intricate causal pathways leading to race differences in CRP.

In line with our hypotheses, African Americans have higher levels of CRP than non-Hispanic whites and reported greater exposure to everyday and lifetime discrimination. Hispanics do not significantly differ from non-Hispanic whites in CRP levels or in their reports or everyday and lifetime discrimination. In unadjusted models, everyday and major lifetime

discrimination are positively associated with CRP. This association remains for lifetime discrimination in fully adjusted models but not for everyday discrimination. Both everyday and lifetime discrimination mediate racial/ethnic and gender differences in inflammation. Lifetime discrimination mediates these differences in and of itself, and in conjunction with other factors, particularly waist circumference. Everyday discrimination also mediates group differences in inflammation in conjunction with waist circumference. Vulnerability to discrimination does not differ by race/ethnicity or by gender, and the coping resources evaluated in this study do not buffer the effects of discrimination on inflammation.

This study supports the presence of racial differences in CRP—an indicator of systemic inflammation and a clinical risk factor for cardiovascular diseases—on a population-level. The findings also suggest that exposure to discrimination, a chronic stressor that disproportionately affects racial minorities, is associated with higher levels of CRP and partially explains racial and gender differences in inflammation. In all, these findings provides further support for the significant contributions of the social environment on health and health disparities.

The dissertation of Uchechi Acholonu Mitchell is approved.

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2014

DEDICATION

Saying “thank you” can never fully express how indebted I am to the many people who have given me unwavering love, support and encouragement throughout this journey. The list is long and I highlight some people at the risk of excluding others, but please know that my feelings gratitude transcends these pages. This work and my success are dedicated to you all, but in particular:

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Saying “hi” when you got off the elevator, when you walked by me in the hallway, and when you were walking up the stairs, undoubtedly is the best thing I have done in the past 10 years! It amazes me how you love me and how dedicated you are to *my* ambitions, as well as your own, and to our love and success as we grow in out relationship. You have brought me so much joy and happiness, and you have shown me that there is no limit to my potential. I thank you, wholeheartedly, for these gifts and for the gifts of your enduring love and companionship.

To My Parents

How do you begin to thank the two individuals who made you who you are today; who showed you a path to success and walked right beside you—without question—the entire way; who showed you the value of a *great* education and of hard work and determination; and who taught you unconditional self-love and loyalty to your goals, ambitions and pursuit of happiness. I am beholden to you and these few lines are just a humble beginning at expressing my true gratitude. I thank you and I look forward to showing you my gratitude everyday.

To My Siblings

I have been blessed with an amazing older sister and two amazing younger brothers. Ugochi, growing up I fought to break free from your “shadow”, from the *years* of dressing alike, from

having the same hairstyles, from being confused for one another (even by our own relatives), and from constantly being referred to as twins (which we're not). Fortunately, I could not and did not escape; and now the shadow I once ran from is a guiding light. You are one of my role models and I am grateful that I have someone like you to look up to. Thank you for being there for me when I needed you the most. Chiedo and Ikenna, growing up you put up with me and tolerated my "I know everything" "you have to listen to me because I'm older that you" attitude. For that alone, I *must* say thank you! But I also thank you for showing me how not to take myself or life too seriously and for being both my family and my friends.

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My friends are an extension of my family and in my heart the line between the two is nonexistent. From elementary school to graduate school, I have met and befriended a phenomenal group of women who have shared in my experiences and provided the proverbial "cherry on top" in the dessert of life. For your friendship I thank my high school friends: Narom and Alice M.; my college friends: Onyi, Stella, Sonia, and Ericka; my college friends and blockmates: Luz, Lacey, Li, Divya, Jessica, Alice Y., Joanie, and JJ; my graduate school friends: Eli, Mienah, Akilah, Kyoko, and Jackie; and friends from other aspects of my life who consistently were there for me when I was in need.

To My Community

Finally, my gratitude is also extended to a larger community of individuals who have directly and indirectly contributed to my success. This list of individuals includes, but is not limited to, my grandparents, aunts and uncles, cousins, extended relatives, and extended family friends. You believed in me and that is more than enough. Thank you.

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Mitchell, U. A., Aneshensel, C. S. Mechanisms of Racial Health Disparities in Inflammation and Blood Pressure: A Test of the Differential Stress Exposure and Differential Stress Vulnerability Hypotheses. Population Association of America (PAA), Boston, MA. May 2014.

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Acholonu, U., Morisky, D. Influence of Active-coping on Medication Adherence in a Low-income Hypertensive African-American Community: A New Application of John Henryism. American Public Health Association (APHA) Annual Meeting, Philadelphia, PA. November 2009.

INTRODUCTION TO THE DISSERTATION

Cardiovascular diseases, such as heart disease and stroke, have been the number one killer in the United States for more than 100 years. They currently affect more than a third of the adult population and by 2030 it is estimated that approximately 40% of Americans will be diagnosed with at least one cardiovascular disease (Roger et al., 2011). Racial minorities are particularly at risk for being diagnosed with and dying from cardiovascular diseases. For example, in 2008, the prevalence of cardiovascular diseases was 44.8% and 47.3% among African American men and women, compared to 37.4% and 33.8% among non-Hispanic white men and women, respectively (Roger et al., 2011). The 2008 death rate attributable to cardiovascular diseases for African American men was 1.35 times the rate for non-Hispanic white men, and the death rate for African American women was 1.38 times the rate for non-Hispanic white women. Additionally, in 2010, the prevalence of hypertension was 33.8% for African Americans, 23.6% among non-Hispanic whites and 22.5% among Hispanics.

The social patterning of cardiovascular diseases is not due to chance; it is rooted in a system of social stratification that consistently and systematically disadvantages minority populations (Adler & Rehkopf, 2008). Racial health disparities are created through residential segregation, differential access to societal goods and services (Williams & Mohammed, 2009), and the unequal distribution of social stressors and the resources needed to cope with these stressors (Pearlin, 1989; Williams & Mohammed, 2009). Therefore, identifying factors that put minority populations at greater risk for cardiovascular diseases has important implications for eliminating racial disparities in cardiovascular health. One such factor is discrimination. Discrimination is a stressor that manifests as discrete life events that significantly hinder

opportunities for status attainment and as day-to-day experiences of unfair treatment (i.e. everyday discrimination), such as being treated with less respect than others or being threatened or harassed (Williams, Yu, Jackson, & Anderson, 1997; Kessler, Mickelson & Williams, 1999). It disproportionately affects racial minorities (Kessler, Mickelson, & Williams, 1999) and has been linked to multiple negative health outcomes including low infant birth weight, elevated blood pressure, hypertension, mental health disorders, such as depressive symptoms, and all-cause mortality (for a review see: (Mays, Cochran, & Barnes, 2007; Pascoe & Richman, 2009; Williams & Mohammed, 2009).

The "black box" linking discrimination to poor cardiovascular outcomes is stress-induced physiological dysfunction. Current research surrounding the health effects of discrimination and other stressors has shifted from clinical measures of illness, as defined by a diagnosis made by a trained medical provider, to markers of physiological functioning (i.e. biomarkers) that are typically free of clinical evaluation but are associated with disease progression. Focusing on biomarkers is advantageous for: 1) early identification of chronic diseases such as atherosclerosis (i.e., screening), 2) understanding the progression of diseases over time, and 3) determining the effectiveness of clinical interventions on a molecular level (Vasan, 2006). One indication of physiological dysfunction is a sustained elevation of circulating inflammatory markers, such as C-reactive protein (CRP), which is referred to as systemic inflammation.

Among other conditions, systemic inflammation is associated with cardiovascular diseases including stroke, atherosclerosis, and myocardial infarction, for example (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997; Ridker, Hennekens, Buring, & Rifai, 2000). As is pertains to the effects of discrimination, epidemiological research has demonstrated a positive association between chronic forms of discrimination and inflammation (Friedman, Williams,

Singer, & Ryff, 2009; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010); one study found racial differences in this association, between African Americans and non-Hispanic whites (Cunningham et al., 2012). Since inquiry into the effects of discrimination on biomarkers in general is a nascent agenda in epidemiological research, studies investigating population-level associations between discrimination and inflammation are relatively limited in number. Furthermore, because there is a plethora of inflammatory markers to investigate, demonstration of the consistency—or lack of consistency—in the effect of discrimination on a particular biomarker is outstanding. Even more scant is literature investigating group differences in the association between discrimination and inflammation and literature illuminating the psychosocial factors that potentially mediate or moderate this relationship.

In light of this gap in the literature, this dissertation investigates racial/ethnic and gender differences in systemic inflammation and the association between discrimination and inflammation. Of particular interest are pathways involving exposure to discrimination that either mediate or moderate racial, ethnic and gender differences in inflammation. Stress buffering by emotional social support and two other coping resources—purpose in life and optimism—also is evaluated. In the end, the findings of this dissertation contribute to a burgeoning line of research investigating systematic differences in health by race/ethnicity and gender, and linking discrimination and other psychosocial factors to biological processes, on a population level. The collective knowledge gained through the identification and explication of the relationships among these factors is a fruitful step forward in understanding the contribution of psychosocial factors in health disparities.

CHAPTER 1

BACKGROUND

1.1 Introduction

This chapter provides an epidemiological overview of cardiovascular diseases and their risk factors. It also includes a review of literature regarding the manner through which social stressors—particularly discrimination—adversely affect health and contribute to health disparities.

1.2 Definition and Epidemiology of Cardiovascular Diseases

Although prevalence and mortality rates are on the decline, cardiovascular diseases have continued to be among the leading causes of death in the United States since the early 1900's. They currently are responsible for more deaths than cancer, chronic lower respiratory disease, and accidents combined, and they accounted for nearly 33% of all deaths in the United States in 2008 (Roger et al., 2011). The fiscal burden of cardiovascular diseases is estimated at more than $297.7 billion in direct and indirect health care costs, a figure that is expected to triple to $818 billion by 2030. Given their immense physical and fiscal burden, the prevention and treatment of cardiovascular diseases are important public health priorities.

Cardiovascular diseases are a collection of diseases affecting the heart and vasculature. This classification of diseases includes coronary heart disease—the leading cause of death in the United States—high blood pressure, myocardial infarction (i.e., a "heart attack"), stroke, and congestive heart failure, for example. Although cardiovascular diseases have a greater prevalence and health impact among older adults, especially those aged 60 years or older, the antecedents and risk factors begin early in life affecting people from infancy into old age. Those aged 60 years or older, however, carry close to 50% of the disease burden (Roger et al., 2011),

while the presence of cardiovascular diseases during childhood is uncommon and typically due to congenital abnormalities (Mensah & Brown, 2007; Roger et al., 2011).

In addition to differences based on age, prevalence of cardiovascular diseases differs by other demographic factors including, importantly, race/ethnicity and gender.[1] For example, men have a greater prevalence and incidence of cardiovascular diseases than women, but among all people diagnosed with at least one cardiovascular disease a greater portion of women die than men (Roger et al., 2011). This pattern of gender differences in morbidity and mortality is present among non-Hispanic whites, African Americans, Hispanics, Asians and Pacific Islanders, and American Indians and Alaska Natives. When looking across racial/ethnic groups instead of genders, cardiovascular diseases particularly affect some minority groups over others. In 2008, based on National Health and Nutrition Examination Survey (NHANES), the prevalence of cardiovascular diseases among adults aged 20 years or older was highest among African American women (47.3%), followed by African American men (44.8%), non-Hispanic white men (37.4%) and non-Hispanic white women (33.8%) (Roger et al., 2011). Mexican American men and women had the lowest prevalence of 30.7% and 30.9% respectively. Additionally, the death rate attributable to cardiovascular diseases among African American men was 35% higher than the rate for non-Hispanic white men and 38% higher among African American women compared to non-Hispanic white women (Roger et al., 2011). A similar pattern of difference is seen when looking at specific cardiovascular diseases such as coronary heart disease. Compared to non-Hispanic white women, African American women are 1.28 times more likely to die from coronary heart disease; the rates among Hispanic and Asian women are 0.82 and 0.58 times those of their non-Hispanic whites counterparts. Among men, the rates among African

[1] Although there is variation in the prevalence of specific cardiovascular diseases across racial/ethnic groups and genders, the following statistics refer the combined prevalence of high blood pressure, coronary health disease, myocardial infarction, angina pectoris, heart failure, stroke, and cardiovascular-related congenital defects.

Americans, Hispanics and Asians are 1.16, 0.74, and 0.55 times those of non-Hispanic white males, respectively. Explanations for these group differences in cardiovascular health lie within a complex mix of risk factors.

1.3 Risk Factors for Cardiovascular Diseases

An intricate and vast list of factors is involved in the development of cardiovascular diseases and in the presence and persistence of racial, ethnic and gender disparities in prevalence and mortality. A family history of cardiovascular diseases is associated with an increased risk of developing cardiovascular disease later in life (Roger et al., 2011), as is being diagnosis with other chronic conditions, such as diabetes, obesity, high cholesterol, and kidney disease. Additionally, lifestyle and behavioral factors such as level of physical activity, dietary habits, smoking status, and alcohol consumption are among the most studied risk factors. Being sedentary and consuming high-caloric, unhealthful meals promotes obesity, a leading risk factor for several cardiovascular diseases and other chronic conditions. Smoking is just as impactful as obesity if not more so. Each year from 2000 to 2004, an estimated 3 million years of potential life lost for males and 2 million years for females is attributed to smoking (Roger et al., 2011). Given the salient role these lifestyle factors play in sustaining "ideal cardiovascular health"—a concept recently coined by the American Heart Association (AHA) (Lloyd-Jones et al., 2010)—promoting optimal levels of physical activity, improved dietary habits, and reduced smoking and alcohol consumption is a central mission of the AHA's 2020 Impact Goals (Roger et al., 2011).

In addition to lifestyle factors and behaviors, psychosocial factors such as depression and exposure to stressors also contribute to cardiovascular diseases. Depression is an affective state characterized by prolonged sadness, fatigue and loss of energy, and a general disinterest in usual activities, among other symptoms (American Psychiatric Association, 2000). In a randomized

study of the effectiveness of antihypertensive therapy, Abramson, Berger, Krumholz, and Vaccarino (2001) found that older adults who presented with depressive symptoms at baseline were more likely to die of heart failure than older adults who did not present with depressive symptoms; this association was present in both unadjusted models and models adjusted for demographic factors, medical history, and time since follow up. Thus, depression is a risk factor for cardiovascular diseases.

Likewise, acute and chronic exposure to stressors increases the risk for atherosclerosis, myocardial infarction, and coronary hearth disease (Steptoe & Kivimäki, 2012). The effect of stressful experiences may lead to the adoption of unhealthy behaviors such as smoking (Slopen et al., 2012) or to the development of depressive and anxiety disorders (Turner & Lloyd, 2004), which increase the risk for adverse cardiovascular events (Abramson et al., 2001). The association between stress exposure and health may be confounded by sociodemographic factors such as age, gender, race, education, and income because these factors play a role in the distribution of stressors (Pearlin, 1989)—a point that will be elaborated upon when discussing the theoretical framework—and they influence risk for cardiovascular diseases at least in part through the behavioral and psychosocial pathways mentioned.

Frequent exposure to stressors can negatively affect health by promoting undesirable changes in stress physiology. In this situation, gradual changes in the function of the immune system, for example, develop over time and lead to dysfunction (i.e., sustained deviation from normal stress physiology). When the dysfunction occurs in biological systems that are central for protecting against physical and psychological harm (e.g., the immune system), processes that were once favorable become detrimental. In this way, physiological dysfunction becomes the mechanism through which stressors undermine health in general and cardiovascular health in

particular. The next section will briefly review the biological pathways linking the central nervous system, the endocrine system and the immune system. For guidance, Figure 1.1, 1.2 and 1.3 are illustrations of the HPA-axis, the inflammation reflex of the autonomic nervous systems and the actual process of inflammation, respectively.

1.4 Stress, Inflammation and Cardiovascular Diseases

Review articles published by Miller, Chen, & Zhou (2007) and Irwin & Cole (2011) offer comprehensive reviews of stress physiology and the reciprocal relationship between the nervous system and the immune system; they are the primary references for the following discussion.

The stress response is coordinated by the central nervous system (CNS). Stimuli from the physical and social environment are received and integrated by certain regions of the brain and signal a cascade of physiological reactions in response to stimuli considered, threatening, challenging or taxing. Two stress signaling pathways stemming from the CNS are involved in the inflammatory stress response: the hypothalamus-pituitary-adrenal (HPA) axis and the inflammatory reflex of the autonomic nervous system (ANS)

The first pathway begins with a brain structure known as the hypothalamus (Step 1, Figure 1.1). The hypothalamus is the coordinating center of the central nervous system; it is responsible for integrating internal and external cues such as changes in hormone levels, blood volume and temperature. It also integrates affective and social cognitive sensations such as fear and anxiety that are associated with stress exposure. The integration of these signals initiates the release of corticotropin-releasing hormone (CRH) (Step 2, Figure 1.1); CRH travels through blood vessels in the hypothalamus to the anterior pituitary gland, which sits right below the hypothalamus. Adrenocorticotropin hormone (ACTH) is released from the anterior pituitary gland (Step 3, Figure 1.1) and subsequently travels through the circulatory system to the adrenal

cortex (Step 4, Figure 1.1), which sits on top of the kidneys, to signal the release of hormones known as glucocorticoids, specifically cortisol (Step 5, Figure 1.1). The anatomical structures involved in this process—from the activation of the hypothalamus to the release of cortisol—are referred to as the HPA axis.

The second signaling pathway begins with activation of the autonomic nervous system, which includes two branches of nerve fibers referred to the sympathetic nervous system (SNS) and the parasympathetic nervous system (PSNS) (Figure 1.2, left-hand panel). In response to perceived stress, nerve fibers form the SNS release neurotransmitters called norepinephrine (NE) that spread throughout the vasculature and organ tissue. The SNS also stimulates the adrenal gland to produce adrenaline (i.e., epinephrine), as well. Norepinephrine and epinephrine travel to lymphoid organs where they interact with structures on immune cells (among other cells of the body) called alpha-adrenergic and beta-adrenergic receptors. Activation of these receptors leads to an increase in the transcription of genes that encode inflammatory markers. In other words, the SNS stimulates the release of noradrenaline and adrenaline, which leads to increases in biomarkers of inflammation.

Inflammation is a biological process designed to protect against harm or injury by recruiting immune cells to the site of an infected area, creating a physical barrier to prevent the spread of infection, and repairing injured tissue (Figure 1.3). For example, if a person is wounded (e.g. cuts his or her finger) chemical signals are released from the damaged tissue that increase blood flow to the site of injury, and activate and signal to immune cells that an injury has been sustained. The immune cells—specifically neutrophils and macrophages, followed by leukocytes (i.e. B cells and T cells)—travel to the injury site, eliminate the pathogen, and contain the spread of infection. During this process, the immune cells are "communicating" with each

other through the release of proteins known as cytokines, or interleukins,[2] and recruiting more cells to the site of injury. The cytokine levels increase rapidly to accelerate the recruitment of additional immune cells that prevent the spread of infection. The physical symptoms associated with inflammation—swelling, redness, increased temperature, and pain—are all due to the increased blood flow and immune activity at the injury site.

Inflammation and the production of cytokines (i.e. interleukins) also occur during times of stress through the two signaling pathways recently described. In this situation, the inflammatory process is a preemptive response initiated in anticipation of physical harm. For example, when a person is faced with a stressor signals originating from the autonomic nervous system activate the HPA-axis to stimulate the production of cortisol. Cortisol then activates immune cells involved in inflammation by binding to glucocorticold receptors on those cells. The immune cells release cytokines, as described before, that travel throughout the body. They travel to the liver to signal the release of acute phase proteins (e.g. C-reactive protein) that help rid the body of bacteria, and they travel to the brain to activate the HPA-axis and further production of cortisol. At later stages of the inflammation process, however, cortisol works to suppress immune cell activity and the release of cytokines. In this way, there is a negative feedback loop between cortisol and cytokines such that cortisol stimulates the production of cytokines during the initial stages of a stress response, and suppresses their production during later stages of the stress response when cytokine levels are substantially increased from baseline levels.

In this way, the HPA-axis is considered more of an immunosuppressive pathway despite its initial effects initiating inflammation. However, in times of chronic stress, the

[2] The terms "cytokine" and "interleukin" are often used interchangeably. I use the term "cytokines" to refer to this group of proteins collectively and the term "interleukin" to discuss a specific protein (e.g. interleukin-6).

immunosuppressive function of cortisol may be compromised, which can lead to the unabated production of inflammatory markers. Cortisol's inability to stop the activation of immune cells and the production of cytokines may be due to glucocorticoid insensitivity. Glucocorticoid insensitivity or resistance refers to a condition where glucocorticoids like cortisol are no longer able to bind to their designated receptors (Miller, Cohen, & Ritchey, 2002; Pace, Hu, & Miller, 2007; Raison & Miller, 2003). Their inability to bind to their immune cells receptors manifests as the inability to "turn off" the inflammatory process. Alternatively, chronic stress exposure may lead to "blunting" of the HPA-axis through its chronic activation. Reductions in HPA-axis activity—and the subsequent reductions in the production of cortisol—prevent the suppression of inflammation. The sustained levels of inflammation can damage blood vessels and various organs in the body (Raison & Miller, 2003).

The signaling pathway through the autonomic nervous system can also fail to "turn off" and lead to constant sympathetic nerve firing and elevation of inflammation, blood pressure and other biomarkers. In contrast to the HPA axis, however, this signaling pathway is more consistently pro-inflammatory (i.e., leading to higher levels of inflammation) (Irwin & Cole, 2011).

Several different cytokines are involved in the inflammatory process. Extensive research has been done on the relationship between stress exposure and interleukin-6, a specific cytokine. In addition to other functions, interleukin-6 triggers the synthesis and release of C-reactive protein (CRP) from the liver during inflammation. C-reactive protein is an acute phase protein that fends off pathogens and clears damaged or dead cells from the body. It is a reliable and stable measure of immune activity because it remains elevated for longer periods of time. Laboratory and population-based studies have provided empirical evidence for the association

between these inflammatory markers and cardiovascular diseases. For example, when comparing tertiles of CRP, Danesh et al. (2004) found that individuals with CRP values within the highest tertile had greater odds of incident coronary health disease compared to individuals with CRP values within the lowest tertile. Taken together, these findings suggest that chronic stress exposure is associated with inflammation and inflammation is associated with greater risk for cardiovascular diseases. The challenge, however, with using biological endpoints to assess the risk of developing cardiovascular diseases is identifying a clear point at which the inflammatory process shifts from protective to pathogenic. For CRP that has been collected using a high-sensitivity assay (i.e. hs-CRP), values above 3.0 mg/L are currently used as an indicator of increased risk for cardiovascular disease (Ridker, 2003).

1.5 Discrimination, Inflammation and Cardiovascular Diseases

A variety of physical, environmental and psychosocial factors can trigger a stress response. Characteristics of these stressors differ based on several factors including frequency of exposure, timing along the life course, severity, and duration (Wheaton, 1994; Wheaton, Young, Montazer, & Stuart-Lahman, 2013). Additionally, stressors vary based on the level of social organization from which they are generated—from the micro-level of interpersonal conflicts in relationships, to the meso- and macro-levels of organization which are exemplified by neighborhood problems and economic recessions, respectively. Variations in these characteristics of stressors can lead to variations in the stress response.

Discrimination is a social stressor (Clark, Anderson, Clark, & Williams, 1999; Harrell, 2000) that disproportionately affects racial and ethnic minorities and women (Hausmann, Kressin, Hanusa, & Ibrahim, 2010; Kessler, Mickelson & Williams, 1999). As opposed to biased beliefs and attitudes (i.e., prejudice), discrimination refers to biased behaviors/actions

towards members of a particular social group who share a common characteristic that is devalued and stigmatized within the social hierarchy (Link & Phelan, 2001). In its chronic form, it manifests as day-to-day experiences of unfair treatment, such as being treated with less respect than others or being threatened or harassed; in its acute form, it manifests as major experiences of unfair treatment, often significantly affecting status attainment or well-being, such as unfairly being denied a job (Williams, Jackson & Anderson, 1997). On the one hand, these major acts of discrimination are formidable stressors because of their intensity and potentially adverse fiscal and social consequences; on the other hand, the subtle, unpredictable, and uncontrollable instances of everyday discrimination are difficult to cope with and may lead to rumination, which potentially prolongs its effects (Harrell et al., 2011; Harrell, 2000). In actuality, both forms of discrimination detrimentally affect mental and physical health. Additionally, discrimination can lead to additional stressors or magnify the effect of co-occurring stressors (Harrell, 2000). It these ways, the stress of discrimination can contribute to the development and progression of chronic conditions like cardiovascular diseases through its impact on stress physiology and health behaviors over an extended period of time.

A clearer understanding of the relationship between everyday and major lifetime discrimination is achieved by using the general stress literature as a point of reference. An important concept discussed in this literature is the process of stress proliferation where a primary stressor within one domain of life can lead to a secondary stressor within a different domain of life (Pearlin, Aneshensel & LeBlanc, 1997); considering a more specific arrangement, an acute stressor can lead to a chronic stressor and, likewise, a chronic stressor can lead to an acute stressor. For example, losing a job is a major life event (i.e., an acute stressor) that can lead to prolonged financial difficulties (i.e., a chronic stressor) and financial difficulties within

the context of a marriage or partnership can lead to divorce, another major life event.[3] Similarly, discrimination in the form of being unfairly stopped or harassed by the police—an acute form of discrimination that frequently occurs to African American and Hispanic males—can lead to situations where an individual is routinely treated with less courtesy or respect than others or treated as though dishonest or "lesser" in status, due to the stereotypes associated with negative interactions with law enforcement. This process of stress proliferation is especially relevant when the altercation with the police occurs in a setting (e.g., neighborhood or workplace) that the individual frequents regularly. Additionally, if pervasive, this chronic form of unfair treatment (e.g., being treated as though dishonest or "lesser" in status) can diffuse into the domain of employment and prevent the same individual from obtaining a job he or she should have rightfully, or it can lead to an unfair job firing.

Finally, the effects of two (or more) stressors, including the two forms of discrimination discussed, can exacerbate one another such that the overall effect of the stressors is greater than the sum of its parts. With respect to the overall effect of discriminatory experiences on health and wellbeing, the effect of everyday discrimination can be exacerbated by a major lifetime experience of discrimination and the effect of major lifetime discrimination can be exacerbated by everyday discrimination. For these reasons, investigations including both forms of discrimination are beneficial and facilitate more accurate estimation of the effect of discriminatory experiences in general.

Although population-based studies looking at clinical outcomes offer support for discrimination's effect on health, investigating biological endpoints that describe the physiological mechanisms through which discrimination and other stressors affect health

[3] Divorce is most often preceded by marital strain (a chronic stressor) that can result from financial difficulties, which highlights the increasing complexity of the stress proliferation process.

provides greater support for causal claims about this association. Accordingly, there has been a recent emergence of research investigating the effect of discrimination on biomarkers related to the stress response and chronic conditions. As reviewed previously, one aspect of the stress response is activation of the immune system, which results in an increase in circulating inflammatory markers. As it pertains to discrimination, Friedman, Williams, Singer & Ryff (2009) used data from Wave I and II of the Survey of Midlife in the United States (MIDUS) to investigate the relationship between racial discrimination and serum concentrations of E-selectin, a cell-adhesion molecule expressed on the surface of endothelial cells during the inflammatory response. Among a predominately non-Hispanic white population, they found that more frequent experiences with discrimination predicted increased serum concentrations of E-selectin but only in men. Similarly, Lewis et al. (2010) used data from the Minority Aging Research Study (MARS) to investigate the relationship between racial discrimination and serum concentrations of CRP among older African Americans. They found that more frequent experiences of discrimination were associated with higher serum levels of CRP. This effect was not mediated by depressive symptoms but was partially mediated by body mass index (BMI).

In contrast to the findings by Friedman et al. (2009) and Lewis et al. (2010), Albert et al. (2008) found no association between racial discrimination and serum CRP levels, among African Americans, Hispanics and non-Hispanic whites. When looking at differences across genders and racial/ethnic groups, Cunningham et al. (2012) found a positive linear association between experiences of racial discrimination and CRP among non-Hispanic white women, but no association among non-Hispanic white men. Among African American women, the association was curvilinear such that those reporting 1-2 experiences of discrimination had the highest levels of CRP, those reporting 3 or more experiences had the lowest, and those reporting no

discrimination had CRP levels between the other two levels. This association was no longer significant after controlling for lifestyle factors such as smoking, alcohol consumption and BMI. Among African American men a negative linear relationship existed but it too was no longer significant after adjusting for lifestyle factors. These finds suggest that the effect of discrimination on inflammation varies by demographic indicators such as race/ethnicity and gender. Taken together, the literature in this area is not consistent, which necessitates further investigation of this relationship. The differences in the patterns of association across and within racial groups may be due to differences in exposure and/or factors that mediate or moderate the relationship between discrimination and inflammation.

1.6 Stress-related Mechanisms of Health Disparities

Because research regarding the effects of discrimination on inflammation is a new and developing line of inquiry, the findings from Cunningham et al. (2012) leave open for discussion (1) what the nature of the discrimination-inflammation association is among different racial groups and genders, (2) whether differences in this association contribute to racial and gender differences in health, and (3) if so, how? As the authors suggested in their discussion, heterogeneity across and within groups may be due to differences in exposure to discrimination and differences in vulnerability, respectively. Both hypotheses are tested in the dissertation Since population-level investigation of inter- and intra-group differences in stress-initiated physiological dysfunction is still emerging, in some instances clinical health outcomes rather than biological markers will be used in the following discussion; and I proceed with the assumption that the patterns and mechanisms we would see using biomarkers as outcomes would be similar to those we would see with clinical outcomes, since changes in biological processes—for the most part—underlay clinical evaluations of disease.

To begin, the fact that society stratifies its members based on certain characteristics, such as race/ethnicity, gender, age, socioeconomic status, and sexual orientation, influences the distribution of material, fiscal and social resources, deficits and threats (Pearlin, 1989; Turner, Wheaton, & Lloyd, 1995). Social stressors, like discrimination, are conditions and experiences that are incompatibly with the needs, abilities and resources of an individual and challenge an individuals ability to adapt to the stressful circumstances (Aneshensel, 1992). These stressors and the resources to cope with them are systematically distributed along social strata and lead to a disproportionate concentration of some stressors among social groups that historically have been marginalized, such as racial/ethnic minority groups. Consequently, individuals belonging to marginalized social groups who experience more frequent or more severe stressors may also experience earlier and more rapid deterioration in health over time. When viewed in the context of racial health disparities, this patterned experience of health deterioration due to concentrated stress exposure and disadvantage is referred to as the weathering hypothesis (Geronimus, 1992).

The weathering hypothesis argues that racial variations in health outcomes are due to the accumulation of disadvantage, which physically manifests as premature deterioration in health. Support for this hypothesis is seen in a recent study of black-white differences in age patterns of allostatic load, a composite measure of biomarkers representing the physiological burden of stress (Geronimus, Bound, Keene, & Hicken, 2007). Baseline allostatic load scores were higher among African American than non-Hispanic whites in all age categories. Age trends showed that African American women had the greatest probability of high allostatic load scores than other racial-gender groups; moreover, the gap in probability projections increased with increasing age. The authors contend that the cumulative exposure to stressors related to the "double jeopardy" of being African American and a women may put this group at greater risk for

poor health outcomes and accelerated aging compared to non-Hispanic whites and African American men.

An additional test of the weathering hypothesis compared Mexican immigrants to U.S.-born Mexican Americans, non-Hispanic whites and African Americans (Kaestner, Pearson, Keene, & Geronimus, 2009). This study produced findings similar to Geronimus et al. (2007) and served as a test of the "Latino Paradox". Immigrants who spent more time in the U.S. experienced greater declines in health as measured by increasing allostatic load scores. The authors interpreted the increase in allostatic load as a consequence of greater exposure to stressors over time due to the dually disadvantaged status of being an immigrant and an ethnic minority. This specific hypothesis, however, was not directly tested by Kaestner et al. (2009) or by Geronimus et al. (2007); the authors did not include a measure of stress exposure in their analyses. Nonetheless, these findings lend some support to the differential stress exposure hypothesis by showing that being a member of a typically marginalized or disadvantaged racial or ethnic group is associated with greater physiological burden or dysfunction, which precipitates disease.

Similar to stress exposure, the personal and social resources needed to effectively cope with stressors may be differentially distributed based on social status (Kessler, 1979; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Turner, Lloyd, & Roszell, 1999). The differential distribution of these resources may lead to within- and between-group differences in vulnerability (Ulbrich, Warheit, & Zimmerman, 1989) that ultimately contribute to group differences in health outcomes (Pearlin, 1989). For this reason, determining whether vulnerability to stress is consistent across race/ethnicity and/or gender, and investigating the buffering capabilities of coping resources are important empirical endeavors. With regard to the

differences in vulnerability, the study by Cunningham et al. (2012) that was reviewed in the previous section is an example of how the effect of discrimination on inflammation can differ across racial/ethnic groups and genders. That study alone provides a good example of how the discrimination-inflammation association can differ in magnitude, direction and shape across race/ethnicity and gender. These variations in vulnerability to discrimination may be due to racial/ethnic and gender differences in coping responses and differences in levels of resources that are advantageous for coping with stressors.

Coping resources are environmental and personal attributes that help individuals overcome or sustain themselves during adverse situations. Although these resources are assumed to occur in conjunction with a stress exposure, they can exist apart from stressors and the cognitive and behavioral responses elicited by stressors (Wheaton, 1985). Social support is one of the most researched resources. In addition to having an independent effect of health, emotional social support can function as either a moderator or mediator of the relationship between stressors and health outcomes (Barrera, 1986; Cohen & Wills, 1985). Arguably, the independent effects of social support on health and its stress buffering effects as a moderator has dominated the stress literature (See Uchino (2006) for a review); far less is known about its role as a stress mediator especially when looking at discriminatory stress.

A recent study by Prelow, Mosher, and Bowman (2006), however, lends support to the often overlooked role of social support as a mediator. Among a sample of African American college students, exposure to discrimination was associated with a decrease in social support, which led to greater reports of depressive symptoms and lower levels of life satisfaction. Models for stress buffering by social support and resources mobilization of social support were tested and not supported by the data. Although the outcomes of this particular study are not

biomarkers, the findings give credence to the role of social support as a mediator of the adverse health effects of discrimination. Therefore, an investigation of both the mediating and moderating function of this resource is advantageous.

It is also advantageous to investigate the mediating and moderating effects of coping resources other than social support and to compare findings across resources. This comparison is useful for ascertaining the specificity of the effect of social support and other resources on the focal relationship. Two coping resources of growing interest in the stress and aging literatures are purpose in life and optimism. Purpose in life is a component of psychological wellbeing (Ryff, 1989) that differs from feelings of pleasure, happiness and satisfaction because it focuses on the realization of one's potential and possessing "goals, intentions, and a sense of directedness" (Ryff, 1989, pp. 43). It is a reflection of successfully negotiating challenges faced over the life course, finding meaning in or purpose from these past experiences, and learning more about oneself in the process (Ryff & Keyes, 1995).

Purpose in life is adversely affected by stress exposures, such as exposure to discrimination (Ryff, Keyes & Hughes, 2003), and—like other coping resources—research has shown that it is differentially distributed by age, gender, race/ethnicity, and education (Ryff, 1989; Ryff, Keyes & Hughes, 2003). As it pertains to health, having a strong sense of purpose in life is associated with good sleep quality (Steptoe et al., 2008), reduced incidence of disability (Boyle, Buchman & Bennet, 2011), and lower all-cause mortality (Boyle et al., 2009). With regard to inflammation, in particular, purpose in life has an inverse association with this biomarker (Friedman et al., 2007; Morozink, Friedman, Coe & Ryff, 2010; Ryff et al., 2006) and other correlates of cardiovascular disease (Morozink, Friedman, Coe & Ryff, 2010). Additionally, a recent study by Friedman and Ryff (2012) showed that among older adults

biological disease risk due to increases in medical comorbidities was attenuated by purpose in life. This study, as well as the other studies reviewed, provides support for investigating the function of purpose in life as a coping resource.

Similar to having a strong sense of purpose in life, being optimistic is beneficial for physical and mental health (See Carver, Scheier & Segerstrom, 2010, for a review). Dispositional optimism refers to having positive expectations for the future and typically it is considered a stable personality trait that directly affects health and moderates the impact of stressors and adversity on health. For example, Achat et al. (2000) found that dispositional optimism was associated with higher levels of self-rated general health among middle-aged and older men, and Kubzansky et al. (2001) found that an optimistic perspective towards life events was associated with lower cardiovascular disease risk. Additionally, optimism is associated with changes in the immune system. Ikeda et al. (2011) found that higher levels of optimism were associated with a decrease in levels of two biomarkers: soluble intercellular adhesion molecule-1 (sICAM-1) and interleukin-6 (IL-6); the latter biomarker is a marker of inflammation while the former is a marker of endothelial function. A study by Roy et al. (2010) revealed a similar association between optimism and CRP, however the association was not significant in fully adjusted models. Therefore, optimism may be consequential for systemic inflammation, which, as described previously, is a marker of immune functioning.

Changes in optimism may also be related to health. Although optimism is a personality trait that is thought to be constant within an individual, recent literature suggests that it might be amenable to social resources (Segerstrom, 2007) and cognitive-behavioral intervention (Antoni et al., 2001) that work towards increasing optimism. Accordingly, if optimism can be increased it seems plausible that optimism can also be decreased by certain exposures and contributes to

poor health due to the loss of its protective effects. Few studies have systematically investigated whether optimism can be adversely affected by stressful experiences and exposures originating from the social environment, such as discrimination, which points to an area of needed research.

CHAPTER 2

THEORETICAL FRAMEWORK & SPECIFIC AIMS

2.1 Introduction

This dissertation is theoretically guided by three models of stress within the sociological and psychological traditions, specifically Pearlin's Stress Process Model (Pearlin & Bierman, 2013; Pearlin et al., 1981), Lazarus' and Folkman's Transactional Model of Stress and Coping (Lazarus & Folkman, 1984, 1987), and Kemeny's Psychobiology of Stress model (Kemeny, 2003b). In combination, these theoretical perspectives contribute to our understanding of how stressors originating in the social environment impact the structure and functioning of physiological factors in the body, thereby affect health. An integrated framework of these three theories is depicted in Figure 2.1 and is elaborated upon below. This discussion begins with a brief description of the conceptualization of stressors and stress, followed by a summary of the three theories and a description of the conceptual model guiding this dissertation.

2.2 Conceptualization of Stressor and Stress

Early stress research tied the conceptualized of stressors to the physiological changes resulting from stress exposure (i.e. the stress response). Hans Selye and Walter Cannon, two pioneers of stress research within the biomedical tradition, examined stressors—such as injury to the body, exposure to cold, and involvement in emergency situations—and tested their ability to trigger physiological processes in animal models. Selye (1950) coined the phrase "general adaptation syndrome" to describe the short and long-term changes that occurred in the hypothalamic-pituitary-adrenocortical axis (HPA axis) in response to stress; Cannon (1932) originated the phrase "fight-or-flight response" to describe similar changes in the sympathetic

and endocrine systems. The ability to bring about the General Adaptation Syndrome or the "fight-or-flight" response became the defining characteristic of a stressor (Wheaton & Montazer, 2010). In other words, the presence of a physiological stress response defined a stressor. Alternative conceptualizations of stressors, particularly within the fields of psychology and sociology, acknowledged that certain stressors had the ability to elicit a physiological stress response but pointed out that the relationship between stressor and stress—i.e. the internal physical sensation—is not consistent (Cohen, Kessler, & Gordon, 1997). Social scientists further asserted that when faced with a circumstance that may objectively appear to be stressful, a corresponding stress response may or may not be prompted, depending upon individual and environmental factors that mediate or moderate the relationship between stressor and stress response.

Thus, the critique by social scientists of the body of stress research presented by Selye and Cannon led to refinements in the conceptualization of stressors. Two specific and important advancements include: 1) the recognition of the social environment as both a source and distributor of stressors and coping resources, and 2) an understanding of the relationship between characteristics of stressors, the cognitive appraisal process and coping, and the stress response. Lazarus and Folkman formally articulated the latter with their Transactional Model of Stress and Coping (Lazarus & Folkman, 1984), and Pearlin articulated the former with his Stress Process Model (Pearlin et al., 1981).

2.3 Stress Process Model

In the sociological tradition of stress research, the definition of a stressor is separated from the occurrence of a stress response. Stressors are conceptualized as the socioenvironmental demands that (1) overburden an individual's capacity/ability to adapt to circumstances, or (2)

hinder the achievement of sought-after ends (Aneshensel, 1992). They can be either acute, isolated events such as a divorce or death of a loved one, or chronic strains and occurrences that persist over long periods of time such as marital conflict of caring for an ailing parent. Stressors are present on all levels of society—from the individual level (e.g. marital conflict) to the neighborhood and institutional levels (e.g. neighborhood crime and organizational restructuring)—and they vary in their degree of severity from daily hassles to traumatic events. The stress process model describes how the social environment affects mental health by functioning as both the source and distributor of social stressors and coping resources.

To elaborate, social hierarchies based on gender, race, social class, religious affiliation, and sexual orientation, for example, are inherent in the social structure. Higher status social groups generally are exposed to fewer or less severe social stressors, compared to lower status social groups, and often have greater access to the resources needed to cope with these stressors (Pearlin, 1989). Additionally, the stress process model describes a system of relationships that includes social and personal resources—for example, coping, mastery, self-esteem, and social support—that function as moderators and/or mediators of the stressor-disease relationship. They can alter the relationship between stressors and health outcomes and they can explain the relationship (Pearlin, 1999). Thus, from the Stress Process Model comes theoretical guidance about how group differences in health are created and sustained.

2.4 Transactional Model of Stress and Coping

Other disciplines such as psychology also have contributed to the explication of the relationship between stress exposure and poor health. The work of Lazarus and Folkman falls within the psychological perspective of stress and emphasizes the role of individual perceptions and interpretations of circumstances in the generation of stress. Similar to the sociological

perspective, stress precipitates from an encounter or enduring situation that overtaxes an individual or demands more from her than she can do (Lazarus & Folkman, 1984). An important point of distinction from the sociological perspective is the Transactional Model of Stress and Coping argues that an encounter or situation first must be perceived to be harmful, threatening, or challenging prior to generating stress. This cognitive appraisal process is a key factor mediating the effects of stressors on health.[4] Two appraisal processes occur: the primary appraisal process determines the stressfulness of the encounter or situation, while the secondary appraisal process assesses the resources needed to cope with the stressor and the options available for preventing harm or overcoming the challenge (Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986).

Within this framework, coping involves cognitive processes and behavioral strategies aimed at managing stressful experiences and minimizing the perceived adverse consequences associated with a stressor (Billings & Moos, 1981; Lazarus, 1990). According to this model of stress, coping is a cyclical process that begins after a situation is appraised to be stressful; this appraisal is followed by a coping response, a re-appraisal of the situation and additional coping responses if previous attempts at resolving the situation are unsuccessful (Folkman et al., 1986; Lazarus & Folkman, 1984). As will be discussed shortly, physiological problems may arise if an individual is unable to successfully cope with or resolve a stressful situation, especially if the presence or awareness of the stressor is reoccurring or persists over long periods of time. In this way the Transactional Model of Stress and Coping informs our understanding of how individual coping processes contribute to inter- and intra-group variability in the adverse effects of stress.

[4] In contrast to this perspective, the sociological perspective suggests that stressors can have an affect even if a person does not consciously appraise the stressor as harmful, threatening or challenging. For example, enduring social circumstances, such as living in an impoverished community, are considered environmental stressors that may not be consciously appraised as stressful by an individual (Cohen, Kessler & Gordon, 1997); nonetheless, it elicits a physiological response that has an effect on the individual's health.

2.5 Psychobiology of Stress

The Psychobiology of Stress model picks up where the Transactional Model of Stress and Coping theoretically ends. Margaret Kemeny (2003b) articulated this model and explained that the mechanistic link between psychosocial risk factors, such as social stressors, and the progression of disease outcomes is mediated by physiological factors involving the central and peripheral nervous systems (Kemeny, 2003a, 2003b). The normal response to a stressor is the activation of the central and peripheral nervous systems and the immune and neuroendocrine systems. However, frequent and persistent exposure to stressors, say from the enduring stress of poverty, and the resulting over-activation of the stress response can compromise the integrity of the regulatory systems that are intended to protect the body from harm. Thus, the Psychobiology of Stress model—also referred to the X-Y-Z model—stipulates that establishing the causal role of psychosocial factors (i.e. the "X") in the etiology and progression of diseases requires identification of the physiological mediators (i.e. the "Y") that produce the specific disease outcome of interest (i.e. the "Z"). The physiological mediators are changes in the neural, immune, and endocrine systems that accompany disease.

The Psychobiology of Stress model is conceptually similar to the allostatic load hypothesis (McEwen & Lasley, 2003; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). This hypothesis puts forth the idea that fluctuations in physiological processes (e.g., blood pressure) due to internal or external stimuli are normal, but frequent and long-term exposure to stressors and other adverse stimuli compromise optimal functioning, which manifests as a resetting of normal physiological set points (e.g., sustained elevations in blood pressure in the absence of a stimulus). The allostatic load hypothesis focuses on a collection of physiological mediators that collectively measure dysfunction across physiological systems. The Psychobiology of Stress

model focuses on the specific relationship between a single or smaller set of physiological mediators that contribute to the etiology of a particular disease. It seeks to explains the process through which psychosocial factors influence physiology and, as a result, contribute to disease. Regardless of the terminology or perspective taken, the Psychobiology of Stress model and the allostatic load hypothesis offer theoretical explanations for the relationship between stress exposure, physiological dysfunction and disease. Therefore, before connecting psychosocial stressors to clinical disease outcomes an association must be established between stressors and physiological mediators (i.e. biomarkers) associated with dysfunction, disease etiology and disease progression.

2.6 Integrated Theoretical Framework of Stress

Taken together, the Stress Process Model, Transactional Model of Stress and Coping and the Psychobiology of Stress model describe a series of relationships linking social structure to disease. Figure 2.1 presents a theoretical framework that integrates these three models of stress.[5] Social characteristics such as race/ethnicity and gender influence relative social status and geographic location of residence.[6] These two factors determine, at least in part, both the distribution of stressors and the personal and social resources used to cope with stressors, and the nature of the stressors and resources individuals are presented with. These stressors influence one another and then directly or indirectly (through the cognitive appraisal process) elicit a set of affective, physiological, and behavioral responses to stressors that subsequently affect the progression of disease. Additionally, social and personal resources serve as moderators and/or mediators of the relationship between stressors and the different stress responses such that the

[5] Additional relationships (e.g., double-headed arrows) between constructs could have been depicted in this figure, such as the reciprocal relationship between disease and stressor. Although these relationships are important to consider, they are not the focus of this dissertation and, for clarity sake, have been omitted.
[6] This aspect of the framework is informed by Gee et al. (2002).

effect of the stressor is explained by the resource or it is altered in a manner that either exacerbates or attenuates the stress. In due course, these responses can lead to additional stressors and/or alter the relationship between stressors and the stress responses.

2.7 Conceptual Model

Informed by the theoretical framework just described and the literature reviewed previously, the conceptual model for this dissertation is depicted in Figure 2.2. This figure is specific to the constructs that are assessed in the dissertation. Moving from left to right on the figure, the focal independent variables are race/ethnicity and gender and the focal dependent variable is systemic inflammation (i.e. concentrations of CRP). It is hypothesized that racial/ethnic minorities and women will have higher levels of inflammation than non-Hispanic whites and men, respectively, and that exposure to everyday and lifetime discrimination will be associated with inflammation and mediate the differences in inflammation seen by race/ethnicity and gender. Furthermore, the relationship between discrimination and inflammation is expected to be moderated by race/ethnicity and by gender, which is indicative of differential vulnerability to this stressor. Coping resources, specifically social support, are hypothesized to buffer the effect of discrimination on inflammation, but they can also function as mediators of the discrimination-inflammation relationship. Finally, alternative risk factors for systemic inflammation are presented on the right-hand side of the figure and include health behaviors and health status. Age, marital status, household income, education, employment status and year of data collection are assessed in this dissertation as control variables that influence inflammation.

The next chapter of this dissertation describes each of these variables in more detail and explains how they were incorporated into the statistical analysis procedures. First, however, is a description of the specific aims of the dissertation, research questions and hypotheses.

2.8 Specific Aims, Research Questions and Hypotheses

The overarching goals of this dissertation are to determine if there are racial/ethnic and gender differences in inflammation, and to ascertain whether differential exposure to discrimination and/or differential vulnerability to discrimination explain any of the differences present. The dissertation assess differences in inflammation by race/ethnicity and/or gender and establishes whether differences in exposure to discrimination are associated with inflammation—as measured by concentration of C-reactive protein (CRP)—and, if so, whether discrimination explains differences in inflammation. The study also tests whether racial minorities and women are more vulnerable to the stress of discrimination, than non-Hispanic whites and men, respectively. Additionally, stress buffering by coping resources such as social support is ascertained. To accomplish the goals of this dissertation, the following aims, questions and hypotheses are investigated:

Specific Aim 1: To determine the extent to which levels of inflammation (i.e., CRP) vary by race/ethnicity and/or gender. Research Questions 1A-1D are:

A. Do levels of inflammation differ across races/ethnicities?
B. Do levels of inflammation differ by gender?
C. To what extent are any racial/ethnic differences in the distribution of CRP contingent on gender?
D. To what extent do these patterns persist when demographic characteristics, socioeconomic factors, health behaviors, and measures of health status are controlled?

Previous studies using this data set have demonstrated racial/ethnic and gender differences in inflammation (Herd, Karraker & Friedman, 2012); therefore, I expect to see the same patterns in this analytic sample. Additionally, given that CRP is a marker of cardiovascular

disease, I would expect the social patterning of this biomarker to follow that of cardiovascular diseases. Therefore, Aim 1 hypothesizes:

H1A. African Americans will have the highest levels of inflammation, while non-Hispanic whites will have the lowest.

H1B. Women will have higher levels of inflammation than men

H1C. Differences in inflammation by race/ethnicity will be contingent on gender, such that African American women will have the highest levels.

H1D. Racial, ethnic and gender differences will remain after controlling for other covariates.

Specific Aim 2: To determine the extent to which differential exposure to discrimination (everyday and lifetime discrimination) accounts for any differences in inflammation by race/ethnicity and/or gender. Research Questions 2A-2C:

A. Does exposure to discrimination vary across race/ethnicity and/or gender?

B. Is greater exposure to discrimination associated with higher levels of inflammation?

C. Does exposure to discrimination mediate racial/ethnic and gender differences in inflammation?

Past literature has shown that exposure to everyday discrimination is associated with markers of inflammation (Cooper, Mills, Bardwell, Ziegler, & Dimsdale, 2009; Elliot M Friedman et al., 2009; Lewis et al., 2010), including elevated CRP (Lewis et al., 2010). The literature has also shown that racial/ethnic minorities and women are more likely to report experiencing discrimination than non-Hispanic whites and men, respectively (Kessler, Mickelson, & Williams, 1999). Therefore it is reasonable to assume that exposure to

discrimination will mediate the differences in inflammation by race/ethnicity and/or gender. Accordingly, the hypotheses for this aim are:

H2A. Racial minorities and women will have more frequent exposure to everyday and lifetime discrimination.

H2B. Greater exposure to discrimination will be associated with higher levels of inflammation.

H2C. Differential exposure to discrimination partially mediates race and gender differences in inflammation.

Specific Aim 3: To determine the extent to which differential vulnerability to discrimination (everyday and lifetime discrimination) by race/ethnicity and/or gender is present for inflammation. Research Questions 3A-3C are:

A. To what extent does the impact of discrimination on inflammation differ by race/ethnicity or gender?

B. If the impact of discrimination is contingent upon race/ethnicity or gender, is discrimination associated with inflammation within each racial/ethnic group?

C. If the impact of discrimination is contingent upon race/ethnicity or gender, are there race or gender differences in inflammation across the full spectrum of exposure to discrimination?

A recent study (Cunningham et al., 2012) suggests that the association between discrimination and inflammation may differ by race and/or gender. Another study (Albert et al., 2008), however, did not find any differences by race in the association between discrimination and inflammation, among other biomarkers. Thus, it is still unclear whether racial and gender differences exist in the discrimination-inflammation relationship. It is important to note that the

expectation of group differences is not based on antiquated ideas about genetic differences between racial groups that influence physiological responses to stress. Rather, differences in the social context of exposure are thought to be the driving factor, such as the availability of resources to cope with discrimination. Additionally, the qualitative experience of discrimination across racial groups may contribute to differences in the strength of its association with health outcomes (Gee, Ryan, Laflamme, & Holt, 2006).

For example, Hispanic and Asian populations may have a proportionally larger immigrant population compared to African American or non-Hispanic white populations. Among immigrant population, experience of discrimination may be perceived and interpreted differently because they are unfamiliar with the manifestations of discrimination in the new country. Therefore, among racial groups with a greater proportion of immigrants, exposure to discrimination may be underreported which may subsequently lead to an underestimate of its association with health outcomes.

The historical patterns of oppression in the United States that have lead to residential segregation that more profoundly affects African Americans than other racial groups (Massey, 2004; Williams & Collins, 2001) may also contribute to differences in the impact of discrimination across groups. Living in a segregated community versus a non-segregated community may have different implications for the nature of discrimination individuals face and the resources available to deal with it. For these reasons, the experience and effect of discrimination on health may differ across racial groups. The hypotheses for Aim 3 are:

H3A. Racial minorities and women will be more vulnerable to the stress of discrimination.

H3B. Even if the effect of discrimination on inflammation is conditional, discrimination will still have an effect within each group.

H3C. Race and gender differences in inflammation will be greater at times of higher levels of exposure.

Specific Aim 4: To determine the extent to which social support and other coping resources (i.e., purpose in life and optimism) buffer the effect of discrimination on inflammation and do so differently by race/ethnicity or gender. Research Questions 4A-4C are[7]:

A. Does social support have a beneficial effect on CRP?

B. Does social support buffer the effect of discrimination on inflammation?

C. Does social support buffer the effect of discrimination equally among racial/ethnic groups and between genders, or is buffering more pronounced among racial minorities and women, relative to non-Hispanic whites and men, respectively?

Studies investigating the effect of social support on inflammation have shown mixed results (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Social support and other resources are traditionally thought to function as moderators of stress exposure that is protective against assaults to health (Cobb, 1976; Uchino). The focus on this role of social support has overlooked the role of this resource at an etiological factor (Thoits, 1982). A recent study by Prelow et al. (2006) attests to the need to look at the role of social support as a mediator. The authors found that the relationship between emotional social support and psychological outcomes is more in line was a support depletion model as opposed to a support mobilization or stress buffering model. In line with the additive stress-buffering hypothesis (Wheaton, 1985), these finding suggest that emotional social support may be depleted by constant exposure to discrimination. The hypotheses for Aim 4 are as follows:

H4A. Social support is inversely associated with inflammation (beneficial).

[7] The research questions and study aims apply to the other coping resources evaluated in the study (i.e., purpose in life and optimism) but, for simplicity, are explicitly stated for social support alone.

H4B. Social support buffers the effect of discrimination on inflammation, such that higher levels of support mitigate the effect of discrimination on inflammation.

H4C. Social support will equally buffer the effect of discrimination on inflammation among races/ethnicities and genders.

CHAPTER 3

METHODS

3.1 Introduction

This chapter describes the data for the dissertation, which come from the 2006 and 2008 Health and Retirement Study (HRS). A brief overview of the history of the HRS is given, followed by a description of its multiple cohorts. The sampling and data collection procedures are described next and then the derivation of the analytic sample is explained. The chapter closes by outlining the measurement and operationalization of the constructs assessed in the study and the statistical procedures used to address the specific aims and corresponding research questions of this dissertation.

3.2 The Health and Retirement Study (HRS)

The data for this dissertation are from the Health and Retirement Study (HRS).[8] The HRS was preceded by the Retirement History Survey (RHS; 1969-1979), which was a national survey of the labor force and retirement experiences of adults aged 58 to 63 years old in the United States. Although the RHS initially was useful for investigating the labor force decisions and economic trajectories of older workers, by the 1980's its utility had diminished due in part to a shift in the demographics of older workers: more women were in the workforce, the number of households with two wage earning adults was increasing, and a greater number of African Americans and Hispanics were participating in the labor market than in the past (Juster & Suzman, 1995). In addition to the demographic changes of the population, the RHS suffered

[8] Unless otherwise noted, the following description of the HRS and the data set relies heavily on information provided by (Juster & Suzman, 1995), the HRS website (http://hrsonline.isr.umich.edu/) and the HRS data documentation.

from design limitations such as the omission of measures of physical and mental health, and inadequate measures of wealth and retirement finances.

In 1988, at the recommendation of a multidisciplinary advisory panel and with fiscal support from the Social Security Administration, the National Institute of Aging (NIA) made the decision to implement the HRS. The goal of the HRS was to address the shortcomings of the previous study and to further our understanding of the physical, social, economic, and personal circumstances that influence and result from retirement among older populations. The target population of the study was non-institutionalized adults age 51 years or older living in households within the contiguous United States. This specific age range was selected for the HRS to effectively capture the pre-retirement and retirement experiences of older Americans. The Institute of Social Research (ISR) at the University of Michigan, Ann Arbor spearheaded this research endeavor and continues to do so.

Since its inception in 1992, six cohorts of eligible individuals have been recruited for the HRS. The original HRS cohort (HRS1) included individuals born between 1931-1941. Subsequent cohorts that were added to HRS1 include: Assets and Health Dynamics of the Oldest Old (AHEAD; birth years: 1890-1923), Children of the Depression Era (CODA; birth years: 1924-1930), War Babies (WB; birth years: 1942-1947), Early Baby Boomers (EBB; birth years: 1948-1953), and Middle Baby Boomers (MBB; birth years 1954-1959). Persons in the AHEAD cohort originally were participants of a companion study of the same name, which started in 1993. The aim of the AHEAD study was to understand the post-retirement and end-of-life experiences of the oldest old (i.e., persons aged 70 or older). Data collection for HRS1 and AHEAD alternated annually from 1992 until 1996 after which, in 1998, data collection for the two studies was merged. The WB and CODA cohorts were added in 1998. The fifth cohort, the

EBB, was added in 2004 while the sixth and most recent cohort, the MBB, was added in 2010. (See Figure 3.1)

This dissertation analyzes data from a combined sample of the first five cohorts: HRS1, AHEAD, WB, CODA, and EBB cohorts, as constituted in 2006 and 2008. The MBB cohort is necessarily excluded because it was recruited after 2008. The sampling procedures for the HRS cohorts are described in the following section.

3.3 Sampling Procedures (Core HRS Sample)

Cohort members were selected using a multi-stage, area probability sampling design (Heeringa & Connor, 1995). Four distinct stages of selection occurred in line with the University of Michigan's Survey Research Center's (SRC) National Sample design and sampling frame. The primary sampling units (PSUs) are Metropolitan Statistical Areas (MSA) and non-MSA counties where the probability of selection into the sample is proportionate to its size, which is the total number of occupied housing units in the PSU as measured by the most recent Census. For the HRS, the Congressional Appropriations Committee gave special attention to geographic areas with high densities and numbers of older Americans. Therefore, additional PSUs from the state of Florida were added to the sampling frame such that Florida residents are oversampled at a rate of 2:1 compared to the national probability sample.

The second-stage sampling units (SSU) are area segments (i.e., census blocks or groups of census blocks) within the MSA and non-MSA counties. Again, the probability of selecting each SSU was proportional to the number of housing units in the area. Additionally, area segments with at least a ten percent African American population or a ten percent Hispanic population were oversampled to improve the precision of survey estimates for these

populations.[9] African Americans and Hispanics were oversampled at a rate of 1.86:1 and 1.72:1, respectively.

During the third stage of sampling, all housing units—which includes houses, apartments, groups of rooms, or a single room occupied as separate living quarters—physically located within selected area segments were enumerated and a subset of units was systematically selected for the study. Prisons, jails, nursing homes, and other institutional facilities were ineligible for the study and excluded from the selection process.

The fourth and final stage of sampling involved the selection of an eligible "household financial unit" within each housing unit; a household financial unit was operationalized as an age-eligible household member (i.e., an individual aged 51 years or older) capable of responding to the interview and knowledgeable about household assets, debts, and retirement finances. The respondent was selected from a list of all age-eligible household members. In households with only one age-eligible person or households with two age-eligible persons who were married to each other (or in an equivalent relationship), the single person or the married couple was designated the household financial unit, respectively, and selected for interview. In households with more than one age-eligible person—persons who were not married to each other—one respondent was selected using procedures described by Kish (Heeringa & Connor, 1995). In partnerships where only one member of the married couple was an age-eligible household member, the spouse was interviewed as a secondary respondent but was not included in the probability sample. Spouses who were too young for a particular HRS cohort (i.e., under the age of 51 years) could enter the study as a member of a subsequent cohort if they became of age (i.e., age 51 or older) by the time data collection began for the subsequent cohort.

[9] To achieve the desired representation of Hispanics, additional PSUs in the West and Southwest of the U.S. were also added.

3.4 Data Collection Procedures

Data on all HRS participants are collected every two years. Face-to-face interviews are conducted at baseline with follow-up interviews completed by phone. A follow-up personal interview is conducted if there is no telephone in the household, or if a respondent has a health condition that would make difficult a phone interview of more than one hour long. Proxy interviews are completed with another household member if the selected respondent is unable or unwilling to complete the interview but agrees to someone else answering questions on his or her behalf. The proxy respondent is expected to be familiar with the financial, health and family situation of the primary respondent.

The core[10] HRS interview collects data on demographic characteristics; housing and family structure; employment, income, assets, and insurance; health care, health status, cognition, and disability; and life expectations. Beginning in 2006, the core interview was supplemented with an enhanced face-to-face interview (EFTF) that was administered to a random half-sample of HRS households; the remaining half-sample completed the EFTF interview in 2008. Data collection for each half-sample has been scheduled to occur every four years until 2016; data collection for the 2006 half-sample last occurred in 2010 and will be repeated in 2014, and data collection for the 2008 half-sample last occurred in 2012 and will be repeated in 2016. In the end, each half-sample will have data from the EFTF for three time points.

The EFTF consists of three study components: (1) a self-administered psychosocial questionnaire (a.k.a., the "leave-behind" questionnaire); (2) the collection of saliva for DNA extraction and blood samples for measuring select biomarkers; and (3) an assessment of physical performance (e.g., lung function, hand grip strength, balance tests, and a timed walk) and

[10] By "core" HRS interview I am referring to the baseline face-to-face interview and the follow-up phone interviews.

physical health, including measures of blood pressure, height, weight, and waist circumference. Interviewers sought consent for the collection of the blood samples and the assessment of the physical measures separate from consent for the core interview and the psychosocial questionnaire. Data from all three components of the EFTF are used to answer the research questions of this dissertation. The remainder section of this chapter describes the specific data collection procedures for each component of the EFTF, with an emphasis on how key biomarkers and physical measures were measured.

Collection of Data on Psychosocial Factors

After completing the in-person core interview, interviewers left a self-administered questionnaire with respondents, which they were asked to complete and mail to the HRS coordinating offices. The questionnaire covered six substantive areas: subjective well-being (e.g., depressive symptoms), lifestyle and stress (e.g., stressful life events), quality of social ties (e.g., positive and negative support), personality (e.g., extraversion), work-related beliefs (e.g., effort-reward balance), and self-rated beliefs and personal resources (e.g., mastery). Respondents were sent reminder notices if the questionnaire was not returned within a given time period. After the second notice, respondents were contacted by telephone for a follow-up interview.

Collection of Blood Samples[11]: C-reactive Protein

After the face-to-face interview, interviewers also collected blood samples using dried blood spots to measure five specific biomarkers: total cholesterol, high-density lipid (HDL) cholesterol, glycosylated hemoglobin (HbA1c), C-reactive protein (CRP), and Cystatin C. The collection of dried blood spots entails cleaning a person's finger with an alcohol swab and then

[11] Since data from saliva samples are not being used in the dissertation, this section only describes the procedures used to collect and process blood samples with a focus on the processing of CRP.

pricking the finger with a sterile lancet (i.e., a small double-edged surgical blade with a sharp point). Droplets of blood are collected on specially treated filter paper within a designated space on the paper. Interviewers attempted to collect six samples but were not always successful. The blood spots air-dried on the paper for 10-15 minutes before being placed in a foil envelope with a desiccant packet (i.e., a pouch containing a substance that absorbs moisture). Then the samples were packaged in mailing containers and shipped to laboratories for analysis.

Different laboratories were used to assay the different biomarkers collected. In 2006, all samples were first sent to facilities operated by BioSafe Laboratories in Chicago for sorting and freezing and then a portion of the blood samples were sent to the University of Vermont for assaying CRP—the biomarker examined in this study. In 2008 the samples were initially sent to BioSafe Laboratories in Chicago, IL, but Biosafe went bankrupt that year in the middle of processing the dried blood spots. The samples were retrieved by HRS staff and sent to the University of Michigan and from then on interviewers sent all samples to the University of Michigan for initial processing (i.e., sorting and freezing). Laboratories at the University of Vermont still completed the assays for CRP, while the assays of other biomarkers were completed at other laboratories in the U.S.

CRP was measured using a BNII nephelometer (Siemens, Inc., Deerfield, IL), which is an instrument that measures the amount of light reflected from particles suspended in a liquid (Figure 3.2). Two blood spots from the filter paper (equivalent to 3 microliters (μL) of blood serum[12]) are hole-punched and mixed with 250 μL of a liquid solution containing antibodies for CRP; the final dilution value is 1:83.3. The solution is run through a nephelometer that shines light through the solution. Rather than shining straight through the vial, the light is scattered by CRP molecules in the solution that are covered with antibodies. This reflection or scattering of

[12] This is the liquid portion of blood that excludes red blood cells *and* clotting factors.

light is proportional to the concentration of CRP. When using blood serum as a control, the range of detection for these immunoassays is 0.63-40 μg/mL and the between-subject coefficient of variation—a measure of the interassay precision of an immunoassay—ranged from 4.36% to 7.96% in 2006 and from 3.82% to 5.84% in 2008. Coefficients of variation less than 15% for an interassay are considered acceptable.

Approximately half of the blood samples had CRP values in a non-detectible range. This means the CRP concentrations were too low for accurate and precise detection by the nephelometer. These samples were assayed again using a sandwich enzyme-linked immunosorbent assay (ELISA; Qantikine Human CRP Immunoassay; R&D Systems #DCRP00, Minneapolis, MN), which is a more sensitive assay (Figure 3.3). In this assay, a solution containing a mixture of blood plasma[13] and a buffering liquid is created. The solution is distributed on a laboratory cell plate where an antibody for CRP is attached to the bottom of each cell well. CRP molecules bind to the immobilized antibodies and any unbound CRP is washed away with a buffer solution. Then, another solution containing an enzyme-linked antibody for CRP is added to each well. The enzyme-linked antibody binds on top of CRP; any unbound enzyme is washed away and a solution containing the substrate for the enzyme (I.e., the molecule upon which the enzyme acts) is added and initiates a chemical reaction that changes the color of the solution in each cell well. The color change is stopped (by adding yet another solution) and measured using a spectrometer, which is an instrument that measures the wavelength of transmitted light. The wavelength of light transmitted is proportional to the concentration of CRP. The detection range for this immunoassay is 0.01-2.5 μg/mL and the interassay coefficient of variability ranged from 7.42% to 9.95% in 2006 and from 9.41% to 15.49% in 2008 using blood serum as a control.

[13] This is the liquid portion of blood that only excludes red blood cells.

NHANES-equivalent CRP Values. Relative to using venipuncture to collect blood samples—a procedure where blood is intravenously withdrawn using a needle—the use of dried blood spots in survey research is a more recent innovation. There are a limited number of dried blood spot assays and the resulting values have greater variability across assays and laboratories.

To facilitate comparison of CRP values measured using dried blood spot versus whole blood samples (i.e., samples collected using venipuncture), the raw CRP values from the HRS were transformed to align with pooled CRP values from the 2005-2006 and 2007-2008 National Health and Nutrition Examination Survey (NHANES) samples. After using sampling weights to adjust for the complex sampling design of the HRS and for any between laboratory differences in dried blood spot assays, CRP values were calculated for each percentile of the data's distribution for the study sample. CRP values for each percentile of the NHANES data were also calculated for the two NHANES samples. Investigators assumed that the CRP values from both studies had similar distributions, arguing that the population distributions should be the same if there are no differences in laboratory procedures. Then, they regressed the HRS CRP values on the NHANES CRP values to convert the HRS values into NHANES equivalent values. In this dissertation, the NHANES-equivalent CRP values are used, as recommended by HRS investigators, for comparability across studies using whole blood samples.

Collection of Physical Measures[14]: Waist Circumference

The HRS physical assessment included measures of lung function, grip strength, balance, walking speed, blood pressure, height, weight, and waist circumference. Before completing any of the measurements respondents were asked if they understood and felt safe completing each procedure. If they answered no, the measurement was not conducted. Consenting respondents

[14] Although other physical measures were collected during this assessment, only the procedures used to measure waist circumference will be described since it is the only one relevant to the study.

were also asked not to eat, drink, smoke, chew gum or brush their teeth during the assessment. Waist circumference was measured by horizontally wrapping an anthropometric measuring tape around the abdominal area of each respondent. The measuring tape was positioned at the level of the navel (i.e. "belly button") and respondents were asked to stand and remove any bulky clothing during the measurement.

3.5 Derivation of the Analytic Sample

As mentioned previously, a random half of the HRS sample was administered the biomarker and physical measures assessments and the psychosocial questionnaire in 2006 and the other half in 2008. The data sets for the two half-samples are combined in this analysis to increase the sample size and statistical power. Constructing the analytic sample in this manner assumes the levels of variables are the same across the two years of data collection. An indicator variable for the year of data collection is included in multivariate analyses to account for differences in inflammation between the two half-samples to test this assumption.

Derivation of the analytic sample involved identifying HRS respondents who were in the study in 2006 and/or 2008 and selected for the EFTF as shown in Figure 3.4. Ineligible respondents and respondents without a valid sampling weight for the biomarker assessment were excluded.[15] In 2012, the total HRS sample size consisted of 37,812 participants.[16] Only sample members belonging to the 2006 and/or the 2008 probability samples were included in the analytic sample for this dissertation. These exclusion criteria resulted in an initial sample size of 22,558 individuals. Among these individuals, 4,721 did not live in a household selected for the EFTF interview in either year (e.g., members of the Middle Baby Boomer (MBB) cohort whose data collection began in 2010). Another 2,929 individuals were dropped from the analytic

[15] A valid sampling weight was non-zero and not missing in the data set.
[16] The sample size is based on the RAND data set, version M that was downloaded in August 2013.

sample because they were not eligible for the EFTF. They were ineligible because they either were younger than 51 years old (n = 940), lived in a nursing home or other institutional facility at the time of data collection (n = 459), completed the interview via proxy (n = 824), and/or completed the core interview by phone (n = 810).[17] Of the remaining 14,937 eligible participants, 12,099 had a valid sampling weight specifically for the biomarker assessment; the other 2,838 were dropped. Individuals who did not self-identify as non-Hispanic white, African American or Hispanic were dropped from the analysis due to small cell sizes (n = 164). As a result, the final analytic sample consists of 11,935 individuals (Figure 3.4).

3.6 Sample Weights and Robust Standard Errors

To account for the complex sampling design of the HRS, household and respondent-level sampling weights were constructed by the HRS. Weights specific to the biomarker assessment are used in the dissertation and respondents are assigned weights that correspond to the year in which they participated in the assessment (i.e., either 2006 biomarker weights or 2008 biomarker weights). Each respondent has only one weight, the 2006 or 2008 weight, but because most respondents were present at both times, applying these weights doubles the estimate of the population size. Therefore the weights were divided in half to correspond to the correct population size. The weights account for differences in the probability of: (1) selection into the core HRS sample, (2) non-response to the baseline HRS survey, (3) geographic differences in response rates, (4) attrition and mortality, and (5) non-response to the biomarker assessment. The weights were also post-stratified by race, gender and age to align with the demographic characteristics of the United States as captured by the American Community Survey that corresponds with the year of data collection for each half-sample.

[17] The reasons for ineligibility are not mutually exclusive

After applying the sample weights, findings from the analytic sample are considered nationally representative of the non-institutionalized, U.S. population born in 1953 or earlier. Extrapolation of the dissertation findings beyond the stated age cohort of persons aged 52 years or older as of 2006 is not recommended. Additionally, because only individuals living within housing units were selected for the HRS, the findings of this dissertation are not generalizable to persons living in institutional facilities or persons not living in housing units within the contiguous United States (e.g., homeless individuals, residents of Hawaii and Alaska).

3.7 Study Measures & Operationalization

As a general overview, the focal dependent variable is systemic inflammation, which is operationalized as concentrations of C-reactive protein (CRP). Because the underlying goal of this dissertation is to explain racial/ethnic and gender disparities in inflammation, these two variables are the focal independent variables. Gender is considered a qualitative moderator of the race-effect on inflammation and, accordingly, race/ethnicity is considered a qualitative moderator of the gender-effect on inflammation; these suppositions will be tested. Exposure to discrimination is evaluated as a potential mediator of race and gender differences in inflammation, and emotional social support is evaluated as a quantitative moderator of the discrimination-inflammation relationship and as a secondary mediator. The mediating and moderating effects of a sense of purpose in life and of optimism are also evaluated, to determine the specificity of coping resources as mediators and moderators of the effect of discrimination. Health behaviors (i.e., smoking, alcohol consumption, vigorous and moderate physical activity), depressive symptoms and waist circumference are alternative predictors of inflammation; the latter two measures—depressive symptoms and waist circumference—are also including in mediation analyses as secondary mediators of the effect of discrimination on inflammation. All

multivariate analyses control for demographic characteristics (i.e., age, marital status) and socioeconomic factors (i.e., education, household income, employment status). Additionally, all analyses include an indicator variable for year of data collection.

Focal Dependent Variable: Inflammation

As discussed previously, systemic inflammation refers to the concentration of cytokines and chemokines (i.e., inflammatory proteins) released by the immune system and circulating the body by way of the vascular system (i.e. the blood vessels). C-reactive protein (CRP) is an acute-phase protein released from the liver in response to increasing levels of certain cytokines. It therefore functions as a general marker of systemic inflammation. CRP was measured from blood samples using a BNII nephelometer (Siemens, Inc., Deerfield, IL). It is a continuous variable measured in units of micrograms per milliliter (μg/mL). Raw CRP values (i.e., untransformed values) are used for descriptive purposes; log-transformed values are used for multivariate analyses because this transformation adjusts for its positively skewed distribution[18].

Figures 3.5 and 3.6 show the distribution of the raw and log-transformed CRP values, respectively. Levels of systemic inflammation ranged from 0.02 μg/mL to 280 μg/mL with approximately 10% of the sample presenting with values above 10 μg/mL. The extremely high CRP levels (i.e., values above 10 μg/mL) may be indications of current infection or illness (Ridker, 2003; Ridker, Hennekens, Buring, & Rifai, 2000). Figure 3.5 shows the frequency distribution of CRP in 1-μg/mL intervals. As stated previously, the graph is positively skewed and resembles exponential decay. Figure 3.6 shows the frequency distribution of CRP after a

[18] Sensitivity analyses were performed to determine the extent to which the findings of the dissertation differ when the variable for CRP is censored at 10 μg/mL (i.e., cut point used to indicate that inflammation is being driven by infection or disease), and when the analytic sample only includes individuals with CRP values less than or equal to 10 μg/mL. Censoring the variable did not affect the substantive findings; limiting the sample to individuals with CRP values less than or equal to 10 μg/mL, however, did change some of the substantive findings (data not shown). Therefore, special attention should be given to the treatment of highly elevated CRP values.

log-transformation. Again, in line with early statements, the log-transformation resulted in a distribution more closely resembling normality. Clinically, the expected concentration of CRP in healthy individuals is less than or equal to 3 μg/mL or 1.099 μg/mL on the log-transformed scale (Ridker, 2003).

Focal Independent Variables: Race/Ethnicity and Gender

The principal motivation of this dissertation is furthering our understanding of racial/ethnic disparities in health by identifying and elaborating differences in inflammation. Therefore, the focal independent variable of the dissertation is self-reported race/ethnicity. During the core interview HRS respondents were asked: "What race do you consider yourself to be: White, Black or African American, American Indian, Alaska Native, Asian, Native Hawaiian, Pacific Islander, or something else?" Multiple racial groups could be selected; if a respondent selected more than one race, a follow-up question asked which racial group the respondent "primarily" considered him or herself. The primary race mentioned is used in this study to measure race. Additionally, respondents were asked if they considered themselves Hispanic or Latino. Based on these two questions, two dummy variables were created comparing non-Hispanic whites, the omitted reference group, to African Americans and to Hispanics. Individuals from other racial or ethnic groups were excluded from the study because they collectively represent less than 3% of the entire HRS sample (e.g., Alaska Natives).

Racial disparities in health may be patterned by gender. Therefore, in addition to evaluating race-based disparities in biomarkers, gender-based disparities and disparities by race and gender are examined. Gender was evaluated during the core interview by the HRS interviewer; the evaluation was based on physical appearance. Any discrepancies in reported gender across waves of data collection were resolved be looking at values of gender-specific

health variables, such as variables indicating whether or not a respondent had a prostate exam or mammogram. Gender is a binary variable where males serve as the omitted reference group.

Mediators: Everyday and Lifetime Discrimination

The mediating effects of everyday discrimination and major lifetime discrimination are evaluated in conjunction with one another. Exposure to everyday discrimination is measured using an instrument from the Detroit Area Study (Williams et al., 1997). This stressor manifests as chronic slights and disparaging treatment on a day-to-day basis that often is ambiguous to its meaning. Only five of the original nine items were included in the 2006 and 2008 waves of the HRS. Respondents were asked the following question: "In your day-to-day life how often have any of the following things happened to you?" This question was followed by a list of experiences including: being treated with less courtesy or respect than other people; receiving poorer service than others at restaurants or stores; people acting as if they think you are not smart; people acting as if they are afraid of you; and being threatened or harassed. The possible responses were 1 = almost every day, 2 = at least once a month, 3 = a few times a month, 4 = a few times a year, 5 = less than once a year, and 6 = never. All items were reverse-coded in the direction of increasing discrimination and rescaled to zero such that the responses ranged from 0 = never to 5 = almost every day. The items were then averaged over the number of items to produce a discrimination score ranging from zero to five. Cronbach's alpha for the scale is 0.80.

Major lifetime discrimination represent incidents that occur across the life course and many have the potential to significantly and negatively affect status attainment (e.g., socioeconomic advancement) (Williams et al., 1997; Kessler et al, 1999). Six of the eleven items from the original instrument were included in the 2006 and 2008 data collection. Respondents were given a list of events and asked to indicate if any of the events happened to

them at any point in their lives. The listed events included: being unfairly dismissed from a job; not being hired for a job for unfair reasons, being unfairly denied a promotion; being unfairly prevented from moving into a neighborhood because the landlord or realtor refuses to sell or rent you a house or apartment; being unfairly denied a bank loan; and, being unfairly stopped, searched, questioned, physically threatened or abused by the police. The possible responses were 1 = Yes and 5 = No, which were recoded to 1 = Yes and 0 = No. The items were summed to create a count of all major experiences of discrimination ranging from zero to six.

Moderators: Emotional Social Support, Purpose in Life and Optimism

Perceived emotional social support is a coping resource that manifests as an individual's perceptions or beliefs about being loved, cared for, understood, or valued by significant others, such as family members or friends (Thoits, 1995). In this dissertation it is conceptualized as a moderator of the discrimination-inflammation relationship and operationalized as a continuous variable. Three questions were used to assess social support from spouses, children, other family members, and friends. Respondents were asked the extent to which each source of support: understands the way you feel about things, can be relied on if there is a serious problem, and is available for you to "open up to" if you need to talk about your worries. Possible response options were 1 = a lot, 2 = some, 3 = a little, and 4 = not at all. The items were reverse coded such that 1 = not at all and 4 = a lot. All items from each of the sources of support were summed and averaged over the number of items for that source, and respondents who indicated that they did not have a spouse, children, other family members, or friends, received a zero value for the corresponding support score for that source. An overall support score was constructed by averaging over the source-specific support scales: spouse support, child support, family support,

and friend support. The final scale ranged from zero to four. The Cronbach's alpha for the support scale is 0.82

Purpose in life is a measure of eudaimonic well-being (striving to realize one's potential in life), as opposed to hedonic well-being (feeling good, content or satisfied with life) (Ryff & Singer, 2008). It refers to the belief that one's life is purposeful and meaningful, and it is a reflection of having life goals and aims, having a sense of direction, and realizing the meaning of present and past experiences (Ryff & Keyes, 1995). Seven items were used to assess this construct. Respondents were asked how strongly they agree with the following statements:

1. I enjoy making plans for the future and working to make them a reality.
2. My daily activities often seem trivial and unimportant to me.
3. I am an active person in carry out the plans I set for myself.
4. I don't have a good sense of what it is I'm trying to accomplish in life.
5. I sometimes feel as if I've done all there is to do in life.
6. I live life one day at a time and don't really think about the future.
7. I have a sense of direction and purpose in my life.

The possible response options are 1 = strongly disagree, 2 = somewhat disagree, 3 = slightly disagree, 4 = slightly agree, 5 = somewhat agree, and 6 = strongly agree. Items 2, 4, 5, and 6 were reverse-coded towards increasing levels of purpose and then all the items were rescaled to range from 0 = strongly disagree to 5 = strongly agree. The scale was constructed by averaging over the items such that scores ranged from zero to five. The Cronbach's alpha for this scale is 0.74.

The conceptualization of optimism is rooted in expectancy-value theories and refers to holding a positive expectation for the future (Carver, Scheier & Segerstrom, 2010). The

measurement of this construct is similar to that of purpose in life. Respondents were asked how strongly they agree with three statements that are indicative of having an optimistic outlook: "I'm always optimistic about my future", "In uncertain times, I usually expect the best", and "Overall, I expect more good things to happen to me than bad". The possible responses were 1 = strongly disagree, 2 = somewhat disagree, 3 = slightly disagree, 4 = slightly agree, 5 = somewhat agree, and 6 = strongly agree, and the scale was created by rescaling the items to zero and averaging over the items. The reliability of the scale (i.e., Cronbach's alpha) is 0.78.

Risk Factors for CRP: Health Behaviors and Health Status

Two questions are used to assess cigarette-smoking behavior. The first asks respondents whether they have ever smoked cigarettes and the second asks if they smoke cigarettes now. Both items are dichotomous (i.e., 0 = No and 1 = Yes) and were used to crease two dummy variables comparing current and former smokers to non-smokers (omitted reference group).

Alcohol consumption was assessed by asking respondents (1) whether they ever drink alcoholic beverages such as beer, wine or liquor, (2) how many days per week they have had alcoholic beverages in the past three months, and (3) how many drinks they usually have on the days that they drink. A single variable was constructed grouping respondents into three categories based on gender: non-drinker (0 drinks/day and 0 drinks/week for men and women), moderate or "low-risk" alcohol consumption (1-3 drinks/day *and* less than 8 drinks/week for women; 1-4 drinks/day *and* less than 15 drinks/week for men), and heavy or "high-risk" alcohol consumption (4 or more drinks/day *or* 8 or more drinks/week for women; 5 or more drinks/day *or* 15 or more drinks/week for men). This classification of alcohol consumption aligns with guidelines set forth by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). Two

dummy variables are used to compare individuals who consume a moderate or heavy amount of alcohol to non-drinkers (omitted reference group).

Physical activity was assessed with a series of question asking about the type and frequency of physical activity respondents engaged in on a daily basis. Separate questions were asked for light, moderate and vigorous physical activity; the current study assesses the effects of vigorous and moderate physical activity on inflammation. Examples of vigorous activities include running or jogging, and examples of moderate activities include brisk walking or dancing. For each type of physical activity, the frequency with which respondents engaged in these or similar activities was measured, where 1 = every day, 2 = more than once a week, 3 = once a week, 4 = one to three times a month, and 5 = hardly ever or never. These response options were reverse-coded and rescaled to zero such that 0 = hardly ever or never and 4 = every day. Respondents were then reclassified into three groups: (1) individuals who hardly ever or never engaged in vigorous/moderate physical activity, (2) individuals who engaged in vigorous/moderate physical activity once a week or 1-3 times a month, and (3) individuals who engaged in vigorous/moderate physical activity more than once a week including every day. There are two dummy variables for each type of physical activity, and for both constructs the omitted reference group is the one including individuals who hardly ever or never engage in that form of physical activity.

Waist circumference[19] is an anthropometric measure reflective of adiposity (i.e. the amount of fat stored in adipose tissue in the body); adipose tissue is a source of inflammatory

[19] Body mass index (BMI) is another anthropometric measure that is associated with inflammation and cardiovascular disease risk (Huxley et al., 2010). The current study only includes waist circumference because of its direct association with central adiposity and because the literature suggests that waist circumference and BMI separately predict inflammation. Supplemental analyses of the dissertation support the latter claim and show that waist circumference and BMI independently affect inflammation. Compared to models only including waist circumference, the substantive findings of models including waist circumference and BMI did not differ.

markers and is associated with increases in both CRP (Yudkin, Kumari, Humphries, & Mohamed-Ali, 2000; Yudkin, Stehouwer, Emeis, & Coppack, 1999; Berg, 2005). Waist circumference was measured by horizontally wrapping a measuring tape around the abdominal area of each respondent, at the level of the navel (i.e. "belly button"). The unit of measurement is inches.

Having depressive symptoms is a measure of psychological distress. It refers to the emotions and behaviors associated with an affective state of extreme sadness. The extent of these symptoms is assessed using a simplified version of the Center for Epidemiologic Studies – Depression (CES-D) Scale (Radloff, 1977), a validated and highly reliable scale that assesses depressive symptomology (i.e. depressed mood) as distinct from major depressive disorder. The original CES-D has 20 items. To simplify the study and decrease the amount of time needed to administer the scale, only eight items were included and the response options were changed from the original four-level rating scale to 1 = Yes and 5 = No (which I recoded to 1 = Yes and 0 = No). Accordingly, the question wording changed to assess only the presence of each depressive symptom as opposed to the extent or frequency of symptoms.

Respondents were asked to indicate if the following were true for them most of the time in the past week: felt depressed, everything was an effort, sleep was restless, happy, felt lonely, enjoyed life, felt sad, and could not get going. Two items were reverse coded in the direction of increasing depressive symptoms, specifically the items assessing whether the respondent was happy and enjoyed life. The items were summed to create a depressive symptoms score ranging from zero to eight. The Cronbach's alpha for the scale is 0.80.

Demographic Characteristics and Socioeconomic Factors

The demographic characteristics that were assessed include age and marital status. Age was calculated by subtracting the date of the HRS core interview from the respondent's self-reported birth date. For this analysis age is measured in years as a continuous variable. For current marital status, there are four categories: married or partnered, separated or divorced, widowed, and never married. This variable is coded into three dummy variables where married or partnered individuals serve as the omitted reference group.

The socioeconomic factors assessed include education, income and employment status. Education is measured in years as a continuous variable; it was assessed by asking respondents the highest grade of school or year of college they completed (up to 17 years). Household income is the combined total of: individual earnings, employer pension and annuities, income from social security, unemployment compensation and other government transfers, and income from other sources, such as alimony, insurance or inheritance. The values were summed across a respondent and his or her spouse; income from other household members was excluded from this sum. To address its positively skewed distribution, household income—measured in thousands of dollars—is log-transformed to improve its distribution. Respondents were asked if they were currently working, temporarily laid off, unemployed and looking for work, disabled and unable to work, retired, working as a homemaker, or something else. Individuals currently working full or part-time are grouped together, as were individuals who stated they were retired. All other employment statuses—unemployed, disabled and not in labor force—are collapsed into an "other" category due to small sample sizes. Two dummy variables were constructed where employed individuals serve as the omitted reference group for retired and "other" employment statuses.

3.8 Data Analysis Procedures

This section presents the analytic techniques used to prepare for the analysis of the research questions (e.g., treatment of missing data) and the statistical strategies used to address the aims and research questions of this dissertation. The strategies used to address the specific aims are guided by the elaboration model (Rosenberg, 1968), as put forth by Aneshensel (2013). The elaboration model outlines a systematic approach to inferential analysis when using observational data. Through the use of a "third-variable", the nature of the relationship between two variables is determined. This dissertation uses the exclusionary and inclusive strategies of this model to elaborate the relationship between racial group membership and inflammation.

Throughout the analyses, regression models were built in a stepwise manner to ascertain the incremental effect of adding new variables to the analysis; variables that are conceptually equivalent were simultaneously added in one step during this process. The contribution of each set of variables to explaining the dependent variable was assessed with an incremental F-test prior to examining individual coefficients. The contribution of the set of variables to explaining the association between race/ethnicity and inflammation was assessed as the change in the unstandardized regression coefficient. Although the primary emphasis of this dissertation is racial/ethnic and gender differences in inflammation, the effect of the other variables in the model is of substantive interest too. In other words, the study seeks to explain inflammation as well as racial/ethnic and gender differences in inflammation.

As a brief overview, in the main analysis (i.e., analyses that address the specific aims of the dissertation) I used regression to determine the distribution of inflammation across race/ethnicity and gender (Aim 1). Then, using a structural equation model (SEM), the mediating function of discrimination was assessed in line with the MacKinnon model when sequential mediation is present (Aim 2). Moderation of the discrimination-inflammation

relationship by race/ethnicity and gender (Aim 3) and by social support, purpose in life and optimism (Aim 4) was assessed with product interaction terms. In the creation of product interactions terms, to reduce multicollinearity and enhance the interpretation of conditional relationships, continuous variables were mean-deviated or "centered" before being used. All analyses are done using Stata® version 12.1. The complex sample design is taken into consideration by using the SVY commands, or survey procedures, offered in this statistical package. The SEM command is used for all regression analyses; this permits the use of full information maximum likelihood (FIML) estimation for missing data, and the estimate of simultaneous equations, which is necessary for the meditation (i.e., path) analysis because complex mediation is tested. Specifically, race/ethnicity is mediated by discrimination, which in turn is independently mediated by health status and coping resources. Sequential mediation of this type is best accomplished in SEM because the multiple dependent variables require multiple equations that should be estimated simultaneously.

Treatment of Missing Data

Of the 11,935 individuals in the CRP sample, only 9,534 had complete data on all variables of interest. A complete case analysis with a 20% reduction in sample size is considered a substantial loss of power. Therefore, to maximize the number of respondents included in the study missing values for all study variables were estimated using full information maximum likelihood (FIML) estimation. This method of estimation is an option in Stata when using the SEM command. FIML is considered to be superior to traditional missing data approaches (e.g., listwise deletion) because it produces unbiased parameter estimates and standard errors assuming the data has a multivariate normal distribution and missing values are either missing at random (i.e., MAR; missing values of the dependent variable may depend on other variables, but do not

depend on its own unobserved values), or missing completely at random (i.e., MCAR; missing values of the dependent variable do not depend on other variables or on its own unobserved values) (Enders, 2001; Enders & Bandalos, 2001).

Another advantage of FIML is that it uses all of the available data in the estimation process. In this process, a likelihood function is estimated for each individual in the data set. This function represents the relative probability (i.e., likelihood) of obtaining a particular parameter value given the observed data.[20] Because the likelihoods tend to be very small, the natural log of the likelihood is taken. This estimation procedure was developed for structural equation modeling but it is general enough for the estimation of means, covariance matrices and multiple regression.

Preliminary Analyses

Univariate and bivariate analyses were conducted to obtain descriptive statistics (e.g., means, standard deviations, proportions) and bivariate associations (e.g., correlations, chi-square statistics) among variables the study variables. The type of statistic calculated depended on the level of measurement of the variable. Scatterplots with fitted predictor lines (for continuous predictors) and connected line graphs (for plotting group means of categorical variables) are used to visual represent the relationship between predictor variables and the dependent variable. If the resulting graph for the relationship between a continuous predictor and any of the dependent variables appears to be curvilinear, a quadratic term was created and tested in regression models.

In line with the steps of the elaboration model (Aneshensel, 2013), the parameter estimates (e.g., regression coefficients) of three-variable regression models are compared to the estimate of the focal relationship. The interpretation of a change in the size of the regression

[20] It is important to note that this procedure does not involve imputation of missing values; only the estimation of parameters.

coefficient depends upon the role of the third variable as stated in the conceptual and analytical models. For example, if a statistically significant bivariate association between race/ethnicity and CRP is no longer significant when a control variable is added, then the focal relationship is considered a spurious one that is confounded by the control variable. If the third variable added is an alternative independent variable, then the loss of a significant focal relationship suggests that the effect of the race/ethnicity on CRP is not separate and district from the effect of the third variable; in other words, there is redundancy in the model because both variables, unlike spuriousness, are associated with CRP and each other, although no causal association is attributed to the latter. If, instead, the third variable is an intervening variable (i.e., a mediator), the decrease in the estimate of the focal relationship suggests that a causal relationship exists between race/ethnicity, the mediator, and CRP that explains the focal relationship.

Similarly, a reduced coefficient that remains statistically significant points to different conclusions: spuriousness with a control variable, redundancy with a rival independent variable, and partial mediation with an intervening variable. At times, the magnitude of the focal independent variable remains the same when a third-variable is added, which indicates that the third-variable does not explain the focal relationship. At other times the magnitude of the regression coefficient for the focal relationship increases, which indicates suppression.

When adding a third variable operationalized as multiple dummy variables, a Wald test was used to determine if the addition of the set of variables to the bivariate model produces a significant increase in R^2, which is equivalent to testing the null hypothesis that the added coefficients all equal zero. If the null hypothesis was rejected (at the 0.05 significance level), then the statistical significance of the singular dummy variables was examined. The same procedure was used to test a set of multiple interaction terms.

Analysis of Aim 1: Distribution of CRP by Race/Ethnicity and Gender

The first aim of this dissertation is to determine the extent to which levels of inflammation differ by race/ethnicity and/or gender. I hypothesize that racial and ethnic minorities—specifically, African Americans and Hispanics—will have higher levels of inflammation than non-Hispanic whites and that the distribution of both biomarkers will depend on gender with women having higher levels than men. I also hypothesize that these group differences in the biomarker will persist after controlling for demographic characteristics, measures of socioeconomic status, health behaviors and measures of health status known to influence levels of inflammatory factors such as CRP.

Regression equations pertaining to Aim 1 are presented in Table 3.1. To test Hypothesis 1A (H1A), I regressed CRP on race/ethnicity, which is operationalized by two dummy variables where non-Hispanic white is the omitted reference group (Model 1A). Hypothesis 1B (H1B) was tested by regressing CRP on gender (Model 1B), and for Hypothesis 1C (H1C) I regress CRP on race/ethnicity, gender and the interaction of race/ethnicity and gender (Model 1C). The interaction is operationalized by two product interaction terms by multiplying the dummy variable for African American race by the gender variable, and multiplying the dummy variable for Hispanics by the gender variable.

Nested regression models were used to test Hypothesis 1D (H1D). Conceptually similar variables were added simultaneously to the three-variable model that includes race/ethnicity and gender. First, demographic characteristics and socioeconomic factors were added (Model 1D), followed by health behaviors (Model 1E) and health status variables (Model 1F). Two additional models were estimated that include all study variables except for race/ethnicity (Model 1G) or gender (Model 1H). Respectively, these models are included to further assess the magnitude of

the race-effect and the gender-effect. From Model 1D, however, we see the net difference in inflammation by race/ethnicity and by gender after accounting for other control and predictor variables.

Analysis of Aim 2: Test of the Differential Exposure to Stress Hypothesis

The second aim of this dissertation is to determine whether differential exposure to discrimination explains differences by race/ethnicity and by gender in inflammation. I hypothesize that African Americans and Hispanics report greater exposure to everyday and major lifetime discrimination than non-Hispanic whites (H2A) and that exposure to discrimination is positively associated with inflammation (H2B). I also hypothesize that racial/ethnic and gender differences in exposure to discrimination explains—at least in part—parallel differences in inflammation (H2C). Table 3.2 presents the regression equations.

To investigate H2A, I separately regressed discrimination on race/ethnicity (Model 2A) and gender (Model 2B). Then I regressed discrimination on race/ethnicity and gender (Model 2C) and on the interaction between race/ethnicity and gender (Model 2D). This series of analyses was conducted separately for everyday discrimination and lifetime discrimination. For H2B, I regressed inflammation on everyday discrimination (Model 2E) and major lifetime discrimination (Model 2F). Then a model including both stressors was estimated (Model 2G), followed by a model including the interaction between everyday and lifetime discrimination (Model 2H). From these last two models we can determine whether the two forms of discrimination have a conditional relationship.

Similar to the analysis conducted for Aim 1, the analysis of Aim 2 proceeded with a nested regression of inflammation on all of the study variables. This analysis, however, differed from that conducted for Aim 1 because it includes everyday and lifetime discrimination, as well

as the coping resources—social support, purpose in life and optimism. Variables were added sequentially beginning with the three variable model of inflammation regressed on race/ethnicity and gender (Model 2I). Then the other variables were added, as conceptually similar sets of variables, in the following order:

1) Everyday and lifetime discrimination (Model 2J)
2) Demographic characteristics and socioeconomic factors (Model 2K)
3) Health behaviors (Model 2L)
4) Health status (Model 2M)
5) Coping resources (Model 2N)

This nested regression is not a formal test of mediation and only provides an indication of whether mediation may be present. Therefore, structural equation modeling was used to conduct a path analysis and test Hypothesis 2C that racial/ethnic and gender differences in inflammation are at least partially explained by differences in exposure to discrimination.

Structural Equation Modeling (SEM). Structural equation modeling (SEM) is a collection of statistical procedures ranging from linear regression to confirmatory factor analysis and simultaneous equations (Weston & Gore, 2006). It is used to determine and test the validity of causal models. This theory-driven process is mathematically based on the general linear model and empirically tests whether a proposed relationship or causal model produces a population covariance matrix that is consistent with the covariance matrix of a sample under study. With SEM, the relationships between one or more independent variables and one or more dependent variables can be examined with a system of equations; competing theories and causal pathways can be tested, and group differences in the relationships among variables can be determined. In addition to observed variables (i.e., manifest or measured variables), SEM

incorporates latent variables (i.e., constructs or factors that are unobserved and not measured) by using multiple measures or indicators of the latent construct to estimate and adjust for error in its measurement. A model relating measured variables—that is, observed indicator variables—to a latent variable is termed a *measurement model*; a model relating latent variables to other latent variables is termed a *structural model*.

In this dissertation, SEM is used to carry out a path analysis. A path analysis is a type of SEM that only includes observed variables. The ability of SEM to simultaneously evaluate a system of equations is advantageous for a model including multiple mediators, especially when the mediators are hypothesized to operate in a specific temporal order. This analysis includes separate equations predicting the dependent variable—systemic inflammation—and each intervening variable or mediator. There are two focal intervening variables (everyday discrimination and lifetime discrimination) and two rival intervening variables (education and household income). Each of these mediators is hypothesized to exert an effect on inflammation through three secondary mediators: waist circumference, depressive symptoms and social support. Purpose in life and optimism are also conceptualized as secondary mediators, but this analysis is more exploratory than confirmatory. A diagram representing the analytic SEM model being tested is presented in Figure 3.7.

Calculation of Indirect Effects. In line with the work of MacKinnon (2008), indirect effects are calculated using the product of coefficients method. Briefly, MacKinnon uses an ***a***, ***b***, ***c***, ***c'*** notation to identify the paths involved in a mediation analysis (See Figure 3.8). Using race differences in inflammation as an example, path ***c in the upper panel*** of Figure 3.8 represents the total effect of the race/ethnicity on inflammation net of the other study variables, except discrimination (i.e., the mediators). When discrimination is added to the model, paths ***a*** and ***b***

are created and respectively represent the effect of race/ethnicity on discrimination (i.e., racial/ethnic differences in discrimination) and the effect of discrimination on inflammation controlling for the focal independent variable and other variables in the model. Path ***c' in the lower panel*** of Figure 3.8 is the remaining direct effect of race/ethnicity on inflammation after accounting for its indirect effect through discrimination. The value of the net direct effect is obtained by regressing inflammation on race/ethnicity, discrimination and all of the other study variables. The magnitude of the indirect effect is calculated by either multiplying path ***a*** and path ***b*** (the product of coefficients method) or subtracting path ***c'*** from ***c***, where ***ab*** = ***c-c'*** (the difference of coefficients method).

Analysis of Aim 3: Test of the Differential Vulnerability to Stress Hypothesis

Aim 3 assesses whether the effect of discrimination on inflammation—net of the other variables in the analytic model—varies by race/ethnicity or gender. Using the model from Aim 2, Aim 3 is addressed by examining interactions between discrimination and race/ethnicity and between discrimination and gender. Table 3.3 presents the regression equations for this moderation analysis. Model 3A is the main effects model, which is estimated first, and Model 3B to 3E are the conditional models. Model 3B tests the interaction between everyday discrimination and race/ethnicity; Model 3C tests the interaction between lifetime discrimination and race/ethnicity; Model 3D tests the interaction between everyday discrimination and gender; and Model 3E tests the interaction between lifetime discrimination and gender.

All interactions were created by centering the discrimination variable at its mean—in order to reduce multicollinearity and enhance interpretation—and multiplying discrimination by each of the dummy variables for race/ethnicity and for gender. For the interaction between race/ethnicity and discrimination, two interaction terms for each form of discrimination were

created because race/ethnicity is a categorical variable that is operationalized by two dummy-coded variables: one comparing African Americans to everyone else, and the other comparing Hispanics to everyone else. Each of these dummy variables must be multiplied by discrimination and included in the regression model to fully assess effect modification of discrimination by race/ethnicity. An incremental F-test was used to compare the fit of the model including the product interaction terms to a model excluding them (i.e. the main effects model). The null hypothesis for this test is that all the coefficients for the interactions equal zero. Failure to reject the null hypothesis suggests that adding the interactions to the regression analysis does *not* improve the fit of the model. In this instance, the more parsimonious main effects model is preferred.

Positive and statistically significant product interaction terms provide support for Hypothesis 3A, which states that differences in vulnerability to discrimination exist, such that racial/ethnic minorities and women are more vulnerable to the stressor than non-Hispanic whites and men, respectively. Based on this finding, Hypothesis 3B is assessed with a simple slope test to determine if discrimination still has an effect within each racial/ethnic group or gender. The magnitude of the difference in inflammation among racial/ethnic groups and between genders are then calculated at minimum, maximum and average levels of discrimination to determine if significant differences persist across the full spectrum of discrimination (Hypothesis 3C)

Analysis of Aim 4: Stress Buffering of the Effect of Discrimination by Social Support

The statistical procedures for assessing stress buffering by social support, and the other coping resources, is similar to those described in the previous section for Aim 3: (1) the main effects model is estimated; (2) product interaction terms with mean-centered variables are created and entered into the model; (3) a t-test (for interactions operationalized by one product

interaction term) or a Wald test (for interactions operationalized by more than one product interaction term) is used to assess improvement in model fit due to the inclusion of the interaction; and (4) simple slopes within groups and the magnitude of the difference in inflammation for different values of discrimination are calculated for regression models with significant interactions. The regression equations for this analysis are presented in Table 3.4 and are organized in manner similar to Table 3.3. Social support is used as an example in Table 3.4.

To determine if social support is inversely associated with inflammation (Hypothesis 4A), for example, inflammation is regressed on support and all of the other study variables (Model 4A). Even in the absence of an independent effect of a predictor on an outcome variable, effect modification may be present. Therefore, Hypothesis 4B is tested to determine if the effect of discrimination on inflammation is buffered by social support by adding the product interaction term (Model 4B). Hypothesis 4C is tested only in the presence of stress buffering by social support. This analysis ascertains whether support equally buffers the effect of discrimination on inflammation across races/ethnicities and genders. A three-way interaction between race/ethnicity, support and discrimination tests equal stress buffering across races/ethnicities; a three-way interaction between gender, support and discrimination tests equal stress buffering across genders. To fully test these higher order interactions, all of the corresponding lower-order interactions must also be included in the regression (Model 4D-4G). Moderation of racial/ethnic and gender differences in inflammation by social support was also assessed. Product interaction terms were created between race/ethnicity and support (Model 4H) and between gender and support (Model 4I). A significant interaction suggests that the race or gender difference in inflammation depends on the levels of social support.

3.9 Data Permissions & Human Subjects Approval

The University of Michigan's Institutional Review Board (IRB) approved all data collection instruments and recruitment procedures used in the HRS. Prior to collecting any data, trained investigators reviewed the rationale for the study and the eligibility criteria with study participants. Eligible participants completed standard informed consent procedures by providing their written or oral consent to participate in the HRS core interview, biomarker assessment, and physical performance assessment. Consent for the psychosocial assessment was implied if the questionnaire was returned to the study coordinating center.

I originally proposed to use a data source other than the HRS for my dissertation. The UCLA South Institutional Review Board reviewed and approved my original proposal on February 22, 2013 under an Expedited status (IRB#: 13-000276). An amendment to use the HRS data instead was submitted and approved in June 2013, and IRB approval for the continuation of the study was granted on January 31, 2014.

HRS data for public use exclude personal identifying information and are available to registered users on the study's website (http://hrsonline.isr.umich.edu/). Access to sensitive data, such as the biomarker and physical measures data, is available to registered users who submit a signed data use agreement to the HRS coordinating center. Therefore, prior to conducting any analyses, I registered as an HRS data user and submitted a signed data use agreement to gain access to the 2006 and 2008 biomarker and physical measures data. The agreement was approved on April 30, 2013.

CHAPTER 4

RESULTS: PRELIMINARY ANALYSES

4.1 Introduction

Before investigating the specific aims of the dissertation, preliminary analyses were conducted to describe the analytic sample and population and to examine bivariate associations among study variables. The findings from these analyses are presented in this initial chapter of study results.

4.2 Sample and Population Characteristics

The demographic, socioeconomic and health-related characteristics of the sample are summarized in Table 4.1. The table presents sample characteristics (i.e., unweighted values) and population parameter estimates (i.e., weighted values)[21]. There are 11,935 individuals in the sample. The average age of the sample is 69 years but there are individuals as young as 52 years and as old as 101 years. The majority of respondents are married, but 20% are widowed and 11% are separated or divorced from their spouse or partner. Only 3% of individuals in the sample say they have never been married.

With regard to socioeconomic factors, the sample, on average, completed at least 12 years of education but this value ranges from zero years to greater than 17 years. The range in household income is even more dramatic. At the lower end, some individuals report annual incomes of zero dollars; at the upper end some individuals report incomes in excess of $10 million. However, the average household income of the sample is slightly less than $63,000. In

[21] The table only includes information on control variables; key variables of the study (e.g., the independent variables) are summarized in Table 4.2.

terms of employment, about 65% of the sample is retired, while nearly 25% are working either full-time or part-time.

For the most part, members of this sample engage in healthy behaviors and practices that affect systemic inflammation. Only 13% of the sample reports that they are current smokers. The remaining 87% of individuals are non-smokers (43%) or former smokers (44%). Nearly half of the sample report never consuming alcoholic beverages, while 45% are moderate drinkers and slightly more than 6% are heavy drinkers. Engaging in vigorous physical activity is more challenging at older ages; therefore, it is no surprise that two-thirds of the sample never engage in vigorous exercises such as jogging, swimming or cycling. That said, at least one in five older adults do engage in these activities more than once a week. An even larger proportion—nearly 55%—engages in moderate forms of activity (e.g. walking, dancing) at least two times a week.

Turning to health status, the average waist circumference of the sample is 40 inches and ranges from 20.5 inches to 74.25 inches. To put this value in context, a waist circumference less that 40 inches for men and less than 35 inches for women, assuming normal weight or body mass index (BMI), is ideal for low disease risk (Rogers et al., 2011). Thus, the average member of this study sample is on the cusp of the distinction between low and high disease risk. Depressive symptoms are relatively low in the sample with most individuals reporting one to two symptoms.

Finally, a relatively equal proportion of participants are from the two years of data collection under study, which is expected given the sampling design for the enhanced face-to-face (EFTF) interview of the HRS.

Overall, the population parameter estimates of the demographic characteristics, socioeconomic factors, health behaviors, and health status, are remarkably similar to the sample statistics. With the exception of age, income and employment status, the sample statistics and

the parameter estimates only differ by less than five percentage points across corresponding variables. The substantial change in age with the application of sample weights arises because recent (i.e., younger) cohorts have had lower selection probabilities than some earlier cohorts. Accordingly, the sampling weights for these cohorts in proportionately larger and, when applied to the sample, manifest as an average age in the population that is lower than that of the sample.

The average household income in the population is near $74,000, a large change from the sample value of approximately $63,000. Similar to the difference in average age between the sample and the population, this increase in income may be due to the larger weights for younger cohorts who are more likely to be employed than early cohorts insofar as income is related to employment. This explanation is consistent with employment status values where we see a larger percentage of the population is estimated to be employed (35%) relative to the sample, and a smaller percentage is estimate to be retired (54%). The proportion of individuals engaging in other forms of employment, 11%, is the same for the sample and the population.

Table 4.2 presents sample statistics and population parameter estimates of the key variables of the study, that is, the focal independent variables, the focal dependent variable, the mediators, and the moderators.

Race/ethnicity and gender are the focal independent variables. In this sample, more than three in four respondents self-identified as non-Hispanic white. African Americans are the second most represented group at 13.1%, followed by Hispanics who make up less than 9% of the sample. In terms of gender, women outnumber men in the sample at a ratio of approximately three-to-two. The population parameter estimates by race/ethnicity and gender somewhat differ from the sample statistics. The proportion non-Hispanic white in the population is slightly larger than that of the sample, and the proportion African American and Hispanic are smaller. The

gender distribution in the population is more equal than that of the sample, with an estimated 54% female and 46% male in the population. The population distribution by race/ethnicity may seem non-representative, so it is important to emphasize that this is an older sample where, for example, the proportion African American in the population will have decreased by these ages due to higher mortality rates.

The focal dependent variable is inflammation. In the sample, average levels of inflammation—as measured by concentrations of C-reactive protein (CRP)—are 4.7 micrograms/milliliter (μg/mL), which is above the clinically significant cut point of 3.0 μg/mL: values less than or equal to 1.0 μg/mL are considered low-risk, values greater than 1.0 μg/mL but less than or equal to 3.0 μg/mL are considered moderate-risk, and values above 3.0 μg/mL are considered high-risk (Ridker, 2003). As mentioned in the previous chapter, CRP values are log-transformed in multivariate analyses to improve the distribution of the variable. On the log-transformed scale, average CRP levels are 0.8 μg/mL. For both measured values of CRP (i.e., raw values and log-transformed values), the population parameter estimates are more of less similar to the sample statistics.

Everyday and lifetime discrimination are evaluated as potential mediators of race and gender differences in inflammation. Average levels of exposure to everyday and major lifetime discrimination are low with a modal value of zero (i.e., no exposure to discrimination). Despite these low averages, the entire range of values is seen in the sample (Table 4.2), which indicates that some respondents encounter frequent exposure to discrimination. For everyday discrimination, this means some people experience discrimination "almost everyday", as discerned from the responses to the Everyday Discrimination instrument; for major lifetime discrimination, this means some people have experienced discrimination from each of four major

social institutions: the labor market, the housing market, the banking system, and law enforcement. The most frequently reported form of discrimination is age discrimination, followed by gender and racial discrimination. The predominance of experiences of age discrimination is a reflection of the older age of the sample. Figure 4.1 is a graph of the three most frequently reported reasons for experiences of discrimination. The categories presented are not mutually exclusive because. Of the 10 possible reasons given for experiencing discrimination, approximately 47% of the sample attributes their experience (at a minimum) to age, gender and/or race/ethnicity.

As for coping resources—the focal moderators of the study—social support from a combination of spouses/partners, children, friends, and other family members are high (Table 4.2). On average, respondents state they receive emotional support from these sources an equivalent of between "some" or "a lot of the time" for all items. The average respondent scores between "slightly agree" or "somewhat agree" on the purpose in life scale, but there are some individuals that "strongly disagree"—and "strongly agree"—with all or almost all statements of this nature. Average levels of optimism are above the midpoint of the scale at a score of 3.5. Like the scale for purpose in life, this value indicates that the average respondent reports that they "slightly agree" or "somewhat agree" with statements that measure optimism. As with the other two resources, the full range of responses for optimism is seen.

4.3 Distribution of Study Variables by Race/Ethnicity

The distribution to each study variable[22] by race/ethnicity is presented in Table 4.3. Significant differences for most of these characteristics exist. On average, non-Hispanic whites are older than African Americans, who are older than Hispanics. A larger percentage of African Americans are female compared to the percentage female among non-Hispanic whites and

[22] With the exception of inflammation (i.e., CRP), which is discussed in the next section.

Hispanics. Nearly 70% of non-Hispanic whites and over 60% of Hispanics are married; African Americans have the lowest proportion of individuals who are married at less than half, and the highest proportion of individuals who are separated or divorced, widowed or never married. Socioeconomic status differs across the three groups such that non-Hispanic whites have the highest levels of educational attainment and household incomes. Compared to African Americans and Hispanics, non-Hispanic whites have completed more than two and five years of education relative to the other groups, respectively. Additionally, the average household income of non-Hispanic whites, which is $80,700, is about two times that of African Americans and Hispanics. As for employment status, the percent employed does not differ much across the three groups. More than half of non-Hispanic whites and African Americans are retired. For Hispanics, employment status is more evenly distributed across the three categories, but not by much.

Non-Hispanic whites, African Americans and Hispanics differ significantly in most of their health behaviors as shown in Table 4.3. Although the percentage of respondents who report never smoking cigarettes does not differ much across racial/ethnic groups, non-Hispanic whites have the largest proportion of former smokers and African Americans have the largest proportion of current smokers and the lowest proportion of former smokers. In terms of alcohol consumption, African Americans and Hispanics are most likely to be non-drinkers, while non-Hispanic whites are most likely to be moderate drinkers. Participation in any vigorous physical activity ranges from 33% among African Americans to 42% among non-Hispanic whites, with the latter group being more likely to engage in vigorous activity at least twice a week (26%). Non-Hispanic whites are also most likely than the other racial/ethnic groups to engage in any moderate physical activity.

Health status, exposure to discrimination and levels of coping resources, significantly differ in their distribution across races/ethnicities, but the magnitude of these differences seem inconsequential for some variables. For example, non-Hispanic whites, African Americans and Hispanics differ significantly in average waist circumference, but the difference between the lowest value—39.7 inches among non-Hispanic whites—and the highest value—40.8 inches among African Americans—is only slightly greater than 1 inch. In contrast, the difference in reports of depressive symptoms is more substantially meaningful given that only eight symptoms are counted: at the low end non-Hispanic whites report an average of 1.3 symptoms; at the high end Hispanics report an average of 2.3 symptoms.

African Americans report the highest average level of exposure to everyday discrimination, and their reports of major lifetime experiences of discrimination are two times those of non-Hispanic whites and Hispanics. Levels of social support are indistinguishable across races/ethnicities but, on average, African Americans have the strongest sense of purpose in life, followed closely by non-Hispanic whites. African Americans and Hispanics do not differ in being optimistic and both groups are more optimistic than non-Hispanic whites.

4.4 Distribution of Study Variables by Gender

Table 4.4 presents the distribution of study variables by gender. On average, women are older than men by a little more than one year. The proportion of women and men who self-identify as non-Hispanic white or African American differ by gender, but the proportion of Hispanics does not. Close to 80% of men are married, compared to only 56% of women. Correspondingly, relatively more women than men are separated or divorced and rates of widowhood among women is nearly four times that of men. This dramatic difference in the proportion of widows by gender may be a reflection of differences in marriage patterns,

specifically the tendency of women to marry older men and the greater longevity of women than men. Men have slightly higher levels of educational attainment, but both men and women average almost 13 years of education. Men also have an average household income of $88,600, which is more than $20,000 higher than the average incomes of women. The proportion of retired individuals does not differ by gender but a greater proportion of men are currently working full- or part-time, while the proportion in the "other" category is greater for women than men.

With the exception of smoking, men more frequently engage in healthful behaviors such as moderate alcohol consumption, vigorous physical activity and moderate physical activity. However, they also have higher rates of being current smokers and heavy drinkers. The average waist circumference of men is three inches larger than that of women. Assuming a normal weight, the average waist circumferences for women (38.4 inches) and men (41.6 inches) are greater than the recommended levels of 35 inches for women and 40 inches for men. The average number of depressive symptoms reported is greater among women, than men.

As for social stressors and coping resources, men report average levels of exposure to everyday and major lifetime discrimination that are greater than those of women. Women experience higher levels of emotional social support, while feelings of having a purpose in life and optimism are similar in magnitude and do not differ significantly by gender. Overall, stress exposure is low (i.e., below the midpoint on the scales) among men and women, and levels of coping resources are high (i.e., above the midpoint on the scales).

4.5 Association between Inflammation and Study Variables

This section discusses the distribution of inflammation and the bivariate association of inflammation with each of the study variables. Previously, in Table 4.2, we saw that the average

concentration of CRP in the study sample was 4.7 μg/mL (SD=9.25). In Figure 4.2 we see the the average level of inflammation by race/ethnicity and gender. Regardless of race/ethnicity women have higher levels of inflammation than men, and regardless of gender African Americans have the highest levels of inflammation. Among all race-gender combinations, African American women standout with inflammation levels more than twice the cut point for clinical significance (3 μg/mL) in terms of cardiovascular disease risk. Thus, at this basic level, we see racial/ethnic and gender differences in inflammation that highlight African American women as a group at elevated risk for cardiovascular diseases.

Table 4.5 also shows differences in average inflammation levels by race/ethnicity and gender; differences across categories of other nominal study variable are also presented. CRP values displayed in this table are log-transformed because findings presented in subsequent chapters use this transformation of the variable as well. As suggested by Figure 4.2, differences in average levels of inflammation are significant across race/ethnicity and across gender. African Americans and women have the highest levels among their respective social groups. Employed individuals have the lowest average inflammation levels relative to retired persons and individuals with other forms of employment, and inflammation among individuals who are separated or divorced from their spouse or partner is higher than levels among individuals with other marital statuses. Current smokers have significantly higher inflammation levels than former smokers and individuals who have never smoked. Individuals who abstain from drinking alcohol have the highest levels of inflammation while individuals who drink alcohol in moderation have the lowest. Engaging in vigorous and moderate physical activity at greater frequencies is associated with lower levels of inflammation. And, lastly, average inflammation

levels are higher among participants of the 2008 biomarker assessment of the HRS, compared to participants on the 2006 assessment.

A correlation matrix is presented in Table 4.6 to show the associations among inflammation and the other continuous study variables. CRP has a statistically significant bivariate association with all variables included in the analysis. It is inversely associated with education, income and all of the coping resources, and positively associated with waist circumference and depressive symptoms. Notably, it is positively associated with both forms of discrimination. The direction of these associations are in line with the expectations that: (1) socioeconomic and psychosocial resources will curtail inflammatory processes, (2) changes in inflammation will coincide with other markers of disease risk, and (3) exposure to social stressors will adversely affect health at a biological level.

Other notable associations include the inverse association between discrimination and age, the positive association between both forms of discrimination and both measures of health status, and the positive association between everyday and major lifetime discrimination, which is expected. With only two exceptions, each of the coping resources are significantly associated with the other variables presented in the table. The exception to this generalization is the absence of an association between age and optimism. The direction of the other associations are as expected. For example, the availability of socioeconomic resources (e.g., education and income) would be expected to be positively correlated with the availability of other types of resources, including psychosocial resources such as social support. This is what is shown in the Table 4.6.

4.6 Summary of Key Findings

The findings from preliminary analyses of the data set the scene for results presented in subsequent analyses. In this chapter we see support for the focal relationships between race/ethnicity and inflammation and between gender and inflammation. Support is also presented for a positive association between discrimination and inflammation, and for significant differences in inflammation by race/ethnicity and by gender. These two findings provide initial—but provisional—support for the role of discrimination as a mediator of race/ethnic and gender differences in inflammation. Thorough modeling of the relationships among all the study variables is needed to strengthen this claim, which will be accomplished in the following three chapters. Chapter 5 will show that racial/ethnic and gender differences in inflammation persist even after controlling for demographic characteristics, socioeconomic factors, health behaviors, and measures of health status; Chapter 6 will present a model of the relationship among all study variables and test for the indirect effect of race/ethnicity and gender through discrimination and other potential intervening variables; and Chapter 7 will describe conditional relationships among race/ethnicity, gender, discrimination, coping resources, and inflammation.

CHAPTER 5

RESUTS: DISTRIBUTION OF CRP

5.1 Introduction

In this chapter I present results for Aim 1 of the dissertation, which examines race and gender differences in inflammation. The objectives of this aim are (1) to determine whether levels of inflammation differ by race/ethnicity and/or gender and (2) to assess the extent to which any race or gender differences in inflammation persist after controlling for demographic characteristics, socioeconomic factors, health behaviors and health status.

As reviewed in the first chapter of this dissertation, African Americans have higher rates of cardiovascular diseases than Hispanics and non-Hispanic whites (Roger et al., 2011). Rates of cardiovascular diseases among Hispanics, however, are similar to or slightly lower than those of non-Hispanic whites. Given that systemic inflammation is associated with cardiovascular diseases (Danesh et al., 2004) and that the prevalence of cardiovascular diseases is greatest among African Americans, I hypothesize that African Americans will have higher levels of inflammation than Hispanics and non-Hispanic whites (Hypothesis 1A). As for the difference between Hispanics and non-Hispanic whites, despite the fact that older Hispanics adults have rates of cardiovascular diseases that are comparable to those of non-Hispanic whites, I contend that Hispanics also will have higher levels of inflammation than non-Hispanic whites, but lower levels than African Americans. This hypothesis is based on the association of systemic inflammation with other chronic conditions that disproportionately affect Hispanics relative to whites.

Gender differences in cardiovascular diseases exist (Rogers et al., 2011) and recent research suggests that levels of inflammation also differ by gender (Khera et al, 2005; Mendelsohn & Karas, 2005); specifically, women have higher levels of inflammation than men. Moreover, when examined by race/ethnicity and gender, African American women have higher levels of inflammation than all race/ethnicity and gender combinations (Khera et al, 2005). In line with these findings, I hypothesize that women will have higher levels of inflammation than men (Hypothesis 1B) with African American women, in particular, having the highest levels (Hypothesis 1C). Adjustments for demographic characteristics, socioeconomic factors, health behaviors, and health status will attenuate these differences in inflammation, but the differences will exist nonetheless (Hypothesis 1D).

This chapter describes group differences in inflammation by race/ethnicity, gender, and race/ethnicity and gender. It also described the extent to which these differences remain after controlling for other factors. The association between inflammation and each of the study variables is discussed, as is the extent with which the covariates explain inflammation. Collectively, the findings from the investigation of Aim 1 offer important insight into the social patterning of inflammation and the factors that influence this biomarker.

5.2 Race and Gender Differences in Inflammation

In Chapter 4, bivariate analyses with cross-tables revealed differences in inflammation by race/ethnicity and gender. Regression models also assessing these relationships are presented in Table 5.1. Consistent with Hypothesis 1A, Model 1 shows that, on average, African Americans and Hispanics have significantly higher levels of inflammation than non-Hispanic whites. The difference in inflammation between African Americans and non-Hispanic whites is more than two times the difference between Hispanics and non-Hispanic whites, which supports the

hypothesis that African Americans will have the highest levels of inflammation, non-Hispanic whites will have the lowest and levels of inflammation among Hispanics will be somewhere in the middle. A control variable for year of data collection is included in the model to account for changes in inflammation between the two time periods. As expected, participants from 2008 have higher CRP levels than participants from 2006.

Model 2 is the regression of inflammation on gender and year of data collection. It provides support for Hypothesis 1B which states that women will have significantly higher levels of inflammation than men. Including race/ethnicity and gender in the same regression negligibly changes the magnitude of their respective coefficients and does not change their statistical significance (Model 3). After examining the individual effects of race/ethnicity and gender on CRP, their conditional effect is considered by adding product interaction terms (Model 4). The interaction between race/ethnicity and gender is not statistically significant, which suggests that—contrary to Hypothesis 1C—racial and ethnic differences in inflammation are not contingent on gender. In other words, the magnitude of race and ethnic differences in inflammation are similar for men and women. Since these analyses show that the conditional association between race/ethnicity and gender is not statistically significant, the more parsimonious main effects model (Model 3) is preferred and used in subsequent analyses. Table 5.1 also shows that year of data collection is a significant predictor of inflammation across all models presented in the table. CRP levels are consistently higher for 2008 HRS participants compared to 2006 participants. This finding coincides with literature showing that inflammation increase over time (Solana, Pawelec & Tarazona, 2006).

Finally, exponentiation of the regression coefficients from the preferred model provides a sense of the clinical significance of these differences in inflammation. In the U.S., average CRP

levels are approximately 1.5 μg/mL and only 25% of Americans have CRP above 3 μg/mL, the cut point for clinical significance (Ridker, 2003). In this study, the difference in raw CRP values between African Americans and non-Hispanic whites is 1.40 μg/mL. The difference between Hispanics and non-Hispanic whites is 1.17 μg/mL, and the difference between women and men is 1.26 μg/mL. These magnitudes are halfway between moderate-risk and high-risk for cardiovascular disease. That said, Model 3 does not account for other factors (e.g., age, income, education, etc.) that influence inflammation and/or racial/ethnic or gender differences in this biomarker. Analyses described in the next section and in the following chapter adjust for additional variables and potentially provide a more exact estimate of these differences.

5.3 *Net* Race and Gender Differences in Inflammation

Table 5.2 presents results from nested models for the regression of inflammation on race/ethnicity and gender (Model 1) plus demographic characteristics and socioeconomic factors (Model 2), health behaviors (Model 3), and measure of health status (Model 4). These models are used to test Hypothesis 1D that differences in inflammation by race/ethnicity and gender exist even after accounting for other factors influencing inflammation. Model 1 is the same as the preferred model from the previous section. In Model 2, variables for age, education, household income (log-transformed), employment status, and marital status are added.

Age is significantly and inversely associated with inflammation such that increasing age is associated with lower levels of inflammation. Education also has an inverse association with inflammation, with is suggestive of a protective effect of this resource against inflammation. Although a similar finding would be anticipated for household income, in this study the data indicate that there is no association between income and inflammation. Employed individuals, have significantly lower levels of inflammation than individuals who are retired or have other

employment statuses (e.g., unemployed, disabled, not in labor force). Net of other variables in the model, married individuals have significantly lower levels of inflammation than individuals who are separated or divorced, but their levels of inflammation do not significantly differ from individuals who are widowed or who have never been married.

After adding the demographic characteristics and socioeconomic factors to the regression, the difference in inflammation between African Americans and non-Hispanic whites decreases by 38% but remains statistically significant. The difference between Hispanics and non-Hispanic whites, however, decreases by more than 100%; in other words, it becomes negative and is not longer statistically significant. These findings suggest that some portion of the association between race/ethnicity and inflammation potentially is due to demographic or socioeconomic intervening variables (i.e., mediators), which is a topic more fully explored in the next chapter. As for gender differences in inflammation, these differences remain after accounting for demographic characteristics and socioeconomic factors and are significant. The magnitude of the difference, however, is reduced by 26%.

Variables for four health behaviors—smoking status, alcohol consumption, vigorous physical activity, and moderate physical activity—are added to the regression and presented in Model 3. All four behaviors are significantly associated with inflammation. Former smokers and current smokers have higher levels of inflammation than individuals who report never smoking, and individuals who are considered moderate or heavy drinkers of alcohol have lower levels of inflammation than non-drinkers. Engaging in vigorous or moderate physical activity, even at a frequency less than or equal to once a week, is protective against inflammation compared to not engaging in any form of moderate or vigorous activity.

The addition of the health behaviors resulted in an additional reduction in the race difference in inflammation (19%), relative to the previous model, but no change in the gender difference. Compared to Model 1 of this table, 50% of the difference in inflammation between African Americans and non-Hispanic whites is explained by the combination of demographic characteristics, socioeconomic factors and health behaviors (Model 3). The effect of age on inflammation remains the same despite the addition of the health behaviors. The effect of education also remains significant but is reduced slightly, as are differences in inflammation between retired and employed persons, and between individuals with other employment statuses and employed persons. The difference in inflammation between individuals who are divorced or separated and individuals who are married or in other partnerships is significant but diminished.

The next model of Table 5.2 (Model 4) adds variables for waist circumference and depressive symptoms. Preliminary analyses during the model building processed revealed curvilinear relationships between both of these variables and inflammation (data not shown). Therefore, quadratic terms for waist circumference and depressive symptoms are included in the model; both variables are centered at their respective means. After adjusting for other variables in the model, these measures of health status are significantly and positively associated with inflammation. Their respective quadratic terms are also significant, but negative. In other words, as waist circumference and depressive symptoms increase, inflammation increases but it does so at a decreasing rate.

Including these measures of health status in the model further reduces the race difference in inflammation. In contrast, the gender difference more than doubles in magnitude relative to the previous model. Both the reduction in the race difference and the increase of the gender difference are indicative of mediation by waist circumference and/or depressive symptoms. The

latter observation—the increase in the gender difference—is specifically interpreted as a suppression effect because including these two health variables revealed a larger effect size that previously was hidden or suppressed when the variables were absent from the model.

Including waist circumference and depressive symptoms also altered the effect of other variables on inflammation. Age is no longer associated with inflammation after accounting for these health measures, which is suggestive of complete mediation of the age effect by waist circumference and/or depressive symptoms. The effect of education is reduced and differences in inflammation by employment status are no longer statistically significant. The difference between married and separated/divorced persons is still significant but lessened. As for behavioral factors, significant differences in inflammation exist for all comparisons except between heavy drinkers of alcohol and non-drinkers. This difference is not significant when waist circumference and depressive symptoms are both in the model. With one exception, the difference in inflammation due to these health behaviors occurs after accounting for measures of health status. The exception to this observation is the difference in inflammation between current smokers and non-smokers, which increased. Thus, the overall difference in inflammation between these groups was suppressed by waist circumference and/or depressive symptoms.

Two final steps were taken to assess the magnitude of the effect of race/ethnicity and the effect of gender on inflammation. Regression models were estimated that exclude each of these variables individually. Model 5 of Table 5.2 presents the regression of inflammation on all of the study variables, except race/ethnicity. The change in R^2 represents the unique contribution of race/ethnicity over and above all the variables in the model. Excluding race/ethnicity from the regression reduced the R^2 value by slightly more than 1% of the value for the fully adjusted model presented in Model 4 (ΔR^2 =-0.002, $F(21,43)=116.10$, $p<0.001$). Thus, its contribution to

explaining the population-level distribution of inflammation is relatively small. The contribution of gender, however, is quite larger. Estimating a regression model without gender (Model 6) reduced the R^2 value by more than 15% (ΔR^2 =-0.026, $F(22,42)=121.27$, $p<0.001$) which suggests that gender is more consequential to variations in inflammation than race/ethnicity. In all, the largest proportion of variance in inflammation that is explained by the models is 16.8% (Model 4).

5.4 Summary of Key Findings

Analyses from this chapter reveal that levels of inflammation differ by race/ethnicity and by gender, but these are independent—not conditional—effects. Racial and ethnic minorities have higher levels of inflammation than non-Hispanic whites and women have higher levels than men. The differences between African Americans and non-Hispanic whites and between women and men persist even after accounting for demographic and socioeconomic factors, health behaviors, and health status. This finding is consistent with Hypothesis 1D and with literature examining racial and gender differences in cardiovascular health outcomes (Khera et al, 2005). Differences between Hispanics and non-Hispanic whites, however, are not significant after adjusting for other variables and they seem to be accounted for by differences in demographic characteristics and/or socioeconomic status. The possibility of a mediating effect by some of these variables will be explored in the next chapter.

Findings from this chapter also suggest that socioeconomic status, health behaviors and health status are consequential for inflammation. Higher levels of education and income are negatively associated with inflammation, being a former or current smoker is positively associated with inflammation, and moderate or heavy alcohol consumption and engaging in vigorous and moderate physical activity is associated with lower levels of inflammation. Waist

circumference and depressive symptoms also adversely affect levels of inflammation, but they do so in a non-linear manner: as waist circumference and depressive symptoms increase, the rate of increase in inflammation decreases. The data also suggest that these two measures of health status may also be important mediators of differences in inflammation by race/ethnicity and gender.

CHAPTER 6

RESULTS: DIFFERENTIAL STRESS EXPOSURE

6.1 Introduction

The previous chapter examined racial/ethnic and gender differences in inflammation and revealed that these differences exist even after controlling for demographic characteristics, socioeconomic factors, health behaviors, and measures of health status. The current chapter seeks to explain the existence of these differences by investigating the role of two social stressors—everyday discrimination and lifetime discrimination—as potential mediators. Mediation by socioeconomic status, health status and coping resources is also ascertained. More specifically, everyday discrimination, lifetime discrimination, education, and household income are considered primary mediators of racial/ethnic and gender differences in inflammation; waist circumference, depressive symptoms, social support, purpose in life, and optimism are considered secondary mediators of racial/ethnic and gender differences in inflammation.

Findings from empirical studies suggest that racial/ethnic minorities and women are more likely to report experiencing discrimination than non-Hispanic whites and men, respectively, as discussed earlier, although in this study women report less discrimination than men. Their disproportionate exposure to discrimination is due to a system of social stratification that marginalizes racial/ethnic minorities and women. Consequently, members of these social groups are faced with negative stereotyping, stigma and discrimination. Hypothesis 2A asserts that African Americans and Hispanics will report greater exposure to discrimination compared to non-Hispanic whites, and women will report greater exposure to discrimination compared to men. I further hypothesize that greater exposure to discrimination will adversely affect

inflammation (i.e., it will be positively associated with inflammation (Hypothesis 2B); moreover, the racial/ethnic and gender differences in inflammation will be explained—at least in part—by parallel differences in exposure to discrimination (Hypothesis 2C).

To test the stated hypotheses, the chapter begins by describing the distribution of everyday and lifetime discrimination by race/ethnicity and gender and then testing the association between both forms of discrimination and inflammation. The chapter continues with a path analysis using structural equation models (SEMs) to formally test the indirect effects of race/ethnicity and gender on inflammation. Intervening pathways linking race/ethnicity and gender to inflammation through education and income are assessed because findings from previously discussed analyses suggest that differences in socioeconomic status—specifically differences in education—drive group differences in inflammation between Hispanics and non-Hispanic whites.

6.2 Race and Gender Differences in Discrimination

Tables 6.1 and 6.2 present regression models estimating racial/ethnic and gender differences in discrimination, controlling for year of data collection. Group differences in everyday discrimination will be taken up first, followed by differences in lifetime discrimination. Model 1 of Table 6.1 shows that relative to non-Hispanic whites, African Americans report more frequent exposure to everyday discrimination. Their level of exposure also significantly differs from that of Hispanics (post-estimation test, $b=0.212$, $SE=0.053$, $p<0.001$). Hispanics, however, do not differ significantly from non-Hispanic whites in reports of discrimination. Model 2 shows that men report significantly greater levels of exposure to discrimination compared to women, and Model 3 shows that the race effect persists net of gender and, likewise, the gender effect persists net of race/ethnicity.

The interaction between race/ethnicity and gender is assessed in Model 4. It was operationalized with two product interaction terms because two dummy variables are used to operationalize race/ethnicity. The collective significance of the two product interaction terms was tested first using an adjusted Wald test, followed by an examination of the individual product interaction terms. The Wald test confirms the significance of the race-by-gender interaction ($F(2, 62)=3.30$; $p=0.044$) and Model 4 shows that the difference between Hispanics and non-Hispanic whites is contingent on gender. Hispanic men, on average, do not differ in reports of everyday discrimination from non-Hispanic white men, but Hispanic women report higher levels than non-Hispanic white women. The models in Table 6.1 also show that the 2008 half-sample, on average, reported less exposure to everyday discrimination than the 2008 half-sample. The magnitude, direction and significant of this difference in exposure were relatively constant across the models.

The patterning of exposure to lifetime discrimination across groups is similar to those seen for everyday discrimination, with the exception of a non-significant race-by-gender interaction (Table 6.2). African Americans report a greater number of major lifetime experiences of discrimination compared to non-Hispanic whites. Hispanics do not differ from non-Hispanic whites in their frequency of exposure (Model 1); they do differ, however, from African Americans who report a significantly higher number of exposures (post estimation test, $b=0.460$, $SE=0.067$, $p<0.001$). Women report fewer lifetime experiences of discrimination than men (Model 2), and this difference is still present after adding race/ethnicity in the model (Model 3). Similarly, the race difference is still present after adding gender. The interaction between race and gender is added in Model 4; it is not significant, as determined by the Wald test ($F(2,62)=0.53$, $p=0.590$). Therefore, the more parsimonious model (Model 3) is preferred.

In contrast to findings presented in Table 6.1, Table 6.2 shows an effect of year of data collection on lifetime discrimination, but in the opposite direction; specifically, members of the 2008 half-sample report a higher level of exposure than members of the 2006 half-sample. Similar to the finding presented in Table 6.1, this difference in lifetime discrimination is relatively constant across the models. If randomization of HRS households was successful, we would expect everyday and lifetime discrimination to increase somewhat because exposures accumulate over time and more time has elapsed, and because the 2008 half-sample is on average 2 years older at the time of data collection than the 2006 half-sample and older persons may experience a greater amount of age discrimination. Therefore, both everyday and lifetime discrimination might increase over time. However, 2008 reports of everyday discrimination are lower than 2006 reports (Table 6.1) while the opposite is the case for lifetime discrimination (Table 6.2). This variable, year of data collection, is included in all subsequent models.

Collectively, the findings presented in these two tables partially support Hypothesis 1A in that African Americans have the highest levels of exposure to discrimination. However, Hypothesis 1A is not confirmed for Hispanics and the opposite association is found for gender. The interaction of race and gender is not significant for lifetime discrimination, and only one of the two product interaction terms is significant for everyday discrimination and only at the 0.05 significance level. Moreover, the interaction between race/ethnicity and everyday discrimination is not significant in subsequent models that adjust for demographic and socioeconomic factors (data not shown). Therefore, the analysis proceeds without the race-by-gender interaction.

6.3 Effect of Discrimination on Inflammation

Results for the regression of inflammation on everyday and lifetime discrimination are presented in Table 6.3; all models presented control for year of data collection. Model 1 includes

everyday discrimination as a predictor of inflammation and Model 2 includes lifetime discrimination. Both models show that discrimination has an adverse effect on inflammation: greater exposure to both stressors is associated with higher levels of inflammation. Model 3 presents the regression of inflammation on both forms of discrimination and shows that everyday discrimination is positively associated with inflammation, net of the effect of lifetime discrimination, and vice versa. The interaction of everyday and lifetime discrimination is added in Model 4 to evaluate the conditional relationship between these two stress exposures. The regression coefficient for the interaction term is not significant; therefore, on the basis of parsimony, Model 3 is the preferred model.

In all, from this series of regressions we see that everyday discrimination and lifetime discrimination have independent adverse effects on inflammation: greater exposure to each stressor is associated with higher levels of systemic inflammation. This finding aligns with Hypothesis 2B of the dissertation. That said, the collective effect of everyday and lifetime discrimination accounts for less than 1% of the variance in inflammation (Model 3: R^2=0.005), which means most of the variance in inflammation is left unexplained by discrimination. While this level of explanation is very low by the standards of social science research, the benchmarks for clinical outcomes like CRP in population-based research on social factors are not well established because of the recent nature of this line of research. Therefore, these associations are sufficient to evaluate whether discrimination mediates racial/ethnic and gender differences in inflammation, and as shown below, discrimination does account for some of these differences.

In Table 6.4, a full model for the regression of inflammation on race/ethnicity, gender and discrimination is presented that accounts for additional demographic characteristics, socioeconomic factors, health behaviors, and health status. The model also includes measures of

social support, purpose in life and optimism. The mediating effect of these coping resources for race and gender differences in inflammation is evaluated. All models presented in the table also control for year of data collection. For reference, the first model in Table 6.4 is the same model presented in Table 5.2, Model 3 and it represents the two focal relationships tested in the dissertation: racial/ethnic differences in inflammation and gender differences in inflammation.

Model 2 adds the focal intervening variables, everyday and lifetime discrimination. Both forms of discrimination have adverse and independent effects on inflammation, and including them in the regression attenuates the difference in inflammation between African Americans and non-Hispanic whites by approximately 15%. The difference between Hispanics and non-Hispanic whites is essentially unchanged and the difference in inflammation between women and men increases by more than 10%. The race and gender differences just described all remain statistically significant despite the inclusion of the discrimination variables. In all, this model explains slightly more than 2% of the variance in inflammation.

Adding the remaining demographic characteristics and socioeconomic factors (Model 3) nearly doubles the explanatory power of the model. Age and education are inversely associated with inflammation, while household income is not significantly related to the biomarker. Retired individuals and individuals with other employment statuses have higher levels of inflammation than individuals who are currently working, and married persons have lower levels of inflammation than those who are divorced or separated from their spouses or partners. Individuals who are widowed or who have never married do not differ significantly from married individuals in their levels of inflammation. Compared to the previous model, accounting for these factors reduces the difference in inflammation between African Americans and non-Hispanic whites and results in a non-significant ethnic difference between Hispanics and non-

Hispanic whites. The gender difference is attenuated somewhat, but remains significant. With regard to discrimination, only the effect of lifetime discrimination on inflammation is significant in this model.

Health behaviors are added in Model 4. All the behaviors are significant and, with one exception, affect inflammation in the expected manner. Smoking is detrimental for inflammation (i.e., increases levels), and all levels of alcohol consumption and vigorous and moderate physical activity are beneficial (i.e., lowers levels). Given the literature on the relationship between alcohol consumption and health, the exception to expected findings is that heavy alcohol consumption is associated with lower levels of inflammation, which means it is beneficial for health. The relationship between alcohol consumption and cardiovascular outcomes is usually "J-shaped" where moderate levels of consumption are most beneficial for health.

All of the other variables that were significant in the previous model (Model 3) are still significant in Model 4 and operate in the same direction (e.g., lifetime discrimination is still positively associated with inflammation). Adding the health behaviors is associated with a reduction in nearly all of the regression coefficients; the exception here is age, which increased its effect size in this model. The variance in inflammation explained by Model 4 is 7.5%.

Models 5 and 6 add health status measures and coping resources, respectively. The addition of the health status variables more than doubles the explanatory power of the model. The R^2 value increased from 7.5% in Model 4 to nearly 17% in Model 5. Waist circumference positively and significantly affects inflammation, but depressive symptoms do not. Again, with heavy alcohol consumption being the exception, all the other health behaviors are significantly associated with inflammation and in the expected direction of effect. This time, however, heavy alcohol consumption is not associated with inflammation, which more closely aligns with the

usual "J-shaped" relationship between alcohol consumption and cardiovascular health. Among the demographic and socioeconomic variables, only education is significant and it has an inverse relationship with inflammation.

As for discrimination, lifetime discrimination has a significant direct effect on inflammation but everyday discrimination does not. Greater exposure to lifetime discrimination is still associated with higher levels of inflammation, but the magnitude of its effect on this biomarker is reduced by 23% after accounting for health status. The magnitude of the race difference has also been attenuated. From its initial value in Model 1, the net difference in inflammation between African Americans and non-Hispanic whites has decreased by 64%, with African Americans having higher levels of CRP. In other words, the collective effects of discrimination, demographic and socioeconomic factors, health behaviors, and health status explain more than two-thirds of the difference in inflammation between these two groups. The gender difference in inflammation also changed dramatically: From its value in Model 1 to its value in Model 5, the difference in inflammation between women and men increased by more than 80%, with women having the higher levels of CRP. This finding for the gender difference is indicative of a suppression effect.

Taken together, the findings presented in Tables 6.1 through 6.4 provide partial support for the hypothesis that race and gender differences in exposure to discrimination mediate race and gender differences in inflammation (Hypothesis 2C). Tables 6.1 and 6.2 show that race/ethnicity and gender (the independent variables) are associated with discrimination (the mediators), Table 6.3 show that discrimination is associated with inflammation (the dependent variable) and Table 6.4 shows changes in the focal relationships when discrimination and other predictors are systematically added to the regression. However, to formally test mediation, the

effects of race/ethnicity and gender on inflammation need to be decomposed into their direct, indirect and overall effects and the significance of these pathways needs to be tested. Accordingly, a path analysis is performed using the suite of commands for structural equation modeling (i.e., the SEM command and the "estat teffects" post-estimation command) available in Stata® 12.1. The findings from this analysis are presented in the remaining sections of this chapter.

6.4 Structural Equation Model, Model Fit and Summary of Direct, Indirect and Total Effects

In Chapter 3, an analytic model for the path analysis was presented (Figure 3.7). This model is a simplified representation of the paths tested in the full structural equation model (SEM).[23] It serves as a guide for the discussion that follows. The arrows printed in bold type are paths operating through exposure to discrimination; dashed arrows are paths operating through socioeconomic factors; and arrows that are dashed and in bold type are path common to intervening mechanisms through discrimination and socioeconomic status. The remaining pathways are presented dash-and-dotted arrows and represent intervening pathways through changes in health status or coping resources and the direct effect of race/ethnicity and gender.

Tables 6.5a-6.5c present regression models for all of the endogenous variables in the SEM (i.e., Figure 3.7), specifically the primary mediators (everyday discrimination, lifetime discrimination, education, and household income), the secondary mediators (waist circumference, depressive symptoms, social support, purpose in life, and optimism) and the dependent variable (discrimination). Regression models for the discrimination and

[23] Additional pathways are not shown to make the relationships that are relevant for answering the research questions clear.

socioeconomic status are presented in Table 6.5a; models for health status are presented in Table 6.5b; and models for the coping resource and inflammation are presented in Table 6.5c.

The coefficients from these regressions are used in the path-tracing process. Following MacKinnon's ***a***, ***b***, ***c***, ***c′*** model for mediation analysis (MacKinnon, 2008), the indirect effects are calculated by multiplying the path (i.e., regression) coefficient of race/ethnicity or gender as given in one of the mediator models (i.e., ***a*** in Tables 6.5a and 6.5b) by the path coefficient of the corresponding mediator as given in the regression model for the inflammation (i.e., ***b*** in Table 6.5c). The indirect effects for each of the mediating pathways for race/ethnicity or gender are summed to calculate the cumulative indirect effect. The residual direct effect (i.e., ***c′***) is the path coefficient of the independent variables (race/ethnicity and gender) as given in the regression model for inflammation (Table 6.5c), and the total effect of race/ethnicity or gender (i.e., ***c***) is the sum of their respective indirect effects and direct effects. Before discussing the findings of the path analysis, the model-building process and the corresponding fit statistics are reviewed.

Indices of Model Fit. To obtain an acceptable fit of the model to the data, covariances among all exogenous variables and between error terms of specific endogenous variables were included. Table 6.6 shows the specific error covariances that were added. Multiple indices of goodness-of-fit were used to determine whether the estimated model appropriately fits the data. Because data from the HRS come from a complex study sample, the fit indices available in Stata 12.1 for survey data are limited to the coefficient of determinant (CD), which is equivalent to the R^2 (i.e., the total explained variance of the dependent variable) and to the standardized root mean squared residual (SRMR). The SRMR, however, cannot be estimated when there are missing data, which is the case for this analytic sample. Therefore, during the model building process I evaluated fit indices for a model assuming a simple random sample (i.e., a model that does not

account for sampling design effects, such as clustering). The fit indices evaluated include the Tucker-Lewis Index (TLI), Comparative Fit Index (CFI), Root Mean Squared Error Approximation (RMSEA), and the p of Close Fit (pclose).[24] Values above 0.95 are desirable for the TLI and CFI, values below 0.05 are desirable for the RMSEA, and values close to 1.000 are desirable for the pclose. For the SEM represented by Figure 3.7, fit indices indicate that the fit of the observed data is excellent: CFI = 1.000, TLI = 1.000, RMSEA = 0.000, and pclose = 1.00. Additionally, based on the R^2-value, the full model accounts for approximately 65.8% of the variance in inflammation. Given the value of these fit indices, it is reasonable to proceed with the path analysis; the next section will discuss its findings.

6.5 Indirect Effect of Race/Ethnicity through Discrimination and Socioeconomic Status

Tables 6.7 and 6.8 show the specific indirect effects of race/ethnicity through each mediator, the sum of these indirect effects, the direct effect, and the total effect. The pathways for the indirect effects are identified using Figure 3.7 by visually tracing the arrows connecting each of the variables to one another. From this process we see that there are nine 1-mediator pathways operating singly through the nine mediators evaluated in the study; ten 2-mediator pathways operating separately through both forms of discrimination and each of the secondary mediators; and ten 2-mediator pathways operating separately through education and income and each of the secondary mediators. These pathways potentially explain differences in inflammation between African Americans, Hispanics, women and their respective reference groups.

Explaining Differences between African Americans and non-Hispanic Whites. Table 6.7 summarizes the intervening pathways explaining differences in inflammation between African

[24] The chi-square test statistic is not presented because with sample sizes greater than 400, this test tends to reject the null hypothesis that the model fits the data even if the fit is acceptable.

Americans and non-Hispanic whites. Unstandardized estimates of the indirect effects are presented along with corresponding standard errors and indication of level of significance (i.e., p-values). At the bottom of the table is a sum of all the indirect effects for the difference between African Americans and non-Hispanic whites, the direct effect (from Table 6.5c), and the total effect, which is the sum of all of the indirect effects and the direct effect. As can be seen, most of these indirect effects are not statistically significant, all of the 2-mediator pathways are very small, and the three statistically significant 1-mediator pathways account for most of the indirect effect across all pathways.

For 1-mediator pathways, the only significant ones are through lifetime discrimination (Path 2), education (Path 3) and purpose in life (Path 8). For inflammation differences between African Americans and non-Hispanic whites, race differences in exposure to lifetime discrimination and race differences in educational attainment contribute to higher levels of inflammation among African Americans. The negative sign for the indirect effect through purpose in life means higher levels of this coping resource work to minimize inflammation differences between African Americans and non-Hispanic whites. That is, this indirect effect is in the opposite direction as the direct and other significant indirect effects of race on inflammation.

Among 2-mediator pathways operating through discrimination there are two significant pathways both of which operate through waist circumference as a secondary mediator (Path 10 and Path 15). They account for only 15.2% of all indirect effects. With regard to socioeconomic mediators, the intervening pathway including education and waist circumference is significant (Path 20), and the pathway including household income and purpose in life is significant (Path 28). They collectively account for just 10.3% of the indirect effect of African American race.

Social support and optimism are not significant mediators of differences in inflammation between African Americans and non-Hispanic whites.

Thus, the primary indirect effects of race on inflammation are through lifetime discrimination and education. That is, most of the explanation of the total effect of race is due to race-related differences in education and in exposure to lifetime discrimination. As shown at the bottom of Table 6.7, the sum total of the indirect effects of race through all mediators is more than half of the size of the direct effect of race. However, there remains a larger and statistically significant direct effect of race. Thus, there is partial not complete mediation. These findings provide partial support for Hypothesis 2C that differential exposure to discrimination mediates race differences in inflammation.

Explaining Differences between Hispanics and non-Hispanic Whites. The lack of a statistically significant direct effect of ethnicity between Hispanics and non-Hispanic whites (See Table 6.4, Model 6) does not negate investigation of intervening pathways between these two groups because direct effects are expected to be explained by the inclusion of intervening variables in the model. Recent simulation studies conducted by Rucker and colleagues (2011) demonstrate that significant indirect effects may exist even in the absence of a significant overall effect and/or significant direct effects. Furthermore, there is a significant gross difference (See Table 6.4, Model 1). Considering the work of Rucker et al (2011) and other researchers (Hayes, 2009; Zhao, Lynch & Chen, 2012), it is reasonable to proceed with the examination of specific indirect pathways explaining differences in inflammation between Hispanics and non-Hispanic whites.

Table 6.8 summarizes the mediation analysis for explaining ethnic differences in inflammation. The table presents the same information as Table 6.7 and is arranged in a similar

fashion. As can be seen, only four of the 29 possible indirect effects are statistically significant. The largest pathway is through education (Path 3), which is the only significant 1-mediator pathway, accounting for 89% of the indirect effect of Hispanic ethnicity on inflammation. It shows that the lower levels of education among Hispanics are associated with higher levels of inflammation. Although considerably smaller in size, there are three 2-mediator pathways that are statistically significant, one through education and waist circumference (Path 20), one through education and purpose in life (Path 23) and one through income and purpose in life (Path 28). The lower levels of education among Hispanics—relative to non-Hispanic whites—is associated with larger waist circumferences and larger waist circumferences are associated with higher levels of inflammation. Additionally, lower household incomes and lower levels of educational attainment among Hispanics are associated were a diminished sense of purpose in life and a diminished sense of purpose in life is associated with higher levels of inflammation, but these effects are negligible relative to the effect of education on inflammation.

In both 1-mediator (Path 1 and Path 2) and 2-mediator pathways (Paths 10-19), differential exposure to discrimination did not explain differences in inflammation between Hispanics and non-Hispanic whites.

Overall, the primary indirect effects of ethnicity involve education and education-related differences in waist circumference. These statistically significant indirect effects are found in the absence of a direct effect of ethnicity. Thus, they help to explain why overall Hispanics have higher levels of inflammation on average than non-Hispanic whites.

6.6 Indirect Effect of Gender through Discrimination and Socioeconomic Status

The specific indirect effects of gender are presented in Table 6.9. Again, this table is similar to Tables 6.7 and 6.8 in the information presented and organization. Findings presented

earlier in this chapter suggested that the net difference in inflammation between women and men was suppressed by waist circumference in that the average waist circumference of women in this sample is significantly smaller than that of men and waist circumference is predictive of inflammation. Accounting for this variable nearly doubled the gender difference. Table 6.9 shows that as a result of these connections, waist circumference generates a substantial indirect effect of gender on inflammations. More than 91% of the indirect effect of gender operates through this specific pathway. Waist circumference is the primary mechanism through which gender influences inflammation. This large indirect effect (Path 5) is opposite to the direct effect of gender. Thus, if it were not for their smaller waist circumference, women would have even higher than observed levels of inflammation relative to men.

The other 1-mediator pathway operates through lifetime discrimination (Path 2). As reported previously, contrary to Hypothesis 2A, women reported less frequent exposure to discrimination than men, and of these two stressors only lifetime discrimination exhibits a significant positive direct effect on inflammation. Considering these two findings, the inference was made that lifetime discrimination is a mediator of gender differences in inflammation. Path 2 of Table 6.9 confirms this inference, although this effect is quite small. Thus, Hypothesis 2C is partially supported by this finding.

Among 2-mediator pathways that operate through discrimination, Path 10, 13 and 15 are significant, but so small relative to Path 5 that they are not substantively meaningful.

Unlike findings for differences between African Americans and non-Hispanic whites and differences between Hispanics and non-Hispanic whites, differences in socioeconomic status do not contribute to gender differences in inflammation (Paths 20-29). Neither education nor

income significantly mediate gender differences in inflammation because on average men and women report similar years of education and household incomes.

Thus, the primary mediator of gender differences in inflammation is waist circumference and it is large and in the opposite direction of the significant direct effect of gender, such that the total effect is smaller than the direct effect and smaller than the indirect effect.

6.7 Summary of Key Findings

In this chapter a path analysis was conducted using SEM. In this analysis, intervening pathways linking race/ethnicity and gender to inflammation were evaluated. Central to the study is determining the extent to which differential exposure to everyday and major lifetime discrimination operates as mediators. Both forms of discrimination significantly contribute to group differences in inflammation by race/ethnicity and by gender, but only lifetime discrimination has a significant direct effect on inflammation in fully adjusted models. However, both forms of discrimination have significant indirect effects either as the sole mediator (lifetime discrimination) or as a primary mediator in a sequential chain of effect (everyday and lifetime discrimination). Waist circumference by itself, or in conjunction with discrimination or socioeconomic status, proved to be an important mediator of differences in inflammation, especially when comparing inflammation levels by gender. Purpose in life was the only coping resource involved in any of the intervening pathways, but the size of the pathways in which it is involved were negligible.

After sifting through the findings discussed in this chapter, there is one key take away message pertaining to the aims of the study: Differential exposure to discrimination is a viable explanation of some of the racial/ethnic and gender differences in inflammation. Because systemic inflammation is associated with cardiovascular diseases, these differences in

inflammation potentially contribute to the race-based and gender-based patterning of cardiovascular disease in old age. To further understand the contribution of these social stressors to differences in inflammation, the next chapter of the dissertation will evaluate two additional hypotheses for racial/ethnic and gender differences: differential vulnerability to social stressors and differential availability of coping resources that mitigate the effects of stress exposure.

CHAPTER 7

RESULTS: DIFFERNTIAL VULNERABILITY & STRESS BUFFERING

7.1 Introduction

At this point in the dissertation, three noteworthy conclusions about this population can be made from the data: (1) levels of systemic inflammation significantly differ between African Americans and non-Hispanic whites and between women and men, but not between Hispanics and non-Hispanic whites; (2) differential exposure to the stress of everyday and lifetime discrimination partially accounts for the disparity between groups; and (3) differences in educational attainment partially mediate differences in inflammation between African Americans and non-Hispanic whites and between Hispanics and non-Hispanic whites. Lifetime discrimination directly and indirectly mediates race and gender differences in inflammation: it has a direct effect on inflammation and an indirect effect that is transmitted through waist circumference. Everyday discrimination does not affect inflammation directly; instead, it mediates race and gender differences in inflammation through its effect on waist circumference.

In this chapter, I further develop and test this model for race and gender differences in inflammation for Aim 3 and Aim 4. Aim 3 is discussed in Part 1 of this chapter; it specifically investigates the extent to which vulnerability to discrimination differs by race/ethnicity and/or gender. I hypothesize that the effect of discrimination on inflammation will be greater among racial/ethnic minorities and women relative to non-Hispanic whites and men, respectively (Hypothesis 3A). Despite these differences in magnitude, discrimination is hypothesized to be a significant predictor of inflammation within each race/ethnicity and both genders (Hypothesis 3B). Additionally, racial/ethnic an gender differences in inflammation are hypothesized to be

present across levels of exposure (Hypothesis 3C), including at minimum and maximum levels of exposure. In this section, I examine whether these patterns are specific to inflammation or generalized to intermediary outcomes in the model, specifically depressive symptoms, waist circumference, social support, purpose in life, and optimism. These analyses help to clarify the role of discrimination as an exogenous antecedent on inflammation versus the health consequences of discrimination (Aneshensel, Rutter, & Lochenbruch, 1991).

Part 2 of this chapter addresses Aim 4, which investigates the extent to which coping resources influence the effect of discrimination on inflammation and/or race and gender differences in inflammation. Stress buffering by social support is the primary moderating effect examined, but moderation by other coping resources—specifically, purpose in life and optimism—is investigated as well. Each resources is hypothesized to have a significant inverse effect on inflammation (Hypothesis 4A) and to buffer the adverse effects of discrimination on inflammation (Hypothesis 4B). The buffering effect of these resources is not expected to differ by race/ethnicity or gender (Hypothesis 4C).

Part 1: Moderation by Race/Ethnicity and Gender

7.2 Conditional Effects of Discrimination on Inflammation

Table 7.1 presents findings for the conditional effect of discrimination on inflammation. Model 1 is the main effects model. This model reproduces Model 6 of Table 6.4 and shows that there is a significant difference in levels of inflammation between African Americans and non-Hispanic whites ($b=0.127$, $SE=0.041$, $p<0.01$) and between women and men ($b=0.426$, $SE=0.027$, $p<0.001$), net of other variables in the model. Differences between Hispanics and

non-Hispanic whites are not significant. This model also shows that lifetime discrimination—but not everyday discrimination—has a significant adverse effect on inflammation (b=0.057, SE=0.016, p<0.001), such that greater exposure to discrimination is associated with higher levels of inflammation.

Models 2 and 3 are conditional models testing moderation of the effect of everyday (Model 2) and lifetime (Model 3) discrimination on inflammation by race/ethnicity (data not shown). The statistical significance of the conditional relationship is assessed first by comparing the fit of the model with the interaction terms to the same model without these terms. From the comparison of Model 1 and 2 we see that the interaction term is not significant (Wald test: F(2,62)=0.86, p=0.428) as is the comparison of Model 1 and Model 3 (Wald test: F(2,62)=0.06, p=0.938), indicating that the effects of everyday and lifetime discrimination on inflammation does not differ by race/ethnicity, respectively. The coefficients for the individual interaction terms also are not significant, therefore, Model 1, the main effects model is preferred on the basis of parsimony.

The interaction between gender and everyday discrimination is presented in Model 4 (data not shown) and it too is not significant (b=-0.017, SE=0.039, p=0.687). Since only one interaction term is added, this is a one degree of freedom test, given by the significance level of the product interaction term. The substantive results for the interaction between lifetime discrimination and gender (Model 5; data not shown) are the same. Lifetime discrimination does not have a significant differential effect on inflammation for males and females (b=0.012, SE=0.027, p=0.658).

Given the lack of statistical significance of the interaction of each form of discrimination with both race/ethnicity and gender, the preferred model based on parsimony is Model 1 in

which race/ethnicity, gender and lifetime discrimination are associated with inflammation net of the other variables in the model (Table 7.1). In this model, education is beneficially associated with inflammation such that more years of education are associated with lower levels of inflammation. Moderate alcohol consumption and vigorous or moderate physical exercise at any frequency above zero are also beneficial, relative to not drinking alcohol at all and not engaging in any form of moderate or vigorous activity. Past and current smoking behavior is associated with greater inflammation than never smoking, and a one inch increase in waist circumference is associated with a 1.07 μg/mL increase in the concentration of CRP in the blood. Finally, among coping resources, only purpose in life is associated with inflammation such that feeling as though one's life is purposeful is associated with reductions in levels of inflammation.

7.3 Conditional Effects of Discrimination on Waist Circumference

As described above, exposure to everyday and lifetime discrimination indirectly affects inflammation through their influence on waist circumference. To further explicate this connection, the conditional effects of both types of discrimination on waist circumference were tested and the findings are presented in Table 7.2. The main effects model is presented first (Model 1) and shows that discrimination is significantly and positively associated with waist circumference (everyday discrimination: $b=0.631$, $SE=0.110$, $p<0.001$; lifetime discrimination: ($b=0.209$, $SE=0.084$, $p<0.05$). Waist circumference does not differ significantly by race/ethnicity but it does differ by gender ($b=-3.476$, $SE=0.163$, $p<0.001$) with women having smaller waists.

Product interaction terms for the conditional relationship between everyday discrimination and race/ethnicity were added in Model 2 (data not shown) and were not significant (Wald test: $F(2,62)=0.92$, $p=0.403$). This suggests that the impact of everyday

discrimination on waist circumference does not differ by race/ethnicity. Similarly, the conditional effect of lifetime discrimination by race/ethnicity is not statistically significant (Model 3; data not shown) (Wald test: $F(2,62)=0.86$, $p=0.430$).

Models 4 and 5 present findings for the gender-contingent effects of discrimination on inflammation. Model 4 includes the interaction between everyday discrimination and gender (data not shown) and Model 5 includes the same interaction for lifetime discrimination. Only the interaction in the latter model (Model 5) is significant ($b=0.308$, $SE=0.132$, $p<0.05$). As can be seen from, the effect of lifetime discrimination on waist circumference is greater among women than men. The magnitude of the difference in effect is given by the coefficient for the interaction term. The coefficient for lifetime discrimination is the effect of discrimination among men. This term is not significant ($b=0.082$, $SE=0.087$, $p>0.05$), which means lifetime discrimination does not influence waist circumference among men. Among women, the effect of discrimination is calculated by summing the coefficients for the interaction term and for the lifetime discrimination term. A simple slope test of this value indicates that among women, lifetime discrimination significantly and positively affects waist circumference ($b=0.390$, $SE=0.129$, $p<0.01$).

The female dummy variable is significant and represents the difference in waist circumference between men and women when discrimination is zero—its average value because it is mean-centered—and net of other variables in the model ($b=-3.50$, $SE=0.164$, $p<0.001$). That is, women, on average, have smaller waist circumferences than men at average levels of exposure to lifetime discrimination. Across the full spectrum of exposure to lifetime discrimination, gender differences in waist circumference persist. The magnitude of this difference is greatest at lower levels of discrimination. For example, the gender difference

among individuals who have not experienced (-0.465) discrimination (b=-3.64, SE=0.176, $p<0.001$) is more than two times that of individuals with the highest (5.53) levels of exposure (b=-1.79, SE=0.747, $p<0.05$). Figure 7.1 is a graph of the interaction; it shows that the average waist circumference of women is consistently smaller than that of men but the difference in circumference diminishes as exposure to lifetime discrimination increases. This reduction in the gender gap is driven by the greater impact lifetime discrimination has among women relative to men.

Considering all of these results, the preferred model for waist circumference is Model 5 because the gender-lifetime discrimination interaction term is significant and because it is the only interaction term that is significant. Everyday discrimination has an independent and adverse effect on waist circumference in this model, where as increasing age and higher levels of education are protective against increasing waist lines, net of other variables in the model. Compared to individuals working full or part-time, retired persons and individuals engaged in other forms of employment have larger waist circumferences. Former smokers have larger waist circumferences than non-smokers, but current smokers have smaller waists than non-smokers. Alcohol consumption, vigorous physical activity and moderate physical activity reduce waist lines and seem to do so in a dose-response manner, and the average difference in waist circumference across the two years of data collection are not significantly different.

7.4 Conditional Effects of Discrimination on Depressive Symptoms

Table 7.3 presents the same series of regressions for depressive symptoms. Five regression models are examined: a main effects model for the independent effects of discrimination, race/ethnicity and gender (Model 1) and four conditional models assessing the race-contingent (Model 2 and 3) and gender-contingent (Model 4 and 5) effects of everyday and

lifetime discrimination on depressive symptoms, respectively. As shown in Model 1, reports of depressive symptoms do not differ significantly between African Americans and non-Hispanic whites. They do, however, significantly differ between Hispanics and non-Hispanic whites ($b=0.352$, $SE=0.111$, $p<0.01$), such that Hispanics report a greater number of depressive symptoms. Depressive symptoms are greater among women, on average, than among men ($b=0.15$, $SE=0.042$, $p=0.001$). Everyday discrimination ($b=0.50$, $SE=0.046$, $p<0.001$) and lifetime discrimination ($b=0.18$, $SE=0.032$, $p<0.001$) are positively associated with depressive symptoms: greater exposure to these stressors is associated with a greater number of reported symptoms.

The next two models in Table 7.3 present the conditional effects of discrimination on depressive symptoms by race/ethnicity. In Model 2 (data not shown) the conditional effects of everyday discrimination are tested and from the two interaction terms in this model we see that the impact of this type of discrimination does not differ across racial/ethnic groups (i.e., the interaction terms are not significant) (Wald test: $F(2,62)=0.39$, $p=0.679$).

Model 3 presents the conditional effects of lifetime discrimination. Unlike everyday discrimination, the interaction between lifetime discrimination and race/ethnicity is significant as determined by a Wald test for the two product interaction terms (Wald test: $F(2,62)=4.48$, $p=0.015$). Of the two interaction terms, only the interaction between lifetime discrimination and African American race is significant ($b=-0.144$, $SE=0.069$, $p<0.05$). The coefficient is negative, which indicates that the impact of discrimination on depressive symptoms is greater among non-Hispanic whites than African Americans. More specifically, the effect of lifetime discrimination is positive and significant among non-Hispanic whites ($b=0.197$, $SE=0.041$, $p<0.001$) but not significant among African Americans (simple slope test: $b=0.053$, $SE=0.062$, $p>0.05$). At

minimum (-0.465) and average (0) levels of exposures, differences in depressive symptoms between African Americans and non-Hispanic whites are not significant, but at the maximum (5.53) level of exposure they are significant (post-estimation test: b=-0.808, SE=0.352, $p<0.05$), with non-Hispanic whites having greater levels of depressive symptoms than African Americans.

For Hispanics, the impact of lifetime discrimination on depressive symptoms is significant (simple slope test: b=0.393, SE=0.095, $p<0.001$), but the magnitude of effect does not differ significantly from that among non-Hispanic whites. The differences between the two groups in reported symptoms is significant, such that Hispanics report a greater number of symptoms relative to non-Hispanic whites at minimum (-0.465) (post-estimation test: b=0.257, SE=0.098, $p<0.01$), average (0) (post-estimation test: b=0.348, SE=0.108, $p<0.01$) and maximum (5.53) levels (post-estimation test: b=1.43, SE=0.654, $p<0.05$). The impact of lifetime discrimination on depressive symptoms is also greater among Hispanics than among African Americans. Graphs of the regression lines for African Americans, Hispanics and non-Hispanic whites are presented in Figure 7.2 and illustrate the conditional effects just described.

The gender-contingent effects of discrimination on depressive symptoms are assessed in Models 4 and 5 (data not shown). Model 4 includes an interaction term for the conditional effect of everyday discrimination on depressive symptoms by gender. The interaction is not significant as determined by the coefficient for the interaction term (b=0.118, SE=0.066, p=0.080). The same substantive finding applies for lifetime discrimination (b=-0.003, SE=0.052, p=0.956). Therefore, Model 3 is the preferred model based on parsimony (Table 7.4). In this model, age, education and income are inversely and significantly associated with depressive symptoms and individuals who are not working full or part-time report more depressive symptoms than other employment statuses, as do individuals who do not have a spouse or partner. Current smokers

report more depressive symptoms than non-smokers, moderate drinkers report less depressive symptoms than non-drinkers, and individuals who engage in both forms of physical activity—at any frequency greater than zero—report less depressive symptoms than individuals who are inactive.

7.5 Conditional Effects of Discrimination on Social Support

Regression models assessing the conditional effects of discrimination on social support are presented in Table 7.4. The main effects model (Model 1) shows a significant difference in support between African Americans and non-Hispanic whites (b=0.099, SE=0.022, p<0.001) and between women and men (b=0.112, SE=0.014, p<0.001). The relationships between each form of discrimination and social support are negative and significant: more frequent exposure to everyday and lifetime discrimination is associated with lower levels of social support (everyday discrimination: b=-0.153, SE=0.010, p<0.00; lifetime discrimination: b=-0.033, SE=0.008, p<0.001). Product interaction terms between race/ethnicity and discrimination were added in Models 2 and 3 to test the conditional effect of discrimination on social support (data not shown). None of the interactions were statistically significant (Lifetime Wald test: F(2,62)=0.05, p=0.950) (Everyday Wald test: F(2,62)=0.42, 0.662).

The interaction between everyday discrimination and gender was added in Model 4, and the interaction between lifetime discrimination and gender was added in Model 5. From these models we see that the impact of *everyday* discrimination on social support does not differ by gender (b=0.014, SE=0.022, p=0.513), but the impact of *lifetime* discrimination does (b=-0.041, SE=0.020, p<0.05). In other words, the effect of lifetime discrimination on social support is contingent on gender. Figure 7.3 graphically displays this interaction. The effect of lifetime discrimination on social support among men is not significantly different from zero (i.e., no

association), but it is significant and negative among women (b=-0.06, SE=0.013, p<0.001): greater exposure to discrimination is associated with lower levels of social support among women. When exposure to lifetime discrimination is at its minimum (-0.465) (b=0.14, SE=0.018, p<0.001) and mean (0) (b=0.12, SE=0.015, p<0.001) values, average levels of social support differ significantly between women and men. At maximum exposure levels (5.53), however, the gender difference is not significant.

Model 5, the conditional model, is preferred. In addition to the gender differences discussion, the model shows that African Americans report greater levels of social support than non-Hispanic whites and Hispanics do not differ from non-Hispanic whites in their feelings of support. Everyday discrimination significantly and adversely affects support, and increasing age and years of education are associated with higher levels of support. Individuals who have never been married have significantly lower levels of support than married persons, and of the health behaviors assessed only physical activity is associated with support: engaging in any frequency (above zero) of moderate or vigorous activity is associated with greater feelings of being supported.

7.6 Conditional Effects of Discrimination on Purpose in Life

The conditional effect of discrimination on purpose in life was assessed and the findings are presented in Table 7.5. In the main effects model we see that African Americans have higher levels of purpose in life than non-Hispanic whites. Their sense of purpose in life is also higher than that of Hispanics (post-estimation test: b=0.342, SE=0.040, p<0.001). Hispanics and non-Hispanic whites do not differ significantly in their levels of purpose in life. Women report a greater sense of purpose than men. Of the two stressors, everyday discrimination is the only one

associated with purpose in life: greater exposure to this form of discrimination is associated with decreases in a sense of purpose in life.

The interaction between discrimination and purpose in life is assessed in Models 2 and 3 (data not shown). In Model 2 the conditional effect of everyday discrimination on purpose in life is not significant (Wald test: $F(2,62)=0.36$, $p=0.699$) and in Model 3 the conditional effect of lifetime discrimination also is not significant (Wald test: $F(2,62)=0.10$, $p=0.907$). Additionally, the interaction between both forms of discrimination and gender are not significant (everyday-x-female: $b=-0.035$, $SE=0.023$, $p=0.142$) (lifetime-x-female: $b=0.010$, $SE=0.020$, $p=0.597$). From these tests of conditional effects we see that the effect of everyday discrimination on purpose in life is independent of race/ethnicity and gender. Therefore the main effects model is preferred.

Model 1, the main effects model, shows that increasing age corresponds with decreasing purpose, while higher levels of socioeconomic factors (i.e., education and income) correspond with higher levels of purpose in life. Respectively, individuals who are not fully or partly employed and who do not have a spouse or partner have significantly lower feelings of purpose than individuals who are employed and individuals with a spouse or partner. The associations between health behaviors and purpose in life are mixed. Past and current smoking is inversely associated with purpose, alcohol consumption is not associated with purpose, and vigorous and moderate physical activity are positively associated with purpose. Lastly, participants from the 2008 half-sample reported having a greater sense of purpose than participants from the 2006 half-sample.

7.7 Conditional Effects of Discrimination on Optimism

The substantive findings for the conditional effects of discrimination on optimism are identical to those of purpose in life (Table 7.6). The interactions between discrimination and

race/ethnicity (everyday Wald test: F(2,62)=0.76, p=0.471) (lifetime Wald test: F(2,62)=1.70, p=0.191) and between discrimination and gender (everyday-x-female: b=-0.053, SE=0.037, p=0.152) (lifetime-x-female: b=-0.012, SE=0.026, p=0.654) are not statistically significant. Therefore, the main effects model is preferred based on parsimony.

In the main effects model (Model 1, Table 7.6), African Americans report significantly higher levels of optimism than non-Hispanic whites and Hispanics (post-estimation test: b=0.023, SE=0.084, p=0.782), and Hispanics report significantly higher levels of optimism than non-Hispanic whites. Men and women do not differ significantly in their levels of optimism. Everyday discrimination is inversely and significantly associated with optimism, while lifetime discrimination is not associated with this resource. Optimism increases with age and education but is unrelated to income and employment status. Individuals who are separated or divorced from their spouse or partner are less optimistic, on average, than married persons; the same relationship is true when comparing individuals who have never been married to individuals who are married. Smoking status is unrelated to optimism and only moderate alcohol consumption is associated with optimism. As seen for other outcomes assessed in this study, engaging in moderate or vigorous physical activity is beneficial, that is, positively and significantly associated with optimism. The two half-samples did not differ in their levels of optimism.

7.8 Summary of Key Findings: Moderation by Race/Ethnicity and Gender

This far, the results presented in this chapter show that the effects of everyday and lifetime discrimination on inflammation do not differ by race/ethnicity or gender. However, significant race- and gender-contingent effects exist for other outcomes, specifically waist circumference, depressive symptoms and social support. For waist circumference and social support there are gender-contingent effects; for depressive symptoms there are race-contingent

effects. The waist circumference of women is consistently smaller than that of men, but exposure to lifetime discrimination is a leveler that more rapidly increases the average waist circumference of women effectively reducing—but not eliminating—the gender difference. With regard to social support, women, again, experience a greater impact of discrimination compared to men. The discrimination-driven decrease in social support among women is greater than that of men. At the lowest level of exposure, women report significantly higher levels of social support than men; at the highest level of exposure, social support does not differ between women and men because emotional support among women substantially decreases to levels similar to those among men.

Race-contingent effects exist for the relationship between lifetime discrimination and depressive symptoms. The effect of this stressor is greater among non-Hispanic whites than among African Americans, but does not differ between non-Hispanic whites and Hispanics. Race differences in depressive symptoms between African Americans and non-Hispanic whites are most pronounced at highest levels of exposure, but the difference in depressive symptoms is not significant at the lowest levels of exposure.

In summary, the data show that the effect of lifetime discrimination is contingent on race/ethnicity or gender depending on the outcome of interest. Relative to men, women are more vulnerable to the effects of lifetime discrimination when the outcome of interest is waist circumference or social support, and relative to African Americans, non-Hispanic whites are more vulnerable to this stressor when the outcome is depressive symptoms. The effect of everyday discrimination does not differ by race/ethnicity or gender for any of the outcomes and, with the exception of inflammation, it is adversely related to the other outcomes. The next part of this chapter assesses stress buffering of the discrimination-inflammation relationship by each

coping resource and whether race and gender differences in inflammation are magnified or minimized, respectively, by the lack or abundance or social support, purpose in life or optimism.

Part 2: Moderation by Coping Resources

The first part of this chapter described findings from tests of the differential stress vulnerability hypothesis. This hypothesis is not support by the data for the relationship between discrimination and inflammation. Part 2 of this chapter assesses stress buffering of the effects of discrimination by social support, purpose in life and optimism. It also tests whether these resources influence the magnitude of race and gender differences in inflammation. At issue is whether the findings for social support generalize to other resources, psychological resources in particular. The main effects model for the analysis of moderation by each resource is the same as Model 1 of Table 7.1. For convenience, it is reproduced in the tables that are discussed in the remainder of the chapter. All conditional models include mean-centered variables for the coping resources and discrimination.

7.9 Moderation by Social Support

Table 7.7 presents results for the moderating effects of social support. Model 1 is the main effects model. In this model we see that lifetime discrimination has a significant effect on inflammation ($b=0.057$, $SE=0.016$, $p<0.01$), but everyday discrimination and social support do not, net of other variables in the model. Significant differences in inflammation between African Americans and non-Hispanic whites ($b=0.12$, $SE=0.041$, $p<0.01$) and between women and men ($b=0.43$, $SE=0.027$, $p<0.001$) exist, as we have seen in previous analyses. In Models 2 and 3 product interaction terms were added for the interaction between support and everyday

discrimination (Model 2) and support and lifetime discrimination (Model 3) (data not shown). Neither interaction term is significant (everyday-x-support: b=-0.021, SE=0.25, p=0.403) (lifetime-x-support: b=-0.002, SE=0.015, p=0.870). Thus, the effect of discrimination on inflammation does not vary by emotional social support. The next two models examine whether race and gender differences in inflammation vary by levels of support. Model 4 includes two product interaction terms to operationalize the conditional effect of social support on inflammation by race/ethnicity. The interactions are not significant (Wald test: F(2,62)=0.40, p=0.675) suggesting that the race difference in inflammation does not vary by level of social support. The interaction between gender and social support is not statistically significant either (female-x-support: b=0.069, SE=0.053, p=0.194). Thus, the preferred model is Model 1, which is presented in Table 7.7.

7.10 Moderation by Purpose in Life

The next set of analyses examines moderation by a sense of purpose in life and the findings are presented in Table 7.8. The hypothesis tested is whether a strong (i.e., higher) sense of purpose in life mitigates the adverse effect of discrimination on inflammation. Purpose in life is a significant predictor of inflammation (Model 1) but, like social support, it does not significantly moderate the effects of everyday (everyday-x-purpose: b=-0.009, SE=0.020, p=0.666) and lifetime (lifetime-x-purpose: b=-0.002, SE=0.015, p=0.870) discrimination on inflammation (Model 2 and Model 3, respectively, data not shown). Model 4 includes product interaction terms for the interaction of race/ethnicity and purpose in life, which tests whether race differences in inflammation are contingent on how strongly people feel their lives have purpose. This interaction is not statistically significant (Wald test: F(2,62)=1.34, p=0.269; data not shown).

Findings for the interaction between gender and purpose in life are presented in Model 5 of Table 7.8. In this model, the interaction is statistically significant, which means gender differences in inflammation vary by purpose in life. The coefficient for the purpose in life variable is negative and significant (b=-0.079, SE=0.018, p<0.001) and represents its effect on inflammation among men. The effect among women is not significant, as determined by simple slope test (b=-0.0004, SE=0.023, p=0.987). Thus, purpose in life is beneficial among men but not among women with regard to inflammation. Figure 7.4 is a graph of the interaction. The graph shows that the women have higher levels of inflammation than men and that the difference between the two groups widens as purpose in life increases. The gender difference at minimum (-3.58) levels of purpose is not significant (b=0.142, SE=0.099, p=0.158), but the difference at maximum (1.42) levels of purpose is significant (b=0.535, SE=0.040, p<0.001).

7.11 Moderation by Optimism

The final coping resource tested is optimism. Table 7.7 presents the findings for this analysis. Model 1 shows that optimism is not significantly associated with inflammation. The interaction between optimism and everyday discrimination (Model 2) is not significant (everyday-x-optimism: b=-0.010, SE=0.012, p=0.411; data not shown), neither is its interaction with lifetime discrimination (Model 3; lifetime-x-optimism: b=-0.003, SE=0.014, p=0.821; data not shown). In Model 4 (data not shown) the conditional effect of optimism on race differences in inflammation is tested with two product interaction terms. A Wald test assessing the significance of the entire interaction—i.e., the significance of both the interaction term for African American race and optimism and the interaction term for Hispanic ethnicity and optimism—is not significant (Wald test: F(2,62)=2.06, p=0.136). Similarly, Model 5 shows that the interaction between female gender and optimism is not significant (b=0.007, SE=0.021,

p=0.741). Collectively, these findings indicate a preference for the main effects model (Model 1).

7.12 Summary of Key Findings: Moderation by Coping Resources

The final part of this chapter assessed buffering of the discrimination-inflammation relationship by social support, purpose in life and optimism. Only purpose in life has a significant independent effect on inflammation, but none of the resources buffer the effect of everyday or lifetime discrimination on inflammation. Race differences in inflammation are independent of the level of any of the resources evaluated. Gender differences in inflammation do not vary by social support or optimism, but they do vary by purpose in life: when purpose in life is low, gender differences in inflammation are not significant; when purpose in life is high, gender differences in inflammation are significant. This change in significance and increase in the gender difference is a consequence of the beneficial effect purpose in life has on inflammation among men. It does not have a similar effect among women, in fact, there is no association between purpose in life and inflammation among women. Thus, purpose in life is a protective psychological resource among men, with regard to inflammation, but not among women.

CHAPTER 8

DISCUSSION

8.1 Introduction

The overarching goal of this dissertation was to describe racial/ethnic and gender differences in systemic inflammation, and to ascertain the extent to which these differences can be attributed to discrimination, and whether psychosocial resources mediate and/or moderate these differences. Systemic inflammation is associated with cardiovascular diseases, such as atherosclerosis and coronary heart disease (Danesh et al., 2004; Ridker, 2003; Ridker et al., 2000). Identifying differences in inflammation by race/ethnicity and gender, and elaborating the pathways that explain or influence these differences may further our understanding of how health disparities develop and persist over time. In this final chapter of the dissertation, the major findings are summarized and discussed, followed by an overview of the study's strengths and limitations. The chapter ends with a statement of conclusions, discussion of the public health implications of the study findings, and recommendations for directions for future research.

8.2 Summary of Major Findings

Using data from the 2006 and 2008 biomarker assessments and enhanced face-to-face interviews of the Health and Retirement Study (HRS), this dissertation addressed four aims: (1) to determine the extent to which systemic inflammation varies by race/ethnicity and/or gender, (2) to establish whether and in what manner differential exposure to discrimination accounts for differences in inflammation across races/ethnicities and between genders, (3) to assess the extent to which members of certain races/ethnicities and/or genders are differentially vulnerable to discrimination, and (4) to investigate the extent to which social and psychological resources

buffer the effects of discrimination on inflammation and do so differently by race/ethnicity or by gender.

As a review, the study sample consisted of 11,935 adults aged 52 to 101 years. More than three-fourths of the sample was non-Hispanic white and approximately 60% were women. C-reactive protein (CRP), the measure of systemic inflammation used in this dissertation, was measured from dried blood samples collected during a clinical assessment. Log-transformed values of this biomarker were used throughout the analysis. Race/ethnicity and gender were self-reported, and measures of discrimination and of social support, purpose in life and optimism—the resources evaluated in this study—were assessed using items from a self-administered questionnaire. To assess the relationships among study variables, structural equation modeling (SEM) was used and multivariate regression models adjusted for demographic characteristics, socioeconomic factors and health behaviors. In this section, I highlight the major findings for each aim of the dissertation and in the subsequent sections I place them within the context of current knowledge about the relationships among these factors.

Key Findings of Specific Aim 1. African Americans and women have the highest levels of inflammation. African Americans have significantly higher levels of inflammation than non-Hispanic whites and Hispanics, net of demographic characteristics and socioeconomic factors, health behaviors, and health status. Although Hispanics have significantly higher levels of inflammation, on average, than non-Hispanic whites, this differences is fully accounted for by differences in socioeconomic status, with education emerging as the key factor. Women have higher levels of inflammation than men, on average, and this difference increases with the addition of control variables. These results support Hypothesis 1A, with the exception of the non-significant difference between Hispanics and non-Hispanic whites.

Key Findings of Specific Aim 2. Greater exposure to discrimination accounts in part for racial and gender differences in inflammation. African Americans report more frequent exposure to everyday and lifetime discrimination than non-Hispanic whites and Hispanics, which supports Hypothesis 2A, but these reports do not differ significantly between Hispanics and non-Hispanic whites, which is counter to this hypothesis. Moreover, men reported more frequent exposure to both forms of discrimination than women, which also is opposite to this hypothesis. Both everyday and major lifetime discrimination are associated with greater inflammation, but only lifetime remains significant when considered in conjunction with everyday discrimination and net of relevant control variables (H2B). Racial and gender differences in inflammation are partially mediated by discrimination, as a simple pathway or as a compound pathway involving multiple mediators (i.e., through both discrimination and waist circumference) (H2C). Differences between Hispanics and non-Hispanic whites are mediated by socioeconomic factors, specifically differences in educational attainment.

Key Findings of Specific Aim 3. There is no evidence to support the differential vulnerability hypothesis for the effects of discrimination on inflammation. The differential vulnerability hypothesis was tested with interaction terms that permit the slope for discrimination to vary by race/ethnicity and by gender. This hypothesis was assessed for inflammation, the focal dependent variable, and for: waist circumference, depressive symptoms, socials support, purpose in life, and optimism. For inflammation, none of the coefficients for the product interaction terms are statistically significant. Thus, the effects of discrimination on inflammation do not seem to differ by race/ethnicity or by gender. The findings for the other outcomes, however, are mixed. With increasing levels of exposure, waist circumferences among women increase more rapidly, compared to men, and social support decreases more quickly.

Additionally, the effect of discrimination on depressive symptoms is greatest among Hispanics and non-Hispanic whites; the mental health of African Americans, with regard to depressive symptoms, is not significantly affected by discrimination. Everyday discrimination, but not lifetime discrimination, affects purpose in life and optimism and the effects are independent of race/ethnicity and gender. In all, discrimination is shown to have an effect on multiple physical, mental and emotional outcomes.

Key Findings of Specific Aim 4. There is no evidence to support the buffering role of social support for the effects of discrimination on inflammation. Other coping resources also do not perform this function, with the sole exception of Purpose in Life. Social support does not buffer the effect of discrimination on inflammation, neither do purpose in life or optimism. Purpose in life, however, does influence gender differences in inflammation. It functions as a protective factor among men, such that a stronger belief in the purpose of one's life is associated with a decrease in inflammation. Among women, purpose in life has no effect: in other words, inflammation among women remains steady as purpose in life increases (holding all other factors constant), while inflammation among men decreases, which results in the widening of the gender difference in inflammation.

8.3 Social Patterning of Inflammation by Race/Ethnicity and Gender (Aim 1)

The racial/ethnic patterning of inflammation found in this study aligns with disparities in cardiovascular diseases (Rogers et al., 2011) and past research investigating race differences in inflammation (Herd, Karraker & Friedman, 2012; Khera et al., 2005). African Americans consistently have higher levels of inflammation than other groups even after accounting for other factors that elevate inflammation and increase risk for poor health. The elimination of the

significant difference in inflammation between Hispanics and non-Hispanic whites after controlling for education is consistent with other studies (Crimmins et al., 2007).

These findings are more than a confirmation of previous research demonstrating the social patterning of diseases and related biomarkers. The current study moves the field forward by substantiating past findings *and* identifying mechanisms that contribute to the patterning of inflammation by race/ethnicity and gender. Moreover, it gives further credence to the contributions of the social environment, in general, and experiences of discrimination, in particular, to poor health and health disparities, and further challenges claims that racial and gender differences in health are primarily driven by genetic or biological differences. A discussion of the findings for gender differences in inflammation is an ideal start point for elaboration.

The literature has shown that a portion of the gender difference in inflammation is based on gender differences in physiology. Although complete details about the physiological process are unclear, women have higher cytokine levels than men (Schuurs, 1990) because female sex hormones (e.g., estrogen) tend to heighten the production of cytokines (Verthelyi & Klinman, 2000; Verthelyi, 2001; Chao 1995; Klein 2000). Additionally, waist circumference is a measure of body mass composition that is positively associated with adiposity (i.e., quantity of adipose cells in the body)., Adipose cells are a primary source of inflammatory markers (Berg & Scherer, 2005), which means adiposity is positively correlated with inflammation. Given that men tend to have larger waist circumferences than women, the increase in the gender difference in inflammation after accounting for waist circumference is understandable and was supported by analyses conducted for Aim 2.

However, this study has shown that physiology does not account for the social patterning of gender difference in inflammation. First, adjustments for demographic characteristics, socioeconomic factors, health behaviors, and health status increased the magnitude of the gender difference to nearly two times the estimate derived from unadjusted models. This change means that, relative to men, the inflammation levels of women would be even greater if women were comparable to men in these characteristics. What, then, is driving the gender difference in inflammation? Investigating systematic differences in the social experiences of men and women—including differences in the experience of social stressors like discrimination—is one means of getting to the bottom of this question.

8.4 Discrimination as a Mediator of Racial and Gender Differences in Inflammation (Aim 2)

Although African Americans have greater exposure to discrimination than non-Hispanic whites, the relatively low rates of exposure among Hispanics and among women were unexpected, in large part because discrimination is disproportionately directed at minorities and women in general. To understand this finding, it is important first to bear in mind several features of this study: the age of the sample, over 51 years, and the cohorts it represents: the AHEAD cohort born prior to 1924 through the early baby boomers, born between 1948 to 1953. Given the age distribution, a large portion of the sample is no longer working, and given the cohorts, a substantial portion of women may not have participated extensively in the labor force. These are important considerations because half of the items used to assess major lifetime discrimination—the stressor associated with inflammation—pertain to employment: being unfairly fired from a job, being unfairly denied a job, and being unfairly denied a promotion.

A related point is the type of discrimination most frequently reported, which is age discrimination. Unlike many other characteristics that are the target of discriminatory acts, the categories of age that elicit discrimination are passed through the transition to adulthood or arrived at relatively late in life. Research by Gee, Spencer, Chen, & Takeuchi (2007) has shown that age, rather than cohort, influences the distribution of age discrimination: around the time of emerging adulthood (i.e., late teens to early twenties) age discrimination is relatively high; then, around age 30 years, it drops to its lowest levels, but peaks again during midlife with in 5 to 10 years of legal retirement age depending on cohort. As such, age discrimination at old age is likely to be the first form of discrimination encountered by some respondents, and therefore may be especially salient to some groups, including non-Hispanic white men, which may contribute to the gender imbalance in experiences of discrimination.

A final consideration for understanding the patterns in reporting of discrimination is that these are measures of perceived discrimination. It is possible that Hispanics and women are exposed to a higher level of discrimination than they report because they do not perceive the acts as discriminatory. The events on the everyday discrimination refer to ambiguous occurrences that could be interpreted as discriminatory or discounted as being something else. Whether this occurs disproportionately for Hispanics and women cannot be sorted out with the available data, but some literature suggests that among Hispanic population with a substantial proportion of foreign-born individuals, socioeconomic and cultural factors may influence their perception and reporting of discriminatory experiences. For example, Perez et al. (2008) found that reports of discrimination among Hispanic population varied by education and ethnic identity, among other socioeconomic and cultural characteristics. With that said, the fewer experiences of discrimination among Hispanics may also be accurate reports of lower than expected levels of

exposure and the lower levels are simply a reflection of the social and cultural geography of where Hispanics live, work and play. Hispanics tend to live in "ethnic enclaves" where the majority of residents are also Hispanic and potentially immigrated to the U.S. from the same regions of their home country. This situation may be beneficial for mitigating the stress of living in a new country and adapting to a new culture; it potentially shields them from exposures because they have less contact with individuals and institutions that engage in discriminatory behaviors and practices. As a result, these ethnically homogeneous communities are sources of protection against being labeled "foreign" or "other" and the disparaging actions these labels can brings upon them.

In interpreting the finding that in models including both forms of discrimination, lifetime discrimination, but not everyday, is related to inflammation, it is important to realize that these are not independent occurrences: the correlation between these two forms of discrimination is $r = 0.29$ ($p < 0.001$). This is contrary to findings pertaining to mental health outcomes, for which everyday discrimination has the greater effect (Kessler, Mickelson & Williams, 1999). Many studies, however, examine only everyday discrimination, based on this earlier research. However, that approach is flawed if both forms are associated with the outcome and with each other, producing "omitted variable bias" and endogeneity. Since these conditions are met in these data, the model with both forms of discrimination is considered the "correct" model. It is important to note that the interaction between lifetime and everyday discrimination on inflammation was not significant, which means the effects of lifetime discrimination on inflammation is not contingent on exposure to everyday discrimination, and vice versa.

Lifetime may be more consequential to this older population than in other more age heterogeneous samples to the extent that it pertains in large part to age discrimination in

employment-related areas of life. Therefore, its occurrence may be recent and hence consequential. Also, the implications of age discrimination in employment settings may be more apparent as people anticipate or enter retirement and assess their financial situations. Furthermore, the nature of discrimination changes over the life course (Gee, Walsemann & Brondolo, 2012) such that major forms of discrimination may be more relevant among older adults than chronic, but less grave, occurrences. The experience of major discrimination may be more of a "shock to the system" for older adults, especially for individuals belonging to social groups that were only minimally exposed to this stressor during earlier stages of in the life course (e.g., non-Hispanic white males). Additionally, older racial and ethnic minorities who faced chronic forms of discrimination early in life may have developed effective coping strategies over time that allow them to adapt to these exposure. As a result they are less reactive to the slights, insults and disrespect that are characteristic of everyday discrimination, but continue to react to lifetime discrimination because of its heterogeneous effects on fiscal, social, physical and psychological well being.

The mediation analysis shows that accounting for the effect of lifetime discrimination on inflammation attenuated differences between African Americans and non-Hispanic whites, but augmented differences between men and women. In other words, the female excess of inflammation increases when discrimination is taken into consideration. This means that women would be at an even greater disadvantage in terms of inflammation and relative to men if it were not for the fact that these women report experiencing less discrimination than men. These results provided some support for Hypothesis 2C insofar as differences in inflammation between African Americans and non-Hispanic whites and between women and men are mediated by lifetime discrimination alone or lifetime and everyday discrimination in conjunction with waist

circumference. For gender, mediation also occurred through purpose in life: women reported fewer experiences of everyday discrimination, everyday discrimination depleted feelings of having a purpose in life, and purpose in life is protective against increasing inflammation. Therefore, in this pathway, the fact that women report experiencing less discrimination than men tends to offset the higher overall levels of inflammation among women. In this way, discrimination independently and in conjunction with other factors adversely affects health by sustaining the process of inflammation and does so differentially based on race/ethnicity and on gender.

Collectively, these findings align with earlier work by Kessler, Mickelson & Williams (1997) as well as more recent studies of other health disparities, although the literature on mediation is not entirely consistent. For example, research by Fuller-Rowell et al. (2012) found that social-class discrimination explained a portion of the association between poverty and allostatic load, a cumulative measure of health status. However, Luo et al. (2012) and Herd et al. (2012) did not find support for the role of discrimination as a mediator of social status differences in health. Rather, among older adults, social status and discrimination independently predicted changes in health over a two-year period. The mixed findings from this body of work speak to the need for more studies of the pathways explaining status-based differences in health.

8.5 Vulnerability to Discrimination is Consistent Across Race/Ethnicity and Gender for Inflammation (Aim 3)

As stated previously, the findings from Aim 2 support the differential exposure to stress hypothesis and contribute to our understanding of racial/ethnic and gender differences in inflammation. An alternate hypothesis for these differences—differential vulnerability to stress—was tested in Aim 3. At the heart of this hypothesis is the argument that when faced with

the same stressor, members of certain social groups will be more reactive to the stressor than members of other social groups. These social groups are typically defined by demographic characteristics or socioeconomic status and differences across these groups in reactivity to a stressor may be due to the nature of the stressor or to the behavioral, emotional and cognitive responses or strategies used to cope with the stressor.

In this study, the strength of the association between discrimination and inflammation was hypothesized to be greater among racial/ethnic minorities and women (Hypothesis 3A). Given the enduring legacies of social movements for women's rights and for civil rights for African Americans and other racial minorities and women, the qualitative experience of discrimination is different across race/ethnicity and gender; it is potentially more severe (i.e., perceived to be more threatening) among these marginalized social groups, which leads to a heightened stress response. Alternatively, differences in vulnerability may be a reflection of differences in coping strategies used across social groups. Some coping strategies attenuate the effects of discrimination on health outcomes and others exaggerate the effects. If the utilization of specific coping strategies varies across groups, then differences by race/ethnicity in coping strategies, for example, could explain differences in the impact of a stressor on health.

The data in this study did not support the differential vulnerability hypothesis. Therefore, the explanations discussed might not applicable to this population. Given the age range of the members of the sample (52 − 101 years) the latter of the two explanations (i.e., differences in coping strategies) seems more relevant. This is an older sample of the population that has faced many stressors over the life course, including discrimination and other adversities. Each exposure leads to the mobilization of available coping resources and the development of effective (and potentially ineffective) strategies for coping with discrimination and other

stressors. In a sense, over time, racial/ethnic minorities and women in this population have learned how to effectively adapt to the stress of discrimination. Unfortunately, at this older age the damaging health effects of discrimination have already been realized, which is reflected in the higher levels of inflammation among these groups, relative to non-Hispanic whites and men, respectively.

8.6 The Effect of Discrimination on Inflammation is not Buffered by Social Support (Aim 4)

The stress-buffering function of social support was not substantiated for the effect of discrimination on inflammation. Other coping resources, specifically a sense of purpose in life and optimism, also failed to moderate the discrimination-inflammation relationship, which means the lack of stress buffering is not specific to support. Although stress theory posits a buffering function for resources, empirical evidence is mixed regarding the role social support plays in relation to the effects of discrimination in existing research.

For example, a study by Prelow et al. (2006) found evidence for a support deterioration model (i.e. social support is depleted by exposure to discrimination) for the relationship among discrimination, emotional social support, depressive symptoms, and life satisfaction; they did not find evidence for a support mobilization model or for a buffering effect of social support on these outcomes. Additionally, in a population of Filipino Americans living in San Francisco or Honolulu, conditional relationships between instrumental support and discrimination and between emotional support and discrimination were not significant (Gee et al., 2006). However, the third-order conditional relationship between instrumental support, discrimination and city was significant: among Filipino Americans living in San Francisco, reports of discrimination were not associated with health conditions at any level of instrumental support; among Filipino

Americans living in Honolulu, reports of discrimination were associated with health conditions and the strength of the association was greater among individuals with low levels of instrument support relative to those with high levels of instrumental support.

Taken together, the role of social support—and other coping resource—appears to vary depending on the outcome under study, the population, the geographic context, or potentially numerous other factors. Thus investigating the competing functions of coping resources, as was done in this dissertation, is advantageous. The findings of the current study are more consistent with a resource depletion model. Everyday discrimination had significant and adverse relationships with each of the coping resources, while lifetime discrimination had an adverse effect on social support alone. Only purpose in life was associated with inflammation, which suggests that the effect of everyday discrimination on inflammation is mediated by the depletion of a sense of purpose in life. Considering that African Americans report greater levels of exposure to everyday discrimination, the subsequent effects of this stressor on their sense of purpose in life is likely a driving factor behind race differences in inflammation. This interpretation is substantiated by the mediation results.

8.7 Other Influences on Inflammation

While the primary goal of this study is advancing our understanding of racial/ethnic and gender difference in inflammation, this is in the service of a more complete understanding of inflammation, which directs attention to other variables in the model beyond the core ones just discussed. Education stands out as a protective factor against inflammation, as does moderate alcohol consumption and vigorous and moderate physical activity. As expected, smoking has an adverse impact on inflammation, as do larger waist circumferences and greater depressive symptoms.

One variable in particular is noteworthy because the sign of its association with inflammation is the opposite of what is expected based on existing research: age. Whereas other studies have found a positive association between age and inflammation, in this study the zero-order association is not statistically significant but the association becomes significantly negative when covariates are added to the model. These findings contradict the idea of "inflammaging" (Franceschi, 2007; Franceschi et al., 2000), which refers to the gradual increase of inflammation overtime (i.e., with increasing age), and with empirical studies demonstrating the positive association between age and inflammation (Finch, 2010; Barlett et al., 2012). What then explains the paradoxical findings in the dissertation?

To begin, the sample has a restricted age range beginning in late middle age and extending through the oldest old; early adulthood through most of middle age is not represented in the sample. The truncated age span may account for differences from previous studies, especially if there is a positive association in the early and middle part of adulthood and a negative one at more advanced ages. Additional analyses (not shown) are suggestive about the role of aging in the later years. Second, when waist circumference is taken into consideration, the direct effect of age was no longer significant), which suggests complete mediation: As people age, they become more frail, which can manifest as a decrease in waist circumference and a corresponding decrease in adiposity. Decreased adiposity means there are fewer adipose cells producing inflammatory markers (although other cell types may continue to do so). Although this interpretation is speculative, age-related changes in SES and in anthropometric measures and physiology warrant further investigation.

8.8 Limitations of the Study

All research endeavors have limitations. As it relates to the current study, interpretation of the findings may call for caution due to specific characteristics of the data, the analytic sample and the data analysis. First, the data are cross-sectional, which preclude unequivocal statements about causality because the temporal ordering of constructs cannot be established. That said, an advantage of researching predictors of a biological marker, such as CRP, is that doubts regarding temporal relationships are partially alleviated. Current knowledge about the biological processes influencing levels of CRP and other inflammatory markers suggests that these biomarkers are predominately influenced by external inputs from the environment. Therefore, it is reasonable to assume that stress exposure predates changes in inflammation. Feedback loops, however, do exist and inflammatory processes can influence cognitive responses and behaviors (Irwin & Cole, 2011) that heighten or diminish stress appraisal, or place individuals in physical and social environments that increase or diminish stress exposure. Additionally, the directional relationships between inflammatory markers and other indicators of physical and mental health, such as depressive symptoms, have yet to be determined definitely, and are quite likely reciprocal. Nonetheless, this cross-sectional analysis of the relationships among CRP and the other study variables is advantageous—especially in an area of public health research that is still emerging—because it helps establish consistency, another criteria for asserting a causal relationships (Hill, 1965).

Using structural equation modeling (SEM) for this research was analytically appropriate and rigorous. This statistical procedure, however, involves a degree of subjectivity in the model building process; the researcher explicitly states the directionality and type of relationships (e.g., covariance versus path) among variables and error terms. When used as a confirmatory process, as is the case in this study, the specified relationships are guided by both theory and the data:

theory provides the foundation for building the model and the data either confirms of refutes the hypothesized relationships. Through the calculation of various indices of model fit, information is provided about possible modifications to the model that potentially improve agreement between what the theory hypothesizes and what the data shows. This iterative process informed the development of the final model of the dissertation (presented in Chapter 6), a model closely aligned with the stated theoretical framework and conceptual model and supported by the data (i.e., acceptable fit indices).

Nevertheless, some researchers might find fault with certain aspects of the model. For example, paths between discrimination and socioeconomic factors were excluded, but correlations between their error terms were included. Additionally, health behaviors were operationalized as exogenous variables when they could also function as endogenous variables that mediate differences in inflammation by race/ethnicity and gender, or mediate the effect of discrimination on inflammation. However, to account for these associations covariances between all exogenous variables—including the health behaviors—were included in the final model. Substituting directed paths for the correlations and covariances mentioned could alter the substantive findings, which is an unknown that can be addressed in future research. Nevertheless, in the end, the construction of the model is always open to debate and the fact that the model is consistent with the data does not preclude the very real possibility that other models also are consistent with the data.

Another limitation of the research that should be mentioned is the increased probability of committing a Type I error when conducting the moderation analysis. For the test of differential vulnerability to discrimination by race/ethnicity and gender (Aim 3), conditional relationships with multiple outcomes were tested: inflammation, waist circumference, depressive

symptoms, social support, purpose in life, and optimism. Also, three coping resources—social support, purpose in life, optimism—were hypothesized to condition racial/ethnic and gender differences in inflammation and to buffer the effect of discrimination on inflammation (Aim 4). Thus, multiple conditional relationships were tested which increases the probability of rejecting the null hypothesis when it is in fact true. Therefore, the findings of the moderation analysis should be interpreted with some caution.

Finally, as operationalized in this study, experiences of discrimination are based on appraisal processes or perceptions of a situation or social interaction. This aspect of the nature of discrimination leads to questions of objectivity—especially given the fact that modern forms of discrimination are more covert and ambiguous than at other times in recent history—but does not refute its relevance or validity: a measurable stress response is exhibited at times of exposure (Harrell, Hall, & Taliaferro, 2003). Moreover, the instruments used to measure discrimination in this study are common to the literature (Clark, Coleman, & Novak, 2004; Kressin, Raymond & Manze, 2008), are psychometrically grounded in qualitative and quantitative research (Shariff-Marco et al., 2011) and have shown with great consistency that exposure to discrimination is detrimental for physical and mental health (Williams & Mohammed, 2009), across races and ethnicities (Ryan, Gee, & Laflamme, 2006) and for men and women (Sims et al., 2012). Therefore, doubts about the relevance and utility of these measures should be assuaged.

8.9 Strengths of the Study

Despite the stated limitations, there are several noteworthy methodological and conceptual strengths of the research. From a methodological perspective, one strength of this research is engrained in the study design of the HRS. The HRS is a large, nationally representative probability sample of the United States. Sample members were selected with a

probability proportionate to the size of the sampling unit (i.e., metropolitan statistical areas, area segments) and weights were constructed to account for the differential probability of selection. Therefore, applying the sampling weights to the analysis yields findings that are generalizable to the larger population. Additionally, the large sample size of the HRS and the oversample of African Americans and Hispanics are advantageous for assessing variations in relationships between subgroups of the population.

Another methodological strength of the research is its formal test of mediation. When following the elaboration model, third variables are systemically added to regression models and their substantive role in the three variable model is inferred from their effect on the focal relationship (i.e., changes in the magnitude or significance of the regression coefficient for the independent variable). However, while this decrease is consistent with mediation it does not demonstrate that mediation has indeed occurred, a conclusion that is better drawn on the basis of an explicit test of the mediated or indirect effect. In addition, since the role of a third variable is defined theoretically, a decrease in race differences in inflammation after accounting for education, for example, may suggest that the focal relationship was confounded by or redundant with education, versus being mediated by education. The choice in interpretation should be informed by theory, but researchers within and across disciplines incorporate theory into their research to varying degrees and possess their own proclivities for the interpretation of changes in effect. Therefore, empirical tests that clarify the role of third variables—such as the mediation analysis done in this dissertation—are necessary for more precisely illuminating the relationship among variables.

From a conceptual standpoint, clarifying the role of third variables may be especially important for health disparities research where, to continue our example, the absence of a race

difference in inflammation after accounting for education may result in the interpretation of the focal relationship as a spurious one—i.e., race differences in health are really socioeconomic differences in health—or as a completely mediated relationship—i.e., race differences in inflammation are caused by parallel differences in education. The latter interpretation offers valuable insight about the drivers of racial health disparities and potential points of interventions, while the former can give rise to arguments for less attention to racial health disparities and more focus on socioeconomic health disparities. Race and socioeconomic status are correlated, however, but solely focusing on socioeconomic health disparities may obscure investigation into alternate drivers of racial health disparities, such as the disproportionate exposure to discrimination among some minority populations. As this study has shown, race/ethnicity and socioeconomic status are more than just variables to control for; rather, they are intimately related such that racial health disparities are fueled by differences in education and income.

A final and central strength of this study is its reliance on a theoretical framework integrating three related perspectives on stress spanning more than 80 years of research in the behavioral and population sciences. Each of the theoretical models used in this dissertation—the Stress Process Model (Pearlin et al., 1981), the Transactional Model of Stress and Coping (Lazarus & Folkman, 1984), and the Psychobiology of Stress model (Kemeny, 2003b)—offer theoretical guidance and support for different aspects of the dissertation. From the Stress Process Model comes guidance on the etiology of group differences in health through the social distribution of stressors and coping resources; from the Transactional Model of Stress and Coping comes guidance on how appraisal processes and coping responses influence whether or not stressors have an effect and the psychological magnitude of their effect; and the Psychobiology of Stress model connects the perception of stress to physiological systems that are

implicated in the development and progression of chronic diseases. In addition to establishing a solid foundation for conceptualizing and implementing the research, the integration of these theoretical models provided a more complete picture than any single theory of how the social environment influences health on a biological level. Such a foundation is necessary for interpreting the study findings and understanding the public health implications of the work.

8.10 Public Health Implications and Future Directions

A defining characteristic of the field of public health is its concern with systematic and avoidable differences in population health, or health disparities (Link & Phelan, 1995). This dissertation specifically speaks to this matter by looking at group differences in stress exposure and vulnerability that potentially lead to group differences in biological risk for poor health. By looking at differences in systemic inflammation by race/ethnicity and by gender, we achieve a greater understanding of parallel differences in cardiovascular diseases and other chronic conditions associated with deviations in immune functioning. Furthermore, by examining the biological effects of exposure to discrimination, greater relevance is given to its role as a social stressor. Public health researchers and other social scientists have identified stigma—and the discrimination that often follows it—as a "fundamental cause of health disparities" (Hatzenbuehler, Phelan, & Link, 2013; Link & Phelan, 2001; Thoits, 2011). Researchers and practitioners outside the social sciences may not fully understand or recognize the formidable role the stress of discrimination plays in health, above and beyond its part in the distribution of material and fiscal resources. This dissertation works against this oversight and sheds light on the deleterious contributions of discrimination to poor health.

Furthermore, elaborating the pathways linking discrimination to adverse health outcomes, such as high levels of systemic inflammation, helps inform potential interventions, interventions

that emphasize changes to our social environment in addition to changes in traditional behavioral risk factors for poor health. From an environmental perspective, efforts to minimize the occurrence of discrimination are important objectives. Anti-discrimination laws, cultural competency and sensitivity initiatives, and social movements fighting for equality for all are illustrations of social movements and macro-level interventions aimed at normalizing the acceptance of diversity and making manifestations of discrimination a thing of the past. Effective and sustained social change, however, takes all too long to achieve and during the intermediate time frame individuals disproportionately exposed to discrimination cannot be left alone to suffer its health damaging consequences. Thus, interventions at other levels, including the individual level, are also needed.

On an individual level, the findings of this dissertation point to interventions that address differences in socioeconomic status, and interventions that identify and bolster coping resources that mitigate the impact of social stressors on health. In addition to discrimination, differences in education were a key driver of differences in inflammation across races/ethnicities. Therefore, early and equal access to *quality* education regardless of race/ethnicity would help minimize disparities in health seen later in life. Improving access to quality education, however, is not enough. This study showed that differences in exposure to discrimination mediates race and gender differences in inflammation and does so independently and in conjunction with a depletion of a sense of purpose in life. It also showed that social support, purpose in life and optimism were not effective buffers of the impact of discrimination. Therefore, mitigating the adverse effects of discrimination on inflammation would require fostering and sustaining a sense of purpose in life, even in the face of adversity, and identifying other resources that function as effective buffers of this stressor is also necessary.

Secondary interventions that seek to rein in systemic inflammation (resulting from stress exposure) may also come into play. These interventions are not specific to discrimination, per say, but they may be beneficial nonetheless. Beyond pharmacological methods of reducing inflammation, cognitive and behavioral techniques should be identified. Yoga and meditation programs have been shown to be effective at suppressing the inflammatory stress response (Kiecolt-Glaser et al., 2010) and attenuate conventional risk factors for cardiovascular diseases, such as high blood pressure (Schneider, Alexander, Salerno, Rainforth, & Nidich, 2005). Although currently these interventions are not considered "mainstream", they are promising especially in combination with the environmental and resource-based interventions highlighted previously. In the end, any intervention that improves the health of the populations affected by discrimination is an important step towards addressing disparities in health.

As health disparities research moves forward, future investigations should seek to assess the extent to which the findings from the current study generalize to other and more diverse samples. Replication studies would be beneficial for ascertaining the degree of consistency in the associations among race/ethnicity, gender, discrimination, coping resources and inflammation. These studies should include better representation of racial/ethnic groups that were excluded from the current study due to their small sample size (e.g., Asian Americans). Separate analyses within racial/ethnic groups would also be advantageous, and would help highlight the collective experiences of these groups while detailing variations among its members. Another reasonable extension of this study would be to refine the resulting model, while also extending some of the pathways tested. The coping resources investigated—social support, purpose in life and optimism—are general, nonspecific resources that are considered useful for protecting against various stress exposures. Buffering the stress of discrimination,

however, may require a distinct set of resources that are specific to this stressor. Additionally, subsequent research could elaborate the current model by (1) investigating stress proliferation due to exposure to discrimination, and (2) including multiple indicators of inflammation and deficiencies in immune function. Finally, longitudinal studies testing how this model unfolds over time would be a reasonable next step. Cohort studies simultaneously assessing trends in stress exposure and trends in inflammation would be informative for understanding the extent with which these trajectories are correlated with each other and predictive of clinical health outcomes and mortality.

8.11 Conclusion

The overall goal of this dissertation was to identify and explain systematic differences in inflammation across race/ethnicity and between genders. This was accomplished by investigating the extent to which exposure to discrimination mediates these differences, the presence of differences in vulnerability by these demographic characteristics, and the buffering effect of coping resources. The study found that exposure to discrimination is a significant mediator of race and gender differences in inflammation, such that it adversely affects African Americans more than non-Hispanic whites, effectively widening the disparity in inflammation between these two groups. The opposite direction of effect holds true for gender differences. The fact that women report fewer experiences of discrimination effectively sustains their sense of purpose in life at levels higher than those of men, which confers more protection against increases in inflammation. That said, race and gender differences in inflammation remain and differences in vulnerability do not account for the residual effect. Moreover, coping resources that are expected to buffer the effects of discrimination do not function in this manner. In the end, this study contributes to our understanding of health disparities and argue for the

development of interventions that (1) counter the disproportionate distribution of stressors among marginalized population, and (2) work towards identifying and sustaining resources advantageous for mitigating the adverse effects of stress.

APPENDIX

Figure 1.1 – Biology of the Stress Response (HPA Axis)

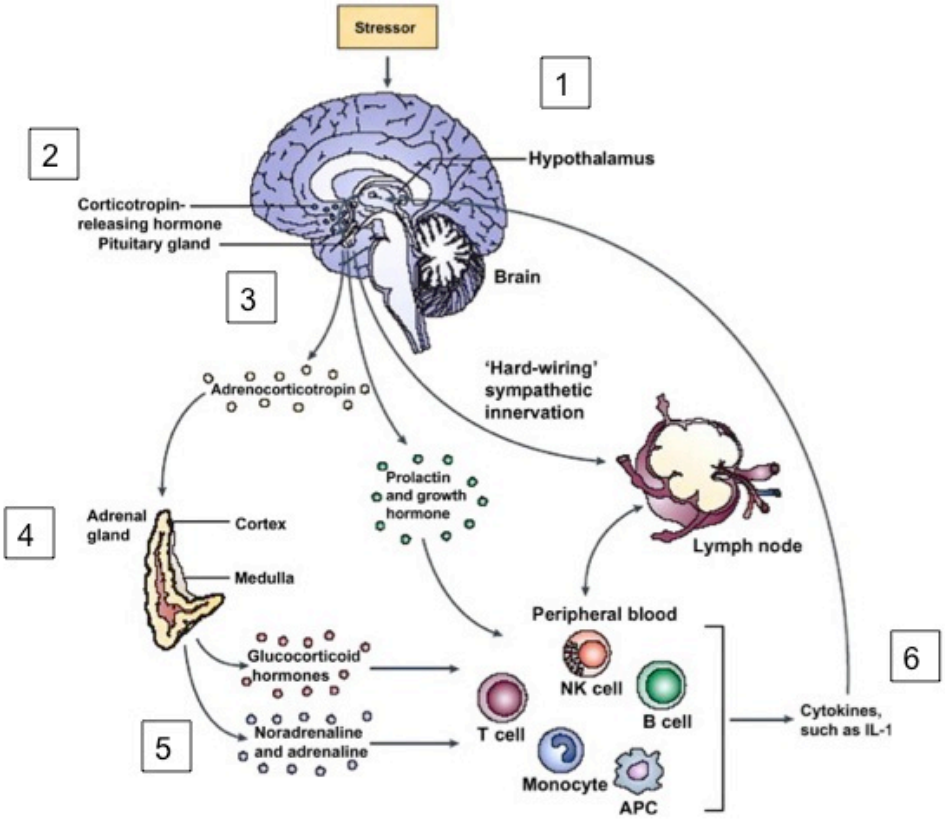


Figure 1.2 – The Inflammatory Reflex of the Autonomic Nervous System

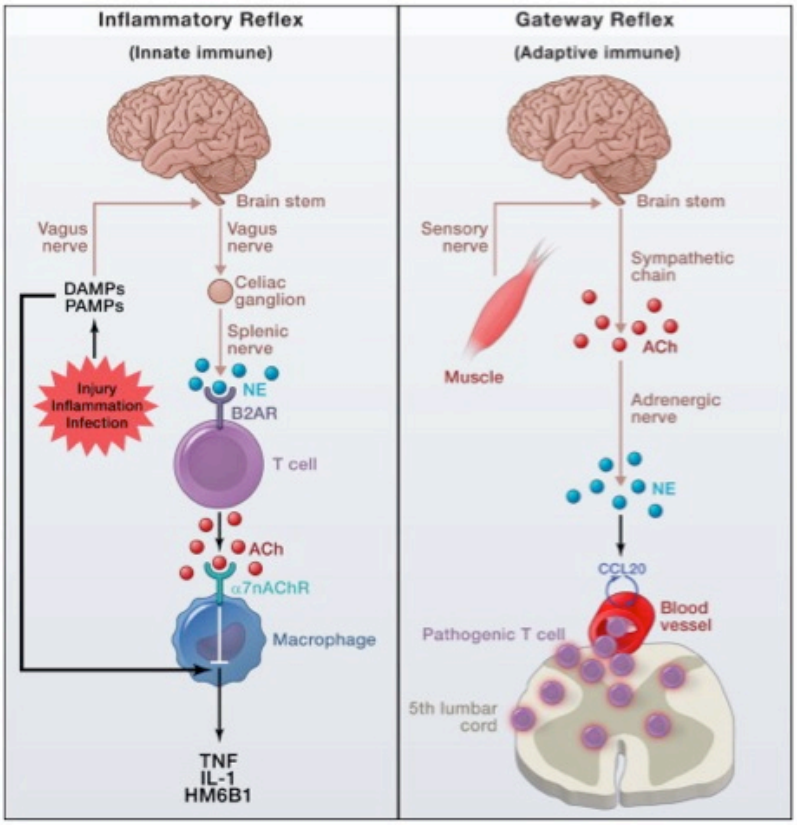


Figure 1.3 – Biology of the Inflammation Process

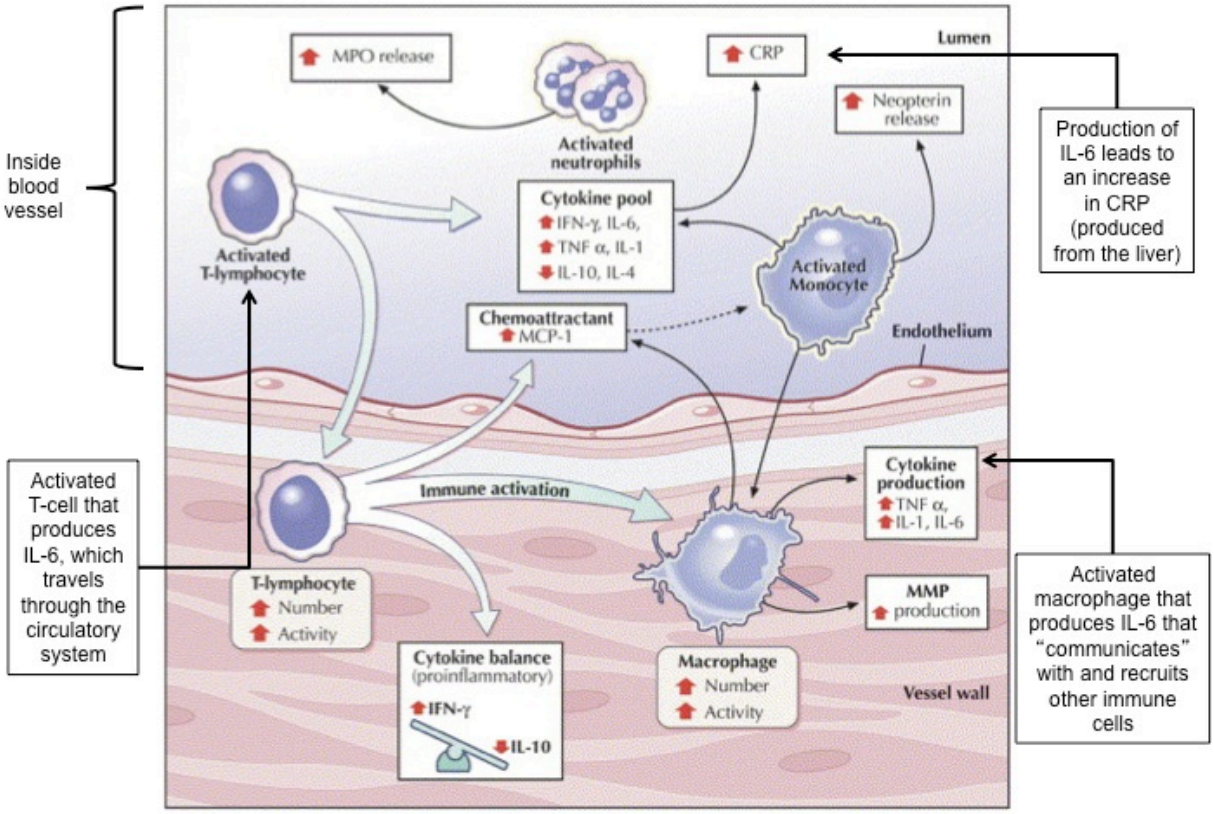


Figure 2.1 – Integrated Theoretical Framework of Stress

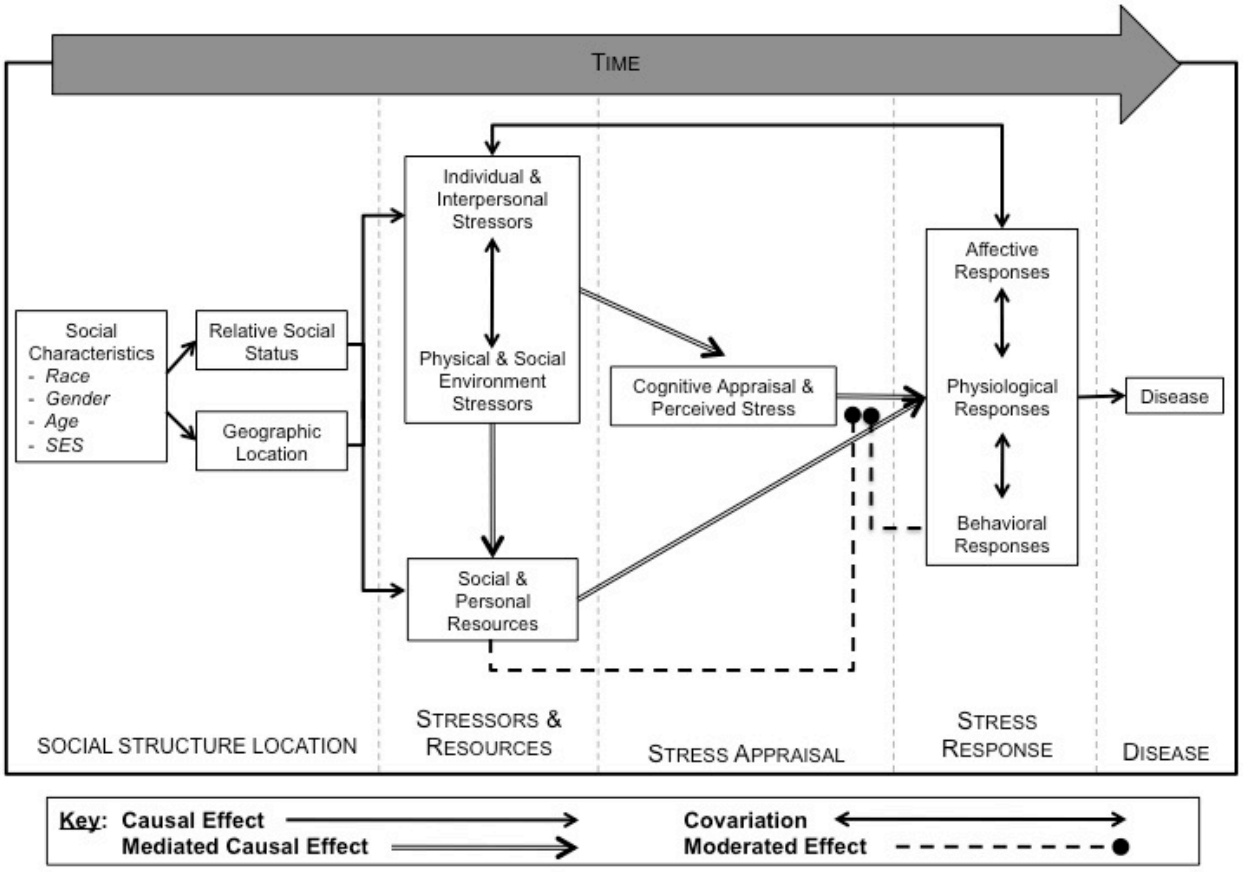


Figure 2.2 – Conceptual Model of the Relationship Between Race/Ethnicity, Gender and Inflammation

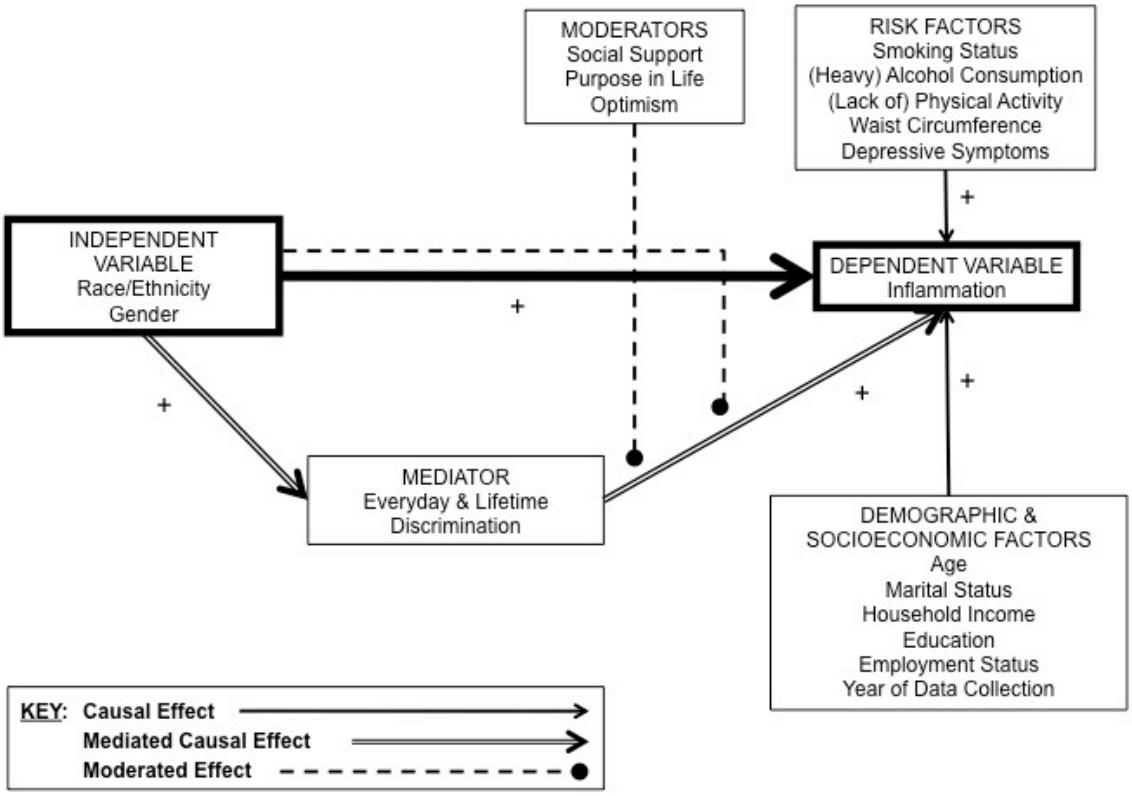


Figure 3.1 – Recruitment of the HRS Cohorts

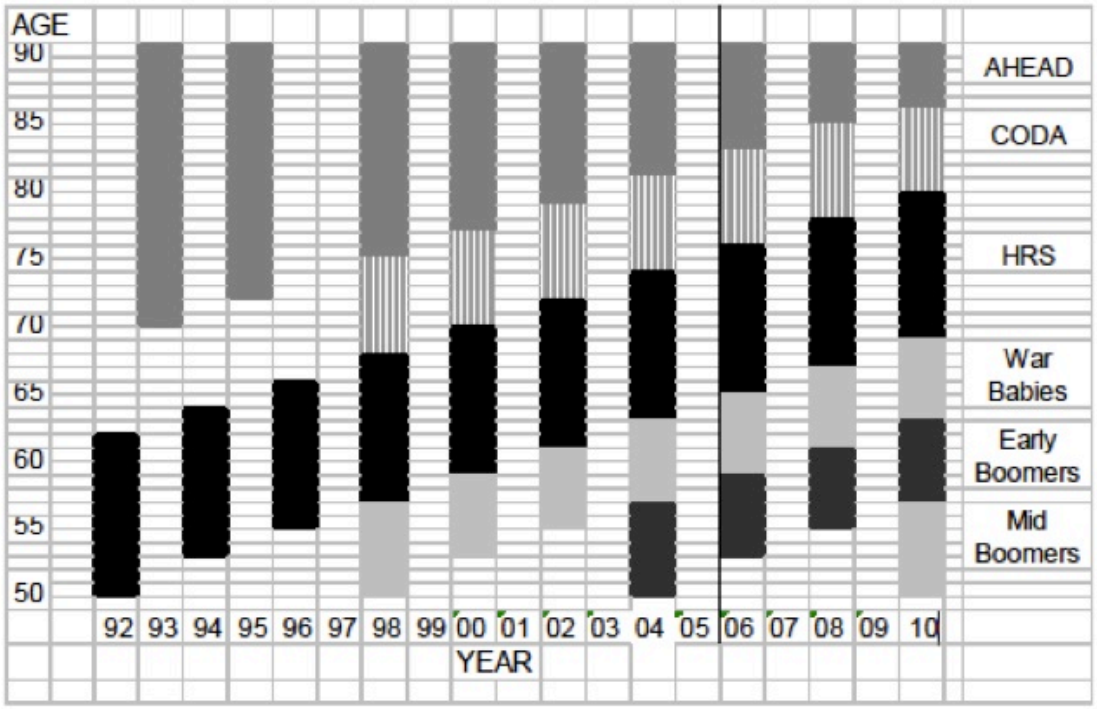


Figure 3.2 – How a Nephelometer Works

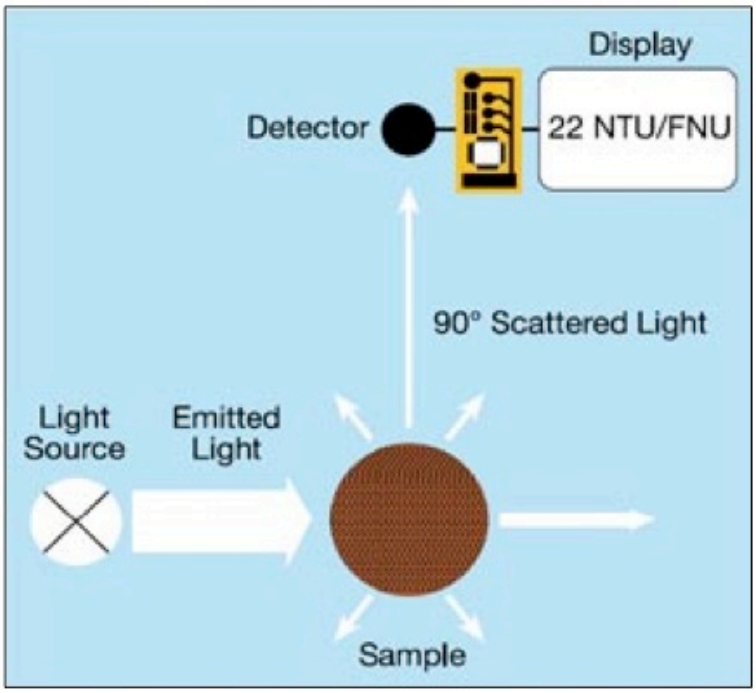
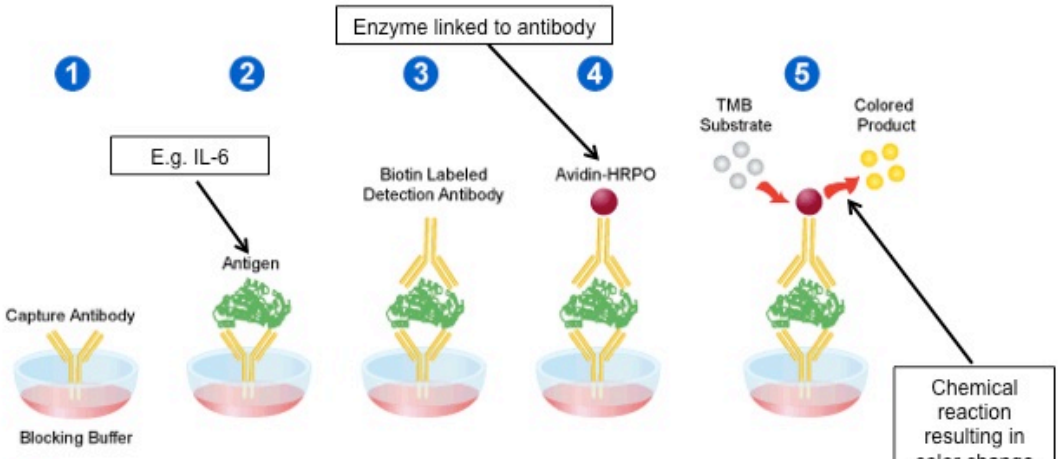
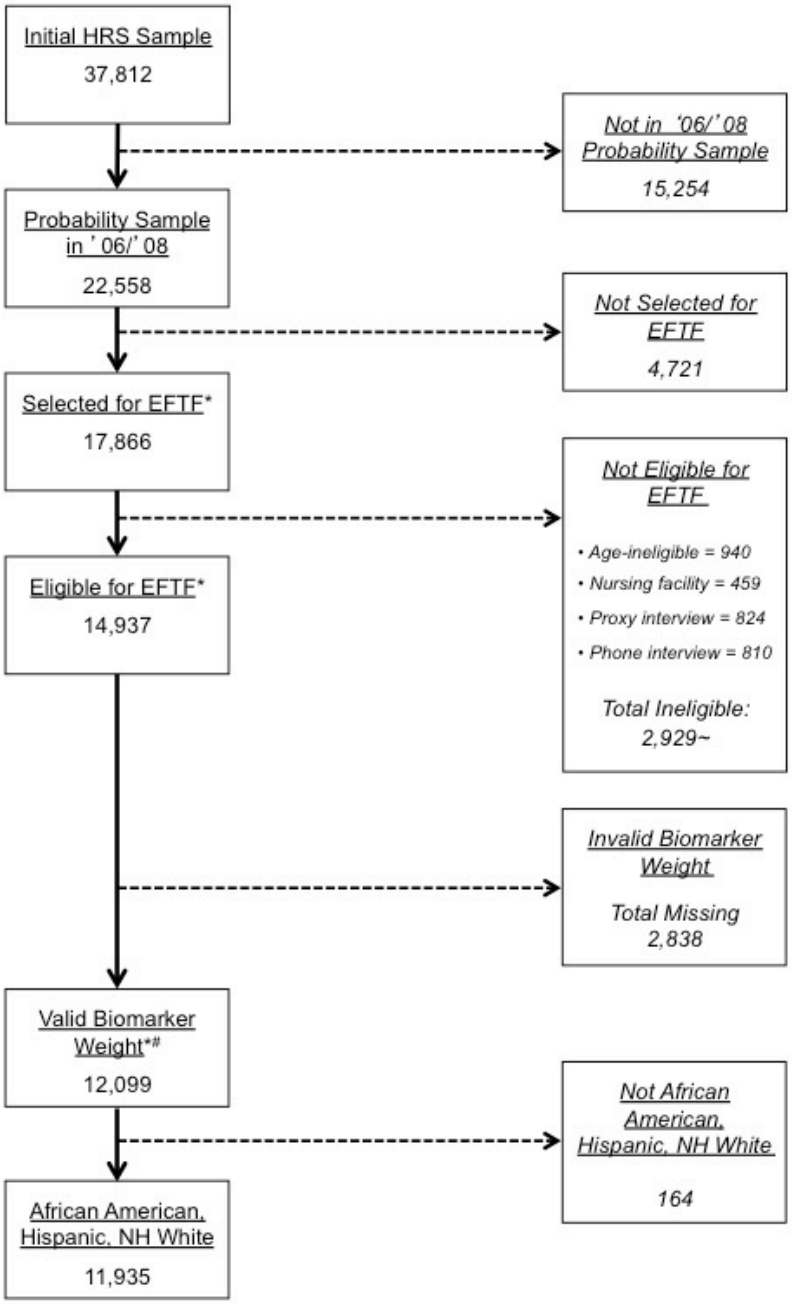


Figure 3.3 – Sandwich Enzyme-Linked Immunoassay (ELISA)



1. **a.)** Plate is coated with a suitable capture antibody. **b.)** Blocking buffer is added to block remaining protein-binding sites on plate.
2. Sample is added to plate and any antigen present is bound by the capture antibody.
3. A suitable biotin labeled detection antibody is added to the plate and also binds to any antigen present in well.
4. UltraAvidin™-HRPO *(Leinco Prod. No. A106)* is added and binds the biotin labeled detection antibody.
5. TMB substrate *(Leinco Prod. No. T118)* is added and converted by HRPO to a detectable form.

Figure 3.4 – Derivation of the Analytic Sample



* Refers to members of 2006/2008 probability sample
~ Numbers do not sum to the indicated total because multiple reasons for ineligibly or incomplete status are permitted
# Valid biomarker and physical measure weights were non-zero and not missing

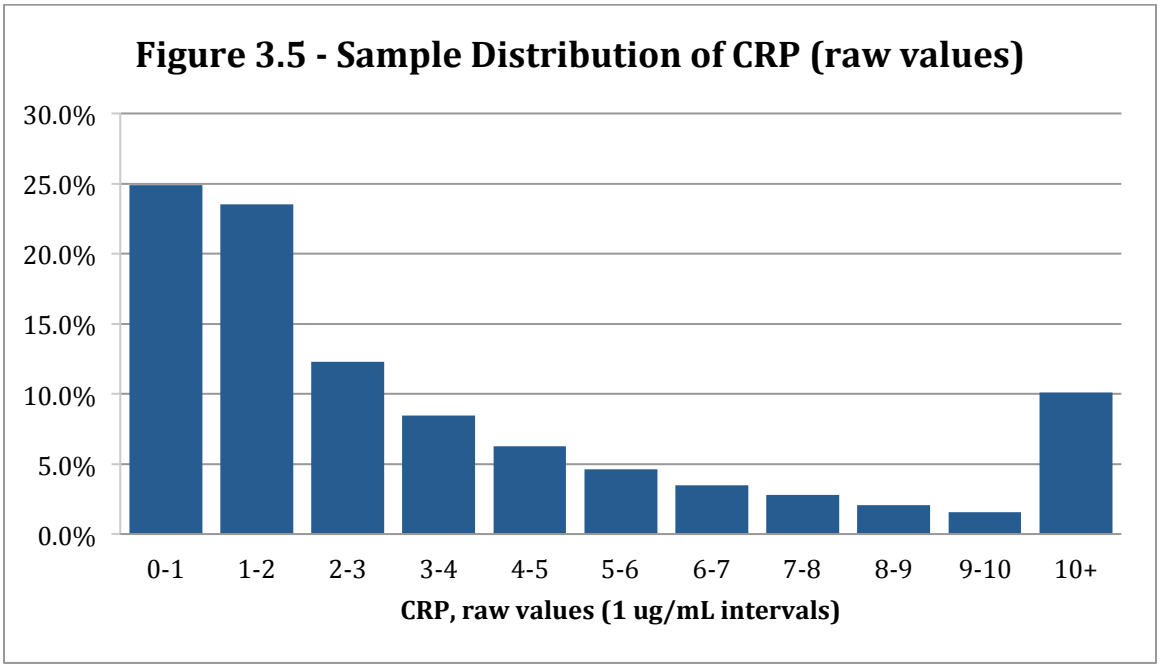


Figure 3.5 - Sample Distribution of CRP (raw values)
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
0-1
1-2
2-3
3-4
4-5
5-6
6-7
7-8
8-9
9-10
10+
CRP, raw values (1 ug/mL intervals)

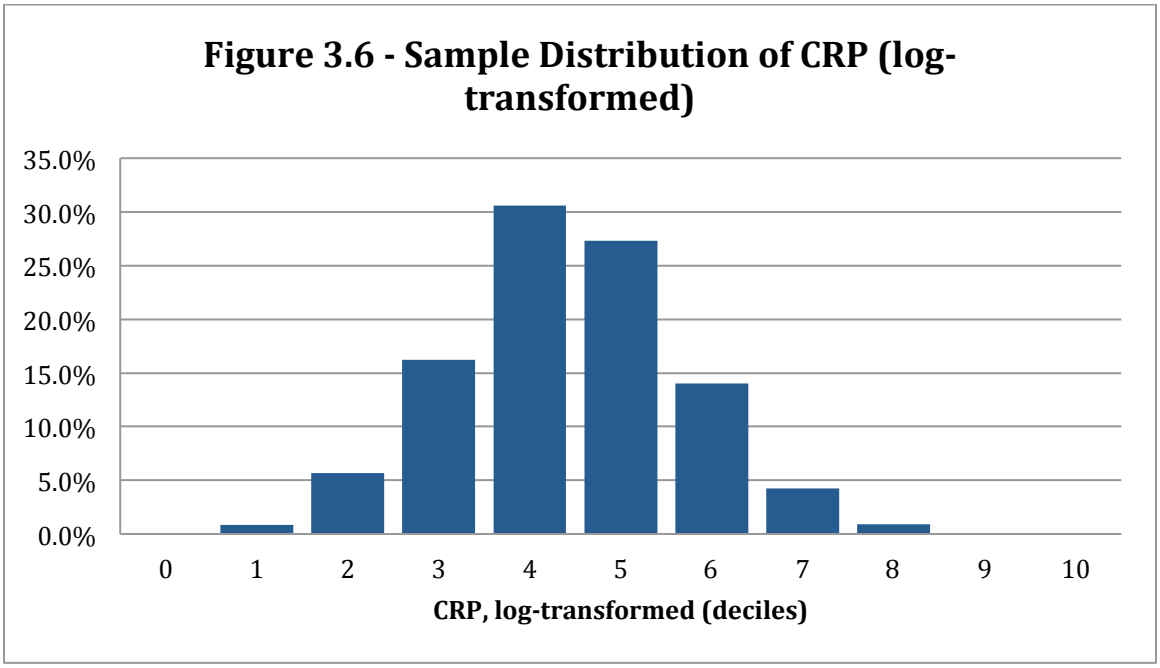


Figure 3.6 - Sample Distribution of CRP (log-transformed)
35.0%
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
0
1
2
3
4
5
6
7
8
9
10
CRP, log-transformed (deciles)

Figure 3.7 Simplified Structural Equation Model Depicted Direct and Indirect Paths between Race/ Ethnicity and Inflammation and Gender and Inflammation

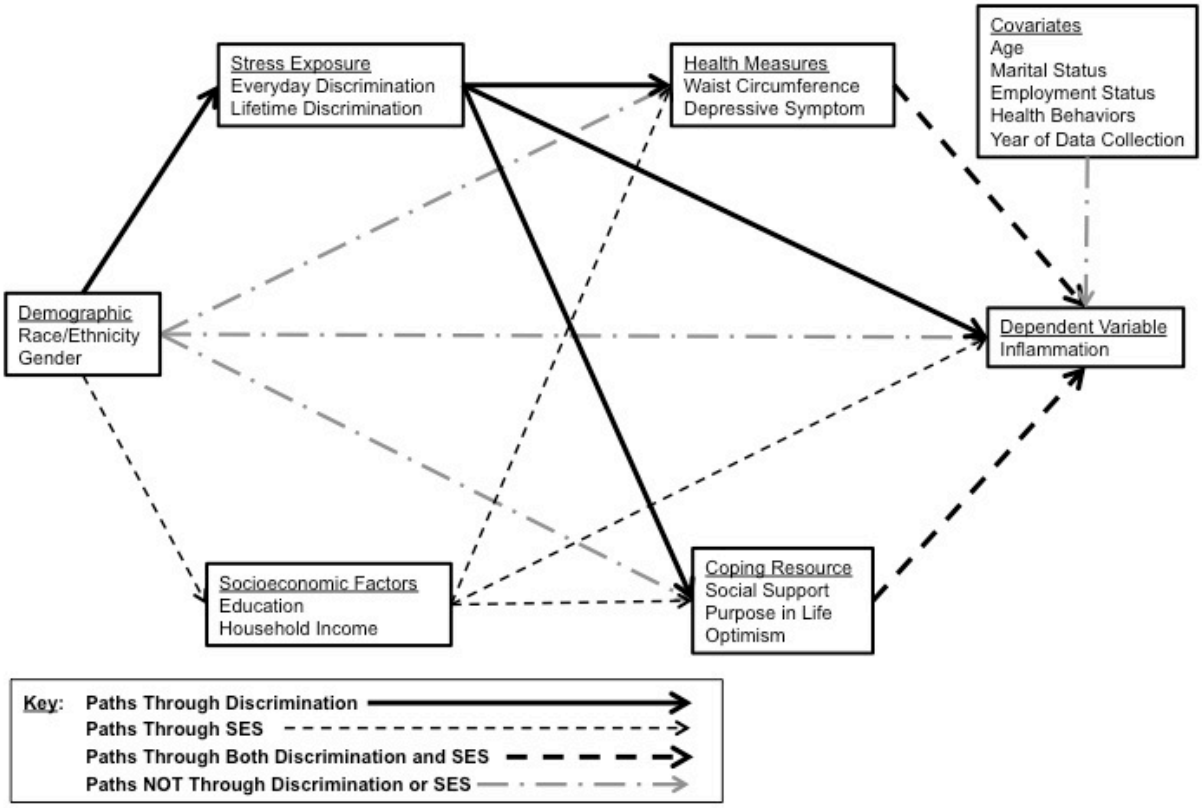
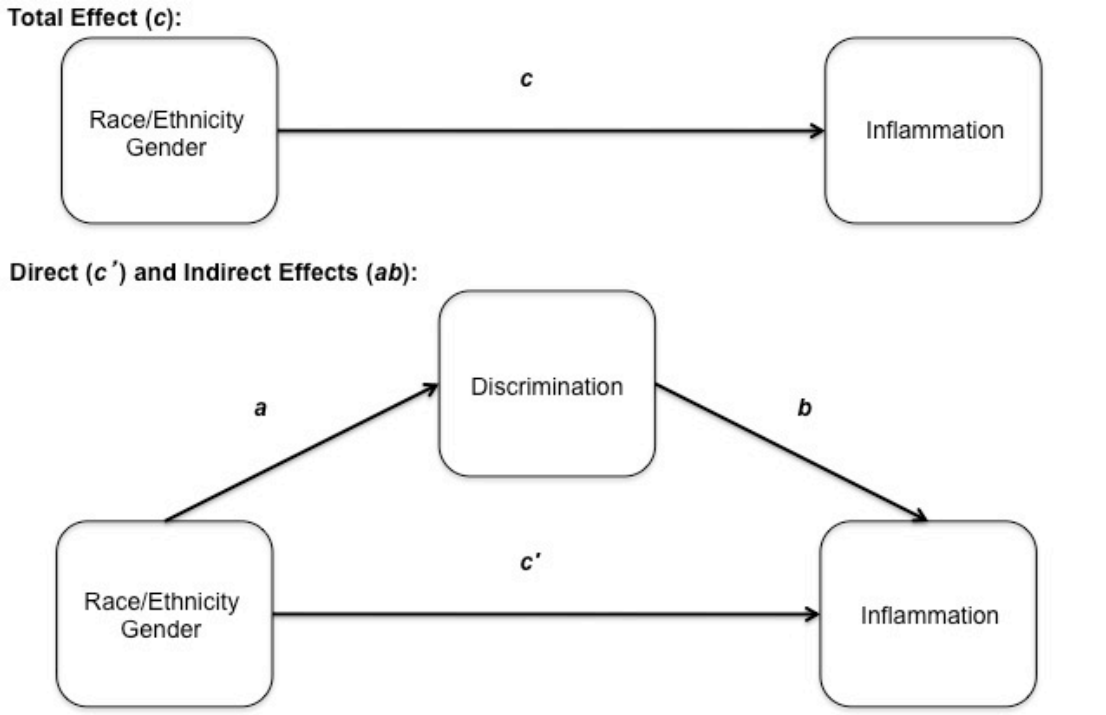


Figure 3.8 – Example of Three Variable Model of Mediation Analysis



Note: Models contain risk factors, coping resources and other control variables, but these variables are not shown for simplicity. Thus, ***c*** in the upper panel is not the total effect of discrimination on inflammation, but the effect net of these other variables.

Table 3.1 Steps in the Analysis of Aim 1

Step	Description of Statistical Procedure	Label	Regression Equation
1)	Total Association between Race/Ethnicity and Inflammation	Model 1A	$Y = a + b_bB + b_hH + e$
2)	Total Association between Gender and Inflammation	Model 1B	$Y = a + b_fF + e$
3)	Conditional Effect of Race/Ethnicity on Inflammation by Gender	Model 1C[1]	$Y = a + b_bB + b_hH + b_fF + b_{b\_f}(B*F) + b_{h\_f}(H*F) + e$
4)	Adjustment for Demographic Characteristics and Socioeconomic Factors	Model 1D	$Y = a + b_bB + b_hH + b_fF + b_cC + e$
5)	Adjustment for Health Behaviors	Model 1E	$Y = a + b_bB + b_hH + b_fF + b_cC + b_{hb}HB + e$
6)	Adjustment for Health Status Risk Factors	Model 1F	$Y = a + b_bB + b_hH + b_fF + b_cC + b_{hb}HB + b_{hs}HS + e$
7)	Model 1F excluding Race/Ethnicity	Model 1G	$Y = a + b_fF + b_cC + b_{hb}HB + b_{hs}HS + e$
8)	Model 1F excluding Gender	Model 1H	$Y = a + b_bB + b_hH + b_cC + b_{hb}HB + b_{hs}HS + e$

NOTE: Y = Inflammation, B = Black dummy variable, H = Hispanic dummy variable, F = Female, C = Control Variables, HS = Health Status, HB = Health Behaviors

a = regression intercept, b = unstandardize regression coefficient, e = error term

[1]The race-x-gender interaction was not statistically significant so the subsequent models exclude this interaction

Table 3.2 Steps in the Analysis of Aim 2

Step	Description of Statistical Procedure	Label	Regression Equation
1)	Total Association between Race/Ethnicity and Discrimination	Model 2A	$ED = a + b_bB + b_hH + e$
2)	Total Association between Gender and Discrimination	Model 2B	$ED = a + b_fF + e$
3)	Adjustment for Demographic Characteristics and Socioeconomic Factors	Model C	$ED = a + b_bB + b_hH + b_fF + e$
4)	Conditional Effect of Race/Ethnicity on Discrimination by Gender	Model 2D[1]	$ED = a + b_bB + b_hH + b_fF + b_{b\_f}(B*F) + b_{h\_f}(H*F) + e$
Step	**Description of Statistical Procedure**	**Label**	**Regression Equation**
5)	Association between Everyday Discrimination and Inflammation	Model 2E	$Y = a + b_{ed}ED + e$
6)	Association between Lifetime Discrimination and Inflammation	Model 2F	$Y = a + b_{ld}LD + e$
7)	Main Effect of Everyday and Lifetime Discrimination on Inflammation	Model G	$Y = a + b_{ed}ED + b_{ld}LD + e$
8)	Conditional Effect of Everyday and Lifetime Discrimination on Inflammation	Model 2H[1]	$Y = a + b_{ed}ED + b_{ld}LD + b_{ed\_ld}(ED*LD) + e$
Step	**Description of Statistical Procedure**	**Label**	**Regression Equation**
9)	Total Association between Race/Ethnicity and Inflammation	Model 2I	$Y = a + b_bB + b_hH + b_fF + e$
10)	Total Association between Gender and Inflammation	Model 2J	$Y = a + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + e$
11)	Conditional Effect of Race/Ethnicity on Inflammation by Gender	Model 2K	$Y = a + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_cC + e$
12)	Adjustment for Demographic Characteristics and Socioeconomic Factors	Model 2L	$Y = a + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_cC + b_{hb}HB + e$
13)	Adjustment for Health Behaviors	Model 2M	$Y = a + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_cC + b_{hb}HB + b_{hs}HS + e$
14)	Adjustment for Health Status Risk Factors	Model 2N	$Y = a + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_cC + b_{hb}HB + b_{hs}HS + b_sS + b_pP + b_oO + e$

NOTE: ED = Everyday Discrimination, LD = Lifetime Discrimination, Y = Inflammation, B = Black dummy variable, H = Hispanic dummy variable, F = Female, C = Control Variables, HS = Health Status, HB = Health Behaviors, S = Social Support, P = Purpose in Life, O = Optimism

a = regression intercept, b = unstandardize regression coefficient, e = error term

[1]The race-x-gender interaction was not statistically significant so the subsequent models exclude this interaction

Table 3.3 Steps in Analysis of Aim 3

Step	Description of Statistical Procedure	Label	Regression Equation[1]
1)	Main Effects Model (Model 1F)	Model 3A	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{id}LD + e$
2)	Effect of Everyday Discrimination Conditioned on Race/Ethnicity	Model 3B	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{id}LD + b_{ed\_b}(ED*B) + b_{ed\_h}(ED*H) + e$
3)	Effect of Lifetime Discrimination Conditioned on Race/Ethnicity	Model 3C	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{id}LD + b_{ed\_b}(LD*B) + b_{ed\_h}(LD*H) + e$
4)	Effect of Everyday Discrimination Conditioned on Gender	Model 3D	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{id}LD + b_{ed\_f}(ED*F)) + e$
5)	Effect of Lifetime Discrimination Conditional on Gender	Model 3E	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{id}LD + b_{ed\_f}(LD*F)) + e$

NOTE: Y = Inflammation, B = Black dummy variable, H = Hispanic dummy variable, F = Female, ED = Discrimination, LD = Lifetime Discrimination
a = regression intercept b = unstandardize regression coefficient
[1] Ellipses (…) = control variables, health behaviors, health status, and coping resources

Table 3.4 Steps in Analysis of Aim 4

Step	Description of Statistical Procedure	Label	Regression Equation[1]
1)	Main Effects Model (Model 1F)	Model 4A	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_sS + e$
2)	Buffering of the Effect of Everyday Discrimination on Inflammation	Model 4B	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_sS + b_{h\_s}(ED*S) + e$
3)	Buffering of the Effect of Lifetime Discrimination on Inflammation	Model 4C	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_sS + b_{h\_s}(LD*S) + e$
4)	Moderation of the Effect of Everyday Discrimination by Race/Ethncity and Social Support	Model 4D	Model 4B + $b_{h\_s\_ed}(B*S*ED) + b_{h\_s\_ed}(H*S*ED) + b_{b\_s}(B*S) + b_{h\_s}(H*S)$
5)	Moderation of the Effect of Lifetime Discrimination by Race/Ethncity and Social Support	Model 4E	Model 4C + $b_{h\_s\_ld}(B*S*LD) + b_{h\_s\_ld}(H*S*LD) + b_{b\_s}(B*S) + b_{h\_s}(H*S)$
6)	Moderation of the Effect of Everyday Discrimination by Gender and Social Support	Model 4F	Model 4B + $bf_{s\_ed}(F*S*ED) + bf_{s}(F*S)$
7)	Moderation of the Effect of Lifetime Discrimination by Gender and Social Support	Model 4G	Model 4C + $bf_{s\_ld}(F*S*LD) + bf_{s}(F*S)$
8)	Moderation by Socual Supprot of the Racial/Ethnic Difference in Inflammation	Model 4H	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_sS + b_{b\_s}(B*S) + b_{h\_s}(H*S) + e$
9)	Moderation by Social Support of the Gender Difference in Inflammation	Model 4I	$Y = a + \ldots + b_bB + b_hH + b_fF + b_{ed}ED + b_{ld}LD + b_sS + b_{f\_s}(F*S) + e$

NOTE: Y = Inflammation, B = Black dummy variable, H = Hispanic dummy variable, F = Female, ED = Discrimination, LD = Lifetime Discrimination, S = Social Support
a = regression intercept b = unstandardize regression coefficient
[1] Ellipses (…) = control variables, health behaviors, health status, and other coping resources

Table 4.1 Characteristics of the Analytic Sample (n=11,935) and Population Parameter Estimates (N=74,522,467)

	Sample (Unweighted)			Population Estimates (Weighted)		
Study Variables	Mean	SD	%	Mean	SE	%
Demographic Characteristics						
Age (range: 52-101; years)	69.2	9.6		66.1	0.2	
Marital Status						
Married			66.3			66.4
Separated/Divorced			11.0			13.4
Widowed			19.9			16.6
Never Married			2.9			3.7
Socioeconomic Factors						
Education (range: 0-17; years)	12.5	3.1		12.9	0.0	
Household Income (range: $0-$13,568; $1,000)	62.7	162.6		73.9	3.6	
Employment Status						
Full/Part Time			24.8			35.4
Retired			64.3			53.6
Other			10.9			11.0
Health Behaviors						
Smoking Status						
Never Smoked			43.0			42.8
Former Smoker			43.8			42.4
Current Smoker			12.8			14.8
Alcohol Consumption						
Non-Drinker			49.0			44.8
Moderate			44.6			47.6
Heavy			6.4			7.6
Vigorous Activity						
Never			62.2			59.0
≤ Once a Week			14.8			16.2
> Once a Week			22.9			24.8
Moderate Activity						
Never			20.8			19.1
≤ Once a Week			24.7			24.4
> Once a Week			54.5			56.5
Health Status Measures						
Waist Circumference (inches)	39.9	6.0		39.9	0.1	
Depressive Symptoms (range: 0-8)	1.4	2.0		1.5	0.0	
Sample Design Indicator						
Year of Data Collection						
2006			51.3	---	---	---
2008			48.7	---	---	---

NOTE: SD = standard deviation; SE = standard error

Table 4.2 Distribution of Key Variables of the Analytic Sample (n=11,935) and Population Parameter Estimates (N=74,522,467)

Study Variables	Sample (Unweighted)			Population Estimates (Weighted)		
	Mean	SD	%	Mean	SE	%
Independent Variables						
Race/Ethncity						
White			77.6			83.1
African American			13.1			9.3
Hispanic			8.3			7.6
Gender						
Female			58.4			54.2
Male			41.6			45.8
Dependent Variable						
CRP (ug/mL, range: 0.02-280)	4.7	9.3		4.5	0.1	
Log-CRP (ug/mL, range: -3.9-5.6)	0.8	1.2		0.8	0.0	
Mediators						
Everyday Discrimination (range: 0-5)	0.6	0.7		0.7	0.0	
Lifetime Discrimination (range: 0-6)	0.5	0.9		0.5	0.0	
Moderators						
Social Support (range: 0-4)	3.1	0.6		3.1	0.0	
Purpose in Life (range: 0-5)	3.6	0.9		3.6	0.0	
Optimism (range: 0-5)	3.5	1.1		4.5	0.0	

NOTE: SD = standard deviation; SE = standard error

Figure 4.1 - Reasons Given for Experiences of Discrimination (any Mention of Age, Race and Gender)

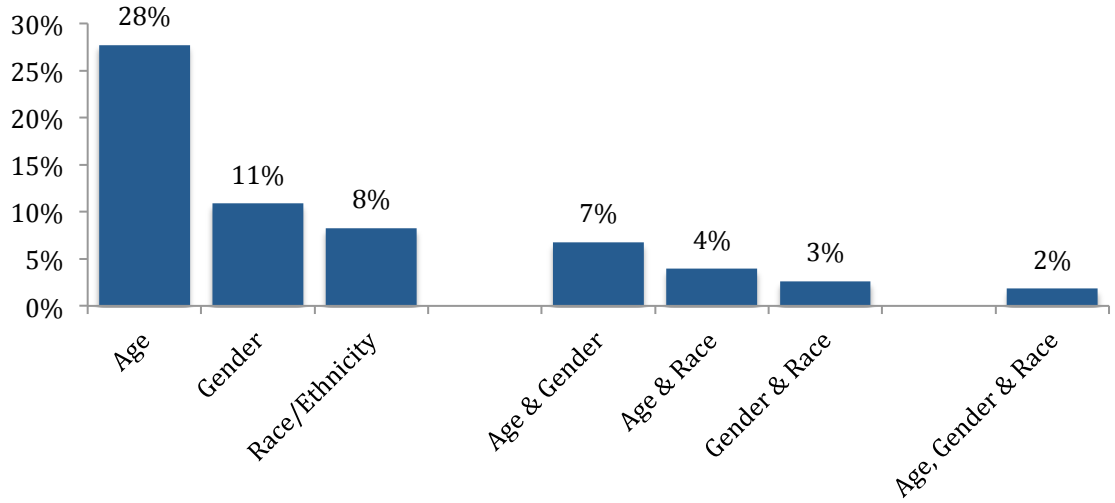


Figure 4.2 - Distribution of CRP by Race and Gender

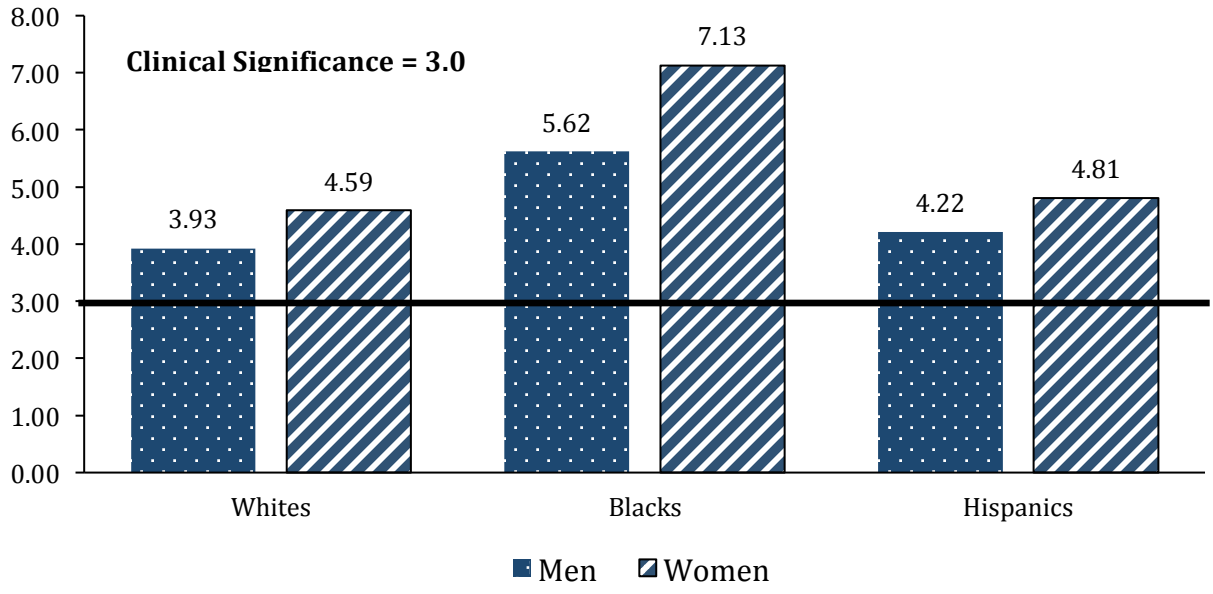


Table 4.3 Distribution of Study Variables by Race/Ethnicity (n=11,935), Weighted

Study Variables	NH Whites (n=9,258)			African Americans (n=1,562)			Hispanics (n=1,115)			p-value
	Mean	SE	%	Mean	SE	%	Mean	SE	%	
Demographic Characteristics										
Age (years)	66.6	0.2		64.5	0.4		63.6	0.8		***
Gender										
Female			53.5			59.5			55.3	***
Male			46.5			40.5			44.7	
Marital Status										
Married			69.0			45.5			62.7	***
Separated/Divorced			11.6			25.3			18.3	
Widowed			16.3			21.2			14.9	
Never Married			3.1			8.0			4.1	
Socioeconomic Factors										
Education (years)	14.3	0.1		11.9	0.1		9.5	0.4		***
Household Income ($1,000)	80.7	4.2		41.1	1.6		39.8	3.1		***
Employment Status										
Full/Part Time			35.6			34.3			35.1	***
Retired			55.2			52.0			38.4	
Other			9.2			13.7			26.5	
Health Behaviors										
Smoking Status										
Never Smoked			42.6			41.7			46.3	***
Former Smoker			43.3			36.7			39.6	
Current Smoker			14.1			21.6			14.0	
Alcohol Consumption										
Non-Drinker			42.1			61.0			54.8	***
Moderate			49.7			34.2			40.0	
Heavy			8.2			4.8			5.2	
Vigorous Activity										
Never			57.9			66.7			61.4	***
≤ Once a Week			16.4			13.6			16.9	
> Once a Week			25.7			19.8			21.8	
Moderate Activity										
Never			18.0			26.4			22.3	***
≤ Once a Week			23.3			30.6			29.0	
> Once a Week			58.7			43.0			48.7	
Health Status										
Waist Circumference (inches)	39.7	0.1		40.8	0.2		40.4	0.3		***
Depressive Symptoms (range: 0-8)	1.3	0.0		2.0	0.1		2.3	0.1		***
Stress Exposures										
Everyday Discrimination (range: 0-5)	0.7	0.0		0.9	0.0		0.7	0.0		***
Lifetime Discrimination (range: 0-6)	0.5	0.0		1.0	0.1		0.5	0.0		***
Coping Resources										
Social Support (range: 0-4)	3.1	0.0		3.1	0.0		3.1	0.0		N.S.
Purpose in Life (range: 0-5)	3.6	0.0		3.7	0.0		3.4	0.0		***
Optimism (range: 0-5)	3.5	0.0		3.7	0.1		3.7	0.1		***

NOTE: SE = standard error
*p<0.05; **p<0.01; ***p<0.001 for F-test of differences by race/ethnicity

Table 4.4 Distribution of Study Variables by Gender (n=11,935), Weighted

Study Variables	Female (n=6,967) Mean	SE	%	Male (4,968) Mean	SE	%	p-value
Demographic Characteristics							
Age (years)	66.8	0.2		65.4	0.2		***
Race/Ethnicity							
NH White (%)			82.0			84.4	***
African American (%)			10.2			8.2	
Hispanic (%)			7.8			7.4	
Marital Status							
Married (%)			55.8			78.9	***
Separated/Divorced (%)			15.7			10.6	
Widowed (%)			25.1			6.6	
Never Married (%)			3.5			3.9	
Socioeconomic Factors							
Education (years)	12.7	0.1		13.1	0.1		***
Household Income ($1,000)	61.4	2.0		88.6	7.1		***
Employment Status							
Full/Part Time (%)			30.5			41.2	***
Retired (%)			53.3			54.0	
Other (%)			16.2			4.8	
Health Behaviors							
Smoking Status							
Never Smoked (%)			51.2			32.8	***
Former Smoker (%)			35.1			51.2	
Current Smoker (%)			13.7			16.0	
Alcohol Consumption							
Non-Drinker (%)			51.6			36.8	***
Moderate (%)			42.1			54.0	
Heavy (%)			6.2			9.2	
Vigorous Activity							
Never (%)			65.8			50.1	***
≤ Once a Week (%)			13.7			19.0	
> Once a Week (%)			20.4			30.0	
Moderate Activity							
Never (%)			22.1			15.5	***
≤ Once a Week (%)			23.9			25.1	
> Once a Week (%)			54.0			59.4	
Health Status							
Waist Circumference (inches)	38.4	0.1		41.6	0.1		***
Depressive Symptoms (range: 0-8)	1.6	0.0		1.2	0.0		***
Stress Exposures							
Everyday Discrimination (range: 0-5)	0.6	0.0		0.8	0.0		***
Lifetime Discrimination (range: 0-6)	0.4	0.0		0.7	0.0		***
Coping Resources							
Social Support (range: 0-4)	3.2	0.0		3.0	0.0		***
Purpose in Life (range: 0-5)	3.6	0.0		3.6	0.0		N.S.
Optimism (range: 0-5)	3.5	0.0		3.5	0.0		N.S.

NOTE: SE = standard error
*p<0.05; **p<0.01; ***p<0.001 for F-test of differences by gender

Table 4.5 - Mean CRP Levels (ug/mL; log-transformed) across Categorical Study Variables (n=11,935), Weighted

Study Variables	Mean	SE	p-value
Independent Variables			
Race/Ethnicity			
NH White	0.72	0.02	***
African American	1.06	0.05	
Hispanic	0.87	0.04	
Gender			
Female	0.87	0.02	***
Male	0.63	0.02	
Demographic & Socioeconomic Variables			
Employment Status			
Full/Part Time	0.64	0.03	***
Retired	0.79	0.01	
Other	0.96	0.04	
Marital Status			
Married	0.69	0.02	***
Separated/Divorced	0.96	0.04	
Widowed	0.84	0.03	
Never Married	0.74	0.09	
Health Behaviors			
Smoking Status			
Never Smoked	0.66	0.02	***
Former Smoker	0.76	0.02	
Current Smoker	1.03	0.04	
Alcohol Consumption			
Non-Drinker	0.90	0.02	***
Moderate	0.63	0.02	
Heavy	0.67	0.04	
Vigorous Activity			
Never	0.91	0.02	***
≤ Once a Week	0.59	0.03	
> Once a Week	0.49	0.02	
Moderate Activity			
Never	1.14	0.03	***
≤ Once a Week	0.82	0.03	
> Once a Week	0.60	0.02	
Control Variable			
Year of Data Collection			
2006	0.73	0.02	*
2008	0.79	0.02	

NOTE: SE = standard error;
*p<0.05; **p<0.01; ***p<0.001 for F-test of differences by each categorical variable

Table 4.6 - Pearson's Correlation Coefficients among CRP & Continuous Study Variables (n=11,935)

Study Variables	A	B	C	D	E	F	G	H	I	J	K
Dependent Variable											
A. CRP (log-transformed)	1.00										
Demographic & Socioeconomic Variables											
B. Age (years)	-0.025**	1.00									
C. Education (years)	-0.118***	-0.139***	1.00								
D. Household Income (log-transformed)	-0.092***	-0.170***	0.394***	1.00							
Health Status											
E. Waist Circumference (inches)	0.284***	-0.061**	-0.069***	-0.031***	1.00						
F. Depressive Symptoms (range: 0-8)	0.112***	-0.010	-0.228***	-0.224***	0.089***	1.00					
Stress Exposures											
G. Everyday Discrimination (range: 0-5)	0.035***	-0.186***	-0.016	-0.022*	0.109***	0.219***	1.00				
H. Lifetime Discrimination (range: 0-6)	0.055***	-0.168***	0.085***	0.002	0.079***	0.115***	0.284***	1.00			
Coping Resources											
I. Social Support (range: 0-4)	-0.022*	0.078***	0.029**	0.041***	-0.077***	-0.175***	-0.235***	-0.130***	1.00		
J. Purpose in Life (range: 0-5)	-0.087***	-0.135***	0.218***	0.200***	-0.093***	-0.346***	-0.206***	-0.022*	0.253***	1.00	
K. Optimism (range: 0-5)	-0.019*	0.010	0.059***	0.051***	-0.041***	-0.201***	-0.160***	-0.022*	0.182***	0.329***	1.00

NOTE: *p<0.05; **p<0.01; ***p<0.001

Table 5.1 Linear Regression of Inflammation on Race/Ethnicity and Gender (n=11,935); Weighted

	CRP (µg/mL, [-3.91-5.63])							
	Model 1		Model 2		Model 3		Model 4	
Study Variables	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Race/Ethnicity (ref. = whites)								
African American	0.351***	0.046			0.338***	0.045	0.275**	0.085
Hispanic	0.157**	0.044			0.153**	0.044	0.126	0.067
Female (ref. = males)			0.239***	0.026	0.233***	0.026	0.220***	0.025
Race & Gender Interacations								
Black x Female Interaction							0.106	0.100
Hispanic x Female Interaction							0.049	0.092
Control Variable								
2008 Data Collection (ref. = 2006)	0.059*	0.025	0.059*	0.025	0.058*	0.026	0.058*	0.026
Constant	0.682***	0.021	0.596***	0.024	0.558***	0.025	0.565***	0.025
Model Statisitcs								
R^2	0.008		0.010		0.018		0.018	
ΔR^2	0.008		0.002		0.008		0.000	
F(d.f.) [Wald Test]	34.19 (2,62)***		82.00 (1,63)***		33.01 (2,62)***		0.61 (2,62)	

NOTE: b = unstandardized regression coefficient; SE = standard error;

* = p<.05; ** = p<.01; *** = p<.001

Table 5.2 Nested Linear Regression Models of Inflammation on All Study Variables (n=11,935); Weighted

	CRP (µg/mL, [-3.91-5.63])											
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
Study Variables	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE
Independent Variable												
Race/Ethnicity (ref. = whites)												
African American	0.338***	0.045	0.22***	0.045	0.172***	0.047	0.14***	0.041			0.134**	0.041
Hispanic	0.153**	0.044	-0.08	0.051	-0.03	0.050	-0.02	0.049			-0.041	0.049
Female (ref. = males)	0.233***	0.026	0.19***	0.024	0.18***	0.025	0.43***	0.028	0.435***	0.028		
Demographic & Socioeconomic Variables												
Age (years)			-0.01***	0.002	-0.01***	0.002	-0.002	0.002	-0.003	0.002	-0.005*	0.002
Education (years)			-0.05***	0.005	-0.03***	0.005	-0.02***	0.005	-0.023***	0.005	-0.024***	0.005
Household Income (log-transformed)			-0.02	0.013	-0.001	0.012	0.004	0.011	0.002	0.010	0.002	0.011
Employment (ref. = full/part time)												
Retired			0.15***	0.036	0.12***	0.036	0.07	0.035	0.071*	0.035	0.093*	0.038
Other			0.16***	0.045	0.12**	0.043	0.08	0.038	0.075	0.039	0.181***	0.039
Marital Status (ref. = married)												
Separated/Divorced			0.17***	0.045	0.13**	0.044	0.10*	0.046	0.110*	0.047	0.167**	0.047
Widowed			0.02	0.034	-0.02	0.034	-0.05	0.033	-0.038	0.034	0.087**	0.032
Never Married			-0.001	0.088	-0.04	0.086	-0.08	0.084	-0.061	0.085	-0.063	0.085
Health Behaviors												
Smoking Status (ref. = never smoked)												
Former Smoker					0.15***	0.029	0.09***	0.025	0.092**	0.025	0.034	0.026
Current Smoker					0.27***	0.042	0.38***	0.039	0.379***	0.040	0.290***	0.039
Alcohol Consumption (ref. = non-drinker)												
Moderate					-0.12***	0.032	-0.08*	0.032	-0.081*	0.031	-0.106**	0.032
Heavy					-0.17***	0.047	-0.07	0.043	-0.079	0.046	-0.113*	0.049
Vigorous Activity (ref. = no activity)												
≤ Once a Week					-0.17***	0.037	-0.10**	0.033	-0.104**	0.033	-0.145***	0.033
> Once a Week					-0.22***	0.030	-0.09**	0.030	-0.089**	0.031	-0.150***	0.029
Moderate Activity (ref. = no activity)												
≤ Once a Week					-0.22***	0.039	-0.11**	0.036	-0.106**	0.036	-0.124**	0.037
> Once a Week					-0.33***	0.046	-0.15**	0.043	-0.154**	0.043	-0.169***	0.043
Health Status												
Waist Circumference (mean-centered)							0.07***	0.002	0.069***	0.002	0.057***	0.002
Waist-squared							-0.001***	0.0002	-0.001***	0.0002	-0.0003	0.0002
Depressive Symptoms (mean-centered)							0.03**	0.011	0.035**	0.011	0.035**	0.012
Depressive Symptoms-squared							-0.01*	0.003	-0.007*	0.003	-0.007*	0.003
Control Variable												
2008 Data Collection (ref. = 2006)	0.058*	0.026	0.06*	0.026	0.06*	0.024	0.05*	0.024	0.054*	0.024	0.059*	0.024
Constant	0.558***	0.025	1.78***	0.173	1.79***	0.176	0.96	0.180	1.026***	0.169	1.416***	0.172
Model Statistics												
R^2	0.018		0.039		0.072		0.168		0.166		0.142	
Δ R^2	0.008		0.021		0.033		0.096		-0.002		-0.026	
F(d.f.) [Wald Test]	33.01 (2,62)***		19.79 (8,56)***		28.66 (8,56)***		379.08 (4,60)***		116.10 (21,43)***		121.27 (22,42)***	

NOTE: b = unstandardized regression coefficient; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 6.1 Regression of Everyday Discrimination on Race/Ethnicity and Gender (n=11,935); Weighted

	Everyday Discrimination (0-5)							
	Model 1		Model 2		Model 3		Model 4	
Study Variables	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Race/Ethnicity (ref. = whites)								
African American	0.247***	0.042			0.258***	0.042	0.289***	0.065
Hispanic	0.036	0.039			0.039	0.039	-0.024	0.045
Female (ref. = males)			-0.163***	0.019	-0.168***	0.019	-0.172***	0.020
Race-x-Gender Interacation								
African American-x-Female Interaction							-0.051	0.074
Hispanic-x-Female Interaction							0.113*	0.048
Control Variable								
2008 Data Collection (ref. = 2006)	-0.100***	0.024	-0.099***	0.024	-0.099***	0.024	-0.100***	0.024
Constant	0.707***	0.017	0.818***	0.019	0.797***	0.020	0.799***	0.020
Model Statistics								
R^2	0.013		0.015		0.025		0.025	
ΔR^2	---		0.002		0.010		0.000	
F(d.f.) [Wald Test]	17.53 (2,62)***		73.46 (1,63)***		19.02 (2,62)***		3.30 (2,62)*	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 6.2 Regression of Lifetime Discrimination on Race/Ethnicity and Gender (n=11,935); Weighted

	Lifetime Discrimination (0-6)							
	Model 1		Model 2		Model 3		Model 4	
Study Variables	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Race/Ethnicity (ref. = whites)								
African American	0.477***	0.057			0.493***	0.057	0.552***	0.093
Hispanic	0.017	0.036			0.024	0.036	0.042	0.063
Female (ref. = males)			-0.230***	0.019	-0.240***	0.019	-0.230***	0.018
Race-x-Gender Interacation								
African American-x-Female Interaction							-0.032	0.100
Hispanic-x-Female Interaction							-0.098	0.069
Control Variable								
2008 Data Collection (ref. = 2006)	0.050*	0.022	0.052*	0.022	0.051*	0.022	0.051*	0.022
Constant	0.458***	0.018	0.623***	0.019	0.586***	0.020	0.581***	0.020
Model Statistics								
R^2	0.023		0.016		0.040		0.040	
ΔR^2	---		-0.007		0.024		0.000	
F(d.f.) [Wald Test]	33.99 (2,62)***		142.45 (1,63)***		37.02 (2,62)***		0.53 (2,62)	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 6.3 Regression of Inflammation on Everyday and Lifetime Discrimination (n=11,935); Weighted

Study Variables	CRP (µg/mL, [-3.91-5.63])							
	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Stress Exposures								
Everyday Discrimination	0.072**	0.023			0.049*	0.023	0.050*	0.023
Lifetime Discrimination			0.077***	0.018	0.065**	0.018	0.067**	0.018
Everyday-by-Lifetime Interaction							-0.005	0.022
Control Variable								
Year of Data Collection (ref. = 2006)	0.070**	0.025	0.058*	0.025	0.065*	0.025	0.065*	0.025
Constant	0.671***	0.023	0.686***	0.022	0.655***	0.024	0.654***	0.023
Model Statistics								
R^2	0.003		0.004		0.005		0.005	
ΔR^2	---		0.001		0.001		0.000	
F(d.f.) [Wald Test]	9.85 (1,63)**		18.25 (1,63)***		9.95 (2,62)***		0.058 (1,62)	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 6.4 Regression of Inflammation on Study Variables (n = 11,935); Weighted

	CRP (µg/mL, [-3.91-5.63])											
	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
Study Variables	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE
Independent Variable												
Race/Ethnicity (ref. = whites)												
African American	0.338***	0.045	0.288***	0.049	0.174**	0.048	0.134**	0.050	0.121**	0.041	0.12**	0.041
Hispanic	0.153***	0.044	0.151***	0.044	-0.081	0.050	-0.032	0.049	-0.015	0.049	-0.02	0.049
Female (ref. = males)	0.233***	0.026	0.259***	0.026	0.214***	0.024	0.202***	0.025	0.426***	0.027	0.43***	0.027
Stress Exposures												
Everyday Discrimination (range: 0-5)			0.056*	0.023	0.043	0.023	0.026	0.023	-0.018	0.021	-0.023	0.022
Lifetime Discrimination (range: 0-6)			0.068***	0.018	0.077***	0.017	0.074***	0.016	0.057**	0.016	0.057***	0.016
Demographic & Socioeconomic Variables												
Age (years)					-0.005**	0.002	-0.007***	0.002	-0.001	0.002	-0.002	0.002
Education (years)					-0.050***	0.006	-0.032***	0.005	-0.025***	0.005	-0.024***	0.005
Household Income (log-transformed)					-0.017	0.013	0.002	0.012	0.003	0.011	0.005	0.011
Employment (ref. = full/part time)												
Retired					0.150***	0.037	0.125**	0.036	0.065	0.035	0.063	0.035
Other					0.154**	0.044	0.110*	0.042	0.069	0.038	0.067	0.038
Marital Status (ref. = married)												
Separated/Divorced					0.145**	0.045	0.112*	0.046	0.086	0.047	0.087	0.046
Widowed					0.005	0.035	-0.024	0.035	-0.048	0.034	-0.049	0.034
Never Married					-0.014	0.089	-0.050	0.086	-0.077	0.086	-0.079	0.088
Health Behaviors												
Smoking Status (ref. = never smoked)												
Former Smoker							0.141***	0.029	0.093**	0.026	0.093***	0.025
Current Smoker							0.256***	0.042	0.372***	0.040	0.368***	0.041
Alcohol Consumption (ref. = non-drinker)												
Moderate							-0.123***	0.032	-0.080*	0.036	-0.080*	0.031
Heavy							-0.169**	0.046	-0.079	0.045	-0.080	0.045
Vigorous Activity (ref. = no activity)												
≤ Once a Week							-0.173***	0.037	-0.102**	0.034	-0.100**	0.034
> Once a Week							-0.216***	0.030	-0.093**	0.030	-0.090**	0.031
Moderate Activity (ref. = no activity)												
≤ Once a Week							-0.214***	0.039	-0.099**	0.036	-0.092**	0.037
> Once a Week							-0.332***	0.046	-0.145**	0.043	-0.138***	0.044
Health Status												
Waist Circumference (inches)									0.065***	0.002	0.065***	0.002
Depressive Symptoms (range: 0-8)									0.009	0.007	0.006	0.007
Coping Resources												
Social Support (range: 0-4)											0.001	0.026
Purpose in Life (range: 0-5)											-0.035*	0.016
Optimism (range: 0-5)											0.015	0.014
Control Variable												
Year of Data Collection (ref. = 2006)	0.058*	0.026	0.063*	0.025	0.056*	0.025	0.058*	0.024	0.048*	0.024	0.051*	0.025
Constant	0.558***	0.026	0.470***	0.028	1.573***	0.160	1.638***	0.166	-1.697***	0.202	-1.62***	0.250
Model Statistics												
R^2	0.018		0.023		0.043		0.075		0.166		0.167	
ΔR^2	---		0.005		0.02		0.032		0.091		0.001	
F(d.f.) [Wald Test]	41.70 (3,61)***		11.52 (2,62)***		20.52 (8,56)***		27.69 (8,56)***		588.62 (2, 62)***		1.56 (3,61)	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 6.5a Regression Equations for Inflammation, Everyday Discrimination, Lifetime Discrimination, Education, Household Income (n = 11,935); Weighted

	Everyday Discrimination		Lifetime Discrimination		Education		Household Income	
Study Variables	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Race/Ethnicity (ref. = whites)								
African American	0.180***	0.040	0.407***	0.060	-1.132***	0.137	-0.441***	0.047
Hispanic	-0.050	0.035	-0.054	0.042	-3.659***	0.318	-0.870***	0.098
Female (ref. = males)	-0.177***	0.019	-0.237***	0.021	-0.020	0.067	-0.025	0.029
Stress Exposures								
Everyday Discrimination (range: 0-5)	---	---	---	---	---	---	---	---
Lifetime Discrimination (range: 0-6)	---	---	---	---	---	---	---	---
Socioeconomic Status								
Age (years)	-0.016***	0.001	-0.016***	0.001	-0.034***	0.004	-0.001	0.002
Education (years)	---	---	---	---	---	---	---	---
Household Income (log-transformed)	---	---	---	---	---	---	---	---
Employment (ref. = full/part time)								
Retired	-0.061**	0.022	0.008	0.029	-0.354***	0.079	-0.643***	0.043
Other	0.041	0.042	0.05	0.049	-1.151***	0.117	-0.894***	0.079
Marital Status (ref. = married)								
Separated/Divorced	0.100**	0.030	0.235***	0.041	0.031	0.108	-0.995***	0.055
Widowed	0.089**	0.029	0.077**	0.026	-0.511***	0.086	-0.829***	0.041
Never Married	0.103	0.058	0.195*	0.089	0.654***	0.147	-0.952***	0.096
Health Behaviors								
Smoking Status (ref. = never smoked)								
Former Smoker	0.054**	0.018	0.037	0.027	-0.382***	0.067	-0.061**	0.022
Current Smoker	0.080*	0.031	0.090*	0.043	-1.052***	0.092	-0.359***	0.047
Alcohol Consumption (ref. = non-drinker)								
Moderate	-0.030	0.024	0.054*	0.026	0.909***	0.068	0.261***	0.034
Heavy	-0.053	0.033	0.076	0.048	0.654***	0.111	0.246***	0.064
Vigorous Activity (ref. = no activity)								
≤ Once a Week	-0.046	0.025	-0.013	0.029	0.272**	0.091	0.149***	0.034
> Once a Week	-0.060*	0.023	-0.028	0.029	0.555***	0.079	0.178***	0.033
Moderate Activity (ref. = no activity)								
≤ Once a Week	-0.074*	0.031	-0.032	0.032	0.211*	0.100	0.011	0.045
> Once a Week	-0.086**	0.025	-0.017	0.032	0.591	0.087	0.091*	0.036
Health Status								
Waist Circumference (inches)	---	---	---	---	---	---	---	---
Depressive Symptoms (range: 0-8)	---	---	---	---	---	---	---	---
Coping Resources								
Social Support (range: 0-4)	---	---	---	---	---	---	---	---
Purpose in Life (range: 0-5)	---	---	---	---	---	---	---	---
Optimism (range: 0-5)	---	---	---	---	---	---	---	---
Control Variable								
2008 Data Collection	-0.080**	0.022	0.063**	0.021	0.039	0.053	0.004	0.030
Constant	1.936***	0.085	1.542***	0.107	15.125***	0.286	11.345***	0.151

Table 6.5b Unstandardized Regression Coefficients for Models Predicting Waist Circumference and Depressive Symptoms (n = 11,935); Weighted

Study Variables	Waist Circumference		Depressive Symptoms	
	b	SE	b	SE
Independent Variable				
Race/Ethnicity (ref. = whites)				
African American	0.266	0.208	-0.075	0.067
Hispanic	-0.240	0.325	0.352**	0.111
Female (ref. = males)	-3.476***	0.163	0.148***	0.042
Stress Exposures				
Everyday Discrimination (range: 0-5)	0.631***	0.110	0.497***	0.046
Lifetime Discrimination (range: 0-6)	0.209*	0.084	0.185***	0.032
Socioeconomic Status				
Age (years)	-0.093***	0.009	-0.019***	0.003
Education (years)	-0.092***	0.023	-0.072***	0.009
Household Income (log-transformed)	-0.0002	0.060	-0.104***	0.022
Employment (ref. = full/part time)				
Retired	0.824***	0.205	0.338***	0.054
Other	0.508	0.261	0.627***	0.076
Marital Status (ref. = married)				
Separated/Divorced	0.274	0.248	0.520***	0.074
Widowed	0.320	0.179	0.501***	0.053
Never Married	0.315	0.286	0.240*	0.114
Health Behaviors				
Smoking Status (ref. = never smoked)				
Former Smoker	0.743***	0.128	0.025	0.050
Current Smoker	-1.870***	0.198	0.267**	0.087
Alcohol Consumption (ref. = non-drinker)				
Moderate	-0.602***	0.149	-0.144**	0.043
Heavy	-1.280***	0.246	-0.093	0.085
Vigorous Activity (ref. = no activity)				
≤ Once a Week	-1.051***	0.183	-0.196**	0.058
> Once a Week	-1.810***	0.139	-0.354***	0.050
Moderate Activity (ref. = no activity)				
≤ Once a Week	-1.672***	0.197	-0.787***	0.070
> Once a Week	-2.791***	0.168	-0.888***	0.080
Health Status				
Waist Circumference (inches)	---	---	---	---
Depressive Symptoms (range: 0-8)	---	---	---	---
Coping Resources				
Social Support (range: 0-4)	---	---	---	---
Purpose in Life (range: 0-5)	---	---	---	---
Optimism (range: 0-5)	---	---	---	---
Control Variable				
2008 Data Collection	0.187	0.158	-0.040	0.043
Constant	50.848***	1.044	4.617***	0.336

Table 6.5c Unstandardized Regression Coefficients for Models Predicting Social Support, Purpose in Life, Optimism, and Inflammation (n = 11,935); Weighted

	Social Support		Purpose in Life		Optimism		Inflammation	
Study Variables	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Race/Ethnicity (ref. = whites)								
African American	0.099***	0.022	0.367***	0.034	0.439***	0.059	0.127**	0.041
Hispanic	0.024	0.025	0.024	0.043	0.416***	0.064	-0.027	0.051
Female (ref. = males)	0.112***	0.014	0.047*	0.018	0.036	0.027	0.426***	0.027
Stress Exposures								
Everyday Discrimination (range: 0-5)	-0.153***	0.010	-0.284***	0.018	-0.248***	0.024	-0.023	0.022
Lifetime Discrimination (range: 0-6)	-0.033***	0.008	-0.010	0.012	0.00005	0.020	0.057***	0.016
Socioeconomic Status								
Age (years)	0.003**	0.001	-0.007***	0.002	0.006**	0.002	-0.002	0.002
Education (years)	0.006*	0.003	0.037***	0.004	0.023***	0.005	-0.024***	0.005
Household Income (log-transformed)	0.014	0.007	0.061***	0.012	0.030	0.015	0.005	0.011
Employment (ref. = full/part time)								
Retired	-0.008	0.019	-0.093**	0.029	-0.248***	0.042	0.063	0.035
Other	-0.015	0.027	-0.165***	0.036	0.000	0.048	0.067	0.038
Marital Status (ref. = married)								
Separated/Divorced	-0.039	0.024	-0.097**	0.035	-0.126*	0.052	0.087	0.046
Widowed	0.011	0.017	-0.164***	0.029	-0.046	0.034	-0.049	0.034
Never Married	-0.295***	0.063	-0.237***	0.055	-0.187*	0.078	-0.079	0.088
Health Behaviors								
Smoking Status (ref. = never smoked)								
Former Smoker	-0.016	0.013	-0.052*	0.025	-0.005	0.034	0.093**	0.025
Current Smoker	-0.002	0.019	-0.159***	0.042	-0.046	0.049	0.368***	0.041
Alcohol Consumption (ref. = non-drinker)								
Moderate	0.025	0.014	0.021	0.022	0.054*	0.023	-0.080*	0.031
Heavy	0.013	0.030	0.026	0.040	0.036	0.050	-0.080	0.045
Vigorous Activity (ref. = no activity)								
≤ Once a Week	0.043*	0.017	0.146***	0.028	0.088*	0.034	-0.100**	0.034
> Once a Week	0.047**	0.015	0.217***	0.024	0.153***	0.035	-0.090**	0.031
Moderate Activity (ref. = no activity)								
≤ Once a Week	0.055**	0.017	0.279***	0.025	0.150**	0.050	-0.092*	0.037
> Once a Week	0.083***	0.015	0.354***	0.032	0.189***	0.047	-0.138**	0.044
Health Status								
Waist Circumference (inches)	---	---	---	---	---	---	0.065***	0.002
Depressive Symptoms (range: 0-8)	---	---	---	---	---	---	0.006	0.007
Coping Resources								
Social Support (range: 0-4)	---	---	---	---	---	---	0.001	0.026
Purpose in Life (range: 0-5)	---	---	---	---	---	---	-0.035*	0.016
Optimism (range: 0-5)	---	---	---	---	---	---	0.015	0.014
Control Variable								
2008 Data Collection	-0.007	0.014	0.102***	0.023	-0.052	0.032	0.051*	0.025
Constant	2.687***	0.122	2.803***	0.191	2.475***	0.257	-1.617***	0.250

Table 6.6 Error Covariance Matrix for the Structural Equation Model

Error Covariance Matrix	e.(Everyday)		e.(Lifetime)		e.(Education)		e.(Waist)		e.(Depressive)		e.(Support)		e.(Purpose in Life)	
e.(Everyday)	---	---												
e.(Lifetime)	0.163***	0.011	---	---										
e.(Education)	-0.067*	0.030	0.169***	0.027	---	---								
e.(Log-Income)	-0.022*	0.010	-0.007	0.014	0.711***	0.053								
e.(Waist)	---	---	---	---	---	---	---	---						
e.(Depressive)	---	---	---	---	---	---	0.293*	0.117	---	---				
e.(Support)	---	---	---	---	---	---	-0.039	0.039	-0.108***	0.012	---	---		
e.(Purpose in Life)	---	---	---	---	---	---	-0.163**	0.050	-0.354***	0.018	0.091***	0.006	---	---
e.(Optimism)	---	---	---	---	---	---	-0.036	0.076	-0.329***	0.076	0.089***	0.007	0.286***	0.012

NOTE: * = p<.05; ** = p<.01; *** = p<.001

Table 6.7 Unstandardized Indirect, Direct and Total Effects of Race on Inflammation: African Americans and Non-Hispanic Whites

	1st Mediator	2nd Mediator	Indirect Effect b	Indirect Effect SE
Single-Mediator Pathways				
(1)	Everyday Discrimination	---	-0.004	0.004
(2)	**Lifetime Discrimination**	**---**	**0.023****	**0.007**
(3)	**Education**	**---**	**0.027*****	**0.005**
(4)	Income	---	-0.002	0.005
(5)	Waist Circumference	---	0.017	0.014
(6)	Depressive Symptoms	---	-0.0005	0.0007
(7)	Social Support	---	0.0001	0.003
(8)	**Purpose in Life**	**---**	**-0.013***	**0.006**
(9)	Optimism	---	0.007	0.006
Two-Mediator Pathways (through Discrimination)				
(10)	**Everyday Discrimination**	**Waist Circumference**	**0.007****	**0.002**
(11)	Everyday Discrimination	Depressive Symptoms	0.001	0.001
(12)	Everyday Discrimination	Social Support	-0.00004	0.0007
(13)	Everyday Discrimination	Purpose in Life	0.002	0.001
(14)	Everyday Discrimination	Optimism	-0.001	0.001
(15)	**Lifetime Discrimination**	**Waist Circumference**	**0.005***	**0.002**
(16)	Lifetime Discrimination	Depressive Symptoms	0.0005	0.0005
(17)	Lifetime Discrimination	Social Support	-0.00002	0.0004
(18)	Lifetime Discrimination	Purpose in Life	0.0001	0.0002
(19)	Lifetime Discrimination	Optimism	0.0000003	0.0001
Two-Mediator Pathways (through Socioeconomic Factors)				
(20)	**Education**	**Waist Circumference**	**0.007****	**0.002**
(21)	Education	Depressive Symptoms	0.001	0.001
(22)	Education	Social Support	-0.00001	0.0002
(23)	Education	Purpose in Life	0.001	0.001
(24)	Education	Optimism	-0.0004	0.0004
(25)	Income	Waist Circumference	0.00001	0.002
(26)	Income	Depressive Symptoms	0.0003	0.0003
(27)	Income	Social Support	-0.00001	0.0002
(28)	**Income**	**Purpose in Life**	**0.001***	**0.0004**
(29)	Income	Optimism	-0.0002	0.0002

Sum of Indirect Effect:	0.079 (38.3%)
Direct Effect[1]:	0.127 (61.7%)
Total Effect[2]:	0.206 (100%)

NOTE: b = unstandardized regression coefficient; SE = standard error;

[2]The direct effect is the regression coefficient for the African American dummy variable in Table 6.4, Model 6;

[3]The total effect is the sum of the indirect effects and the direct effect.
* = p<.05; ** = p<.01; *** = p<.001

Table 6.8 Unstandardized Indirect, Direct and Total Effects of Race/Ethnicity on Inflammation: Hispanics and Non-Hispanic Whites

	1st Mediator	2nd Mediator	Indirect Effect b	Indirect Effect SE
Single-Mediator Pathways				
(1)	Everyday Discrimination	---	0.001	0.001
(2)	Lifetime Discrimination	---	-0.003	0.003
(3)	**Education**	**---**	**0.089*** **	**0.018**
(4)	Income	---	-0.004	0.010
(5)	Waist Circumference	---	-0.016	0.021
(6)	Depressive Symptoms	---	0.002	0.002
(7)	Social Support	---	0.00003	0.0006
(8)	Purpose in Life	---	-0.001	0.002
(9)	Optimism	---	0.006	0.006
Two-Mediator Pathways (through Discrimination)				
(10)	Everyday Discrimination	Waist Circumference	-0.002	0.002
(11)	Everyday Discrimination	Depressive Symptoms	-0.0002	0.0002
(12)	Everyday Discrimination	Social Support	0.00001	0.0002
(13)	Everyday Discrimination	Purpose in Life	-0.0005	0.0004
(14)	Everyday Discrimination	Optimism	0.0002	0.0002
(15)	Lifetime Discrimination	Waist Circumference	-0.001	0.001
(16)	Lifetime Discrimination	Depressive Symptoms	-0.0001	0.0001
(17)	Lifetime Discrimination	Social Support	0.000002	0.00005
(18)	Lifetime Discrimination	Purpose in Life	-0.00002	0.00003
(19)	Lifetime Discrimination	Optimism	-0.00000004	0.00002
Two-Mediator Pathways (through Socioeconomic Factors)				
(20)	**Education**	**Waist Circumference**	**0.022*** **	**0.006**
(21)	Education	Depressive Symptoms	0.002	0.002
(22)	Education	Social Support	-0.00003	0.0006
(23)	**Education**	**Purpose in Life**	**0.005***	**0.002**
(24)	Education	Optimism	-0.001	0.001
(25)	Income	Waist Circumference	0.00001	0.003
(26)	Income	Depressive Symptoms	0.001	0.001
(27)	Income	Social Support	-0.00002	0.0003
(28)	**Income**	**Purpose in Life**	**0.002***	**0.0009**
(29)	Income	Optimism	-0.0004	0.0004
		Sum of Indirect Effect:	0.101 (136.5%)	
		Direct Effect[1]:	-0.027 (-36.5%)	
		Total Effect[2]:	0.074 (100%)	

NOTE: b = unstandardized regression coefficient; SE = standard error;

[2]The direct effect is the regression coefficient for the Hispanic dummy variable in Table 6.4, Model 6;

[3]The total effect is the sum of the indirect effects and the direct effect.

* = p<.05; ** = p<.01; *** = p<.001

Table 6.9 Unstandardized Indirect, Direct and Total Effects of Gender on Inflammation

	1st Mediator	2nd Mediator	Indirect Effect b	Indirect Effect SE
Single-Mediator Pathways				
(1)	Everyday Discrimination	---	0.004	0.004
(2)	**Lifetime Discrimination**	**---**	**-0.014****	**0.004**
(3)	Education	---	0.0005	0.002
(4)	Income	---	-0.0001	0.0003
(5)	**Waist Circumference**	**---**	**-0.224*****	**0.012**
(6)	Depressive Symptoms	---	0.001	0.001
(7)	Social Support	---	0.0001	0.003
(8)	Purpose in Life	---	-0.002	0.0009
(9)	Optimism	---	0.001	0.001
Two-Mediator Pathways (through Discrimination)				
(10)	**Everyday Discrimination**	**Waist Circumference**	**-0.007*****	**0.001**
(11)	Everyday Discrimination	Depressive Symptoms	-0.001	0.001
(12)	Everyday Discrimination	Social Support	0.00004	0.0007
(13)	**Everyday Discrimination**	**Purpose in Life**	**-0.002***	**0.0008**
(14)	Everyday Discrimination	Optimism	0.0007	0.0006
(15)	**Lifetime Discrimination**	**Waist Circumference**	**-0.003***	**0.001**
(16)	Lifetime Discrimination	Depressive Symptoms	-0.0003	0.0003
(17)	Lifetime Discrimination	Social Support	0.00001	0.0002
(18)	Lifetime Discrimination	Purpose in Life	-0.0001	0.0001
(19)	Lifetime Discrimination	Optimism	-0.0000002	0.00007
Two-Mediator Pathways (through Socioeconomic Factors)				
(20)	Education	Waist Circumference	0.0001	0.0004
(21)	Education	Depressive Symptoms	0.00001	0.00003
(22)	Education	Social Support	-0.0000002	0.000004
(23)	Education	Purpose in Life	0.00003	0.0001
(24)	Education	Optimism	-0.00001	0.00002
(25)	Income	Waist Circumference	0.0000003	0.0001
(26)	Income	Depressive Symptoms	0.00002	0.00003
(27)	Income	Social Support	-0.0000005	0.00001
(28)	Income	Purpose in Life	0.0001	0.0001
(29)	Income	Optimism	-0.00001	0.00002
		Sum of Indirect Effect:	'-0.246 (-136.7%)	
		Direct Effect[1]:	0.426 (236.7%)	
		Total Effect[2]:	0.18 (100%)	

NOTE: b = unstandardized regression coefficient; SE = standard error;

[2]The direct effect is the regression coefficient for the female dummy variable in Table 6.4, Model 6;

[3]The total effect is the sum of the indirect effects and the direct effect.

* = p<.05; ** = p<.01; *** = p<.001

Table 7.1 Regression of Inflammation on Discrimination and Other Study Variables: Main Effects Model (n = 11,935), Weighted

Study Variables	CRP (ug/mL; [-3.##,5.56]) Model 1: Main Effects Model b	SE
Independent Variable		
Race/Ethnicity (ref. = whites)		
African American	0.127**	0.041
Hispanic	-0.027	0.051
Female (ref. = males)	0.426***	0.027
Stress Exposures		
Everyday Discrimination (range: 0-5)	-0.023	0.022
Lifetime Discrimination (range: 0-6)	0.057***	0.016
Socioeconomic Status		
Age (years)	-0.002	0.002
Education (years)	-0.024***	0.005
Household Income (log-transformed)	0.005	0.011
Employment (ref. = full/part time)		
Retired	0.063	0.035
Other	0.067	0.038
Marital Status (ref. = married)		
Separated/Divorced	0.087	0.046
Widowed	-0.049	0.034
Never Married	-0.079	0.088
Health Behaviors		
Smoking Status (ref. = never smoked)		
Former Smoker	0.093**	0.025
Current Smoker	0.368***	0.041
Alcohol Consumption (ref. = non-drinker)		
Moderate	-0.080*	0.031
Heavy	-0.080	0.045
Vigorous Activity (ref. = no activity)		
≤ Once a Week	-0.100**	0.034
> Once a Week	-0.090**	0.031
Moderate Activity (ref. = no activity)		
≤ Once a Week	-0.092*	0.037
> Once a Week	-0.138**	0.044
Health Status		
Waist Circumference (inches)	0.065***	0.002
Depressive Symptoms (range: 0-8)	0.006	0.007
Coping Resources		
Social Support (range: 0-4)	0.001	0.026
Purpose in Life (range: 0-5)	-0.035*	0.016
Optimism (range: 0-5)	0.015	0.014
Control Variable		
2008 Data Collection	0.051*	0.025
Constant	-1.617***	0.250
Indicator(s) of Model Fit		
R^2	0.656	
ΔR^2	---	
F(d.f.) [Wald Test]	---	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 7.2 Regression of Waist Circumference on Discrimination and Other Study Variables: Main Effects and Conditional Model (n = 11,935), Weighted

	Waist Circumference (inches)			
	Model 1: Main Effects Model		Model 5: Conditional Model	
Study Variables	b	SE	b	SE
Independent Variable				
Race/Ethnicity (ref. = whites)				
African American	0.266	0.208	0.249	0.209
Hispanic	-0.240	0.325	-0.242	0.325
Female (ref. = males)	-3.476***	0.163	-3.497***	0.164
Stress Exposures				
Everyday Discrimination (mean-centered)	0.631***	0.110	0.627***	0.110
Lifetime Discrimination (mean-centered)	0.209*	0.084	0.082	0.087
Gender-x-Discrimination Interaction				
Lifetime-x-Female	---	---	0.308*	0.132
Socioeconomic Status				
Age (years)	-0.093***	0.009	-0.093***	0.009
Education (years)	-0.092***	0.023	-0.092***	0.023
Household Income (log-transformed)	-0.0002	0.060	-0.001	0.060
Employment (ref. = full/part time)				
Retired	0.824***	0.205	0.828***	0.207
Other	0.508	0.261	0.539*	0.265
Marital Status (ref. = married)				
Separated/Divorced	0.274	0.248	0.254	0.251
Widowed	0.320	0.179	0.332	0.180
Never Married	0.315	0.286	0.297	0.284
Health Behaviors				
Smoking Status (ref. = never smoked)				
Former Smoker	0.743***	0.128	0.731***	0.130
Current Smoker	-1.870***	0.198	-1.874***	0.200
Alcohol Consumption (ref. = non-drinker)				
Moderate	-0.602***	0.149	-0.603***	0.148
Heavy	-1.280***	0.246	-1.284***	0.246
Vigorous Activity (ref. = no activity)				
≤ Once a Week	-1.051***	0.183	-1.050***	0.182
> Once a Week	-1.810***	0.139	-1.804***	0.139
Moderate Activity (ref. = no activity)				
≤ Once a Week	-1.672***	0.197	-1.665***	0.196
> Once a Week	-2.791***	0.168	-2.795***	0.168
Control Variable				
2008 Data Collection	0.187	0.158	0.192	0.158
Constant	50.848***	1.044	51.348***	1.033
Indicator(s) of Model Fit				
R^2	0.656		0.673	
ΔR^2	---		0.017	
F(d.f.) [Wald Test]	---		5.44 (1,63)*	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

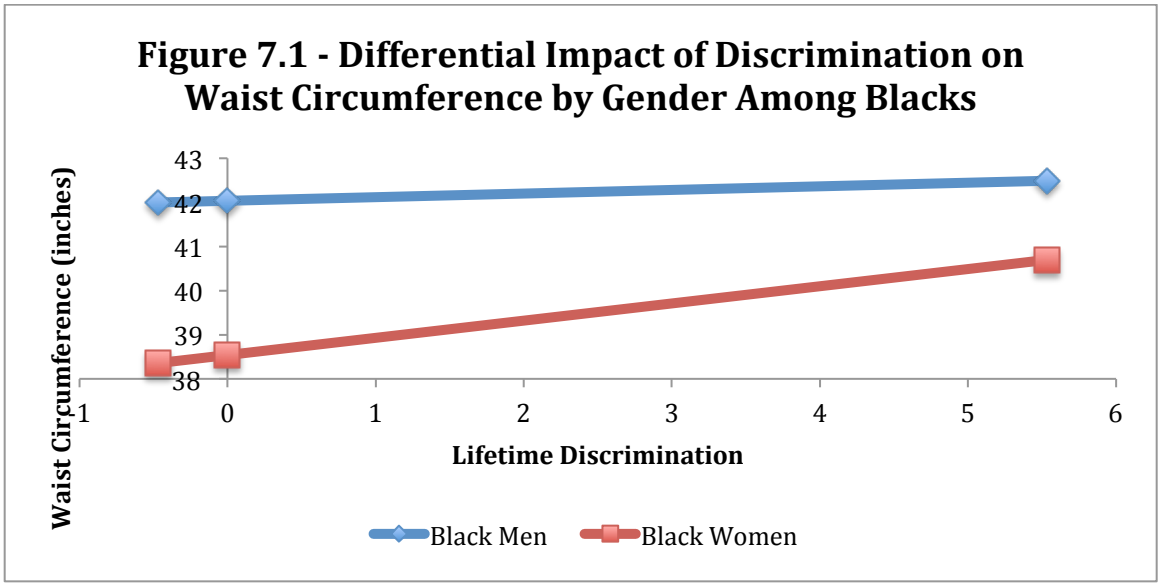


Figure 7.1 - Differential Impact of Discrimination on Waist Circumference by Gender Among Blacks
Waist Circumference (inches)
43
42
41
40
39
38
-1
0
1
2
3
4
5
6
Lifetime Discrimination
Black Men
Black Women

Table 7.3 Regression of Depressive Symptoms on Discrimination and Other Study Variables: Main Effects and Conditional Model (n = 11,935), Weighted

	Depressive Symptoms			
	Model 1: Main Effects Model		Model 3: Conditional Model	
Study Variables	b	SE	b	SE
Independent Variable				
Race/Ethnicity (ref. = whites)				
African American	-0.075	0.067	-0.009	0.075
Hispanic	0.352**	0.111	0.348**	0.108
Female (ref. = males)	0.148***	0.042	0.150**	0.042
Stress Exposures				
Everyday Discrimination (mean-centered)	0.497***	0.046	0.496***	0.046
Lifetime Discrimination (mean-centered)	0.185***	0.032	0.197***	0.041
Race-x-Discrimination Interaction				
Lifetime-by-Black	---	---	-0.144*	0.069
Lifetime-by-Hispanic	---	---	0.196	0.108
Socioeconomic Status				
Age (years)	-0.019***	0.003	-0.018***	0.003
Education (years)	-0.072***	0.009	-0.072***	0.009
Household Income (log-transformed)	-0.104***	0.022	-0.103***	0.022
Employment (ref. = full/part time)				
Retired	0.338***	0.054	0.340***	0.053
Other	0.627***	0.076	0.631***	0.077
Marital Status (ref. = married)				
Separated/Divorced	0.520***	0.074	0.522***	0.075
Widowed	0.501***	0.053	0.500***	0.053
Never Married	0.240*	0.114	0.239*	0.113
Health Behaviors				
Smoking Status (ref. = never smoked)				
Former Smoker	0.025	0.050	0.023	0.05
Current Smoker	0.267**	0.087	0.263**	0.088
Alcohol Consumption (ref. = non-drinker)				
Moderate	-0.144**	0.043	-0.145**	0.043
Heavy	-0.093	0.085	-0.097	0.085
Vigorous Activity (ref. = no activity)				
≤ Once a Week	-0.196**	0.058	-0.198**	0.059
> Once a Week	-0.354***	0.050	-0.353***	0.049
Moderate Activity (ref. = no activity)				
≤ Once a Week	-0.787***	0.070	-0.785***	0.069
> Once a Week	-0.888***	0.080	-0.887***	0.079
Control Variable				
2008 Data Collection	-0.040	0.043	-0.038	0.043
Constant	4.617***	0.336	4.984***	0.330
Indicator(s) of Model Fit				
R^2	0.657		0.673	
ΔR^2	---		0.016	
F(d.f.) [Wald Test]	---		3.72 (2,62)*	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

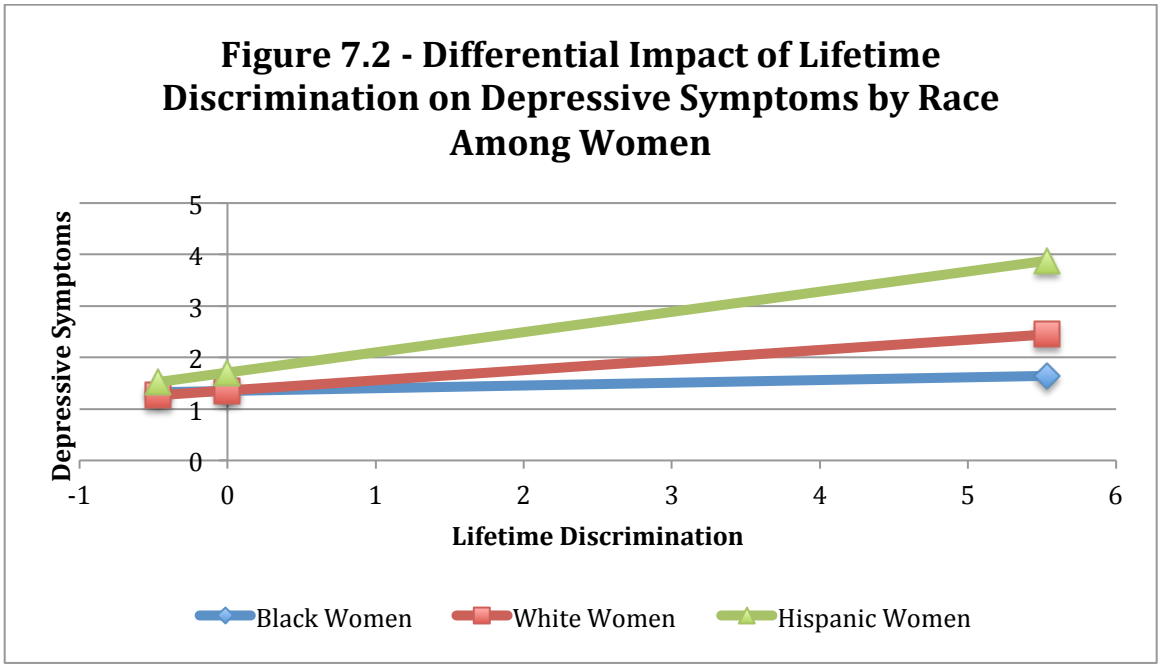


Figure 7.2 - Differential Impact of Lifetime Discrimination on Depressive Symptoms by Race Among Women
Depressive Symptoms
5
4
3
2
1
0
-1
0
1
2
3
4
5
6
Lifetime Discrimination
Black Women
White Women
Hispanic Women

Table 7.4 Regression of Social Support on Discrimination and Other Study Variables: Main Effects and Conditional Model (n = 11,935), Weighted

	Social Support			
	Model 1: Main Effects Model		Model 5: Conditional Model	
Study Variables	b	SE	b	SE
Independent Variable				
Race/Ethnicity (ref. = whites)				
African American	0.099***	0.022	0.101***	0.022
Hispanic	0.024	0.025	0.025	0.025
Female (ref. = males)	0.112***	0.014	0.116***	0.015
Stress Exposures				
Everyday Discrimination (mean-centered)	-0.153***	0.010	-0.153***	0.010
Lifetime Discrimination (mean-centered)	-0.033***	0.008	-0.015	0.013
Gender-x-Discrimination Interaction				
Lifetime-by-Female	---	---	-0.041*	0.020
Socioeconomic Status				
Age (years)	0.003**	0.001	0.003**	0.001
Education (years)	0.006*	0.003	0.006*	0.003
Household Income (log-transformed)	0.014	0.007	0.014	0.007
Employment (ref. = full/part time)				
Retired	-0.008	0.019	-0.008	0.019
Other	-0.015	0.027	-0.019	0.028
Marital Status (ref. = married)				
Separated/Divorced	-0.039	0.024	-0.037	0.024
Widowed	0.011	0.017	0.009	0.017
Never Married	-0.295***	0.063	-0.293***	0.063
Health Behaviors				
Smoking Status (ref. = never smoked)				
Former Smoker	-0.016	0.013	-0.014	0.012
Current Smoker	-0.002	0.019	-0.001	0.019
Alcohol Consumption (ref. = non-drinker)				
Moderate	0.025	0.014	0.025	0.014
Heavy	0.013	0.030	0.014	0.030
Vigorous Activity (ref. = no activity)				
≤ Once a Week	0.043*	0.017	0.043*	0.017
> Once a Week	0.047**	0.015	0.046**	0.015
Moderate Activity (ref. = no activity)				
≤ Once a Week	0.055**	0.017	0.054**	0.017
> Once a Week	0.083***	0.015	0.083***	0.015
Control Variable				
2008 Data Collection	-0.007	0.014	-0.008	0.014
Constant	2.687***	0.122	2.573***	0.120
Indicator(s) of Model Fit				
R^2	0.656		0.673	
ΔR^2	---		0.017	
F(d.f.) [Wald Test]	---		4.27 (1,63)*	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

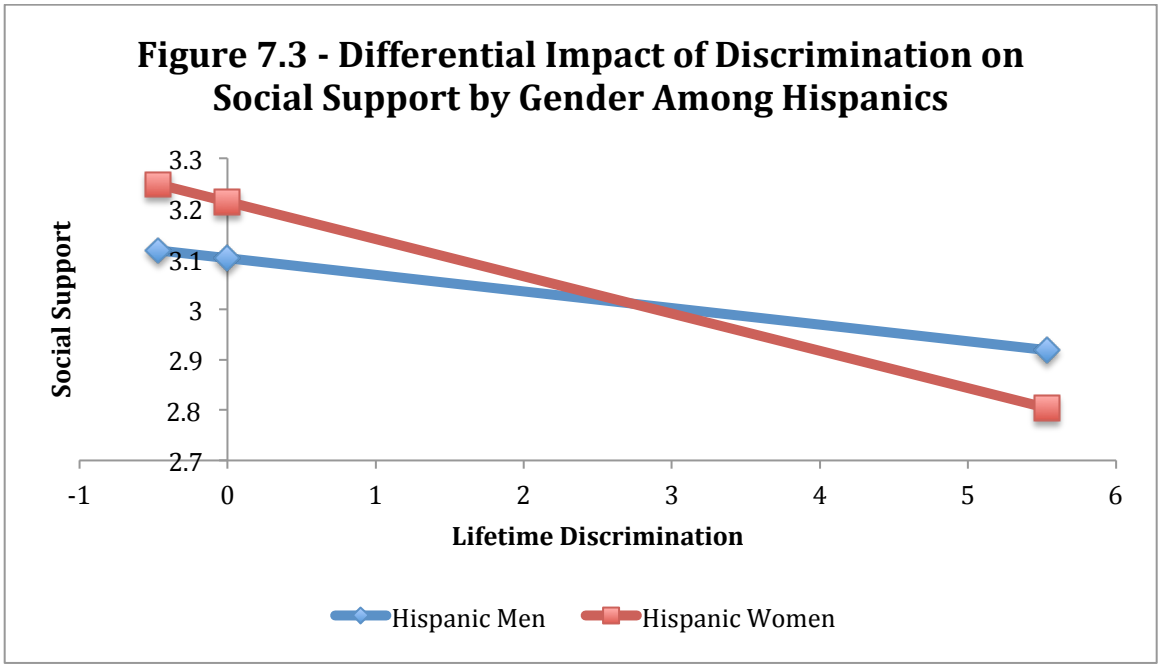


Figure 7.3 - Differential Impact of Discrimination on Social Support by Gender Among Hispanics
Social Support
3.3
3.2
3.1
3
2.9
2.8
2.7
-1
0
1
2
3
4
5
6
Lifetime Discrimination
Hispanic Men
Hispanic Women

Table 7.5 Regression of Purpose in Life on Discrimination and Other Study Variables: Main Effects and Conditional Model (n = 11,935), Weighted

Study Variables	Purpose in Life Model 1: Main Effects Model b	SE
Independent Variable		
Race/Ethnicity (ref. = whites)		
African American	0.367***	0.034
Hispanic	0.024	0.043
Female (ref. = males)	0.047*	0.018
Stress Exposures		
Everyday Discrimination (range: 0-5)	-0.284***	0.018
Lifetime Discrimination (range: 0-6)	-0.010	0.012
Socioeconomic Status		
Age (years)	-0.007***	0.002
Education (years)	0.037***	0.004
Household Income (log-transformed)	0.061***	0.012
Employment (ref. = full/part time)		
Retired	-0.093**	0.029
Other	-0.165***	0.036
Marital Status (ref. = married)		
Separated/Divorced	-0.097**	0.035
Widowed	-0.164***	0.029
Never Married	-0.237***	0.055
Health Behaviors		
Smoking Status (ref. = never smoked)		
Former Smoker	-0.052*	0.025
Current Smoker	-0.159***	0.042
Alcohol Consumption (ref. = non-drinker)		
Moderate	0.021	0.022
Heavy	0.026	0.040
Vigorous Activity (ref. = no activity)		
≤ Once a Week	0.146***	0.028
> Once a Week	0.217***	0.024
Moderate Activity (ref. = no activity)		
≤ Once a Week	0.279***	0.025
> Once a Week	0.354***	0.032
Control Variable		
2008 Data Collection	0.102***	0.023
Constant	2.803***	0.191
Indicator(s) of Model Fit		
R^2	0.656	
ΔR^2	---	
F(d.f.) [Wald Test]	---	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 7.6 Regression of Optimism on Discrimination and Other Study Variables: Main Effects Model (n = 11,935), Weighted

Study Variables	Optimism Model 1: Main Effects Model b	 SE
Independent Variable		
Race/Ethnicity (ref. = whites)		
African American	0.439***	0.059
Hispanic	0.416***	0.064
Female (ref. = males)	0.036	0.027
Stress Exposures		
Everyday Discrimination (range: 0-5)	-0.248***	0.024
Lifetime Discrimination (range: 0-6)	0.00005	0.020
Socioeconomic Status		
Age (years)	0.006**	0.002
Education (years)	0.023***	0.005
Household Income (log-transformed)	0.030	0.015
Employment (ref. = full/part time)		
Retired	-0.084	0.042
Other	-0.063	0.048
Marital Status (ref. = married)		
Separated/Divorced	-0.126*	0.052
Widowed	-0.046	0.034
Never Married	-0.187*	0.078
Health Behaviors		
Smoking Status (ref. = never smoked)		
Former Smoker	-0.005	0.034
Current Smoker	-0.046	0.049
Alcohol Consumption (ref. = non-drinker)		
Moderate	0.054*	0.023
Heavy	0.036	0.050
Vigorous Activity (ref. = no activity)		
≤ Once a Week	0.088*	0.034
> Once a Week	0.153***	0.035
Moderate Activity (ref. = no activity)		
≤ Once a Week	0.150**	0.049
> Once a Week	0.189***	0.047
Control Variable		
2008 Data Collection	-0.052	0.032
Constant	2.475***	0.257
Indicator(s) of Model Fit		
R^2	0.656	
ΔR^2	---	
F(d.f.) [Wald Test]	---	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 7.7 Regression of Inflammation on Social Support and Other Study Variables: Main Effects and Conditional Model (n = 11,935); Weighted

Study Variables	CRP (ug/mL) (range: -3.9-5.5)	
	Main Effects Model	
	b	SE
Independent Variable		
Race/Ethnicity (ref. = whites)		
African American	0.127**	0.041
Hispanic	-0.027	0.051
Female (ref. = males)	0.426***	0.027
Stress Exposures		
Everyday Discrimination (range: 0-5)	-0.023	0.022
Lifetime Discrimination (range: 0-6)	0.057***	0.016
Socioeconomic Status		
Age (years)	-0.002	0.002
Education (years)	-0.024***	0.005
Household Income (log-transformed)	0.005	0.011
Employment (ref. = full/part time)		
Retired	0.063	0.035
Other	0.067	0.038
Marital Status (ref. = married)		
Separated/Divorced	0.087	0.046
Widowed	-0.049	0.034
Never Married	-0.079	0.088
Health Behaviors		
Smoking Status (ref. = never smoked)		
Former Smoker	0.093**	0.025
Current Smoker	0.368***	0.041
Alcohol Consumption (ref. = non-drinker)		
Moderate	-0.080*	0.031
Heavy	-0.08	0.045
Vigorous Activity (ref. = no activity)		
≤ Once a Week	-0.100**	0.034
> Once a Week	-0.090**	0.031
Moderate Activity (ref. = no activity)		
≤ Once a Week	-0.092*	0.037
> Once a Week	-0.138**	0.044
Health Status		
Waist Circumference (inches)	0.065***	0.002
Depressive Symptoms (range: 0-8)	0.006	0.007
Coping Resources		
Social Support (range: 0-4)	0.001	0.026
Purpose in Life (range: 0-5)	-0.035*	0.016
Optimism (range: 0-5)	0.015	0.014
Control Variable		
2008 Data Collection	0.051*	0.025
Constant	-1.617***	0.250
Indicator(s) of Model Fit		
R^2	0.656	
ΔR^2	---	
F(d.f.) [Wald Test]	---	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

Table 7.8 Regression of Inflammation on Purpose in Life and Other Study Variables: Main Effects and Conditional Model (n = 11,935); Weighted

	CRP (ug/mL; -3.9,5.5)			
	Model 1: Main Effects Model		Model 5: Conditional Model	
Study Variables	b	SE	b	SE
Independent Variable				
Race/Ethnicity (ref. = whites)				
African American	0.127**	0.041	0.129**	0.041
Hispanic	-0.027	0.051	-0.022	0.051
Female (ref. = males)	0.426***	0.027	0.423***	0.027
Coping Resources				
Social Support (range: 0-4)	0.001	0.026	0.003	0.026
Purpose in Life (mean-centered)	-0.035*	0.016	-0.079***	0.018
Optimism (range: 0-5)	0.015	0.014	0.015	0.014
Gender-x-Resource Interaction				
Purpose-by-Female	---	---	0.079**	0.025
Stress Exposures				
Everyday Discrimination (range: 0-5)	-0.023	0.022	-0.023	0.021
Lifetime Discrimination (range: 0-6)	0.057***	0.016	-0.057**	0.016
Socioeconomic Status				
Age (years)	-0.002	0.002	-0.002	0.002
Education (years)	-0.024***	0.005	-0.024***	0.005
Household Income (log-transformed)	0.005	0.011	0.004	0.011
Employment (ref. = full/part time)				
Retired	0.063	0.035	0.062	0.035
Other	0.067	0.038	0.068	0.038
Marital Status (ref. = married)				
Separated/Divorced	0.087	0.046	0.087	0.046
Widowed	-0.049	0.034	-0.041	0.034
Never Married	-0.079	0.088	-0.083	0.089
Health Behaviors				
Smoking Status (ref. = never smoked)				
Former Smoker	0.093**	0.025	0.093**	0.025
Current Smoker	0.368***	0.041	0.366***	0.041
Alcohol Consumption (ref. = non-drinker)				
Moderate	-0.080*	0.031	-0.081*	0.031
Heavy	-0.080	0.045	-0.082	0.045
Vigorous Activity (ref. = no activity)				
≤ Once a Week	-0.100**	0.034	-0.099**	0.034
> Once a Week	-0.090**	0.031	-0.089**	0.031
Moderate Activity (ref. = no activity)				
≤ Once a Week	-0.092*	0.037	-0.095*	0.037
> Once a Week	-0.138**	0.044	-0.137**	0.043
Health Status				
Waist Circumference (inches)	0.065***	0.002	0.065***	0.002
Depressive Symptoms (range: 0-8)	0.006	0.007	0.007	0.007
Control Variable				
2008 Data Collection	0.051*	0.025	0.052*	0.025
Constant	-1.617***	0.250	-1.743***	0.227
Indicator(s) of Model Fit				
R^2	0.656		0.672	
ΔR^2	---		0.016	
F(d.f.) [Wald Test]	---		9.80 (1,63)**	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

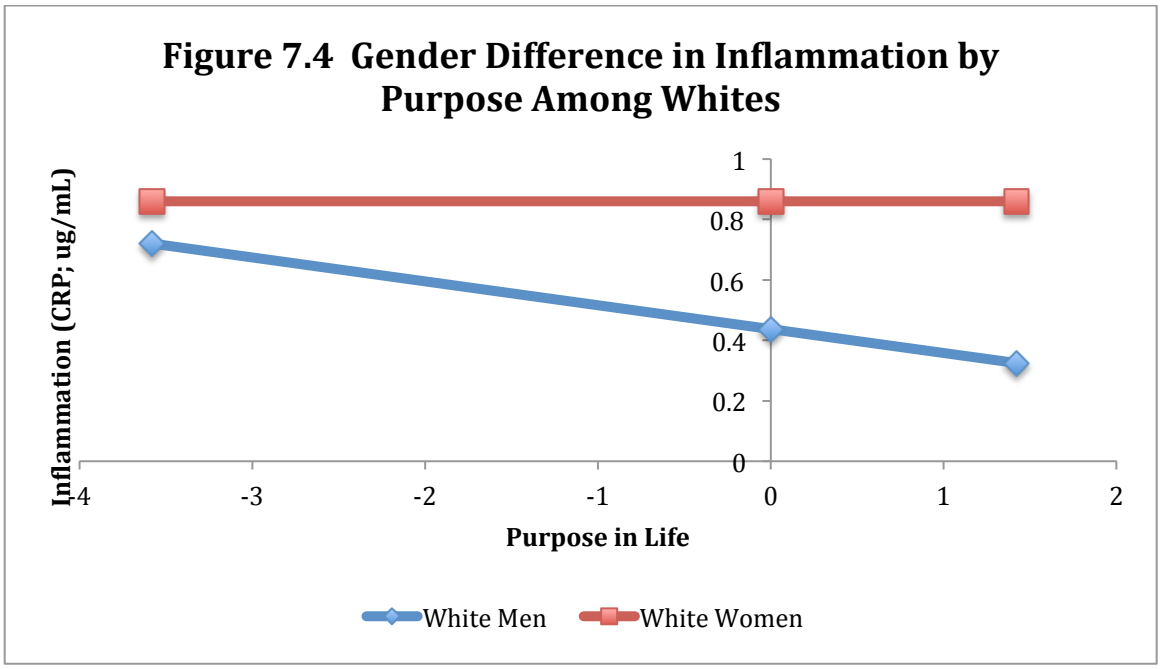


Figure 7.4 Gender Difference in Inflammation by Purpose Among Whites
Inflammation (CRP; ug/mL)
1
0.8
0.6
0.4
0.2
0
-4
-3
-2
-1
0
1
2
Purpose in Life
White Men
White Women

Table 7.9 Regression of Inflammation on Optimism and Other Study Variables: Main Effects and Conditional Model (n = 11,935); Weighted

Study Variables	CRP (ug/mL) (range: -3.9-5.5) Main Effects b	SE
Independent Variable		
Race/Ethnicity (ref. = whites)		
African American	0.127**	0.041
Hispanic	-0.027	0.051
Female (ref. = males)	0.426***	0.027
Coping Resources		
Social Support (range: 0-4)	0.001	0.026
Purpose in Life (mean-centered)	-0.035*	0.016
Optimism (range: 0-5)	0.015	0.014
Gender-x-Resource Interaction		
Purpose-by-Female	---	---
Stress Exposures		
Everyday Discrimination (range: 0-5)	-0.023	0.022
Lifetime Discrimination (range: 0-6)	0.057***	0.016
Socioeconomic Status		
Age (years)	-0.002	0.002
Education (years)	-0.024***	0.005
Household Income (log-transformed)	0.005	0.011
Employment (ref. = full/part time)		
Retired	0.063	0.035
Other	0.067	0.038
Marital Status (ref. = married)		
Separated/Divorced	0.087	0.046
Widowed	-0.049	0.034
Never Married	-0.079	0.088
Health Behaviors		
Smoking Status (ref. = never smoked)		
Former Smoker	0.093**	0.025
Current Smoker	0.368***	0.041
Alcohol Consumption (ref. = non-drinker)		
Moderate	-0.080*	0.031
Heavy	-0.080	0.045
Vigorous Activity (ref. = no activity)		
≤ Once a Week	-0.100**	0.034
> Once a Week	-0.090**	0.031
Moderate Activity (ref. = no activity)		
≤ Once a Week	-0.092*	0.037
> Once a Week	-0.138**	0.044
Health Status		
Waist Circumference (inches)	0.065***	0.002
Depressive Symptoms (range: 0-8)	0.006	0.007
Control Variable		
2008 Data Collection	0.051*	0.025
Constant	-1.617***	0.250
Indicator(s) of Model Fit		
R^2	0.656	
ΔR^2	---	
F(d.f.) [Wald Test]	---	

NOTE: b = unstandardized regression coefficients; SE = standard error;
* = p<.05; ** = p<.01; *** = p<.001

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