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AN INTERSECTIONAL PERSPECTIVE ON THE RELATIONSHIPS AMONG SOCIAL STATUS, SELF-REPORTED DISCRIMINATION, AND LOW-GRADE C-REACTIVE PROTEIN IN THE HEALTH AND RETIREMENT STUDY

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ABSTRACT

Background: A broad literature has documented social patterning of health, such that those with lower social status (e.g., racial minorities, women, and people of lower socioeconomic status) bear a disproportionate burden of morbidity and mortality relative to those with higher status. Disparities may be larger for some individuals, particularly those who are socially disadvantaged in more than one status. Variations in stress exposure, like experiences of discrimination, and resulting low-grade inflammation, may explain such disparities. A limited body of literature has examined the social distribution of low-grade inflammation, which is implicated in chronic diseases that are responsible for excess deaths in the United States. Limited work has examined how social statuses might interact to predict inflammatory markers like C-reactive protein (CRP), or tested psychosocial mechanisms responsible for the social distribution of CRP. **Objective:** The present study will examine the relationship between race, gender, and SES; everyday and lifetime discrimination exposure; and CRP. Aim 1 will test whether an interaction of race, gender, and SES are associated with CRP levels. Aim 2 will assess how race, gender, and SES shape exposure to lifetime and everyday discrimination, and whether this discrimination exposure mediates the relationship between race x gender x SES and CRP. **Methods:** Data were drawn from 5,486 respondents in the Health and Retirement Study, a nationally representative sample of midlife and older adults, to analyze the relationships between social status (e.g., race, gender, SES), everyday discrimination, lifetime discrimination, and CRP levels at baseline and after a four-year follow-up. **Results:** Aim 1 study results demonstrate that race, gender, and SES interact to produce differential CRP levels at baseline and follow-up. The results suggest that there are incremental benefits for each additional level of SES for all race and gender groups except Black women. Significant three-way interactions of race, gender, and SES also indicate that Black women experience higher CRP levels with increases in SES, and that Black men with low SES are have

the highest levels of CRP. Aim 2 study results show that everyday and lifetime discrimination exposure varies across social status groups. However, there were no significant interactions among race, gender, and SES on discrimination exposure. Further, results showed that both everyday and lifetime exposure to discrimination were significantly associated with CRP levels at baseline but not with change in CRP over four years. **Implications:** In clarifying the complexity inherent in disparities in low-grade inflammation, as well as potential psychosocial mechanisms responsible for these mechanisms, this work will contribute to a greater understanding of the factors underlying major causes of excess morbidity and mortality in the United States, and may identify potential intervention points for addressing health disparities.

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Thank you!

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CHAPTER 1

INTRODUCTION

The United States is undergoing rapid growth in its midlife and older adult population (those aged 50+), but especially in those aged 65 and older; this population is projected to dramatically increase over the next fifty years (Ortman, Velkoff, & Hogan, 2014). In addition to this growth, the United States will also have its most racially diverse older population (Ortman, Velkoff, & Hogan, 2014). A recent publication by the United States Census Bureau showed that about 8.8% of the older population was Black in 2012; projections show that by 2050, this percentage will increase to 12.3% (Ortman, Velkoff, & Hogan, 2014). With the increasing diversity of the older population, it is imperative to better understand and characterize the unique experiences of older minorities in order to help them age optimally, reduce premature morbidity and mortality, as well as to reduce excess costs to the economy. Widely studied psychosocial factors that contribute to the wellbeing, morbidity, life expectancy, and mortality of Black compared to White adults throughout their lives include the disproportionate burden of the effects of racial segregation, low socioeconomic status, and more frequent exposure to discrimination (Williams & Jackson, 2005).

Cardiovascular disease (CVD) is currently the leading cause of mortality in the United States for adults aged 65 and older (Heron, 2015), and presents a major economic burden to the country. Projections show that by 2030, CVD will cost the United States a total of over $1 trillion in both direct and indirect costs (Heidenreich et al., 2011). Although these issues affect many people, some are disproportionately affected: a broad literature consistently shows that health is patterned along social lines, with extant research indicating that there are social status (e.g., racial, gender, and socioeconomic) disparities in the prevalence (Mensah & Brown, 2007; Mensah, Mokdad, Ford, Greenlund, & Croft, 2005), risk factors (Ailshire & House, 2011;

Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009; Kanjilal et al., 2006; Karlamangla, Merkin, Crimmins, & Seeman, 2010; Kurian & Cardarelli, 2007; Richardson & Brown, 2016; Romero, Romero, Shlay, Ogden, & Dabelea, 2012), and mortality (Mensah & Brown, 2007) for CVD.

Thus, the long-term value of the following work is that it can provide information that will allow us to better address the needs of the midlife and older population and, in turn, to reduce the costs to society. This dissertation aims to clarify the social distribution of health in midlife and older populations and add to the literature on the social patterning of biological and psychosocial mechanisms responsible for health problems, such as CVD, by addressing the following questions:

(1) Do social statuses (e.g., race, gender, and socioeconomic status) interact and relate to disparities in low-grade C-reactive protein, a stress-associated biological risk factor for CVD-related morbidity and mortality?

(2) Is exposure to everyday and lifetime discrimination a psychosocial mechanism responsible for these disparities?

In Chapter 2, a literature review will provide a summary of the existing research on: (1) three indicators of social status (race, gender, and socioeconomic status [SES]) and their relationship to overall health in midlife and older ages; (2) C-reactive protein (CRP), a predictor of disease and biomarker that captures chronic stress exposure; (3) and the distribution of CRP across three social statuses—race, gender, and SES. It also summarizes the relationship between discrimination and health, the social distribution of discrimination exposure, and biological pathways that link social status and resulting exposure to discrimination to negative health outcomes. In Chapter 3, I discuss the four theoretical frameworks that inform this work, then I present and describe the conceptual model for this dissertation, which integrates these four

existing frameworks to form a new approach to conceptualize health disparities. In addition, the two aims guiding this dissertation and the research questions are posed. In Chapter 4, I detail the data source for this work and describe the methods that were used to examine the research questions. The results of the proposed aims are presented in Chapter 5. Lastly, Chapter 6 will conclude with a summary and comprehensive discussion of the findings and their relation to the current literature, and then I review the implications of this work, discuss the limitations of this study, address areas for future research to expand on the knowledge that results from this work, and finally, I address points of intervention and areas that may inform policy regarding health disparities.

CHAPTER 2

LITERATURE REVIEW

This literature review will provide a detailed definition of social status and will then describe how race, socioeconomic status (SES), and gender are key facets of social status that are linked to poor health outcomes. Then, I will highlight the role of stress exposure as an important mechanism linking social status to negative health outcomes, particularly focusing on everyday and lifetime discrimination exposure as stressors that disproportionately affect those who belong to lower social status groups. Finally, I will describe the way that C-reactive protein, an indicator of systemic inflammation, may play a role in linking social status and discrimination exposure to adverse health outcomes.

SOCIAL STATUS

Social status is a broad and multidimensional term often used to describe a set of power relations in society based on social and physical characteristics that can be described either as ascribed or achieved. *Ascribed* factors are those attached to a person at birth, (e.g., race/ethnicity, gender, and age). *Achieved* factors are those earned when one is provided with sufficient access to opportunities and resources, and encompass measures of socioeconomic status (SES) such as education, occupation, income, and wealth (Alwin & Wray, 2005).

Social status is an important predictor of health status because it serves as a mechanism for inequality: it provides individuals with a differential set of experiences, opportunities, and resources that can either protect or damage health (Alwin & Wray, 2005; Link & Phelan, 1995; Williams & Mohammed, 2009). Lower status groups (e.g., racial/ethnic minorities, women, and

those with lower SES) are disproportionately exposed to stressors (Turner & Avison, 2003; Williams & Mohammed, 2009), which suggests that stress exposure is potentially a major pathway linking social status to adverse health outcomes, particularly those seen in midlife and older ages where the cumulative effect of stressors on health is more apparent. This is hypothesized to work primarily through cumulative exposure to stressors and lack of protective resources to mitigate the negative effects of stress on health (Alwin & Wray, 2005; Dannefer, 2003; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin, 1989; Singh-Manoux, Adler, & Marmot, 2003; Turner, 2013; Turner & Avison, 2003). As a result, social status is inextricably linked to a wide array of health outcomes, such as cardiovascular disease, physical disability, and type 2 diabetes (Alwin & Wray, 2005; Graham, 2015; Hatch & Dohrenwend, 2007; Kung, Hoyert, Xu, & Murphy, 2008; Ski, King-Shier, & Thompson, 2014; Williams & Mohammed, 2009). There is mounting evidence that belonging in more than one lower social status group may result in even poorer health than belonging in one or no lower status groups (Grollman, 2012; 2014); and a growing but limited literature has suggested that social statuses may interact to shape exposure to stressors and, thus, increase risk of subsequent health outcomes (Ailshire & House, 2011; Brown & Hargrove, 2013; Richardson & Brown, 2016; Khera et al., 2005).

Race

Large and pervasive health disparities exist between non-Hispanic Black/African-American and non-Hispanic White/Caucasian people (hereafter referred to as Black and White) for a wide variety of physical problems, including heart disease, type 2 diabetes, physical disability, and stroke (Graham, 2015; Kung, Hoyert, Xu, & Murphy, 2008; Ski, King-Shier, & Thompson, 2014). For several of these conditions, evidence demonstrates that disparities have endured or grown over the past fifty years. While some Black-White gaps are decreasing (e.g.,

mortality), Blacks still experience excess morbidity and mortality compared to Whites and these disparities exist at all levels of education (Case & Deaton, 2015; Jemal, Ward, Anderson, Murray, & Thu, 2008; Williams & Jackson, 2005; Williams & Sternthal, 2010). In addition to disparities in disease prevalence, incidence, and mortality, Blacks (and Black women in particular) have more risk factors for CVD, such as hypertension, chronic low-grade inflammation, and lifestyle-related risk factors like obesity (Geronimus, Bound, Keene, & Hicken, 2007; Jackson et al., 2013; Kurian & Cardarelli, 2007; Khera et al., 2005; Lu et al., 2016; Nazmi & Victora, 2007; Richardson & Brown, 2016).

Despite early ideologically-based speculations that racial differences in health were primarily due to biological inferiority of racial minorities compared to Whites (see Williams & Sternthal, 2010 for review), race groups and the differences in their health status are now widely recognized as the result of the diverging social and economic contexts of racial minorities versus Whites rather than due to innate biological differences (Du Bois, 1899; Krieger, Rowley, Herman, & Avery, 1993; Link & Phelan, 1995; Williams & Sternthal, 2010). The leading mechanisms believed to shape contemporary racial disparities in health include: socioeconomic status, residential racial segregation, interpersonal and structural discrimination, increased exposure to other stressors, as well as increased exposure to hazardous living and occupational settings which may be more likely to expose Blacks to pollution, violence, and toxins (Krieger et al., 1993; Williams, 2012; Williams & Jackson, 2005; Williams & Mohammed, 2009; Williams, Mohammed, Leavell, & Collins, 2010).

For example, Blacks are overrepresented in lower SES groups. Further, for any given indicator of SES, Blacks receive fewer returns for higher achieved status (Williams & Mohammed, 2009). Compared to Whites, Blacks receive less income for similar levels of education, have higher unemployment levels and lower status positions at the same level of education, and have less wealth at every level of income. All of these differences likely contribute

to the poorer health observed for Blacks compared to Whites (Farmer & Ferraro, 2005; Krieger et al., 1993; Williams & Collins, 1995). Many respected scholars argue that these observations are a function of racialized inequality resulting in racial hierarchies because it is embedded into the very structure of the United States; and racism represents a critical factor shaping Blacks' achieved status and subsequent health (Geronimus, 1992; Geronimus, Hicken, Keene, & Bound, 2006; Williams, 1999; Williams, Priest, & Anderson, 2016).

Although SES differences account for race gaps in some health outcomes, a growing, though inconsistent, body of work shows that there are residual race gaps remain at each level of SES. This suggests that additional factors must account for Black-White gaps in health (Do, Frank, & Finch, 2012; Williams, Mohammed, Leavell, & Collins, 2010). In a recent study examining the effects of race and SES on C-reactive protein (CRP), a measure of systemic inflammation, Dinwiddle, Zambrana, Doamekpor and Lopez (2016) found that SES, measured as income and education, was important in explaining differences between Black and White adults. However, income and education did not fully account for Blacks' higher CRP. In addition, this study found that adjustment for traditional anthropometric risk factors and lifestyle risk factors reduced, but still did not explain the residual gap in CRP between Blacks and Whites. Krieger and colleagues (1993) suggest that assuming that SES is a mediator rather than a moderator of racial disparities in health is a major limitation of the current literature, and recommend that additional literature explore the role of SES as a moderator in race-health relationships.

In addition to experiencing higher rates of morbidity and mortality, Blacks also show evidence of advanced physiological deterioration at earlier ages than Whites, and earlier onset of chronic diseases compared to Whites. Geronimus (1992; 2006) refers to this phenomenon as 'weathering,' which she suggests results largely due to cumulative exposure to stressors, social and economic adversity, and other hazards arising from the disadvantaged social contexts that many Blacks live, work, and reside in, as a function of racialized inequality. Geronimus and

colleagues (2006) also point out that in addition to living in a race-conscious society with the attending social, economic, and psychological disadvantages associated with being a racial minority, Black women may be vulnerable to physiological deterioration because of the synergistic effects associated with being a woman of color. As a result, Black women may experience additional burdens (such as consequent gender- and race-based discrimination, but also gendered race-based discrimination) that contribute to their poorer health relative to Black men, White women, and White men. Geronimus and colleagues (2006) argue that the cumulative impact of these unequal social conditions lead to early physiological deterioration, earlier onset of disease, and contribute to gaps in morbidity and mortality between Blacks and Whites, and particularly for women. In their work, they made two major discoveries: (1) Blacks who were not poor showed evidence of greater physiological deterioration than did poor Whites, and (2) poor Black women were the most disadvantaged group. These results point to the need to explore the cumulative role that stressors such as discrimination may play in explaining poorer health outcomes experienced by lower SES Black women.

Socioeconomic Status (SES)

Link & Phelan (1995) describe SES as a "fundamental cause" of health, wellbeing, and mortality. In their seminal paper, they explain that SES is a multidimensional construct that encompasses one's access to key health-sustaining resources such as knowledge, prestige, power, and opportunities, all of which have the potential to shape health, regardless of the outcome or geographical, historical, or social context (Link & Phelan, 1995). Indeed, a large body of research supports this claim: people with lower SES (regardless of whether it is measured by education, occupation, income, wealth, etc.) show greater morbidity and mortality on a wide array of health indicators, including but not limited to higher prevalence of and mortality from CVD (Ski, King-

Shier, & Thompson, 2014), incidence of and mortality due to stroke (Addo et al., 2012), allostatic load (Dowd, Simanek, & Aiello, 2009), systemic levels of C-reactive protein (Nazmi & Victora, 2007) and prevalence of physical health problems throughout their lives (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010).

Although measures of SES have been consistently linked to negative physical and mental health outcomes, few studies to date have explored whether SES interacts with other social statuses to differentially produce health outcomes, and particularly, biological indicators of current and future health, like low-grade systemic inflammation, such as CRP. This remains an important area for future lines of inquiry, and may help researchers identify specific subgroups of individuals who are at particularly high risk for both stress exposure and subsequent chronic health problems.

Studies have shown that when considered with other social statuses, the effect of SES on health may vary. Farmer and Ferraro (2005) documented a phenomenon termed "diminishing returns," where there was a strong and consistent education gradient in self-rated health for Whites, such that with each increase, they also experienced an improvement in self-rated health. In contrast, they found nearly no improvement in self-rated health for Blacks with increasing levels of education. Similarly, a recent study by Holmes and Zajacova (2014) found that education benefitted Whites' health more than Blacks using data from the National Health Interview Survey. Finally, Hinze and colleagues (2012) found that being a Black woman with less than a high school education was associated with poorer self-rated health than any other combination of race, gender, and education in a nationally representative sample of older adults. What remains to be understood are the biological and psychosocial mechanisms that might underlie these complex relationships. For example, strong and consistent inverse SES gradients exist for CRP, a major risk factor for future CVD and diabetes, but whether SES interacts with other social status variables like race and/or gender to produce social variations in CRP, and the

potential psychosocial mechanisms responsible for the association between SES and CRP have received less empirical attention (Nazmi & Victora, 2007).

Extant work shows that health risks also follow a social gradient, where people of lower SES are more likely to engage in health-damaging behaviors such as smoking compared to those of higher SES. Using data from the Health and Retirement Study, Wray, Alwin, & McCammon (2005) found that having more years of education and more wealth decreased the likelihood of being a heavy drinker. They also found that having greater wealth was associated with a decreased risk of engaging in low levels of physical activity. Despite the relationship between SES and health risks, the available literature suggests that they do not fully account for SES gradients in health and mortality. Two studies conducted by Lantz and colleagues (1998; 2001) and using the Americans' Changing Lives study, found that SES (measured by income and education) was positively associated with all-cause mortality, physical functioning, and self-rated health. Both studies concluded that while risky behaviors (smoking, alcohol use, low levels of physical activity, and body weight), partially accounted for the relationships between education and income on three health outcomes, these risky behaviors only explained a moderate portion of the SES differential in all three health measures (Lantz et al., 1998; Lantz et al., 2001). This literature suggests that mechanisms apart from but associated with SES, like chronic stress exposure, may account for the socioeconomic gradient in health.

An area receiving increasing research interest involves the social patterning of stress exposure, and the role that greater exposure to stressors may play in accounting for SES gradients in health (Aneshensel & Mitchell, 2014; Baum, Garofalo, & Yali, 1999; Kristenson, Erikson, Sluiter, Starke, & Ursin, 2004; Sapolsky, 2005; Steptoe & Marmot, 2002; Turner, 2013). Turner's stress process model (2013) explains that health disparities in social status measures such as SES arise due to the unequal patterning of stress exposure and the psychosocial resources used to

prevent or buffer the negative effects of stress. This model will be discussed at length in Chapter 3.

A key study examining SES differentials in stress exposure came from Turner and Avison (2003), who found that in a sample of 899 Black and White women and men, stressors accounted for nearly 50% of the SES-psychological distress relationship. In a test of the stress process model, Turner & Lloyd (1999) found that stress exposure, along with measures of psychosocial resources (e.g., mastery) explained about 91% of the SES difference in depressive symptoms. Of stressors, those which are chronic appear to be the most pathogenic, and abundant work suggests that chronic stress significantly contributes to advanced physiological wear and tear, a process referred to as allostatic load, which also shows a SES gradient (Dowd, Simanek, & Aiello, 2009; McEwen, 1998). Mulder and colleagues (2011) recently reported that the relationship between education and the use of risky behaviors was partially explained by increased exposure to stressors (e.g., financial strain, self-rated health, and psychological distress) and fewer social resources (e.g., social support, social cohesion, and sense of control). Thus, high stress coupled with fewer resources with which to cope with these stressors may explain the SES-health relationship.

Research is still required to examine whether the benefits of SES vary by social statuses such as race and gender, and the relationship that social statuses have with biological markers of disease risk and progression, such as CRP. In exploring their relationship with CRP, researchers may gain the advantage of understanding how social processes “get under the skin” to produce disparities in health.

Gender

Gender refers to a set of societally-based expectations and roles ascribed to people based on their biological sex at birth (Alwin & Wray, 2005). The relationship between gender and health is complicated and depending on the health outcome measured, sometimes paradoxical—decades of research have shown that men have higher rates of mortality for nearly all of the leading causes of death and have higher mortality rates in every age group, but it also shows that women have greater morbidity, disability, and spend more years with non-life threatening chronic health problems than do men (Alwin & Wray, 2005; Rieker & Bird, 2005). These paradoxical findings suggest a need to employ interdisciplinary work to clarify what pathways may differentially drive patterns of morbidity and mortality for men and women.

Less evidence supports the notion that solely biological differences underlie the differential rates of morbidity and mortality for men and women, while mounting evidence suggests that psychosocial mechanisms related to the socially-constructed nature of gender may provide an additional explanation for the apparent gender-health paradox (Rieker & Bird, 2005). Instead of focusing on either biology or social environmental factors, scholars such as Rieker and Bird (2005) and Alwin and Wray (2005) have called for an integration of the biological, psychological, and sociological literatures to gain a better understanding of the way that men and women are different. They suggest that men and women's patterns of health vary because of the social context which shapes their lives, including the economic resources, and argue that there may also be differences in the frequency and type of stressors encountered, such as discrimination. Specifically, Rieker and Bird (2005) argue for the constrained choice model to better understand patterns of health across gender, because it explains that the choices made by men and women are largely determined by their access to opportunities and resources, both of which are differentially distributed across genders, as well as the ways in which men and women

are socialized. This socialization may place men and women on different health trajectories by exposing them to different types of stressors and shaping their individual responses to stressors, for example (Alwin & Wray, 2005). This may explain the differences in rates of morbidity and mortality where women live longer than men do, but with a greater burden of disease.

The constrained choice framework may get more complicated, however, when other social statuses such as race/ethnicity are considered with gender simultaneously. As Geronimus asserts (1992; 2006), women of color are at an even greater risk for early physiological deterioration because they are more likely to be faced with a lifetime of stress exposure arising from both structural and interpersonal discrimination on the basis of both their race *and* gender, along with other social statuses that form their identity, such as SES. Similar to Geronimus (2006), scholars like Crenshaw (1991) have argued that women of color face an added disadvantage in society because their location at the bottom of two social hierarchies, which provides them with even fewer resources and opportunities than they would face if they were at the bottom of one or no social hierarchies, a perspective coined as "intersectionality" by Crenshaw (1989). Greater detail on intersectionality will be presented in Chapter 3. Data from intersectional studies support the argument that women of color face added disadvantages, even to their health, relative to White men and White women, and men of color. Brown and Hargrove (2013) utilized data from the Health and Retirement Study (HRS) to examine whether the effects of race/ethnicity and gender on physical disability and functional limitations were additive or multiplicative. Using multilevel models, they found that the effect of being a Black woman was larger than the additive combination of being Black and being a woman combined, net of sociodemographic, behavioral, and health-related factors. More recently, also using HRS data, Richardson and Brown (2016) found that hypertension risk was greatest in Black women, and that an additive model obscured the differential risk for hypertension—it showed that Blacks had greater risk for hypertension, but that women did not have a significantly greater risk of

hypertension than men. These findings suggest that women of color may face greater adversity—which has potentially major implications for their health because of their location at the bottom of multiple social hierarchies—and that mechanisms like stress exposure may help explain their health status. They also suggest that examining race, gender, and SES using an intersectional approach may reveal disparities that may be obscured in models examining the independent effects of social statuses.

Although the existing literature suggests that psychosocial mechanisms like stress exposure may underlie the complex patterns of morbidity and mortality by gender, the results on these pathways are mixed (Hatch & Dohrenwend, 2007). For instance, using a sample of men and women from ages 18 to 65, Matud (2004) found no significant differences in the total number of stressors encountered by men and women in the past year, but the type and intensity of the stressors that men and women were exposed to differed. Women reported more negative and uncontrollable life events than men, and they reported more family and health-related stressors than men did. A study by Meyer, Schwartz, and Frost (2008) similarly found that men reported significantly more instances of prejudice than women.

Currently, few studies have employed an intersectional framework in their examinations of the gender-health relationship and of stress exposure, and even fewer explore whether there are important differences in stress exposure and the health effects of stress exposure when multiple social statuses are considered simultaneously. While the literature indicates that Blacks and men both report more exposure to discrimination, there may be an interaction between race and gender on discrimination exposure that reveals that Black women experience greater exposure to discrimination because they belong to two lower status groups. Studies show that Black women have the highest levels of systemic inflammation of all race/gender groups, but traditional biological and behavioral risk factors do not explain these rates (Albert, Glynn, Buring, & Ridker,

2004; Khera et al., 2005). These findings suggest that the added burden of discrimination may account for the higher systemic inflammation in Black women.

Stress Exposure

Increased stress exposure represents one major pathway hypothesized to link lower social status to poorer health outcomes (Adler & Stewart, 2010; Alwin & Wray, 2005; Krieger et al., 1993; Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009). The original work on the stress process paved the way for two major hypotheses regarding the effect of stress on shaping health: the stress exposure hypothesis and stress vulnerability hypothesis (Aneshensel & Mitchell, 2014). The stress exposure hypothesis states that social patterning of stress exposure may account for the poorer health of lower status groups, and, in turn, the stress vulnerability hypothesis states lower status groups may be more vulnerable to the negative effects of stress than others. Turner and Avison's (2003) findings indicate that the distribution of stressors varies across social status groups, and that it largely depends on the type of stressor. For example, women report significantly less frequent discrimination than men do but report more instances of recent stress exposure than men do.

Despite the established social patterning of stressors, few studies have explored the extent to which stress exposure, particularly discrimination, explains variations in health. To date, only a handful of studies have explicitly tested the hypothesis that stress exposure accounts for social status-related health disparities. A study by Sternthal, Slopen, and Williams (2011) indicated that Blacks relative to Whites experience a greater burden of recent and lifetime exposure to stressors in eight domains (i.e., traumatic experiences excluding discrimination; lifetime, job, and everyday discrimination; early life adversity; relationship stressors; and community stressors), as well as more clustering of stressors. While they did not find support for the hypothesis that Blacks were

more vulnerable to stressors, they did find that the increased exposure to stressors accounted for much of the variance in the poorer physical and mental outcomes of Blacks relative to Whites.

Another major gap in the literature is that biological mechanisms responsible for the relationships linking social status to health are understudied. In particular, there is a growing need for studies that examine the multiplicative effect of social statuses and how this complex interaction may affect biological processes that are implicated in chronic disease pathology. Studying objective outcomes such as biomarkers may provide additional insight into the way that social and psychological factors "get under the skin" and provide a new lens to study health disparities. Biomarkers are particularly useful because they are not subject to bias due to social desirability or forgetfulness that subjective measures are susceptible to, amongst other issues. In addition, biomarkers such as C-reactive protein (CRP) are critically important to study because they provide insight into subclinical processes underlying the development of diseases such as cardiovascular disease and type 2 diabetes. In measuring CRP at multiple points in the life course, we might be able to better understand the sometimes complex and paradoxical findings within and across social groups. Various researchers argue that social distributions of health vary across the life course: some studies suggest that disparities widen as people age (Dannefer, 2003), while some demonstrate that disparities remain stable (Kelley-Moore & Ferraro, 2004), and others show that disparities decreazse due to leveling processes (Dupre, 2007). In addition, by examining multiple measures of CRP, we may be better able to address issues related to reverse causality.

In utilizing multidimensional approaches such as intersectionality—which focus on the simultaneous relationship among social statuses (such as race, SES, and gender)—research can answer multifaceted questions on the distribution of stressors within and across social groups—such as major race and SES differences in the frequency and type of stressors that women are exposed to, as well as the biological mechanisms underlying these links. Despite studies showing that poor Black women experience greater physiological deterioration relative to other social

groups (Geronimus, 2006), few studies have examined the psychosocial mechanisms responsible for this link. In particular, a fruitful area for future research would be in assessing the complex patterns of exposure to everyday and lifetime discrimination and how these exposures may account for the advanced physiological wear and tear experienced by women of color, as suggested in the intersectionality and stress process literatures (Bauer, 2014; Bowleg, 2012; Crenshaw, 1991; Turner, 2013). The stress process model (Aneshensel & Mitchell, 2014; Turner, 2013) suggests that there may be direct and indirect effects of psychosocial stressors like discrimination on health: discrimination may reduce availability of psychosocial resources known to buffer the adverse effects of stressors such as sense of control, and discrimination may also work to generate secondary stressors (Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009).

Discrimination as a Mechanism Linking Social Status to Poor Health

A growing literature suggests that reports of discrimination, defined as experiences of unfair and differential treatment, are significantly associated with worse mental and physical health (Williams & Mohammed, 2009). There are two broad types of discrimination: lifetime and everyday. Lifetime discrimination refers to experiences that are acute, meaning they occur at infrequent intervals, often occurring unexpectedly, are relatively uncommon but may interfere with achieving higher social status, including being denied a bank loan or housing. Everyday discrimination, on the other hand, refers to experiences of discrimination that are chronic, meaning that they are persistent and endure over long periods of time; this includes being treated with less respect or courtesy than others, or being treated as inferior (Kessler, Mickelson, & Williams, 1999).

In particular, if experiences of discrimination are chronic, such as repeated exposure to everyday discrimination, it may result in "wear and tear" across multiple physiological systems, including the immune and cardiovascular systems (McEwen & Seeman, 1999). In response to a large literature documenting the relationship between discrimination and physical health outcomes, including cardiovascular disease (CVD), studies have begun to explore how exposure to discrimination may be related to cardiovascular, neuroendocrine, and immune functioning, given that each system is implicated in both the stress response and in the development of chronic disease (Lewis, Williams, Tamene, & Clark, 2014; Pascoe & Richman, 2009). A greater understanding of the relationship between discrimination and stress-related biomarkers may provide insight into specific psychosocial and biological mechanisms responsible for observed social gradients in health, where Blacks, women, and people with low socioeconomic status (SES) experience poorer health compared to their higher status counterparts (Adler & Stewart, 2010).

Available evidence suggests that lower social status groups (e.g., racial minorities and those of lower SES) are disproportionately more likely to report everyday and lifetime discrimination, and discrimination exposure may cluster with other stressors to create greater psychological and physical burden for those who already belong to lower social status groups (Kessler, Mickelson, & Williams, 1999; Grollman, 2014; Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009). The early literature on the racial variation in discrimination exposure shows that Blacks are more likely to report everyday and lifetime discrimination than Whites, and also more likely to report everyday discrimination when that experience is attributed to race (Kessler, Mickelson, & Williams, 1999). This initial finding has been substantiated in subsequent work, where Blacks consistently report more frequent exposure to various forms of discrimination, and considerably more discriminatory experiences attributed to race than Whites

(Barnes et al., 2004; Hatch & Dohrenwend, 2007; Turner & Avison, 2003; Williams, Yu, Jackson, & Anderson, 1997).

Despite continued findings that discrimination is more commonly reported by Blacks, few studies have sought to explore whether discrimination exposure contributes to racial disparities in health (Williams & Mohammed, 2009). One of the few studies in existence was conducted by Slopen, Sternthal, & Williams (2011), who demonstrated that stress exposure, including everyday and lifetime discrimination, plays an instrumental role in shaping Black-White disparities in health. Despite mixed evidence on the salience of attributing race for exposure to discrimination, it is clear that race is an important predictor of discriminatory experiences and that frequent exposure to discrimination is associated with poorer health. What remains to be understood is whether interactions of race, gender, and SES differentially contribute to discrimination exposure.

Individuals of lower SES may also report more frequent exposure to discrimination, but work demonstrates that the relationship varies based on both the measure of SES and the measures of discrimination used. For example, Kessler, Mickelson, & Williams (1999) found that education was not significantly associated with everyday discrimination, but it was positively associated with lifetime discrimination; and that income was inversely associated with everyday discrimination, but it was not associated with lifetime discrimination. In another study, Barnes and colleagues (2004) found no consistent relationship linking SES (measured by education) to discrimination exposure. Both Turner and Avison (2003) and Hudson and colleagues (2012) found an inverse association between SES and exposure to both everyday and lifetime discrimination. However, the link between SES and discrimination may also depend on other social statuses, such as race. Some work suggests that there are SES differences within race groups when it comes to discrimination: Blacks of higher SES tend to report more frequent discrimination (especially race-based discrimination) than Blacks of lower SES (Borrell, Kiefe,

Williams, Diez-Roux, & Gordon-Larsen, 2006; Dailey, Kasi, Holford, Lewis, & Jones, 2010; Hudson, Neighbors, Geronimus, & Jackson, 2016; Hunt, Wise, Jipguep, Cozier, & Rosenberg, 2007; Krieger et al., 2011; Lewis, Cogburn, & Williams, 2015; Taylor, Kamarck, & Shiffman, 2004). However, there is also evidence to the contrary: Williams and colleagues (2012) found that low SES Blacks and Whites reported more instances of discrimination than those with moderate or higher SES. It remains unclear whether discrimination exposure varies across intersections of multiple social status combinations, and whether discrimination is a salient link between intersecting social statuses and health.

The evidence is inconclusive when it comes to gender and exposure to discrimination: some studies indicate that gender may be associated with discrimination, but the findings on this are inconsistent. Some work suggests that the link between gender and reporting discrimination depends on the type of discrimination, and in particular, the attribution for said experience (Kessler, Mickelson, & Williams, 1999). Kessler and colleagues (1999) found no substantial gender differences in reporting of lifetime discrimination but found that men reported more everyday discrimination in the Midlife in the United States (MIDUS) sample. They also found no discrimination-related vulnerability to mental health problems between men and women. In contrast, two earlier studies demonstrated that men reported more frequent exposure to everyday and lifetime discrimination than women (Turner & Avison, 2003; Turner & Lloyd, 2004). Available studies show that there is no gender-related vulnerability to discrimination: for example, Lewis and colleagues (2010) did not find that there were significant gender differences in the effect of everyday discrimination on CRP within a sample of older Blacks.

While there may not be discrimination-related vulnerability for women versus men, prior theoretical models and empirical work point to the role that stress exposure, such as persistent exposure to various forms of discrimination, and the accumulation of being exposed to stressors like discrimination due to multiple attributions (e.g., race and gender) may account for racial-

gender inequities in health (Bowleg, 2012; Brown & Hargrove, 2013; Crenshaw, 1989; Crenshaw, 1991; Cunningham et al., 2012; Geronimus et al., 2010; Grollman, 2014; Pearlin, 1989). It is also unclear whether there are gender differences in exposure to discrimination within social status such as race and/or SES (Lewis, Cogburn, & Williams, 2015). According to intersectionality and the weathering hypothesis, Black women may be exposed to more adversity, such as discrimination, throughout their lives, and this may account for their poorer health in some domains, such as cardiovascular disease risk factors (Richardson & Brown, 2015).

Thus, an understanding of the way that discrimination can "get under the skin" may provide an additional insight above and beyond other known psychosocial factors associated with being in a lower status group or multiple lower status groups, and further inform research on the contribution of psychosocial stressors to health disparities. This understanding may be particularly useful given research showing that racial disparities in health continue to exist even after controlling for indicators of SES (Williams & Mohammed, 2009).

C-Reactive Protein

The immune system is a critical component of human functioning. Its primary job is to protect the body from infection and disease through its numerous roles such as detecting and repairing damaged tissue. Many processes are involved in immune functioning, which can be divided into two broader divisions: natural and specific immunity. The main difference between these two forms of immunity is that natural immunity is the body's most basic, primitive, and immediate response to an infection, injury, challenge, or stressor (Segerstrom & Miller, 2004). In contrast, specific immunity takes longer to mount, and it is generated by the immune system to target each specific or unique pathogen that invades the body (Segerstrom & Miller, 2004). As part of the immune response to injury or illness, cells that arrive at the site of infection called

macrophages initiate the production of a pro-inflammatory cytokine called interleukin-6 (IL-6). IL-6 is a key regulator of the acute phase response to assist in the prevention of infection or further damage as well as to direct the repair of damaged tissue (Segerstrom & Miller, 2004).

The acute phase response consists of organized responses by a set of proteins that are critical in fighting off infection and regulating inflammation. Inflammation is a process defined as a coordinated set of physical responses that typically include swelling, redness, fever, and pain, all of which indicate increased blood flow to the damaged or infected area; it has a critical role in orchestrating the migration of other immune cells to the area (Hänsel, Hong, Cámara, & von Känel, 2010). Upon production of IL-6 and chemical messages that IL-6 sends out, the liver begins to produce proteins for the acute phase response; whether a given protein is upregulated or downregulated during this process depends on the specific protein (Black, 2003). The acute phase response involves a variety of proteins, including C-reactive protein and serum amyloid A, which are upregulated to a maximum of about 1,000 times their basal levels when there is presence of injury or infection (Black, 2003). The production of acute phase proteins begins after approximately six hours from the initial chemical messages from IL-6 and continues until the infection or injury has subsided, and overall production of acute phase proteins is a process that can last from a week or take up to 12 days for levels to return to normal (Gabay & Kushner, 1999; Pepys & Hirschfield, 2003).

However, inflammation is also evident when there is no clear indication of infection or acute illness (Glaser & Kiecolt-Glaser, 2005; Miller, Cohen, & Ritchey, 2002). This is the case with chronic stress: the immune response becomes a deleterious rather than protective process (Glaser & Kiecolt-Glaser, 2005; Miller, Cohen, & Ritchey, 2002; Pearson et al., 2003), and chronic stress-associated dysregulation can lead to heightened levels of systemic inflammation which contribute to elevated risk for health problems and mortality. The effects of lifelong exposure to chronic stress are particularly prominent in middle and older ages when the long-term

effects of low-grade inflammation accumulate and give way to physiological wear and tear (Glaser & Kiecolt-Glaser, 2005; Pearson et al., 2003). There is evidence that hormones in the stress response can directly trigger the acute phase response (Black, 2003). Glucocorticoids, such as cortisol, which are produced by the hypothalamic-pituitary-adrenal axis (HPA-axis) in response to stress, are known to have an anti-inflammatory role, which means that they suppress the production of pro-inflammatory cytokines like IL-6 (Sapolsky, Romero, & Munck, 2000). When a person is chronically exposed to stress, this process can be dysregulated, meaning that with prolonged exposure to stress, the body has a decreased ability to adequately and appropriately respond to a stressor (McEwen, 1998). For example, cortisol, a key hormone released in the HPA-axis's response to stress, is important in the HPA-axis's negative feedback loop, which halts the secretion of stress hormones, thus allowing the body to return to its usual state of functioning after a challenge, threat, or stressor has ended (McEwen, 1998). In response to chronic stress, the HPA-axis can become dysregulated, thereby resulting in *increases* in circulating inflammatory levels through a process called glucocorticoid receptor resistance (Miller, Cohen, & Ritchey, 2002). Glucocorticoid receptor resistance occurs when high levels of glucocorticoids such as cortisol cause white blood cells to reduce their expression of receptors that glucocorticoids bind to in order to effectively shut down the stress response (Miller, Cohen, & Ritchey, 2002). With fewer glucocorticoid receptors expressed on the immune cells, glucocorticoids are unable to bind to their receptors on immune cells, reducing their anti-inflammatory function, which then leaves an individual with higher inflammation (Miller, Cohen, & Ritchey, 2002).

Low-grade elevation of C-reactive protein (CRP) ranges from 0-10.0 mg/L, with a value of 0 mg/L indicating no inflammation, and higher levels indicative of greater inflammation. Low-grade CRP levels are prospectively linked to the diagnosis, prognosis, and risk for a number of chronic diseases for which there are known disparities, including cardiovascular disease (CVD)

and type 2 diabetes (Pearson et al., 2003). CRP is a robust marker of subclinical atherosclerotic processes which take a long time to progress to a clinical diagnosis of CVD, as well as a strong independent predictor of clinically relevant outcomes such as incidence or recurrence of type 2 diabetes, CVD, and CV-related mortality (Pearson et al., 2003). Further, CRP is an attractive candidate to capture the long-term risk of morbidity and mortality, particularly because it provides additional insight above and beyond other existing risk measurements, such as the Framingham Risk Score (Lloyd-Jones, Liu, Tian, & Greenland, 2006). Thus, its use by health practitioners is highly recommended by the Centers for Disease Control and Prevention (CDC) and the American Heart Association (AHA). CRP can offer additional insight above and beyond traditional biological risk factors for CVD, such as cholesterol. According to a joint statement released by the CDC and AHA, CRP levels can be stratified into three risk categories: low (< 1.0 mg/L), moderate (1.0 – 3.0 mg/L), and high (3.0 – 10.0 mg/L), and these levels reflect tertiles of CRP, based on the distribution of CRP in over 40,000 individuals (Pearson et al., 2003).

Extant work shows that CRP also varies across social status, where lower status groups such as Blacks, women, and those of lower SES have higher CRP levels (Nazmi & Victora, 2007). The reasons for these associations are not clearly defined: traditional biological and lifestyle risk factors do not fully account for them, and neither do indicators of health status. There is also some evidence of within-group variations in CRP, which reveals that social statuses interact to produce CRP levels, such that Black women have the highest levels of CRP (Khera et al., 2005). This has led to further speculation that exposure to stressors may account for the residual effects linking social status to higher CRP (Johnson, Abbasi, & Master, 2013; Nazmi & Victora, 2007). In addition, low-grade elevations in CRP may indicate physiological dysregulation in other systems (e.g., glucocorticoid receptor resistance), and as a result, is an ideal biomarker to examine the way that stressors like discrimination influence health processes. A recent review by Johnson, Abbasi, & Master (2013) demonstrated that experiences of

discrimination were positively related to CRP levels. Due to these characteristics, CRP represents a major underlying biological pathway that may link social status to discrimination exposure and consequently, health disparities.

Variations in CRP by Social Status

Much of the work on the relationship between social status and CRP has utilized additive models of social status, where social statuses are viewed as independent of one another. To date, few available studies have examined the intersection of social statuses on CRP, and none have based their conceptualizations on intersectionality.

The available findings generally show that those in lower status groups, such as racial minorities, women, and people of low socioeconomic status (SES) have higher CRP compared to those in higher status groups. A literature review published in 2007 by Nazmi and Victora found that overall, regardless of whether the sample was younger or olderand regardless of the measures of SES utilized, there existed can inverse relationship between various indicators of SES and CRP. These studies showed that higher SES was consistently linked to lower CRP and follows the well-established SES gradient in health (Link & Phelan, 1995; Nazmi & Victora, 2007). Of the 15 studies examining race/ethnicity and CRP in Nazmi and Victora's 2007 review, all but one found that Blacks had significantly higher CRP compared to Whites. In models that included adjustment for potential biobehavioral characteristics, racial/ethnic minority status remained significantly associated with CRP in six out of eight studies. Recently, a study by Mitchell and Aneshensel (2016) found that CRP was significantly higher for racial minorities, women, and those with less education using data from the HRS.

Khera and colleagues (2005) conducted one of the first studies showing that CRP levels varied by both race and gender using a sample of 2,749 Black and White men and women aged

30 to 65 from the Dallas Heart Study. Their results indicated that Black women had the highest levels of CRP, followed by White women, Black men, and White men, respectively. They also found that, even after adjustment for potential confounding, Black women continued to have the highest CRP relative to other groups. This work points to the potential importance of examining whether psychosocial factors, like discrimination, account for these differences, as well as the role of SES in moderating the race-gender relationship with CRP.

A study conducted in 2008 used data from 3,154 participants in the Study of Women's Health Across the Nation (SWAN) to show that Black women had the highest CRP concentrations, followed by Hispanics, and Whites, respectively (Kelley-Hedgepeth et al., 2008). Their results also demonstrated that the association remained after controlling for anthropometric, lifestyle, and sociodeomographic variables. Because the sample was comprised exclusively of women, it is unclear whether the effect of being Black and a woman were independent or interactive, but the results from this study certainly suggest that there is an added disadvantage associated with being a Black woman, relative to being a woman from another race/ethnicity. That the effect of being Black was not completely accounted for by confounders as well as other traditional risk factors, suggests that other factors may underlie the complex association among race, gender, and CRP.

In 2012, Herd, Karraker, and Friedman explored the independent contributions of race, income, and education on CRP in a sample of older adults ages 57-85 in the National Social Life, Health, and Aging Project. They noted that few studies have explored the interaction between race and gender, and suggested that there may be important gender differences in CRP due to the unequal distribution of risk factors known to increase CRP, such as obesity in Black women. Thus, they ran analyses stratified by gender to examine whether the relationships between race and SES with CRP varied by gender. They found that for both women and men, Blacks had higher CRP than Whites, and both education and income were significantly and inversely

associated with CRP for both genders. After accounting for SES, however, the effect of race for women was reduced to marginal significance, while the effect of race for men was largely unchanged. These results demonstrate that there are complex relationships linking various indicators of social status to CRP. These complexities may have been obscured if the researchers had not stratified by gender. Additionally, the results from this study suggest that different mechanisms explain the race-CRP link across genders.

Except where noted, the majority of studies have focused on additive models that assumed that the effects of social statuses such as race, gender, and SES do not interact with one another to produce differential risk for health problems within social groups. Intersectionality offers a theoretical framework useful for addressing health disparities by examining the complex relationships underlying combinations of social statuses and how the social construction of identity may influence health through a number of ways. Rather than assuming an additive model, intersectionality suggests that we take into account the ways that resources, such as power, and risk factors may be socially patterned across combinations of social status. It allows scholars to use a new lens to understand existing issues and relationships in disparities research. Most importantly, intersectionality raises the possibility that the relationships among social status variables such as race, gender, and SES are more complex than they initially appear to be in additive models, and as a result, they may simultaneously shape the experiences, including the exposure to discrimination, of people located at the bottom of multiple systems of social stratification. Greater detail on this perspective is offered in the next chapter.

Mechanisms Linking Social Status to CRP: Discrimination Exposure

The earliest study on the relationship between discrimination and inflammation was conducted by Albert and colleagues in 2008. Their study assessed the relationship between a

variety of cardiovascular disease biomarkers with having ever experienced perceived racial/ethnic discrimination using a single-item question, “Have you ever been discriminated against due to your race/ethnic background?”. The study examined the relationship using data from the Dallas Heart Study, a collection of 1,475 Black, White, and Hispanic men aged 40+ and women aged 45+ from Dallas. CRP was dichotomized into: ≥ 3.0 mg/L or less than 3.0 mg/L, because this indicates those with high risk of future cardiovascular incidents. No significant association was found, even after stratification by gender and race. Major limitations of this study were the use of a single item measuring ever experiencing discrimination, and the question was worded to focus exclusively on discrimination attributed to race/ethnicity.

In 2010, Lewis and colleagues examined how everyday discrimination was associated with CRP (modeled continuously) in a cross-sectional study of 296 Black men and women aged 65 and over from the Minority Aging Research Study. The results showed a positive association between everyday discrimination and CRP, and the association attenuated after adjusting for BMI. In unadjusted models, a dose-response relationship was observed, where individuals in the highest tertile of everyday discrimination had the highest CRP. This study was one of the first to identify that everyday discrimination was a stressor that might contribute to racial disparities in health through low-grade inflammation.

Cunningham and colleagues (2012) evaluated the association between racial/ethnic discrimination and CRP in analyses using data from 3,336 young and middle-aged Black and White men and women from the Coronary Artery Risk Development in Young Adults (CARDIA) study. The association between racial/ethnic-based discrimination and CRP varied by both race and gender. In Black women, a curvilinear relationship was observed after adjusting for physiological factors, where 1 or 2 experiences of discrimination were associated with higher CRP levels and those reporting no experiences and those reporting 3+ experiences had lower CRP levels than those reporting none. In fully adjusted models, discrimination was no longer

significantly associated with CRP. In Black men, in contrast, a negative linear association was observed when adjusting for physiological confounders, where those reporting 3+ experiences of discrimination had lower CRP than those who reported no experiences. In adjusted models, the association was no longer significant. Interestingly, among White women, they found a positive linear association between discrimination and CRP, where women who reported 3+ experiences of discrimination had significantly greater CRP than those reporting no experiences of discrimination, and the association remained significant in fully adjusted models. In White men, no association was found between discrimination and CRP. This study showed that there might be both race- and gender-specific effects of discrimination on CRP, but that the issue warrants further investigation.

Beatty Moody and colleagues (2014) found a significant longitudinal association between everyday discrimination and CRP across seven years in a sample of 2,490 diverse women (Black, White, Chinese, Hispanic, and Japanese) in the Study of Women's Health Across the Nation (SWAN). Everyday discrimination was measured at baseline and CRP was measured at baseline, then again after 1, 3, 4, 5, 6, and 7 years of participation. The authors also tested an interaction of everyday discrimination x race and of everyday discrimination x BMI. Results indicated that more everyday discrimination predicted higher CRP across seven years and a significant everyday discrimination x BMI interaction revealed that the relationship was moderated by BMI, such that the everyday discrimination-CRP relationship was observed among nonobese but not among obese women. The everyday discrimination x race interaction term was not significant. Taken together, these findings were especially important because they assert that everyday discrimination was associated with CRP over time in women regardless of racial/ethnic background.

Mitchell (2014) used data from the HRS to examine the cross-sectional effects of everyday and lifetime discrimination on CRP levels, as well as whether this exposure to

discrimination mediated the relationships between race and gender on CRP. She demonstrated that both everyday and lifetime discrimination were positively associated with CRP, and that they remained significant predictors of CRP, net of one another. In addition, this work revealed that exposure to both lifetime and everyday discrimination significantly mediated the relationship between being Black and having higher CRP, with the race coefficient being reduced by nearly 15%. Of note, exposure to everyday and lifetime discrimination strengthened the gender coefficient by nearly 10%. In regards to mediation, Mitchell found that only lifetime discrimination mediated the relationship between gender and CRP. While this study was the first of its kind and instructive, a limitation is that it did not focus on low-grade levels of CRP, which are indicative of future CVD risk, and those with values above 10.0 mg/L were included in all analyses. This may be problematic because those with values 10.0 mg/L may have an infection or ongoing illness that may increase their circulating CRP levels for an acute period of time. Thus, it is imperative to examine the effects of social status and discrimination exposure on low-grade levels of CRP, as well. This work also suggests that the social status-discrimination-CRP relationship exists and that a closer investigation of whether these relationships vary across levels of SES is warranted.

In a more recent study, Goosby and colleagues (2015) found that everyday discrimination was positively associated with CRP in a sample of low-income children with ages ranging from 10 to 15 years who participated in the Omaha Urban Research on Health Study. This was the first study to find evidence that everyday discrimination had negative effects on CRP in a younger sample.

Kershaw and colleagues (2016) examined the cross-sectional relationship between both everyday and lifetime discrimination on two measures of inflammation: IL-6 and CRP. Their sample consisted of 6,567 men and women from multiple race/ethnic groups participating in the Multiethnic Study of Atherosclerosis. Their goal was to test whether the relationship between

everyday and lifetime discrimination varied across race/ethnicity, gender, and specific attributions of discrimination. Their results indicated that none of the measures of discrimination were related to CRP in men or women. However, they found that higher everyday, lifetime, and racial/ethnic-based discrimination was associated with increased IL-6 in women; in men, everyday discrimination was *inversely* associated with IL-6; and lifetime and racial/ethnic-based discrimination were not significantly associated with IL-6. This study had null findings for the relationship between discrimination and CRP, but it did show that IL-6, a precursor to CRP, was associated with multiple measures of discrimination for men and women. This suggests that there is a link between discrimination and inflammation, but it requires more investigation.

Finally, a 2017 paper by Stepanikova, Bateman, and Oates found that reports of lifetime but not everyday discrimination were positively associated with measures of systemic inflammation in models adjusted for a wide variety of anthropometric, lifestyle, and health covariates in a sample of 1,054 middle-aged adults in the MIDUS sample. In particular, the results demonstrated that when included in a model simultaneously, everyday discrimination was not a significant predictor of systemic inflammation, but lifetime discrimination remained a significant predictor of three measures of inflammation (fibrinogen, e-selectin, and IL-6), net of everyday discrimination and other covariates. In analyses stratified by race, there was not a significant relationship between everyday or lifetime discrimination with any of the markers of inflammation for Blacks, but for Whites, lifetime discrimination was positively associated with all markers of inflammation except CRP. These findings reveal the often inconsistent relationship between measures of discrimination with CRP, and suggest the need for additional research on the relationship among race, discrimination, and CRP.

Based on this review of the literature, it appears that overall, everyday discrimination and lifetime discrimination may be positively associated with CRP, but there is mixed evidence regarding these links. Further, race and gender may play a role in the relationship between

discrimination and CRP, but it is unclear whether discrimination mediates the social status-CRP link. Some work suggests that race does not play a moderating role, as the stress vulnerability hypothesis would suggest. Rather, it appears that exposure to everyday and lifetime discrimination may be one pathway linking those in lower status groups to higher CRP, rather than their status(es) making them more vulnerable to the effects of discrimination. However, there is limited work that has specifically examined whether exposure to everyday and lifetime discrimination mediates the social status-CRP link overall, and no work has been conducted on how multiple social statuses may be linked to higher CRP through increased exposure to discrimination. Several leading theoretical frameworks provide insight into the complex ways that race, gender, and SES may interact to shape CRP levels and the mediating role that exposure to everyday and lifetime discrimination may play in this relationship. The following chapter will describe the four frameworks that inform this dissertation.

CHAPTER 3

THEORETICAL & CONCEPTUAL FRAMEWORKS

The social patterning of CRP is complex, and the mechanisms responsible for this association are underexplored. In order to gain more insight into how these relationships play out, four leading theoretical frameworks from various disciplines are used to guide the hypotheses regarding the way that race, gender, and SES may interact in their association with CRP and the role that discrimination may play in accounting for this interaction. This chapter will review, compare, and contrast each of these frameworks and identify their unique contributions to the conceptual framework underlying this dissertation.

THEORETICAL FRAMEWORKS

Lifespan Developmental Perspective on Social Status and Health

The lifespan developmental perspective on social status and health describes why and how people in various social status groups are differentially exposed to stressors and adversity or protective factors compared to people who are in higher status groups (Alwin & Wray, 2005). Its strengths and uniqueness comes from its expansion on previous research in that it focuses on the social structural underpinnings of health inequalities (e.g., Link & Phelan, 1995), but builds on gaps in the literature on social status and health by describing the crucial role that social status has in shaping health disparities: previous work typically used restrictive terminology to describe social status, sometimes referring to one dimension of status, without acknowledging that social

status is a multidimensional term that can be broken down into *ascribed* and *achieved* status; the previous literature did not often focus on how social status can affect health throughout life through various processes such as the timing of exposure to stressors or protective factors, and the historical context in which a person is embedded that may have important implications for how social experiences are interpreted and understood, which the authors acknowledge are key to understanding how social inequalities in health in midlife and older ages arise.

Ascribed status refers to social statuses that are quite stable and attached at birth because of biological or group-related characteristics, and includes race and gender; whereas *achieved* status refers to statuses that can be attained through access to resources and opportunities, including socioeconomic measures such as education, income, occupation, and wealth. These terms are vital in understanding the differences in social status and how they may be differentially related to health status through a number of processes throughout life, including differential exposure to stressors like discrimination. Alwin and Wray (2005) provide an explanation as to how each of the most commonly used measures of social status are related to health and regard social and environmental factors as mechanisms that may place individuals at differential risk for health problems. For example, a long line of research has documented that racial minorities experience greater morbidity, disability, and mortality compared to Whites (Williams & Mohammed, 2009). The lifespan developmental perspective explains that in cases where achieved status cannot explain racial variations in health, research should focus on examining other social and contextual factors, including social environmental factors that may accumulate throughout the lives of minorities to influence their health, such as childhood economic and health circumstances, disproportionate experiences of both institutional and interpersonal discrimination, along with increased exposure to adversity throughout their lives, limited access to resources and opportunities for health-promotion, and increased exposure to detrimental living

and working conditions relative to Whites (Alwin & Wray, 2005; Hayward, Miles, Crimmins, & Yang, 2000; Krieger, Rowley, Herman, Avery, & Phillips, 1993; Williams & Mohammed, 2009).

Gender refers to socially ascribed roles, expectations, and attributions based on one's assigned sex at birth. These factors may adversely and differentially affect the health of men and women in a number of ways, such as placing them on different life-course pathways because of the often constrained choices of women compared to men, the different socialization for how men and women should engage in risky or protective behaviors, as well as the way that men and women are socialized to react to stress and interact with others (Alwin & Wray, 2005; Matud, 2004; Rieker & Bird, 2005). Given current research findings that suggests that men have higher mortality at younger ages than women, and that women live longer than men but live in poorer health and greater disability for a longer duration than men do, the literature has pointed that this may be partially due to psychosocial in addition to biological factors. While some work has speculated that differences may be at least in part due to biological differences between the sexes, more often, social differences appear to account for differences between men and women largely because of the social pathways shaped by the differential and constrained experiences, exposures to opportunities and resources, and role expectations of women and men (Alwin & Wray, 2005; Krieger, Rowley, Herman, Avery, & Phillips, 1993; Rieker & Bird, 2005).

The socioeconomic gradient in health is a well-documented phenomenon that exists for a number of health outcomes and behaviors, and is demonstrated across an array of geographical, historical, and social contexts. Extant work shows a gradient effect of socioeconomic status (SES) on health, meaning there is an incremental increase in health and longevity for each increase in level of SES, and this is not merely a threshold effect (Adler & Ostrove, 1999; Adler, Boyce, Chesney, Folkman, & Syme, 1993; Adler et al., 1994; Marmot, 2005). The lifespan developmental perspective argues that education gives way to other achieved statuses because it is causally prior to occupation and income, for example, and these achievements in social status

can be reached with access to opportunities and resources (such as economic and social) that provide opportunities for success.

To summarize, the lifespan developmental perspective on social status and health provides a better understanding of why and how social status-related disparities may arise, and it especially emphasizes that exposures to adversity and access to resources occur from birth to death—though some points in time may be more critical or sensitive than others (Ben-Schlomo & Kuh, 2002). The lifespan developmental perspective also explains that lower social status may give rise to poor health largely through differential exposure to risk factors (stressors, negative health behaviors) and protective resources (sense of control, opportunities for advancement, and economic and social capital). It suggests that socially-patterned experiences and psychosocial risks, such as exposure to discrimination, may provide a better understanding of social status-related inequalities in health.

The Stress Process

The stress process is a framework developed by Pearlin and colleagues (1981; 1989) that aimed to understand how society shapes the health of its members, such as why lower status social groups experience poorer health than higher status groups (see Figure 1). In particular, it shares concepts from the lifespan developmental perspective, but elaborates on the specific structural origins and identifies psychosocial mechanisms responsible for observed social gradients in health. It describes a stress process that has origins in social structural conditions and its view of stress is dynamic and complex (Pearlin, Menaghan, Lieberman, & Mullan, 1981)

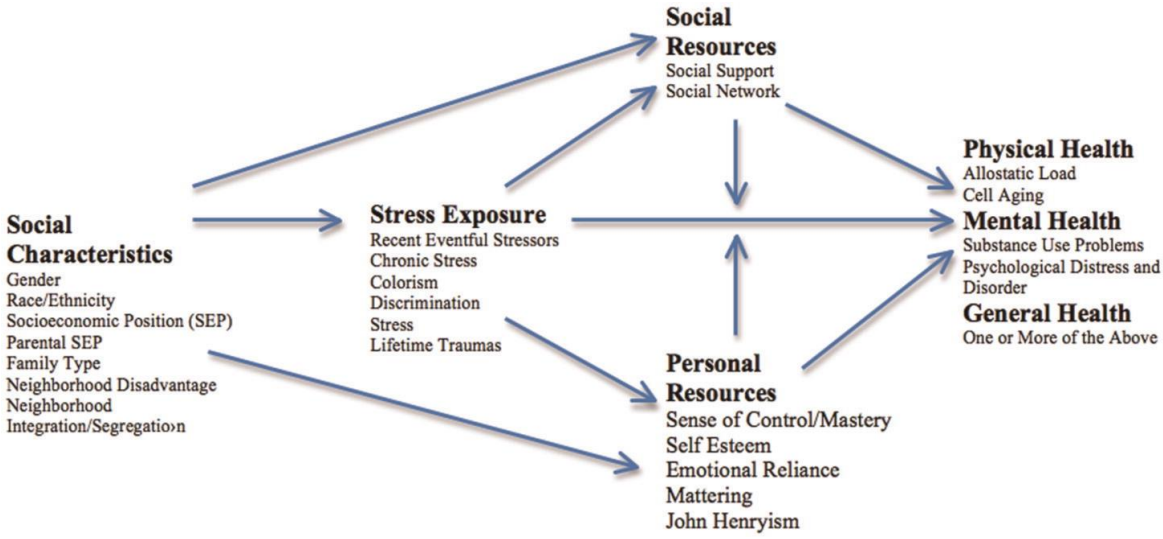


Figure 1. The stress process. Image from (Turner, 2013).

This framework evolved from the observation that much of the stress literature at the time had not focused on the social distribution of stress exposure, the social origins of stressors, and the social and psychological resources necessary to cope with said stressors. It is these processes, Pearlin and colleagues asserted, that are key to understanding why lower status groups are at higher risk of morbidity and mortality than higher status groups. Put simply, individuals are embedded within unique social contexts and their social statuses shape the type of and frequency of exposure to stressors they encounter and the resources they may have access to draw upon that have important implications for their health. For example, a person's exposure to a stressor depends on their location within a system (or systems) of stratification in society, and this location also determines how they perceive and respond to a potential stressor, as well as the way that a stressor manifests in their health (Pearlin, 1989; Aneshensel & Mitchell, 2014).

Subsequent research has shown that stress exposure varies across systems of social stratification, such as race, gender, and SES (Pearlin, 1989; Turner & Avison, 2003), and that more frequent exposure to stressors contributes to differing rates of health outcomes, such as depressive symptoms, across social status groups.

The stress process goes into further detail on the specific types of stressors people may encounter: it distinguishes life events from chronic stressors, noting that there are major differences in the way they have traditionally been conceptualized, measured, and the way they may affect health. Life events are major events that are discrete, and they can be positive or negative, while chronic stressors are more persistent, repetitive, and negative, often occurring throughout day-to-day life over long periods of time (Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin, 1989). Much of the work prior to this pioneering paper on the stress process utilized life event inventories to measure stress exposure. However, Pearlin believed that it was critical to distinguish between life events and chronic stressors, and to focus on the context in which each stressor occurs (including the desirability, unexpectedness, and normative nature of a given stressor), and emphasized that stressors may also cluster together (Pearlin, Menaghan, Lieberman, & Mullan, 1981).

Where the lifespan developmental perspective outlines the different social status-health relationships and explains how exposures may unfold over one's life, the stress process focuses on lower status groups as a whole, and broadly focuses on how the set of power relations and prestige associated with both ascribed and achieved social status gives way to differential exposure to stressors and protective resources that have critical implications for health. The major contribution of the stress process is detailing the specific types of stressors that may affect health, the social origin and distribution of exposure to stressors, and protective resources (e.g., social support, mastery) that can combat the negative effects of stressors on health. While it is mentioned in passing, the stress process focuses less on the way that stressors may unfold over historical and biographical time, and may shape the timing and sequencing of life-course trajectories, which the lifespan developmental perspective discusses in greater detail. While both perspectives offer solid explanations for the social origins of health disparities and mechanisms linked to disparate health outcomes, neither describes the way that being located at the bottom of

more than one system of social stratification may create distinctive and additional experiences and strains that are altogether different and go beyond the sum of their individual parts, nor do they expand on the underlying biological processes that explain how lower status individuals come to experience poorer health than their counterparts.

Intersectionality

Intersectionality emerged from early black feminist scholarship, much of which was qualitative at the time of its conception. In the beginning, it primarily reflected the personal experiences of several scholars and activists who felt that the experiences of women of color were underacknowledged in the literature, and often unaccounted for through traditional frameworks that examined race or gender separately (Bowleg, 2012; Dill & Zambrana, 2009; McCall, 2005). In 1991, scholar Kimberlé Crenshaw coined the term "intersectionality" to reflect the unique lived experiences of women at the intersection of multiple systems of social stratification, such as experiences of women of color compared to the experiences of women or Blacks. She pointed out that previous conceptual frameworks on gender were inherently flawed in that they failed to account for within-gender heterogeneity, in terms of the experiences of discrimination women from varying racial backgrounds and classes faced, for example (Dill & Zambrana, 2009). This work challenged earlier work on social status (namely gender at first) by asserting that social statuses were not mutually exclusive and that belonging at the intersection of social statuses creates unique and diverging experiences that often remain unaccounted for by utilizing additive approaches that examine facets of social status, such as gender or race, separately (Bowleg, 2012; Crenshaw, 1991).

Similar to Pearlin's stress process and the lifespan developmental perspective, intersectionality suggests that social status is a crucial determinant of experiences, the

interpretation of events, exposure to opportunities, resources, and stressors, and posits that power relations between social groups primarily drives the differential experiences of social status groups. Intersectionality departs from previous work on social status in that it places individuals within multiple systems of social stratification defined by power and prestige, and situates individuals within a social context based on the joint impact of their social statuses, which then shapes their experiences. It captures how oppression may be due to overlapping social statuses, and provides a lens to examine how having multiple lower social status positions shapes the experiences and life chances of groups at the intersection of multiple systems of oppression, such as the experiences of women of color (Brown & Hargrove, 2013; Crenshaw, 1991; Ore, 2003). It explains that one's lived experiences that arise due to their social statuses shape their perceptions of the world, their social experiences, their responses to those experiences, and as a result, their social statuses can shape their life chances. In sum, intersectionality suggests that the effect of social statuses should not be assumed to be additive, as much research traditionally does, but rather consider that social statuses might be interactive and dynamic: they are not mutually exclusive and can combine to create new and distinct social experiences that are entirely exclusive to a specific set of social statuses; a major limitation is that these experiences are often unaccounted for in additive models (Brown & Hargrove, 2013; Dill & Zambrana, 2009).

However, intersectionality falls short in that it was not conceptualized in terms of health and health risks; it does not explain how unique social experiences may translate to risk of negative health outcomes for women of color, for instance. Recently, scholars have advocated for the widespread use of intersectionality theory within public health research in order to better conceptualize and address health disparities, primarily because it can provide scholars with an explanation for how interlocking systems of oppression (or privilege) and power relations between groups can adversely affect health for lower status groups, such as women of color (Bauer, 2014; Bowleg, 2012). These scholars argue that intersectionality is well-suited to address

health disparities, in that it draws attention to the social structural determinants of health and may provide more explanatory power than additive models (Bauer, 2014; Bowleg, 2012). Bowleg (2012) also suggests that intersectionality is apt for public health researchers because it can provide a theoretical framework for those who may already be studying intersectional relationships and health, but are unaware of the framework, and this may provide public health researchers a larger, multidisciplinary literature to draw upon to better understand the ways that intersecting social statuses translate to differential health outcomes. She also suggests that intersectionality may be particularly useful in helping researchers think through and interpret unexpected results.

The lifespan developmental perspective, stress process, and intersectionality provide detailed explanations for why and how people in different social status groups may be differentially exposed to risk and protective factors, which predisposes lower status groups to premature morbidity and mortality. However, they do not provide an explicit description of how these processes can ultimately "get under the skin" to produce health disparities.

Allostatic Load

Allostatic load was a term introduced by McEwen (1998) to explain how adverse life experiences, and particularly chronic stress, may place individuals at greater risk for health-related pathology (see Figure 2). Like Pearlin's stress process framework (1989), McEwen made important distinctions between major life events (acute stressors) and chronic stressors, but explained that both can have long-term adverse effects on health.

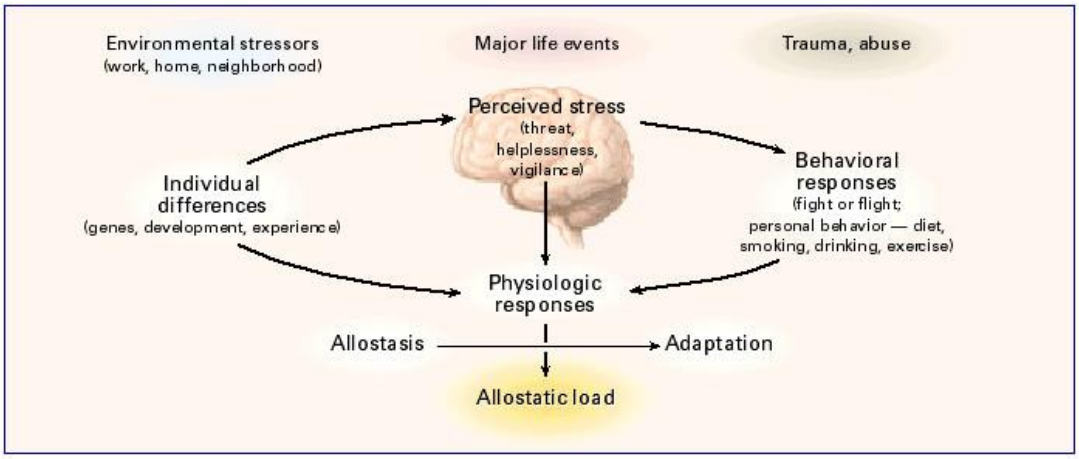


Figure 2. **The stress response and development of allostatic load. Image from (McEwen, 1998)**.

According to McEwen (1998), life experiences and threatening or challenging situations have the potential to generate stress if they are perceived by an individual as being harmful, challenging, or threatening. If perceived as a threat, the person behaves accordingly, by fighting or fleeing, for example, and their physiological stress response is then triggered. This is a complex process that involves the triggering of a cascade of multiple regulatory biological systems that work largely through the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis (HPA-axis). These systems react to the perceived stressor by releasing catecholamines (from the sympathetic-adrenal-medullary axis) and, in instances of chronic stress, glucocorticoids (from the HPA-axis; McEwen, 1998), which trigger the activation or deactivation of other systems, such as the immune response, to actively deal with the threat (McEwen, 1998). Once the threat has ended, the systems are deactivated through a negative feedback process, enter a stage of recovery, and shortly return to their baseline levels. These adaptive processes of the sympathetic nervous system and HPA axis may become dysregulated over time due to persistent, enduring (chronic) stress exposure over prolonged periods of time (e.g., months, years, and even

decades), and this results in the wear and tear of the body's systems, as they may be unable to operate within their normal baseline levels, and this then results in increased vulnerability to a wide range of health problems (McEwen, 1998). Once this occurs, individuals who experience chronic stress are repeatedly exposed to stress hormones and this process contributes to allostatic load, a key contributor to age-related pathology such as cardiovascular disease and type 2 diabetes.

Similar to the lifespan developmental perspective, allostatic load is viewed as a cumulative, lifelong process, in that it does not occur within a short or defined period of time (McEwen, 1998; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Instead, it is the resulting collective and repeated exposure to stress hormones that gives way to allostatic load, and thus, morbidity and mortality. Available evidence indicates that allostatic load is socially patterned, where lower status individuals (e.g., those with low SES, racial minorities) experience greater allostatic load (Geronimus, Hicken, Keene, & Bound, 2006; Gruenewald et al., 2012).

What makes the concept of allostatic load so appealing and unique from the previously reviewed theoretical and conceptual frameworks is that it explains how social experiences affect biological responses that then give way to health problems. Unlike the lifespan developmental perspective, stress process, and intersectionality, McEwen's description of stress is broad, and while he presents social context (e.g., work, home, and neighborhood) as factors that contribute to perceptions of stress and resulting allostatic load, he does not provide a detailed account of how social structural arrangements and systems of social stratification work together to predispose some individuals to greater duration, types of, and frequency of stressors, and how an individual's location within a system (or systems) of social stratification contributes to their perception of whether a stimulus is a threat or not. The concept of allostatic load is most similar to the stress process, in that it describes a process whereby stressors exert deleterious effects on health through

various mechanisms, but McEwen takes a largely biological approach whereas Pearlin takes a sociological approach to understanding the stress-health relationship.

CONCEPTUAL FRAMEWORK

Figure 3 presents a conceptual model based on the available literature that will guide the research questions (RQs) in this dissertation. In this model, the solid lines are used to indicate key analytic variables for this dissertation and dashed lines are used to indicate key mechanisms for the process linking social status to health but will not be explored in this dissertation.

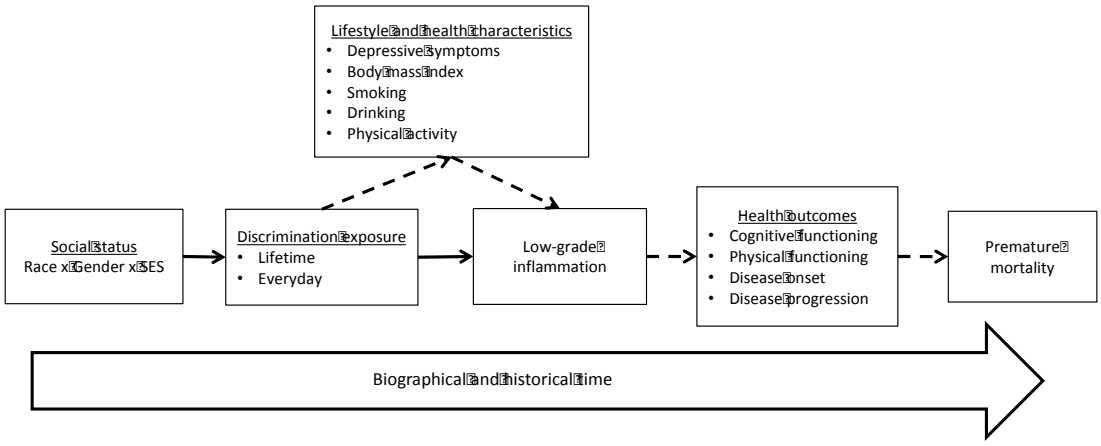


Figure 3. Conceptual model linking social status, discrimination exposure, and C-reactive protein.

These four approaches may have differences in their conceptualization of the way that the social environment produces health differentials, but are certainly not mutually exclusive. In fact, they can be integrated to provide a thorough explanation of: (1) the social patterning of stressors and health problems; (2) psychosocial mechanisms responsible for health problems; and (3) a guideline for how these processes may cumulate over a lifetime and produce disparate health outcomes in the population at midlife and older ages. Given the relative strengths of each of these perspectives, none of these is fully sufficient to explain the experiences and health consequences of intersecting social statuses, and a stronger understanding of these complex processes linked to health disparities can be gained through an incorporation of each of these approaches.

An integration of these four frameworks will more fully develop researchers' understanding of the origins of health disparities in that social status contributes to both greater exposure to stressors and allocates fewer protective psychosocial resources for buffering stressors' effects on health. Intersectionality can offer a new perspective to examine social status with, mainly by considering the extent to which exposure to stressors may vary across and within social groups. Intersectionality also offers a distinct conceptualization of what it means to be a member of an oppressed social group, as well as the implications of belonging to multiple systems of oppression or privilege. By integrating intersectionality with the stress process and lifespan developmental perspectives, scholars can better understand how these may translate to health, either directly or indirectly through risk factors like stress and discrimination exposure that are not race- or gender- or SES-specific, but exposures may be a unique combination of the three attributions of discrimination simultaneously. This approach will also clearly elucidate how the above processes unfold over the lifespan and historical time to influence how one perceives and understands threats and challenges that have the potential to cause stress. Through increased physiological wear and tear as a result of the cumulative insults from exposure to a variety of stressors, individuals who are most disadvantaged (in terms of belonging to the most lower social

status groups) are likely to be those who experience early and greater rates of morbidity and mortality. While beyond the scope of this paper, in addition to the effect of stress exposure, stress exposure and psychosocial resources may also work indirectly and partially through negative lifestyle factors to adversely affect health.

As shown in Figure 3, social statuses—s pecifically race, gender, and SES—are conceptualized as simultaneous but not necessarily equal contributors to acute and chronic exposure to discrimination throughout an individual's lifespan, as indicated by the lifespan developmental perspective and stress process theory (Aneshensel & Mitchell, 2014; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin & Bierman, 2013). These models suggest that those belonging to lower status groups have distinct experiences which contribute to their health in different ways, compared to higher status groups (Alwin & Wray, 2005; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin, 1989; Pearlin & Bierman, 2013). For example, Turner and Avison (2003) report that stress exposure is socially patterned but may depend on the stressor. They find that there is an unequal distribution of stressors, where Blacks and those in low SES groups disproportionately reported more exposure to both acute and chronic stressors. Other studies show that Blacks report more frequent exposure to both everyday and lifetime discrimination (Kessler, Mickelson, & Williams, 1999; Williams, Yu, Jackson, & Anderson, 1997).

The literature is less clear when it comes to the combination of social statuses because few studies have specifically examined how social statuses might interact to shape stress exposure, but two recent studies suggest that being in overlapping low social status groups (defined as being a racial/ethnic minority, woman, sexual minority, and obese) were more likely to be exposed to general stress, both everyday and lifetime discrimination, and more likely to be exposed to discrimination attributed to multiple factors (Grollman, 2012; 2014). While much of the stress and health literature views these social status categories as separate and independent of

one another, the intersectionality literature suggests that one's social statuses are not necessarily mutually exclusive—they can and often do work together to shape people's life experiences and social context (Crenshaw, 1991), such as discrimination exposure, and in turn, this exposure has the capability to negatively influence health (Bauer, 2014; Bowleg, 2012), such as through increasing C-reactive protein (CRP) levels.

In considering the simultaneous impact of social statuses on discrimination exposure and, in turn, CRP, this will allow for a multidimensional perspective on how psychosocial mechanisms contribute to health disparities in later life by examining within-group differences: the application of intersectionality will test whether there is an interaction among race, gender, and SES in exposure to lifetime and everyday discrimination and CRP levels. Given that race, gender, and SES are each individually considered critical determinants of social experiences, exposure to stress, health, and wellbeing, it is imperative to further examine how these three social statuses may interact to differentially affect experiences of discrimination and CRP, two mechanisms linked to health.

Based on previous work by Sternthal, Slopen, and Williams (2011) and Turner and Avison (2003), there tends to be greater exposure to stressors, like discrimination, in lower status groups, particularly when an individual belongs to a racial minority group in the United States. Indeed, Grollman's recent work (2012, 2014) demonstrated that those who are multiply disadvantaged (through belonging to multiple lower social status groups) experience significantly greater exposure to discrimination, and report more attributions of discrimination, and his work showed that these differences account for differences in both mental and physical health status of those who are in multiple low status groups compared to those with only one or no low social status.

Next, the model shows that over time, chronic psychosocial stressors such as discrimination exposure can lead to a dysregulated stress response, which is best described in the

allostatic load model (McEwen, 1998). Because the negative effects of stressors are theorized to accumulate over a lifetime, they are thought to contribute to physiological wear and tear, which places people who are disproportionately exposed to chronic stressors at an increased risk for health problems and premature mortality.

When a person is exposed to and perceives a stressor or a threat, the stress response is activated—this includes activation of a feedback loop involving the sympathetic-adrenal-medullary (SAM) axis and the HPA-axis (Glaser & Kiecolt-Glaser, 2005). Once each of these axes is triggered, each releases specific stress hormones that work to help the body maintain proper functioning and appropriately respond to the stressor (Glaser & Kiecolt-Glaser, 2005). Acute, or short-term stress (e.g., major life events), signals the SAM-axis, which triggers the adrenal medulla to release the catecholamines epinephrine and norepinephrine. If a stressor is chronic, the HPA-axis is then signaled, which triggers the hypothalamus to release coricotropin-releasing hormone (CRH), which triggers the release of adrenocorticotropic hormone (ACTH) from the pituitary gland, and this process triggers the release of cortisol from the adrenal cortex (Glaser & Kiecolt-Glaser, 2005; McEwen, 1998). Cortisol, one of the hormones released through this response, has an anti-inflammatory effect by binding to glucocorticoid receptors on immune cells, a process which causes decreased inflammatory cytokine production (Bennett, Fagundes, & Kiecolt-Glaser, 2013; Chrousos, 1995; McEwen, 1998; Miller, Cohen, & Ritchey, 2002). After the threat or stressor has subsided, the secretion of these hormone levels are gradually reduced through a negative feedback process: in the HPA-axis, cortisol production signals the hypothalamus and pituitary glands to halt the secretion of CRH and ACTH, respectively, which then reduces the amount of cortisol being produced, allowing the body's levels of the hormones to return to their normal circulating levels (McEwen, 1998). However, McEwen explains that when a person is exposed chronically to stressors, this process does not properly function as it should, leading to physiological wear and tear through a dysregulated stress response. In the case

of inflammation, a critical response to injury, infection, or tissue damage, this may be especially pronounced. Miller, Cohen, & Ritchey (2002) explain that while glucocorticoids (e.g., cortisol) normally have an anti-inflammatory effect in response to stress, chronic stress may lead to higher circulating levels of glucocorticoids in turn, contribute to a *pro-inflammatory* rather than anti-inflammatory response through glucocorticoid receptor resistance, a process that occurs when glucocorticoids cannot bind to their receptors on immune cells to downregulate inflammation as it should (Bennett, Fagundes, & Kiecolt-Glaser, 2013; Cohen et al., 2012; Miller, Cohen, & Ritchey, 2002). The ability of cortisol to bind to its receptors is a key mechanism involved the negative feedback loop to halt the production of cortisol and other stress hormones, and a disruption in this process can result in chronically elevated levels of inflammatory markers, such as CRP (Bennett, Fagundes, & Kiecolt-Glaser, 2013; Cohen et al., 2012; Miller, Cohen, & Ritchey, 2002).

Research Questions and Hypotheses Addressed in this Dissertation

This dissertation will focus on the way that social status combinations contribute to health disparities via their influence on discrimination exposure and CRP levels in midlife and older ages. The following research questions (RQs) will be addressed by testing the hypotheses presented following each RQ:

RQ #1: How are social statuses linked to low-grade CRP levels in midlife and older adults?

H1. Non-Hispanic Black adults will have higher CRP than non-Hispanic Whites.

H2. Women will have higher CRP than men.

H3. Those with lower SES will have higher CRP than those with higher SES.

H4. Black women will have higher CRP than White women, Black men, and White men.

H5. There will be a significant three-way interaction between race, gender, and SES on CRP levels, where Black women with lower SES will have the highest levels of CRP.

RQ #2: Does discrimination exposure (everyday and lifetime) vary across and within social status groups by race, gender, and SES?

H6. Discrimination exposure (everyday and lifetime) will be significantly higher for Blacks.

H7. Discrimination exposure (everyday and lifetime) will be significantly higher for men.

H8. Discrimination exposure (everyday and lifetime) will be significantly higher for those of lower SES.

H9. Discrimination exposure (everyday and lifetime) will be significantly higher for Black women.

H10. Discrimination exposure (everyday and lifetime) will be significantly higher for Black women of lower SES compared to combinations of other social status groups.

RQ #3: Is discrimination exposure (everyday and lifetime) associated with CRP in midlife and older adults?

H11. Everyday discrimination will be positively associated with CRP.

H12. Lifetime discrimination will be positively associated with CRP.

RQ #4: Does discrimination exposure (everyday and lifetime) account for the relationship between race x gender x SES and CRP?

H13. Everyday discrimination will attenuate the three-way interaction among race, gender, and SES on CRP (as tested in Aim 1).

H14. Lifetime discrimination will attenuate the three-way interaction among race, gender, and SES on CRP (as tested in Aim 1).

The dissertation will consist of sets of research analyses that build upon one another in a stepwise manner to understand the mechanisms involved linking social status to negative health outcomes. The first aim will focus on the social distribution of CRP levels at baseline and over a four-year period of time—specifically, whether CRP varies not only across race, gender, and SES groups independently, but also at the intersection of these groups, by testing a race x gender x SES interaction term on CRP. Then, Aim 2 will examine whether the distribution of everyday and lifetime discrimination exposure varies by combinations of race, gender, and SES. If there are significant variations in exposure to everyday and lifetime discrimination, then the final step of this dissertation will explore whether everyday and lifetime discrimination help explain the relationship between race x gender x SES and CRP. The following chapter will provide details regarding the data, measures, and methods used to address these aims. Chapter 5 will then present the results of the analyses related to Aims 1 and 2.

CHAPTER 4

DATA, MEASURES, AND METHODS

This chapter begins with an overview of the source of the dataset for this dissertation: the Health and Retirement Study (HRS). It then details the HRS sampling and collection procedures, provide details on how the specific waves of data for this dissertation were put together. The measures used for each aim is provided, with information on how each variable was coded. Finally, the statistical analysis plan for each aim is described.

DATA SOURCE

Overview of the Health and Retirement Study

This dissertation utilizes panel data from the Health and Retirement Study (HRS). The HRS is an ongoing nationally representative panel study of more than 20,000 non-institutionalized midlife and older adults ages 51 and older living in the United States and their spouses. It has an average response rate ranging from 87-90% (HRS staff, 2017; Juster & Suzman, 1995). It is sponsored by the National Institutes on Aging (NIA U01AG009740) and the Social Security Administration and conducted by the Institute for Social Research (ISR) at the University of Michigan.

The HRS was initially designed as an effort to gain a better and more thorough understanding of the retirement process, and, in particular, to provide more data on the aging population, as a response to its rapid growth, in order to better prepare for policy changes that may arise due to the changing age composition of the United States (Juster & Suzman, 1995;

Sonnega et al., 2014). Surveys have been routinely collected every two years since 1992, when the HRS was initiated. The current HRS sample was initially comprised of two separate cohorts: the HRS cohort (collected in 1992, 1994, and 1996), a sample of adults who were born during 1931 to 1942 (aged 51-61) and their spouses; and a second study, called the Asset of Health Dynamics Among the Oldest Old (AHEAD), collected data in 1993 and 1995 on cohorts born from 1890-1923 (ages 70+) to better understand the oldest-old, an age group where data at the time were sparse (Sonnega et al., 2014). The AHEAD cohort was sampled in an attempt to investigate older age individuals without waiting for 20 years for the HRS cohort to reach these ages (Juster & Suzman, 1995). Thus, the data collected on each of these cohorts was similar and in some cases, identical, except that the AHEAD cohort dealt more with functional health rather than the labor market (Juster & Suzman, 1995). In 1998, the HRS and AHEAD cohorts were merged in order for researchers to examine the population aged 51+ more comprehensively, with the new sample retaining the name: HRS (HRS staff, 2017; Sonnega et al., 2014). This new HRS sample would then be interviewed at the same time, beginning in 1998 (HRS staff, 2017). At the time that the two cohorts were merged, two additional birth cohorts were added to the HRS sample: the Children of the Depression Era (CODA), which consisted of respondents born from 1924-1930; and the War Babies (WB), which consisted of respondents born from 1942-1947 (HRS staff, 2017). These two new cohorts were added to maintain the sample size of the HRS. From this point on, every six years, a new birth cohort is added to retain the overall sample size of the HRS (Sonnega et al., 2014).

The primary aim of the current HRS is to understand characteristics of the midlife and adult population in the United States, and the HRS routinely collects information at the individual and household level for demographic background, socioeconomic status indicators, health risks, and health conditions, among other topics through the use of core interviews, which are conducted every two years (Juster & Suzman, 1995).

HRS Sampling Procedures

The HRS sample consists of non-institutionalized adults ages 51+ living in the United States. In order to ensure that the sample was racially/ethnically diverse and nationally representative, HRS researchers regularly oversampled Black and Hispanic populations, in addition to oversamples of Florida residents (Heeringa & Connor, 1995). The HRS provides several sample weights in order to account for the differential selection into the sample and oversampling processes, and the specific weight used for analyses depends on the sample (e.g., whether at the household or respondent level; whether core, leave-behind questionnaire, or biomarker collection; Heeringa & Conor, 1995).

Respondents were chosen through a multi-stage area probability sample design that consisted of four stages of selection. The respondents were first selected based on geographical location, which were then narrowed down into smaller segments, where specific households were selected for participation within these smaller segments (Heeringa & Connor, 1995). Finally, respondents were selected into the sample if their household financial unit was eligible for the study, and this was determined whether there was at least one age-eligible respondent, who could be single or unmarried.

To account for the complex design of the HRS, additional variables were required in survey-specific procedures for statistical analysis. In particular, the HRS provides two variables—stratum and cluster variables—that must be used in conjunction with a sampling weight. The sample weight used in analyses varies depending on whether analyses focus on the household or respondent and whether analyses include respondents who participated in special modules (e.g., biomarker data).

HRS Data Collection Procedures

The data from this dissertation come from the core interviews (2006 and 2008 waves), the psychosocial leave-behind questionnaire (2006 and 2008 waves), and the biomarker data (2006-2012 waves). At the respondent's baseline interview, data was typically collected through an in-person interview; and every two years (referred to as a wave) thereafter, the respondents were asked to complete a series of questions for the core interview, which took place in person, over the phone, or through a designated proxy (Sonnega et al., 2014). The majority of follow-up interviews were completed through phone or an enhanced face-to-face format (EFTF), depending on whether the respondent was randomly assigned to an EFTF, and the only exception to this is that respondents who were 80 years of age or older were offered an in-person interview for all follow-up waves (Sonnega et al., 2014).

Initially, the HRS consisted of the core interviews only, but over time, HRS investigators began to routinely include modules during specific waves to examine additional topics of interest to researchers in other disciplines, such as social, psychological, and health scientists (Clarke, Fisher, House, Smith, & Weir, 2008; Servais, 2010; Smith et al., 2013). In 2004, the HRS released a pilot aimed to collect preliminary data on psychosocial measures through a self-administered, in-person leave-behind questionnaire after completion of the core interview. After the success of this pilot data collection, the HRS researchers began to collect additional information from respondents through enhanced face-to-face interviews (EFTFs) starting in 2006, including anthropometric, biomarker, and psychosocial data (Servais, 2010).

The EFTF was attained through randomly placing respondents' households into one of two rotating groups consisting of approximately 50% of the total sample, with the first half of households receiving the EFTF in 2006 and the second half in 2008 (Clarke, Fisher, House, Smith, & Weir, 2008; Smith et al., 2013). Respondents then completed interviews every other

wave for follow-up EFTF data—the first half of respondents completed their second EFTF survey in 2010, and the second half of respondents completed their second EFTF survey in 2012, given that they met the eligibility criteria for the EFTF (Clarke, Fisher, House, Smith, & Weir, 2008; Smith et al., 2013). A respondent was considered eligible for the EFTF if they were non-institutionalized and completed their core interview on their own (rather than through using a proxy).

Psychosocial data were not initially an area of focus in the HRS, but HRS researchers began collecting psychosocial measures after the release of a report on the importance for future research to investigate the psychosocial factors related to health, well-being, and retirement in midlife and older ages (Smith et al., 2013). In order to determine which measures should be included in their investigation of psychosocial factors, HRS investigators created a scientific review committee designed to review the available literature and make recommendations for measures to be used based on this literature (Smith et al., 2013). The psychosocial measures to be included in the leave-behind questionnaire (LB) were adapted from successful existing studies, such as the English Longitudinal Study of Ageing (Smith et al., 2013).

After the 2004 pilot LB was completed, the HRS scientific review committee reconvened to discuss the possibility of renewing the LB for future waves (Smith et al., 2013). They concluded that the LB would require additional grounding in psychological theory and that the National Institutes on Aging would have a subcommittee on HRS Data Monitoring to consult regarding these changes, along with additional experts in psychology of aging (Smith et al., 2013). These consultations led to the revised versions used in subsequent waves of data collection, including six key domains: subjective well-being, lifestyle and experiences of stress, quality of social ties, personality traits, work-related beliefs, and self-related beliefs. The LB questionnaire covers an assortment of stressors, including measures of everyday and lifetime

discrimination. In 2006, the LB had a 90% response rate, with an overall response rate of 74% after factoring in those who completed the core interview (Clarke et al. 2008; Smith et al. 2013).

Beginning in 2006, the HRS began to collect biological samples from respondents who were asked to complete the EFTF (Crimmins et al., 2013). The HRS utilized the dried blood spot method for collecting blood because there are no temperature control requirements for preserving the blood with this method, which makes transportation, storage, and processing of blood-based samples easier (Crimmins et al, 2013). A randomly selected 50% of respondents were assigned to biomarker collection in 2006, referred to as panel A, and the second half were selected for collection 2008, referred to as panel B. Each subset was reinterviewed every other wave (every four years), meaning that panel A had biomarker data collected in 2006 and 2010, while panel B had biomarker data collected in 2008 and 2012. Respondents had to complete their interviews on their own and not via proxy, could not reside in a nursing home, and had to complete their interview in person (Crimmins et al., 2013). If the respondent provided their consent, then an HRS interviewer explained the procedures for collection and briefly demonstrated how the measure would be collected. The respondents were also asked to explain whether they understood the directions for collection and whether they felt safe to have their sample collected (Crimmins et al, 2013). When a respondent did not answer affirmatively, then the sample was not collected. The consent rate for blood spots was: 83% in 2006; 87% in 2008; 85% in 2010; and 87% in 2012 (Crimmins et al., 2013; Crimmins, Faul, Kim, & Weir, 2015).

According to the procedures outlined by Crimmins and colleagues (2013), conditional on consent, the HRS interviewer asked the respondent to rub their hands together or to massage them quickly to get blood flowing. After this, blood was drawn by pricking the respondent's finger with a sterile lancet after the finger was cleaned with an alcohol swab. An initial pool of blood was wiped away with a piece of gauze, and then the next pool of blood was used on the blood spot card (Crimmins et al., 2013). The HRS interviewer instructed the respondent to put droplets

of blood directly on a piece of specially treated filter paper with six circles printed across it (Crimmins et al., 2013). Ideally, all of the circles should be filled, but this was not always successful (Crimmins et al., 2013). Once the respondents' blood was collected on the filter paper, the paper was placed in a special foil envelope with a desiccant packet and shipped to specific laboratories for assay (Crimmins et al., 2013).

HRS Data Preparation

The data used for this dissertation were accessed through the HRS website, where users must first register, and are then able to download and analyze publicly available data (such as the core interviews). Additionally, data for this dissertation were prepared by locating the specific section (e.g., leave-behind, health, biomarker) of the HRS wave to be utilized. In addition to wave-specific data files, there is also a tracker file that records the HRS respondents' details, including demographic characteristics (e.g., age at each interview, race/ethnicity), EFTF assignment, completion of specific modules (e.g., biomarker data), as well as household and respondent weights for each wave and specific modules (e.g., LB questionnaire and biomarker data).

In order to increase accessibility of the wide variety of data offered by the HRS, as well as to encourage longitudinal research in aging studies, the RAND Center for the Study of Aging, along with support and funding from the National Institute on Aging and the Social Security Administration, released a series of HRS data files that have been cleaned and reorganized to create consistency across waves where measures and their wording may vary slightly (Sonnega et al., 2014). RAND collects variables that are asked of respondents at all (or nearly all) waves, and combines them into a single data file which consists of a similar naming convention across waves, since some variable names changed over time, along with new variable names that

specifically refer to whether the measure is from the household or respondent level, which wave of HRS collection the variable was in, and an easily identifiable name (Servais, 2010). In addition, RAND cleaned up the data and imputed values for variables, such as wealth, where there were large amounts of missingness (RAND, 2016).

Table 1 includes all variables to be included in this dissertation, along with the method the HRS used to gather the data, the selection of responses that a respondent could choose from, which of the HRS datasets the variable is located in, and whether the variable was available for all waves of collection or a subset of waves.

Table 1. **Constructs and information on measurement using data available in the HRS (2006-2012)**.

Construct	How obtained	How Measured	Location in Datasets	Wave(s) Available
C-reactive protein	Dried blood spot sample	C-reactive protein level (mg/L)	HRS Biomarker File	Half of respondents has data in 2006, the other half has data in 2008; Repeated collection after four years
Race	Self-report selection of one racial category	Non-Hispanic White Non-Hispanic Black	HRS core survey	All waves
Gender	Self-report	Men Women	HRS core survey	All waves
Education	Self-report of years of education completed	Number of years of education completed	RAND data file	All waves
Income	Self-report of the sum of all income in the last calendar year by respondent and spouse	A summary of all sources of income from the respondent and spouse were calculated	RAND data file	All waves
Net Worth	Self-report value of all assets and debts	Variable was calculated by summarizing all assets and subtracting all debts from this value	RAND data file	All waves
Lifetime Discrimination	Self-report	At any point in life, has the respondent been unfairly: 1. Dismissed from a job? 2. Not been hired for a job? 3. Denied a promotion? 4. Prevented from moving into a neighborhood because the landlord or	HRS Leave-Behind Psychosocial Survey	Half of respondents has data in 2006, the other half has data in 2008; Repeated collection after four years

		realtor refused to sell or rent the respondent a house or apartment? 5. Denied a bank loan? 6. Stopped, searched, questioned, physically threatened, or abused by the police?		
Everyday Discrimination	Self-report	Within the past two years, how frequently has the respondent reported: 1. Being treated with less courtesy or respect than other people 2. Receiving poorer service than other people at restaurants or stores 3. People acting as if they think the respondent is not smart 4. People act as if they are afraid of the respondent 5. Being threatened or harassed	HRS Leave-Behind Psychosocial Survey	Half of respondents has data in 2006, the other half has data in 2008; Repeated collection after four years
Age	Self-report of date of birth	Age in years	HRS core survey	All waves
Marital Status	Self-report of current marital status	Never married Married Separated/Divorced Widowed	HRS core survey	All waves
Smoking	Self-report of current smoking status	Not a current smoker Past smoker Currently smoke	RAND data file	All waves
Alcohol Use	Self-report	In the past 3 months, on the days you drink, about how many drinks do you have?	RAND data file	All waves
Physical Activity	Self-report 1. more than once a week 2. once a week 3. one to three times a month 4. hardly ever or never 5. every day	Moderate Activity - how often do you take part in sports or activities that are moderately energetic such as, gardening, cleaning the car, walking at a moderate pace, dancing, floor or stretching exercises Vigorous Activity - how often do you take part in sports or activities that are vigorous, such as running or jogging, swimming, cycling, aerobics or gym workout, tennis, or digging with a spade or shovel	RAND data file	All waves
CESD	Self-report of whether or not the respondent reported any of the feelings within the past week	Much of the time during the past week: You felt depressed You felt that everything you did was an effort Your sleep was restless You were happy You felt lonely You enjoyed life You felt sad You could not get goings	RAND data file	All waves

BMI	Self-report of height at study entry and weight each wave	Variable calculated as weight converted to kilograms divided by square of height in meters; height from study entry is carried over to each subsequent wave	RAND data file	All waves
Chronic Conditions	Self-report of doctor diagnosis for sum of total conditions	High blood pressure Diabetes Cancer Lung disease Heart disease Stroke Arthritis	RAND data file	All waves

Sample Selection

The data from this dissertation include pooled HRS data derived from the core interview (2006 and 2008 waves), the tracker file, the LB (2006 and 2008 waves), and the biomarker data (2006, 2008, 2010, and 2012 waves). A random rotating subset of respondents completed LB and biomarker data in 2006 and 2010; the remaining respondents completed the LB and biomarker data in 2008 and 2012. The final analytic sample included 5,486 HRS respondents who were eligible for and completed both the LB and biomarker component in addition to the core interview in either 2006 or 2008. The respondents in this study were ineligible if they had a missing CRP value at baseline or follow-up; if they had a CRP value over 10.0 mg/L at baseline or follow-up (because this is indicative of ongoing injury or infection, rather than chronic, low-grade inflammation); were Hispanic ethnicity or a race other than Black or White; and if they had missing data on independent variables. The flowchart provided (Figure 4) shows how the final sample for this dissertation was derived.

Separate sets of analyses were conducted to assess whether respondents who were excluded from this study were significantly different from included respondents on all variables. These analyses revealed that there were significant differences between excluded and included respondents: excluded respondents were significantly more likely to be Black, have lower

income, have less wealth, have significantly higher CRP at baseline and follow-up, and report more frequent everyday discrimination. They were also significantly more likely to be older, non-married, more likely to be a past smoker, less likely to be a current smoker, less likely to report being a moderate or heavy drinker, less likely to engage in moderate or vigorous physical activity, and have more depressive symptoms. This means that this study's findings may yield conservative estimates.

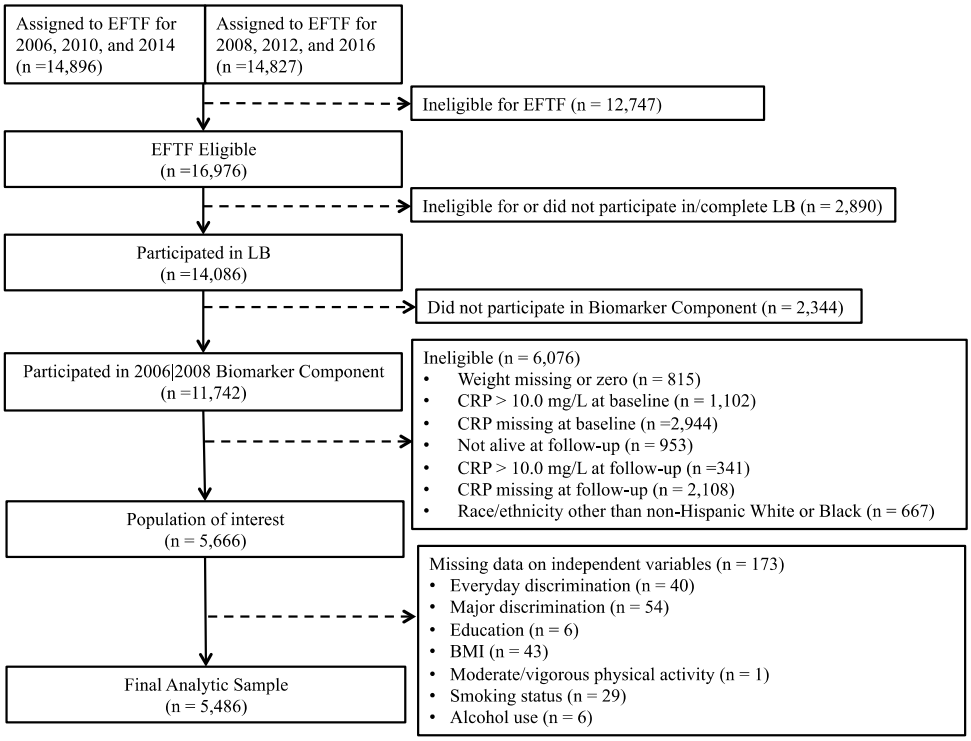


Figure 4. Flowchart of sample selection criteria and sample size at each stage of sample selection.

HRS Respondent Data Protection Procedures

From its inception, HRS researchers have made concerted efforts to preserve the privacy and protect the confidential data provided to them by its respondents. This was done through several steps. First, the HRS requires that anyone who wishes to access public data files must formally register through their website. In addition, researchers who wish to access sensitive data (e.g., biomarkers) must also receive special permission, and to complete a user agreement form. Along with this form, once a registered user is approved to access these data, they are provided with a unique set of passcodes in order to download and open the sensitive data files.

Prior to public distribution, all HRS respondents' personal identifying information (e.g., name, address, etc.) is removed from the data files, and each respondent is assigned specific identifying codes, HHID and PN (Weir, 2017). These codes correspond to their household identification number and person number, respectively.

All respondents are required to provide their consent for their participation in the study; and additional written consent is required when the respondent is selected for additional modules (e.g., biomarker data collection). The HRS is approved by the University of Michigan's Institutional Review Board (Weir, 2017).

MEASURES

This section will consist of details on the measures used for this dissertation. It includes details on the type of measure (as presented in Table 1), what part of the HRS the data were located (e.g., LB, tracker) if it came from a location other than the core survey, how the measure was obtained (e.g., self-report), and how the variable was measured. Additional details, such as psychometric properties and coding, are also described.

Study Variables

This section will provide a description of the dependent variable, C-reactive protein (CRP), the independent and covariate variables. It will first describe each of the primary analytical variables, race, gender, and socioeconomic status, and then each of the additional variables will be explained. Each of the covariates included in models were chosen due to their existing relationships between social status and CRP.

Dependent Variable

C-Reactive Protein (CRP). CRP is a measure of systemic inflammation that increases in response to various stimuli, including chronic stress. Respondents' blood was collected in 2006 and 2010 or in 2008 and 2012 by a trained HRS interviewer using the dried blood spot (DBS) process and shipped to either Biosafe or the University of Vermont for CRP (2006) the University of Vermont (2008), and the University of Washington (2010-2012). The CRP sample was measured with a BNII nephelometer (Siemens, Inc., Deerfield, IL) and assayed with a dilution of 1:83.3. If the sample's CRP levels were too low to be detected through this method, then it was rerun using a sandwich Elisa test (R&D Systems # DCRP00, Minneapolis, MN) and assayed with a dilution of 1:50 using two of the spaces on the DBS card (Crimmins et al., 2013). Because consent rates vary across social characteristics, particularly race, the HRS takes special steps to routinely oversample racial minorities and to retain minority respondents (Ofstedal & Weir, 2011). The blood-based consent rates for Blacks was significantly lower than for Whites and in both 2006 and 2008, leading the HRS to meet with interviewers, improve interview and collection processes, and take additional steps to ensure the comfort, safety, and privacy of its minority respondents (Ofstedal & Weir, 2011).

The HRS released a variable called the "NHANES equivalent value" which was employed for all analyses because it adjusted DBS values on a distribution aligned with the NHANES (Crimmins et al., 2013; Crimmins et al., 2014). This variable also transforms DBS values to serum values, as research shows that there is a slight difference in the values when collected via DBS or serum (Crimmins et al., 2014). Finally, all CRP values were logarithmically transformed for all analyses due to skewness.

As recommended by the AHA/CDC, all cases with CRP values exceeding 10 mg/L were excluded from analyses as these may indicate ongoing infection or acute illness (Pearson et al., 2003). Analyses were conducted to assess whether those who had high CRP at baseline, follow-up, or at both baseline and follow-up were significantly different (Table A4 in Appendix). Several sociodemographic, lifestyle, and health characteristics were associated with the likelihood of having very high (>10 mg/L) levels of CRP. These analyses revealed that being Black, being a woman, have lower SES, more everyday discrimination, more major discrimination, fewer protective lifestyle factors, and worse overall health were associated with increased odds of having very high CRP at baseline and follow-up. The 200 respondents who had very high CRP at both baseline and follow-up were more likely to be younger, be Black, be women, have lower SES, report more major discrimination, be unmarried, be overweight/obese, have higher depressive symptoms, have more chronic health conditions, not engage in moderate or vigorous physical activity, and report never drinking. This means that individuals who were had acutely elevated CRP might also be those who had low-grade elevations of CRP had their CRP been tested within two weeks; and this suggests that the estimates derived from this work might be conservative due to the exclusion of these potential respondents.

Independent Variables

Race. The respondents were asked to report their race and ethnicity. The present study includes respondents who reported being non-Hispanic Black/African American (coded as 1), or non-Hispanic White/Caucasian (coded as 0).

Gender. Respondents provided their self-reported gender, with 0 = man and 1 = woman.

Education. Respondents' educational attainment was assessed through self-report and operationalized in two ways: first, a continuous variable for years of education was created (range, 0-17); and then another variable used a categorization of education coded as 2 = less than high school education, 1 = high school education/GED, and 0 = some college or more.

Income. A variable for total household income in self-reported dollars was provided from the RAND data, and is the sum of the respondent and, if they have one, their spouse's reported income at baseline. It includes a sum of all income in the respondent's household from the respondent's earned income, their spouse's earned income, household capital income (e.g., income from self-employment, stocks, bonds, savings, CDs, rental properties, checking and savings account), income from the respondent and spouse's pension or annuity, income from social security disability or supplementary security, income from social security retirement, unemployment or worker's compensation, and other sources of income. The original income value had a constant value of one added to it, and was then logarithmically-transformed to achieve a normal distribution.

Wealth. A variable for net assets were included as a measure of total wealth minus total debt was drawn from the RAND file, which includes the net value of mortgages/land contracts (primary and secondary residences); value of other home loans; net value of real estate (not primary residence); net value of vehicles; net value of businesses; net value of stocks, mutual funds, and investment trusts; IRAs; value of checking, savings, or money markets; value of CD,

government savings bonds, and T-bills; net value of bonds and bond funds; value of other debt; and all other savings. Due to a non-normal distribution and negative values, the variable was logarithmically-transformed after adding a constant value of one to the respondent's value for wealth. All log-transformed values of wealth with a value less than 0 were multiplied by -1 to maintain the negative values of wealth.

Everyday discrimination was evaluated using a well-established 5-item questionnaire (Williams, Yu, Jackson, & Anderson, 1997) that measures the frequency of discrimination in the respondent's day-to-day life in the LB. The scale consists of the following five items: (1) You are treated with less courtesy or respect than other people; (2) You receive poorer service than other people at restaurants or stores; (3) People act as if they think you are not smart; (4) People act as if they are afraid of you; and (5) You are threatened or harassed. Responses were coded as: 0 = never, 1 = less than once a year, 2 = a few times a year, 3 = a few times a month, 4 = at least once a week, and 5 = almost every day. This variable was calculated by taking the average of the score of the five items (range, 0-5, with higher scores suggesting more everyday discrimination in the past year). If the values of more than three items were recorded as missing, the final score was coded as missing. The psychometrics of this scale were tested in 2006, $\alpha = 0.80$ and again in 2008, $\alpha = 0.82$ (Smith et al., 2013).

Lifetime experiences of discrimination were measured through a 6-item questionnaire in the LB (Williams, Yu, Jackson, & Anderson, 1997) designed to capture whether or not respondents reported major experiences of unfair treatment at any point in their life. This questionnaire includes the following items: (1) At any time in your life, have you ever been unfairly dismissed from a job?; (2) For unfair reasons, have you ever not been hired for a job?; (3) Have you ever been unfairly denied a promotion?; (4) Have you ever been unfairly prevented from moving into a neighborhood because the landlord or realtor refused to sell or rent you a house or apartment?; (5) Have you ever been unfairly denied a bank loan?; and (6) Have you ever

been unfairly stopped, searched, questioned, physically threatened or abused by the police? The responses were coded as 1 if the event occurred, and 0 if the event did not occur, and the responses were then summed.

Covariates.

The following variables were included in analyses based on prior literature indicating they may play a confounding or mediating role in the relationships among social status, discrimination, and CRP.

Age. Age included as a continuous measure of the respondents' age in years at the beginning of the baseline interview for this dissertation, as located in the Tracker file (range, 52-95).

Marital Status. Using data from the Tracker file, a variable for the respondent's marital status at baseline was created. This variable was coded as 1 = married and 0 = not married.

Drinking. To assess for alcohol use at baseline, a measure of self-reported alcohol use was drawn from the RAND data file. This question asked how many drinks per day the respondent had when they would drink and was a continuous measure. Never drinkers reported not drinking any alcoholic beverages per day; moderate drinkers reported drinking one or two drinks per day; and heavy drinkers were coded as those drinking two or more drinks per day. The final variable for respondent's drinking status was coded as: 0 = never drinker, 1 = moderate drinker, and 2 = heavy drinkers.

Smoking. Two self-reported measures of smoking were also collected from the RAND data file and recoded to capture the respondent's smoking status at baseline. The first question asked whether the respondent had ever smoked, which could be answered with yes, no, or refused/missing. Then, a second measure of smoking assessed the respondent's current smoking

status, which indicated whether the respondent smoked at the time of the survey or not. These two variables were then combined into a three-level variable for smoking status: 0 = never smokers, 1 = former, but not current smokers, and 2 = current smokers.

Physical Activity. Self-reported measures of moderate and vigorous physical activity were drawn from the RAND file. These measures assessed the frequency that the respondent engaged in moderate and/or vigorous physical activity at baseline. The values were recoded into a new variable with three levels: 0 = respondent never engaged in moderate or vigorous physical activity, 1 = respondent sometimes engaged in moderate or vigorous physical activity, and 2 = respondent frequently engaged in moderate or vigorous physical activity.

Health Characteristics. Physical and mental health variables in analyses included a continuous measure of body mass index (BMI, calculated as weight divided by squared height); and a measure of depressive symptomatology within the past week using the Center for Epidemiological Studies Depression short eight-item scale (Radloff 1977; CESD; range 0-8). A chronic health condition variable was also included to indicate a count of diagnoses of: hypertension, arthritis/rheumatism, diabetes, cancer (excluding skin cancer), lung problems, stroke/transient ischemic attack, and heart problems (heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems; range, 0-7).

STATISTICAL ANALYSES

Data Management

Data were managed using Stata SE, version 14.2, which included dataset merging, data cleaning, the recoding and creation of variables, and data analysis. All analyses adjusted for the complex design of the HRS using Stata's *svy* commands; stratum and cluster variables provided

by the HRS to accurately estimate standard errors; and all analyses were weighted to account for differential probability of being selected into the sample. The use of these variables with the *svy* command allowed the results to be generalizable to the non-institutionalized population of adults over 50 years old residing in the contiguous United States.

Descriptive Statistics

Prior to testing the hypotheses for this dissertation, descriptive analyses were generated to evaluate the characteristics of the sample (n = 5,486). This included means and standard deviations (SD) for all continuous variables and frequencies for all categorical variables. Bivariate analyses (using *t* tests, adjusted Wald tests, and correlation matrices) were used to evaluate statistical significance ($p \leq .05$), along with other characteristics, such as the magnitude and direction of the association between variables to be included in models (variables shown in Table 1).

CRP levels were compared within and across combinations of race and gender: descriptives were first generated for the sample stratified by race, and significance tests were conducted to assess significant race differences ($p \leq .05$) for all variables of interest. T-tests were run to examine whether there was a significant race difference in continuous variables; and chi-square tests were used to assess race differences in categorical variables.

Next, descriptive analyses were generated for the sample stratified by gender within race. Following this, a series of tests were run to assess for significant differences from White men ($p \leq .05$) across race-gender groups; and whether there were gender differences within race groups. This was completed using the *lincom* command available in Stata, following guidelines described on the Survey Data Analysis with Stata 14 by the University of California Los Angeles' Institute for Digital Research and Education, Statistical Consulting Group.

Aim 1 Analytical Plan

Ordinary least squares (OLS) regression models tested Aim 1, which explored how social statuses (e.g., race, gender, and SES) are linked to continuous measures of low-grade C-reactive protein (CRP) at baseline and over a four-year span using a nationally-representative sample of midlife and older adults. In particular, it examines whether the effects of race, gender, and SES are independent or interactive. This was first assessed by regressing log-CRP on race, gender, SES, age, and marital status. Next, a two-way interaction term, race x gender, was added to the model. Third, to examine whether social statuses interact to produce differential levels of CRP, a three-way interaction term, race x gender x [measures of SES], was added to the model. Separate models were run for each measure of SES. Interpretation of a significant interaction term was facilitated with graphical representation. A fourth model adjusted the above model for lifestyle characteristics (drinking status, smoking status, and physical activity); and a final model adjusted for mental and physical health conditions (CESD, BMI, and chronic health conditions), in addition to lifestyle and sociodemographic characteristics. Identical models were run to examine the distribution of CRP over four years by regressing log-CRP at follow-up on all baseline models described above; the only difference was that a control variable for CRP at baseline was included in each of these models.

Aim 2 Analytical Plan

The following analyses were conducted to address Aim 2, which investigated whether everyday and lifetime discrimination exposure accounted for race, gender, and SES variations in low-grade C-reactive protein (CRP) at baseline and over a four-year span. In order to examine whether discrimination exposure accounts for social status variations in CRP, mediation analyses

were planned through a joint significant test (MacKinnon et al., 2002).

Prior to mediation analyses, descriptive statistics were generated to evaluate the characteristics of the sample, namely the social distribution of lifetime and everyday discrimination across combinations of race and gender; and the bivariate association between lifetime and everyday discrimination with CRP. A series of *t* tests, adjusted Wald tests, and correlation matrices were run to compare lifetime and everyday discrimination with study variables. Then, additional models assessed the significance, strength, and magnitude of the association between everyday and lifetime discrimination with CRP.

In order for mediation to be present, criteria must be met. According to MacKinnon and colleagues (2002), a joint significance test can determine evidence of mediation if the path linking the predictor to the mediator (α path) is significant and if the path linking the mediator to the outcome (β path) is significant, while controlling for the predictor. Thus, the α path in this model is the path linking race x gender x SES to each measure of discrimination exposure. The β path is the path linking each measure of discrimination exposure to CRP.

If the analyses in Aim 1 reveal statistically significant interactions, then a series of regression analyses will be used to assess for mediation using the joint significance test. In the first set of models, each measure of discrimination will be separately regressed on the three-way interaction between race, gender and SES, controlling for age and marital status, and assessed for statistical significance ($p \leq .05$). Following this, a second set of models will regress CRP at follow-up on the race x gender x SES interaction and everyday and lifetime discrimination in separate models, controlling for CRP at baseline, race x gender x SES, age, and marital status. Evidence of statistical mediation was assessed through comparison of the coefficients, standard errors, and *p*-values in the models measuring the total effect (the race x gender x SES interactions from Aim 1) to the results from Aim 2 to assess whether the indirect effect of the race x gender x SES interaction

on CRP at follow-up through each measure of discrimination significantly differs from 0. Two additional models will be run in blocks, as in Aim 1, with the next model adjusted for lifestyle characteristics (e.g., drinking status, smoking status, and physical activity); and the final model adjusting for health conditions in addition to all variables in previous models.

SUMMARY

This chapter summarized information on the dataset that will be used to address the aims of this dissertation, the HRS' sampling and collection procedures, and variables included and their sources within the HRS. It then laid out the key analytic variables and covariates that were included in models, and it provided a summary of the statistical analyses that were conducted to test each aim of this dissertation. All analyses for this dissertation were run in Stata using HRS-issued *svy* variables to account for the complex design of the HRS and to allow the results to be generalizable to the non-institutionalized midlife and older population in the United States. The following chapter will describe the relevant sample statistics and present the findings for each aim, as outlined above.

CHAPTER 5

RESULTS

This chapter presents sample descriptive characteristics and the results from Aims 1 and 2. Aim 1, which answered Research Question (RQ) #1, explored how social statuses (e.g., race, gender, and SES) are linked to low-grade C-reactive protein (CRP) at baseline and over a four-year span using a nationally-representative sample of midlife and older adults. Aim 2, which answered RQs #2-4, investigated whether increased everyday and lifetime discrimination exposure accounted for race, gender, and SES variations in low-grade C-reactive protein (CRP) at baseline and over a four-year span. For Aim 2, the analyses first tested whether race, gender, and SES interacted to produce differential exposure to everyday and lifetime discrimination. Then, based on the results from the analyses, the decision was made to further explore whether everyday and lifetime discrimination were associated with CRP at baseline or over the four-year follow-up, regardless of whether or not there were race, gender, and SES variations in exposure to discrimination.

The sample is first described overall; then, it is described by race, and within race by gender simultaneously. All log-transformed variables are presented in their original, non-transformed format for ease of interpretation in the descriptives and figures. The regression findings for Aim 1 are presented separately for each indicator of SES and further broken down by analyses examining CRP at baseline and follow-up. Then, the regression findings for Aim 2 are presented separately for each indicator of SES and additionally, by each measure of discrimination exposure (everyday and lifetime discrimination). A summary of the major findings is provided at the end of this chapter.

PRELIMINARY ANALYSES

In Table A1, a correlation matrix of all variables is presented (Appendix). Briefly, log-CRP levels at baseline and follow-up were significantly associated with all key variables of interest. Race and gender were significantly and positively associated with CRP levels at baseline and at follow-up, suggesting that Blacks and women had higher CRP levels than their higher status counterparts. There was a significant negative association between all three indicators of SES (education, income, and wealth) with CRP at baseline and follow-up. Of the three measures of SES, education was the most strongly correlated with both CRP at baseline and follow-up, followed by wealth. In addition, there was a positive association between everyday discrimination and CRP levels at baseline and at follow-up, as well as a positive association between lifetime discrimination and CRP levels at baseline and follow-up.

SAMPLE CHARACTERISTICS

Descriptive statistics for the sample (n = 5,486), as well as stratified first by race, and then by race and gender are presented in Table 2. Raw values (non-transformed) are presented for CRP, income, and wealth, to ease interpretation. As shown, the total sample was 7.23% Black, and slightly over half women (53.38%), with an average age of 64.93 (*SD* = 9.00), and about 67% of the sample was married. The average CRP at baseline was 2.37 mg/L (*SD* = 2.14). At follow-up, CRP levels were 2.14 mg/L (*SD* = 1.96). The sample had an average of 13.51 years of education (*SD* = 2.49), an income of $82,138 (*SD* = $128,293), and a wealth of $607,528 (*SD* = $1,080,413).

Table 2. Weighted means (SD) and percentages (%) by race and gender within race, Health and Retirement Study (n=5,486).

		White (n = 4,904)		Black (n = 582)		
	Total	**Men (n = 2,088)**	**Women (n = 2,816)**	**Men (n = 203)**	**Women (n = 379)**	***p***
CRP (mg/L), baseline	2.37(2.14)	2.08(1.82)	2.52(2.24)*†	2.65(2.67)*	3.42(3.42)*†	+
CRP (mg/L), follow-up	2.14(1.96)	1.94(1.70)	2.24(2.05)*†	2.28(2.37)	2.95(2.95)*†	+
Age	64.93(9.00)	64.27(8.06)	65.75(9.60)*†	63.09(8.69)	63.54(10.23)	+
Black	7.23%	–	–	–	–	
Women	53.38%	–	–	–	–	+
Adult Socioeconomic Status						
Education (years)	13.51(2.49)	13.83(2.40)	13.38(2.36)*†	12.43(3.34)*	12.43(3.33)*	+
Income	$82,138.51($128,293.50)	$95,713.08($141,112.40)	$75,783.54($113,868.70)*†	$54,443.40($50,525.37)*	$35,367.64($60,862.30)*†	+
Wealth	$607,528.10($1,080,413)	$678,100.90($1,058,060)	$608,678.40($1,110,867)*†	$199,986.20($500,023.10)*	$154,988.10($435,546.10)*	+
Everyday Discrimination	0.67(0.74)	0.75(0.71)	0.58(0.70)*†	0.93(0.97)*	0.76(1.07)	+
Major Discrimination	0.53(0.91)	0.62(0.90)	0.40(0.78)*†	0.95(1.47)*	0.83(1.47)*	+
Married	66.96%	78.54%	60.47%*†	56.77%*	30.58%*†	+
Health Status						
Overweight/Obese	71.36%	77.46%	64.88%*†	72.07%	82.77%*†	+
CES-D Score	1.14(1.76)	0.95(1.46)	1.24(1.88)*†	1.30(1.95)	1.91(2.83)*†	+
Health Conditions	1.68(1.25)	1.61(1.16)	1.69(1.27)	1.83(1.55)	2.12(1.62)*†	+
Lifestyle Characteristics						
Moderate/Vigorous Activity						
Never	11.32%	8.13%	12.77%*†	13.82%	25.26%*†	+
Sometimes	22.36%	21.66%	22.12%	27.31%	28.80%*	+
Frequent	66.32%	70.20%	65.11%*†	58.86%*	45.94%*†	+
Smoking						
Never Smoked	45.26%	36.62%	53.92%*†	26.13%*	47.09%†	+
Former Smoker	42.61%	51.37%	35.24%*†	51.58%	31.65%*†	
Current Smoker	12.13%	12.01%	10.84%	22.29%*	21.26%*	+
Alcohol Use						
Never	58.06%	48.14%	64.57%*†	65.74%*	79.26%*†	+
Moderate	32.59%	36.31%	31.23%*†	21.84%*	17.58%*	+
Heavy	9.36%	15.55%	4.21%*†	12.41%	3.16%*†	

Note. Weighted descriptives using pooled 2006|2008 and 2010|2012 data from the Health and Retirement Study.
+ Sig diff $p < .05$ between race groups
* Sig diff $p < .05$ from White men
† Sig diff $p < .05$ between genders within race

As shown in Table 2, results from descriptive analyses demonstrated that the sample significantly differed by race on all variables. Black respondents had significantly higher CRP levels, both at baseline and follow-up, compared to White respondents. The average CRP at baseline for Black respondents was 3.12 mg/L (*SD* = 3.16), compared to an average of 2.31 mg/L (*SD* = 2.14) for White respondents. While CRP levels were lower at follow-up for both Black and White respondents, Black respondents continued to have significantly higher values than White respondents, 2.69 mg/L (*SD* = 2.75) compared to 2.10 mg/L (*SD* = 1.90), respectively. Baseline values of CRP for Blacks fell in a higher risk category for future CVD, 3.12 mg/L, relative to Whites' 2.31 mg/L.

Blacks also had significantly lower SES than Whites—they had significantly fewer years of education, less income, and less wealth. The most marked differences were found for education and wealth: Blacks reported an average of 12.43 (*SD* = 3.35) years of education compared to 13.59 (*SD* = 2.40) for Whites; and Blacks' average wealth was $172,827 (*SD* = $468,496) compared to Whites' $641,419 (*SD* = $1,089,017). Nearly 69% of Whites were married, while almost 41% of Blacks reported being married. Blacks also reported significantly more frequent exposure to both everyday and lifetime discrimination. Blacks' average everyday discrimination score was 0.83 (*SD* = 1.03), while Whites' respective score was 0.66 (*SD* = 0.72). There was more exposure to lifetime discrimination for Blacks than Whites: 0.88 (*SD* = 1.48) compared to 0.50 (*SD* = 0.86), respectively. Black respondents also experienced more depressive symptomatology, had more chronic conditions, and were more likely to be classified as overweight or obese. Regarding lifestyle characteristics, there were also marked differences: 51.06% of Blacks compared to 67.51% of Whites reported frequently engaging in moderate or vigorous physical activity; about 11.40% of Whites versus 21.67% of Blacks reported being current smokers, while 45.76% of Whites and 38.78% of Blacks reported being never smokers;

and 9.55% of Whites and 6.83% of Blacks reported being heavy drinkers, and 33.62% of Whites and 19.27% of Blacks reported being moderate drinkers.

Within race groups, there were significant differences by gender for almost all variables. Within Blacks, women had significantly higher CRP at both baseline and follow-up compared to men. At baseline, Black women had a CRP of 3.42 mg/L (*SD* = 3.42), in contrast to Black men, whose CRP was 2.65 mg/L (*SD* = 2.67). At follow-up, both Black women and men had lower CRP levels than at baseline, but Black women continued to have higher levels than men, 2.95 mg/L (*SD* = 2.95) versus 2.28 mg/L (*SD* = 2.37), respectively. There were no gender differences in years of education for Blacks, but Black women had significantly less income and less wealth than Black men. There were no statistically significant differences in exposure to either everyday or lifetime discrimination between Black men and women. Black women were less likely to be married (30.58%), had greater depressive symptomatology, and had more health conditions than Black men. There were also significant differences for all three lifestyle characteristics between Black women and men: more Black women (25.26%) reported never engaging in moderate or vigorous physical activity, and fewer Black women (45.94%) reported engaging in frequent moderate or vigorous physical activity than Black men (58.86%). Black women (47.09%) were more likely to report never smoking compared to Black men (26.13%) and less frequently were former smokers than Black men (31.65% compared to 51.58%, respectively). Significantly more Black men than women (12.41% versus 3.16%) were heavy drinkers; and more Black women than men reported being never drinkers (79.26% versus 65.74%).

White women had significantly higher CRP levels at baseline and follow-up compared to White men, 2.52 mg/L (*SD* = 2.24) versus 2.08 mg/L (*SD* = 1.82) at baseline, and 2.24 mg/L (*SD* = 2.05) versus 2.10 mg/L (*SD* = 1.90) at follow-up, respectively. White women were slightly older than White men, 65.75 years compared to 64.27 years and less likely to be married than White men: 60.47% compared to 78.54%, respectively. As expected, White men fared better than

White women on SES measures: White men had more years of education and more income than White men, but there were no statistically significant differences in wealth between White women and men. White men reported more frequent everyday discrimination compared to White women, 0.75 (*SD* = 0.71) versus 0.58 (*SD* = 0.70) and more lifetime discrimination, 0.62 (*SD* = 0.90) versus 0.40 (*SD* = 0.78), respectively. A larger proportion of White men than White women were categorized as overweight or obese, and White women had significantly lower depressive symptoms than White men. There were also significant gender differences for lifestyle characteristics among Whites: for instance, a larger proportion of White women reported never engaging in moderate or vigorous physical activity, a smaller proportion of White women reported engaging in frequent moderate or vigorous physical activity (65.11% compared to 70.20% of White men); and a larger proportion of White women reported being never smokers (53.92% compared to 36.62% of White men).

There were notable differences between Black women and Black men compared with White men, as well. Black women had significantly higher levels of CRP at both baseline and follow-up compared to White men: at baseline, Black women's CRP was 3.42 mg/L and White men's was 2.08 mg/L; at follow-up, Black women had an average CRP of 2.95 mg/L compared to White men's average of 1.94 mg/L. Black men had significantly higher CRP at baseline than White men, but there were no significant differences between Black men and White men at follow-up. Black women and men had significantly lower SES than White men on all measures. Black women had significant differences from White men in health status, but Black men did not. To illustrate, a greater proportion of Black women were overweight or obese (82.77%), and Black women had more depressive symptoms and chronic health conditions compared to White men.

RESULTS: AIM 1

This section describes the results from Aim 1, which sought to investigate how social statuses (e.g., race, gender, and SES) are associated with low-grade C-reactive protein (CRP) at baseline and over a four-year follow-up in a nationally-representative sample of midlife and older adults (RQ #1). The analyses run predicting CRP at baseline are presented first, broken down by each measure of SES; then, analyses predicting CRP at follow-up are presented, similarly broken down by each measure of SES.

Baseline CRP

Independent Effects

The results from OLS models regressing log-CRP at baseline on the independent effects of race, gender, and SES are presented in Table 3. In model 1 of the OLS models, which regressed log-CRP (hereafter referred to as CRP) at baseline on race, gender, education, income, wealth, age, and marital status revealed that nearly all of the proposed social status variables were significantly associated with CRP at baseline (Table 3). Being Black (b = 0.07, SE = 0.02, $p \leq$ .001) and being a woman (b = 0.08, SE = 0.01, $p \leq .001$) were both positively associated with CRP. Antilog transformations of these coefficients suggest that those who were Black had 17% higher baseline CRP than Whites, and that women had approximately 20% higher baseline CRP than men. Compared to Whites, Blacks had Conversely, both education (b = -0.02, SE = 0.003, $p \leq .001$) and wealth (b = -0.01, SE = 0.004, $p \leq .01$) were negatively associated with baseline CRP. Thus, those who had more years of education and more wealth had lower levels of CRP at baseline. A one percent increase in wealth was associated with a 0.01% reduction in baseline

CRP; and one additional year of education was associated with a 0.02% reduction in baseline CRP. However, income was not significantly associated with CRP at baseline.

Table 3. Linear regression models testing independent relationship between social status (race, gender, and SES) and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.62***	0.11	0.53***	0.11	0.11	0.11
Race						
Black	0.07***	0.02	0.06*	0.02	0.05*	0.02
Gender						
Woman	0.08***	0.01	0.08***	0.01	0.11***	0.01
SES						
Education	-0.02***	0.003	-0.02***	0.003	-0.01***	0.003
Log-income	-0.02	0.02	-0.005	0.02	-0.0001	0.02
Log-wealth	-0.01**	0.004	-0.01*	0.004	-0.01	0.004
Age	-0.001	0.001	-0.001	0.001	0.001	0.001
Married	-0.01	0.02	0.01	0.02	0.004	0.02
Lifestyle Characteristics						
Smoking						
Former			0.04**	0.01	0.02	0.01
Current			0.09***	0.02	0.12***	0.02
Drinking						
Moderate			-0.05**	0.02	-0.03†	0.02
Heavy			0.005	0.02	0.02	0.02
Moderate/Vigorous Physical Activity						
Sometimes			-0.04†	0.02	-0.03	0.02
Frequent			-0.11***	0.02	-0.07***	0.02
Health Conditions						
Overweight/Obese					0.25***	0.01
CESD					0.003	0.004
Chronic conditions					0.01	0.01
R^2	0.037		0.0528		0.1210	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.

Key. † p< .10, *p < .05, **p < .01, ***p < .001

In Model 2, which adjusted for lifestyle characteristics, the effects of the social status variables on CRP remained unchanged, with one exception: the association between race and CRP was attenuated in both strength and significance, although it continued to be a significantly and positively associated with baseline CRP ($b = 0.06$, $SE = 0.02$, $p \leq .05$). The fully adjusted Model 3 showed that inclusion of health conditions reduced the effects of both race and education, although each remained significantly associated with CRP at baseline. The effect of wealth was reduced to non-significance upon inclusion of health conditions.

Interactive Effects of Race, Gender, and SES on Baseline CRP

The results for the effects of race x gender x SES on CRP at baseline are presented below. A series of four models was run with interaction terms separately for each measure of SES: education, income, and wealth. The tables for each set of analyses were set up in the same manner: Model 1 tested a two-way Black x woman interaction, controlling for age and marital status. Model 2 introduced a three-way Black x woman x [measure of SES] interaction to Model 1. Model 3 added lifestyle characteristics (smoking, drinking, and moderate/vigorous physical activity). Lastly, Model 4 added health conditions.

Race, Gender, and Education. In Model 1 of the OLS regression models, which regressed CRP at baseline on two-way interactions between race and gender, controlling for income, wealth, age, and marital status, the Black x woman interaction was not statistically significant. This suggests that the relationship between race and CRP was not conditional on gender. This model revealed a significant effect of being a woman ($b = 0.08$, $SE = 0.01$, $p \leq .001$), and that both education ($b = -0.02$, $SE = 0.003$, $p \leq .001$) and wealth ($b = -0.01$, $SE = 0.004$, $p \leq .01$) were negatively associated with CRP. Thus, being a woman was associated with a nearly

20% increase in baseline CRP, one additional year of education was associated with 4.71% lower baseline CRP, and a 10% increase in wealth was associated with a 0.01% decrease in CRP. By way of example, a person with a CRP value of 3.00 mg/L would have approximately 0.2823 mg/L lower CRP with two additional years of education, but with a 10% increase in their wealth, they would have 0.003 mg/L lower CRP. Next, *contrast* and *pwcompare* commands were included to conduct pairwise comparisons of each of the race-gender groups to one another. The results from this comparison showed that Black women had significantly higher CRP than White men, and that White men had significantly higher CRP than White women, but there were non-significant associations for all other combinations of race-gender.

As shown in Model 2, the Black x woman x education interaction term in Model 2 was not statistically significant. This indicates that the effects of race, gender, and education on CRP were not conditional on one another, and that their effects on CRP were independent rather than interactive. The effect of being a woman was no longer statistically significant, and the effects of education and wealth remained stable.

The addition of lifestyle characteristics did not affect the previously described interactions, as shown in Model 3. The effects of education (b = -0.02, SE = 0.003, $p \leq .001$) and wealth (b = -0.01, SE = 0.004, $p \leq .05$) remained stably and negatively associated with baseline CRP. In addition, these analyses revealed that being a former smoker (b = 0.03, SE = 0.01, $p \leq .05$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were positively associated with baseline CRP. Moderate drinking (b = -0.05, SE = 0.02, $p \leq .01$), and reporting both sometimes (b = -0.04, SE = 0.02, $p \leq .10$) and frequently (b = -0.11, SE = 0.02, $p \leq .001$) engaging in moderate or vigorous physical activity were negatively associated with baseline CRP. These results suggest that lifestyle characteristics, particularly not being a former smoker, not being a current smoker, moderate drinking, and reporting engaging in moderate or vigorous physical activity sometimes or frequently are associated with lower baseline CRP.

In the fully adjusted model, which further adjusted for health characteristics, education was negatively associated with baseline CRP (b = -0.02, SE = 0.003, $p \leq .001$), but wealth was no longer significantly associated with CRP. The effects of being a former smoker, being a moderate drinker, or sometimes engaging in moderate or vigorous physical activity were also reduced to non-significance. However, being a current smoker (b = 0.11, SE = 0.02, $p \leq .001$), frequently engaging in moderate or vigorous physical activity (b = -0.07, SE = 0.02, $p \leq .001$), and being overweight or obese (b = 0.26, SE = 0.01, $p \leq .001$) were significantly associated with CRP. These results suggest a strong education-CRP relationship, that persists net of potential confounding, and indicate that there may be other mechanisms that link education to CRP.

Table 4. Linear regression models testing relationship between race x gender x education and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.62***	0.11	0.64***	0.12	0.55***	0.13	0.18	0.12
Race								
Black	0.05	0.04	0.01	0.11	0.01	0.11	-0.003	0.11
Gender								
Woman	0.08***	0.01	0.09	0.07	0.08	0.07	0.01	0.06
SES								
Education	-0.02***	0.003	-0.02***	0.004	-0.02***	0.004	-0.02***	0.003
Log-income	-0.01	0.02	-0.01	0.02	-0.004	0.02	-0.001	0.02
Log-wealth	-0.01**	0.004	-0.01**	0.004	-0.01*	0.004	-0.01	0.004
Interactions								
Black x woman	0.03	0.05	-0.23	0.17	-0.18	0.17	-0.11	0.18
Black x education			0.003	0.01	0.002	0.01	0.01	0.01
Woman x education			-0.001	0.005	0.0004	0.005	0.01	0.004
Black x woman x education			0.02	0.02	0.01	0.01	0.01	0.01
Age	-0.001†	0.001	-0.001	0.001	0.02	0.01	-0.001	0.001
Married	0.001	0.02	0.001	0.02	-0.001	0.001	0.003	0.01
Lifestyle Characteristics								
Smoking								
Former					0.03*	0.01	0.01	0.01
Current					0.09***	0.02	0.11***	0.02
Drinking								
Moderate					-0.05**	0.02	-0.03	0.02
Heavy					0.004	0.02	0.01	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.04†	0.02	-0.03	0.02
Frequent					-0.11***	0.02	-0.07***	0.02
Health Conditions								
Overweight/Obese							0.26***	0.01
CESD							0.003	0.004
Chronic conditions							0.01	0.01
R^2	0.0375		0.0383		0.0534		0.1219	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Race, Gender, and Income. Table 5 presents the linear regression models which tested whether there were significant two- and/or three-way interactions among race, gender, and income on CRP at baseline. In Model 2, as shown in Figure 5, a Black x woman x income interaction term was statistically significant and positive (b = 0.19, SE = 0.07, $p \leq .01$). There was also a significant two-way negative Black x woman interaction (b = -0.80, SE = 0.31, $p \leq .01$) and a significant negative Black x income interaction (b = -0.10, SE = 0.05, $p \leq .05$). Model 2 also showed a significant effect of being Black (b = 0.51, SE = 0.23, $p \leq .05$), education (b = -0.02, SE = 0.003, $p \leq .001$), and wealth (b = -0.01, SE = 0.004, $p \leq .01$). These findings suggested that the relationship between income and baseline CRP was inverse for all race and gender groups, with the exception of Black women, who had higher CRP levels with increasing income (Figure 5). Further, at lower levels of income, Black men had higher CRP than any race and gender group, including Black women with higher income.

Model 3 shows that upon inclusion of lifestyle characteristics, the Black x woman x income interaction was attenuated, but remained significantly associated with CRP at baseline (b = 0.17, SE = 0.07, $p \leq .01$). The two-way Black x woman interaction was also attenuated (b = -0.73, SE = 0.30, $p \leq .01$), but remained statistically significant. The two-way Black x income interaction remained stable in Model 3. As hypothesized, nearly all lifestyle characteristics were significantly associated with CRP levels at baseline: being a former smoker (b = 0.04, SE = 0.01, $p \leq .01$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were positively associated with CRP. Moderate drinking (b = -0.05, SE = 0.02, $p \leq .01$), as well as reporting both sometimes (b = -0.04, SE = 0.02, $p \leq .10$) and frequently (b = -0.11, SE = 0.02, $p \leq .001$) engaging in moderate or vigorous physical activity were negatively associated with CRP. Of note, race (b = 0.48, SE = 0.22, $p \leq .05$), education (b = -0.02, SE = 0.003, $p \leq .001$), and wealth (b = -0.01, SE = 0.004, $p \leq .05$) were also significantly associated with CRP. This suggests that the interactive

relationships among race, gender, and income on CRP were not largely explained by lifestyle characteristics.

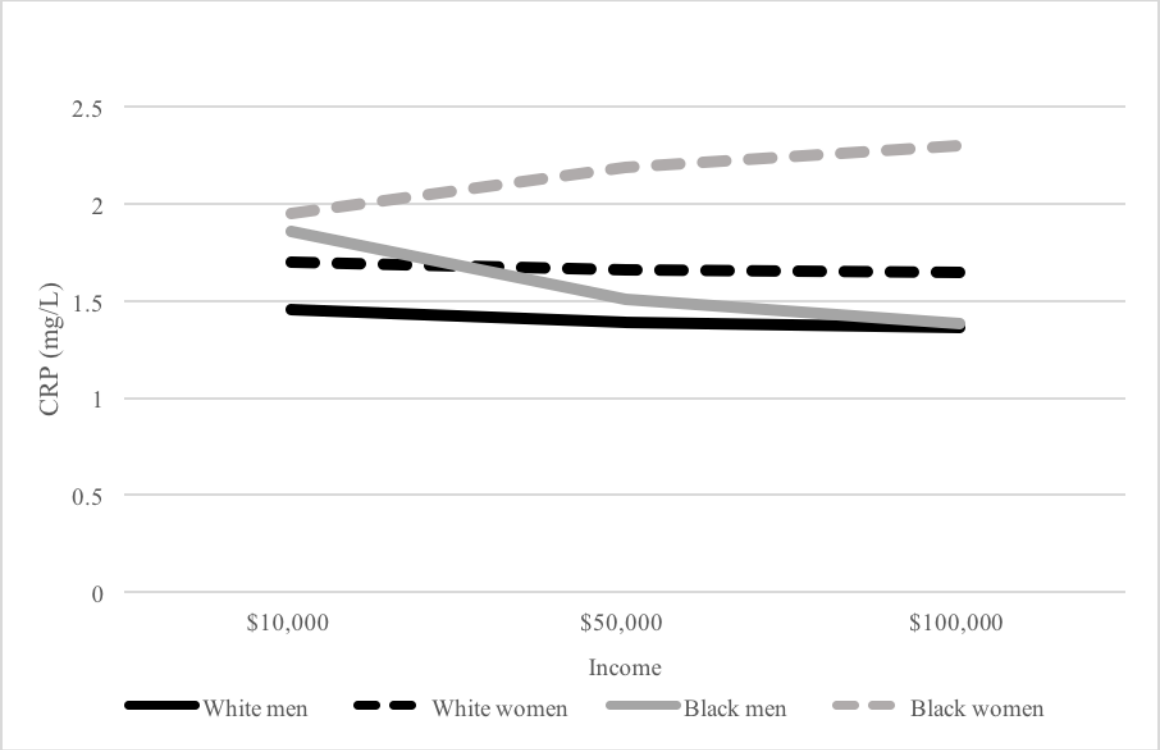
When health conditions were added in Model 4 of Table 5, the interaction terms were further attenuated, but remained significantly associated with CRP: the three-way Black x woman x income interaction remained positively associated with CRP ($b = 0.16$, $SE = 0.08$, $p \leq .05$), and the Black x woman interaction was negatively associated with CRP ($b = -0.71$, $SE = 0.35$, $p \leq .05$). The Black x income two-way interaction was no longer significantly associated with CRP. This suggests that health conditions and lifestyle characteristics only play a partial role in accounting for the interactions among race, gender, and income. Being Black was marginally associated with CRP ($b = 0.51$, $SE = 0.28$, $p \leq .10$), the effect of education was halved ($b = -0.01$, $SE = 0.003$, $p \leq .001$), and the effect of wealth was reduced to non-significance. The inclusion of health conditions also reduced the effects of being a former smoker, being a moderate drinker, and sometimes engaging in moderate or vigorous physical activity to non-significance. The effects of being a current smoker ($b = 0.12$, $SE = 0.02$, $p \leq .001$) and engaging in frequent moderate or vigorous physical activity ($b = -0.07$, $SE = 0.02$, $p \leq .001$) were slightly changed, but remained statistically significant. Finally, being overweight or obese was positively associated with CRP ($b = 0.26$, $SE = 0.01$, $p \leq .001$), but neither CESD nor chronic conditions were significantly associated with CRP. This suggests that the effect of lifestyle characteristics on CRP are in part due to their influence on health conditions (Table 5).

Table 5. Linear regression models testing relationship between race x gender x income and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.62***	0.11	0.69***	0.13	0.60***	0.14	0.27†	0.14
Race								
Black	0.05	0.04	0.51*	0.23	0.48*	0.22	0.51†	0.28
Gender								
Woman	0.08***	0.01	0.01	0.14	0.01	0.14	-0.15	0.14
SES								
Education	-0.02***	0.003	-0.02***	0.003	-0.02***	0.003	-0.01***	0.003
Log-income	-0.01	0.02	-0.03	0.03	-0.02	0.02	-0.03	0.03
Log-wealth	-0.01**	0.004	-0.01**	0.004	-0.01*	0.004	-0.01	0.004
Interactions								
Black x woman	0.03	0.05	-0.80**	0.31	-0.73**	0.30	-0.71*	0.35
Black x log-income			-0.10*	0.05	-0.10*	0.05	-0.10	0.06
Woman x log-income			0.01	0.03	0.02	0.03	0.06†	0.03
Black x woman x log-income			0.19**	0.07	0.17**	0.07	0.16*	0.08
Age	-0.001†	0.001	-0.001	0.001	-0.001	0.001	0.001	0.001
Married	0.001	0.02	0.001	0.02	0.01	0.02	0.002	0.02
Lifestyle Characteristics								
Smoking								
Former					0.04**	0.01	0.02	0.01
Current					0.09***	0.02	0.12***	0.02
Drinking								
Moderate					-0.05**	0.02	-0.03	0.01
Heavy					0.004	0.02	0.02	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.04†	0.02	-0.03	0.02
Frequent					-0.11***	0.02	-0.07***	0.02
Health Conditions								
Overweight/Obese							0.26***	0.01
CESD							0.004	0.004
Chronic conditions							0.01	0.01
R^2	0.0375		0.0392		0.0543		0.1237	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, **p* < .05, ***p* < .01, ****p* < .001

Figure 5. Association between Black x woman x income and C-reactive protein at baseline, controlling for sociodemographic characteristics.



Race, Gender, and Wealth. Table 6 presents the linear regression models which tested whether there were significant two- and/or three-way interactions among race, gender, and wealth on CRP at baseline. As shown in Model 2, there was a significant positive Black x woman x wealth interaction term (b = 0.03, SE = 0.01, $p \leq .05$). As depicted in Figure 6, the relationship between wealth and CRP was negative for all race and gender groups, except for in Black women, who had higher baseline CRP with greater wealth. At lower levels of wealth, there appeared to be similar CRP levels across all groups except for Black men who had nearly twice the log-CRP of other race and gender groups, particularly at the lowest levels of wealth. The effect of being Black was marginally significant (b = 0.10, SE = 0.05, $p \leq .10$), and there was a significant negative relationship between wealth and CRP (b = -0.02, SE = 0.01, $p \leq .05$). Of note, education was also significantly and negatively associated with CRP (b = -0.02, SE = 0.003, $p \leq .001$), which corresponds to a 4.70% increase in CRP for one fewer year of education.

Figure 6. **Association between Black x woman x wealth and C-reactive protein at baseline, controlling for sociodemographic characteristics**.

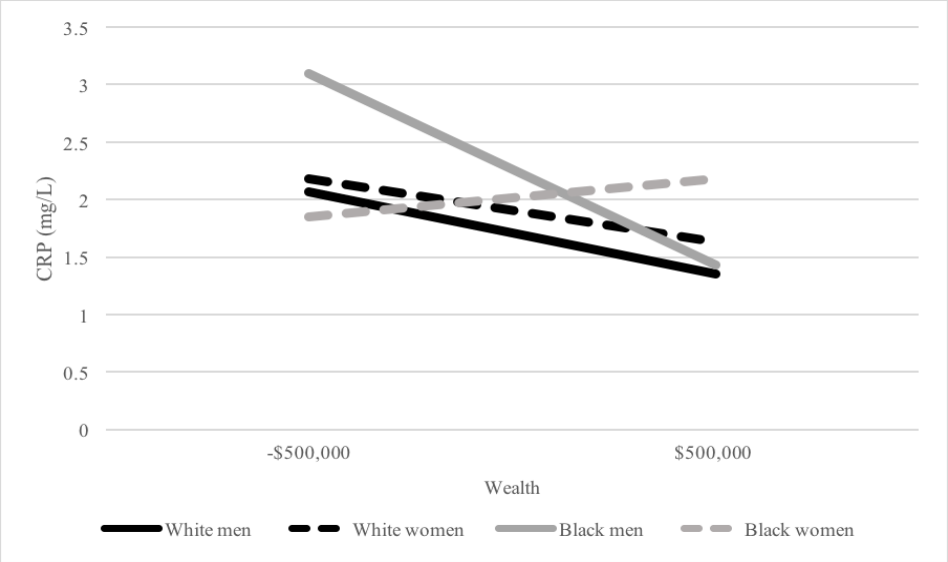


Table 6. Linear regression models testing relationship between race x gender x wealth and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*B*	*SE*	*b*	*SE*
Constant	0.62***	0.11	0.64***	0.11	0.56***	0.11	0.15	0.11
Race								
Black	0.05	0.04	0.10†	0.05	0.11*	0.05	0.11*	0.05
Gender								
Woman	0.08***	0.01	0.05	0.04	0.06	0.04	0.05	0.04
SES								
Education	-0.02***	0.003	-0.02***	0.003	-0.02***	0.003	-0.01***	0.003
Log-income	-0.01	0.02	-0.01	0.02	-0.004	0.02	0.0005	0.01
Log-wealth	-0.01**	0.004	-0.02*	0.01	-0.01†	0.01	-0.01*	0.01
Interactions								
Black x woman	0.03	0.05	-0.07	0.08	-0.09	0.08	-0.11	0.08
Black x log-wealth			-0.01	0.01	-0.02†	0.01	-0.01	0.01
Woman x log-wealth			0.01	0.01	0.01	0.01	0.01†	0.01
Black x woman x log-wealth			0.03*	0.01	0.03*	0.01	0.03†	0.01
Age	-0.001†	0.001	-0.001	0.001	-0.001	0.001	0.001	0.001
Married	0.001	0.02	0.0003	0.02	0.01	0.02	0.003	0.02
Lifestyle Characteristics								
Smoking								
Former					0.04**	0.01	0.02	0.01
Current					0.09***	0.02	0.12***	0.02
Drinking								
Moderate					-0.05**	0.02	-0.03	0.02
Heavy					0.004	0.02	0.01	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.04†	0.02	-0.03	0.02
Frequent					-0.11***	0.02	-0.07***	0.02
Health Conditions								
Overweight/Obese							0.25***	0.01
CESD							0.004	0.004
Chronic conditions							0.01	0.01
R^2	0.0375		0.0387		0.0542		0.1230	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

As shown in Model 3, after adjustment for lifestyle characteristics, the Black x woman x wealth interaction term remained significantly and positively associated with baseline CRP (b = 0.03, SE = 0.01, $p \leq .05$). In addition, there was a marginally significant two-way Black x wealth interaction (b = -0.02, SE = 0.01, $p \leq .10$). This suggests that lifestyle characteristics do not account for the three-way interaction among race, gender, and wealth on baseline levels of CRP. Being Black (b = 0.11, SE = 0.05, $p \leq .05$), and having less education (b = -0.02, SE = 0.003, $p \leq .001$) and less wealth (b = -0.01, SE = 0.01, $p \leq .10$) were associated with higher CRP at baseline. This suggests that lower ascribed and achieved social status are associated with higher baseline CRP. Of the lifestyle characteristics, being a former smoker (b = 0.04, SE = 0.01, $p \leq .01$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were associated with higher CRP; while moderate drinking (b = -0.05, SE = 0.02, $p \leq .01$), and reporting sometimes (b = -0.04, SE = 0.02, $p \leq .10$) or frequently (b = -0.11, SE = 0.02, $p \leq .001$) engaging in moderate or vigorous physical activity were associated with lower CRP. This suggests that not smoking, moderate drinking, and engaging in moderate or vigorous physical activity at least sometimes are associated with lower baseline CRP.

The fully adjusted model, which adjusted for health conditions in conjunction with sociodemographic and lifestyle characteristics, showed that the Black x woman x wealth interaction term was reduced to marginal significance, but the strength of the association remained the same (b = 0.03, SE = 0.01, $p \leq .10$). This suggests that health conditions may explain part of the effect that the Black x woman x wealth interaction had on CRP. There was no longer a significant Black x wealth interaction, but a marginally significant woman x wealth interaction emerged in this model (b = 0.01, SE = 0.01, $p \leq .10$). The main effect of wealth remained significantly and inversely associated with CRP (b = -0.01, SE = 0.003, $p \leq .001$), and the effect of being Black remained significantly associated with higher CRP (b = 0.11, SE = 0.05, $p \leq .05$). This suggests that lower social status is associated with higher CRP. The addition of health conditions reduced the effects of former smoking, moderate drinking, and sometimes engaging in moderate or vigorous physical activity to

non-significance. There was a positive association between being a current smoker (b = 0.12, SE = 0.02, $p \leq .001$) and being overweight or obese (b = 0.25, SE = 0.01, $p \leq .001$) with CRP. Frequently engaging in moderate or vigorous physical activity was negatively associated with CRP (b = -0.07, SE = 0.02, $p \leq .001$). This suggests that the effects of lifestyle characteristics on baseline CRP work in part due to their influence on health conditions.

CRP at Four-Year Follow-Up

Independent Effects

In Model 1 of the lagged OLS models, which regressed log-CRP at follow-up on the independent effects of race, gender, measures of SES, age, marital status, and CRP at baseline (Table 7), only wealth (b = -0.01, SE = 0.002, $p \leq .01$) and baseline CRP (b = 0.57, SE = 0.01, $p \leq .001$) were negatively associated with follow-up CRP. In particular, this suggests that each dollar increase in wealth was associated with a 0.01% decrease in CRP levels at follow-up.

In Model 2, which adjusted for lifestyle characteristics, the effects of the social status variables remained unchanged: there was a significant negative relationship between wealth and CRP at follow-up (b = -0.01, SE = 0.003, $p \leq .01$). Being a former (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.08, SE = 0.02, $p \leq .001$) were positively associated with CRP at follow-up, while being a moderate drinker (b = -0.01, SE = 0.002, $p \leq .01$) was negatively associated with CRP at follow-up. This suggests that more wealth is associated with lower CRP at follow-up, but that the effect of wealth on CRP does not work through lifestyle characteristics. This also suggests that being a former smoker and being a current smoker are associated with higher follow-up CRP, net of baseline CRP levels.

The fully adjusted model showed that inclusion of health conditions did not affect any of the previously described relationships between measures of social status and CRP at follow-up. This suggests that neither lifestyle characteristics nor health conditions account for the negative relationship between wealth and follow-up CRP. In this model, several lifestyle characteristics were significantly associated with CRP at follow-up. Being a former smoker (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were positively associated

with CRP at follow-up, as was being overweight or obese (b = 0.06, SE = 0.01, $p \leq .001$) and having more chronic conditions (b = 0.01, SE = 0.01, $p \leq .10$). This suggests that being a former smoker or being a current smoker, being overweight or obese, and having more chronic conditions are associated with having higher follow-up CRP.

Table 7. Lagged effect linear regression models testing independent relationship between social status (race, gender, and SES) and C-reactive protein over four years (n = 5,486).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.25*	0.11	0.17	0.11	0.10	0.11
Log-CRP at baseline	0.57***	0.01	0.56***	0.01	0.54***	0.01
Race						
Black	-0.01	0.02	-0.01	0.02	-0.01	0.02
Gender						
Woman	0.01	0.01	0.01	0.01	0.02	0.01
SES						
Education	-0.004	0.003	-0.002	0.003	-0.002	0.003
Log-income	-0.02	0.02	-0.02	0.02	-0.02	0.02
Log-wealth	-0.01**	0.002	-0.01*	0.003	-0.01*	0.003
Age	0.0001	0.001	0.0002	0.001	0.0003	0.001
Married	-0.02	0.01	-0.01	0.01	-0.01	0.01
Lifestyle Characteristics						
Smoking						
Former			0.04***	0.01	0.04***	0.01
Current			0.08***	0.02	0.09***	0.02
Drinking						
Moderate			-0.02†	0.01	-0.02	0.01
Heavy			-0.03	0.02	-0.02	0.02
Moderate/Vigorous Physical Activity						
Sometimes			-0.003	0.02	-0.004	0.02
Frequent			-0.01	0.01	-0.01	0.01
Health Conditions						
Overweight/Obese					0.06***	0.01
CESD					-0.003	0.004
Chronic conditions					0.01†	0.01
R^2	0.3674		0.3720		0.3765	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.

Key. † p< .10, *p < .05, **p < .01, ***p < .001

Interactive Effects of Race, Gender, and SES on CRP at Four-Year Follow-Up

Below, the results for the effects of race x gender x SES on log-CRP at follow-up are presented. A series of four models were run with interaction terms for each measure of SES: education, income, and wealth. The tables for each set of analyses were set up in the same manner: Model 1 tested a two-way Black x woman interaction, controlling for age, marital status, and CRP at baseline. Model 2 introduced a three-way Black x woman x [measure of SES] interaction to Model 1. Model 3 included lifestyle characteristics (smoking, drinking, and moderate/vigorous physical activity). Lastly, Model 4 further adjusted for health conditions.

Race, Gender, and Education. Table 8 presents the lagged linear regression models which tested whether there were significant two- and/or three-way interactions among race, gender, and education on CRP at follow-up. The first model, which tested a Black x woman interaction was not significantly associated with CRP at follow-up. This suggests that the effect of race on CRP over four years was not conditional on gender. Wealth was the only social status variable that was significantly associated with CRP at follow-up (b = -0.01, SE = 0.002, $p \leq .01$). The findings suggest that a one-dollar increase in wealth is associated with 0.01% lower CRP at follow-up. As shown in Model 2, which included a three-way Black x woman x education interaction term, there was a marginally significant and positive Black x woman x education interaction (b = 0.03, SE = 0.01, $p \leq .10$). Figure 7 provides a graphical representation of the Black x woman x education interaction. It shows that incremental increases in education were protective against higher CRP at follow-up for all race and gender groups except for Black women, who had higher CRP at follow-up with more years of education. Of all groups, Black women had the lowest follow-up CRP at lower levels of education.

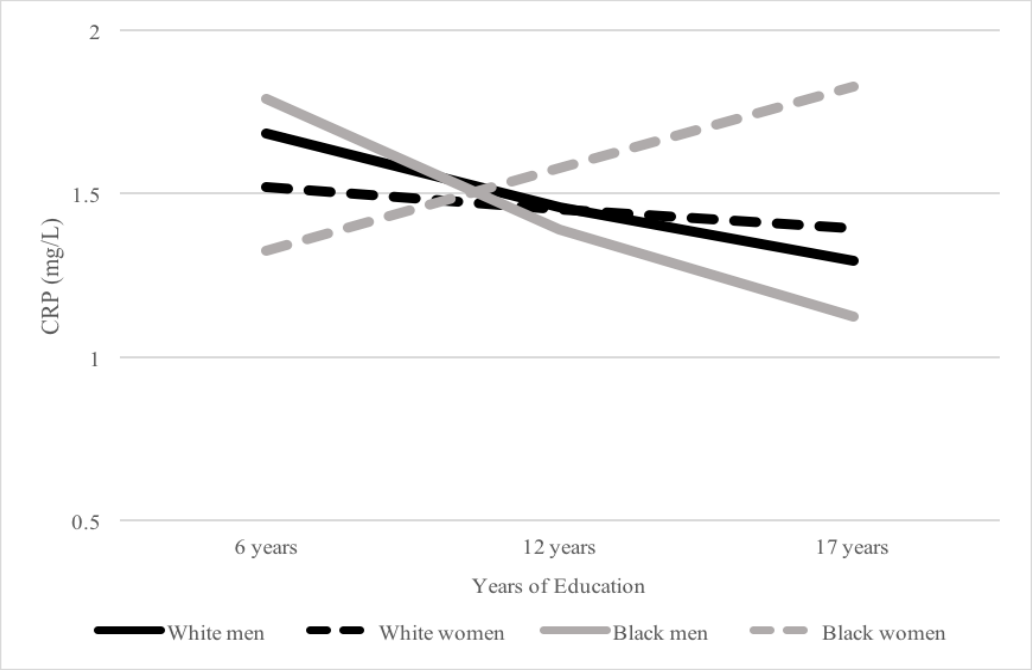
Table 8. Lagged effect linear regression models testing relationship between race x gender x education and C-reactive protein over four years (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.25*	0.11	0.31**	0.11	0.23*	0.11	0.17	0.11
Log-CRP at baseline	0.57***	0.01	0.57***	0.01	0.56***	0.01	0.54***	0.01
Race								
Black	-0.03	0.03	0.06	0.13	0.06	0.13	0.06	0.13
Gender								
Woman	0.004	0.01	-0.11	0.07	-0.09	0.07	-0.11	0.07
SES								
Education	-0.004	0.003	-0.01*	0.003	-0.01†	0.003	-0.01†	0.003
Log-income	-0.02	0.02	-0.02	0.02	-0.02	0.02	-0.02	0.02
Log-wealth	-0.01**	0.002	-0.01***	0.002	-0.01*	0.003	-0.01*	0.003
Interactions								
Black x woman	0.05	0.04	-0.26	0.18	-0.25	0.18	-0.23	0.18
Black x education			-0.01	0.01	-0.01	0.01	-0.01	0.01
Woman x education			0.01	0.005	0.01	0.005	0.01†	0.005
Black x woman x education			0.03†	0.01	0.02†	0.01	0.02	0.01
Age	0.0001	0.001	0.0001	0.001	0.0003	0.001	0.0003	0.001
Married	-0.02	0.01	-0.02	0.01	-0.01	0.01	-0.02	0.01
Lifestyle Characteristics								
Smoking								
Former					0.04***	0.01	0.04**	0.01
Current					0.08***	0.02	0.09***	0.02
Drinking								
Moderate					-0.02†	0.01	-0.02	0.01
Heavy					-0.03	0.02	-0.03	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.01	0.02	-0.01	0.02
Frequent					-0.01	0.01	-0.01	0.01
Health Conditions								
Overweight/Obese							0.06***	0.01
CESD							-0.003	0.004
Chronic conditions							0.01†	0.004
R^2	0.3677		0.3692		0.3736		0.3782	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

When lifestyle characteristics were introduced to the analyses, as shown in Model 3, the Black x woman x education interaction was reduced in magnitude, but the effect remained marginally associated with follow-up CRP (b = -0.02, SE = 0.01, $p \leq .10$). The effects of education (b = -0.01, SE = 0.003, $p \leq .10$) and wealth (b = -0.01, SE = 0.003, $p \leq .05$) were attenuated but remained significant. Of the lifestyle characteristics, being a former smoker (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.08, SE = 0.02, $p \leq .001$) were strongly and positively associated with CRP at follow-up.

Figure 7. **Association between Black x woman x education and C-reactive protein at follow-up, controlling for sociodemographic characteristics and CRP at baseline**.



In Model 4, which further adjusted for health conditions, the Black x woman x education interaction was no longer statistically significant. The woman x education interaction emerged as marginally significant (b = 0.01, SE = 0.005, $p \leq .10$). This suggests that the Black x woman x education interaction was partially accounted for by more health conditions. Education remained negatively and marginally associated with CRP at follow-up (b = -0.01, SE = 0.003, $p \leq .10$). Wealth was also remained negatively associated with follow-up CRP (b = -0.01, SE = 0.003, $p \leq .05$). This suggests that the relationship between wealth and CRP at follow-up is independent of sociodemographic, lifestyle characteristics, and health conditions. It also suggests that there are other mechanisms that account for the relationship between wealth and CRP at follow-up.

Being a former smoker (b = 0.04, SE = 0.01, $p \leq .01$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were positively associated with CRP at follow-up, but the effect of moderate drinking was not statistically significant. Being overweight or obese (b = 0.06, SE = 0.01, $p \leq .001$) and having more health conditions (b = 0.01, SE = 0.004, $p \leq .10$) were positively associated with CRP at follow-up. This suggests that being a former smoker, being a current smoker, being overweight or obese, and having more health conditions are associated with higher CRP at follow-up.

Race, Gender, and Income. Table 9 presents the lagged linear regression models which tested whether there were significant two- and/or three-way interactions among race, gender, and income on CRP at follow-up. Controlling for CRP at baseline, age, and marital status, Model 2 shows that there was not a statistically significant Black x woman x income interaction. However, there were significant effects of both income (b = -0.06, SE = 0.02, $p \leq .01$) and wealth (b = -0.01, SE = 0.002, $p \leq .01$) on follow-up CRP. This suggests that the relationships among race, gender, and income on CRP at follow-up are independent of one another, rather than conditional on one

another. It also suggests that having more income and having more wealth are associated with lower follow-up CRP.

In Model 3, which adjusted for lifestyle characteristics, there was a non-significant three-way interaction for Black x woman x income. Further, there was a slightly attenuated effect of income (b = -0.05, SE = 0.02, $p \leq .05$), and a stable effect of wealth (b = -0.01, SE = 0.003, $p \leq .01$). This suggests that the effect of income on follow-up CRP works partly through lifestyle factors, but that the effect of wealth on follow-up CRP does not work through lifestyle factors. Being a former smoker (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.08, SE = 0.02, $p \leq .001$) were positively associated with CRP at follow-up. Moderate drinking (b = -0.02, SE = 0.01, $p \leq .10$) was negatively and marginally associated with CRP at follow-up. This suggests that being a former smoker and being a current smoker are associated with higher follow-up CRP levels, and that moderate drinking is associated with lower follow-up CRP (Table 9).

Table 9. Lagged effect linear regression models testing relationship between race x gender x income and C-reactive protein over four years (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*B*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.25*	0.11	0.43**	0.15	0.34*	0.15	0.29*	0.14
Log-CRP at baseline	0.57***	0.01	0.57***	0.01	0.56***	0.01	0.54***	0.01
Race								
Black	-0.03	0.03	-0.38	0.49	-0.38	0.49	-0.37	0.46
Gender								
Woman	0.004	0.01	-0.23	0.15	-0.22	0.15	-0.25	0.15
SES								
Education	-0.004	0.003	-0.004	0.003	-0.002	0.003	-0.002	0.003
Log-income	-0.02	0.02	-0.06**	0.02	-0.05*	0.02	-0.06**	0.02
Log-wealth	-0.01**	0.002	-0.01**	0.002	-0.01*	0.003	-0.01*	0.003
Interactions								
Black x woman	0.05	0.04	0.14	0.53	0.14	0.53	0.13	0.51
Black x log-income			0.07	0.10	0.07	0.10	0.07	0.10
Woman x log-income			0.05	0.03	0.05	0.03	0.06†	0.03
Black x woman x log-income			-0.01	0.11	-0.01	0.11	-0.01	0.11
Age	0.0001	0.001	0.00004	0.001	0.0002	0.001	0.0003	0.001
Married	-0.02	0.01	-0.02	0.01	-0.01	0.01	-0.02	0.01
Lifestyle Characteristics								
Smoking								
Former					0.04***	0.01	0.04**	0.01
Current					0.08***	0.02	0.09***	0.02
Drinking								
Moderate					-0.02†	0.01	-0.02	0.01
Heavy					-0.03	0.02	-0.02	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.005	0.02	-0.01	0.02
Frequent					-0.01	0.01	-0.01	0.01
Health Conditions								
Overweight/Obese							0.06***	0.01
CESD							-0.003	0.004
Chronic conditions							0.01†	0.004
R^2	0.3677		0.3694		0.3739		0.3786	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Race, Gender, and Wealth. Table 10 presents the lagged linear regression models which tested whether there were significant two- and/or three-way interactions among race, gender, and wealth on follow-up CRP. The model which tested a Black x woman x wealth interaction term, controlling for CRP at baseline, age, and marital status, showed a non-significant relationship. There was a marginal main effect of wealth (b = -0.01, SE = 0.005, $p \leq .10$), as shown in Model 2. This suggests that the relationships among race, gender, and wealth on follow-up CRP are independent of one another, rather than conditional on one another. This also suggests that more wealth is associated with lower follow-up CRP. With the addition of lifestyle characteristics in Model 3, the Black x woman x wealth interaction remained non-significant, and there was no longer a significant effect of wealth on CRP at follow-up. This suggests that lifestyle characteristics might be a pathway linking wealth to follow-up CRP. Being a former smoker (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.08, SE = 0.02, $p \leq .001$) were both significantly and positively associated with CRP at follow-up, and moderate drinking (b = -0.02, SE = 0.01, $p \leq .10$) was marginally and negatively associated with CRP at follow-up.

In the fully adjusted model, as shown in Table 10, there were no statistically significant interactions among race, gender, and wealth on CRP at follow-up, but there was a marginal main effect of wealth (b = -0.01, SE = 0.01, $p \leq .10$). The effects of being a former (b = 0.04, SE = 0.01, $p \leq .001$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were significant, and moderate drinking was no longer associated with CRP at follow-up. Further, being overweight or obese (b = 0.06, SE = 0.01, $p \leq .001$) and having more chronic conditions (b = 0.01, SE = 0.004, $p \leq .10$) were associated with higher CRP at follow-up. This suggests that being a former smoker, being a current smoker, being overweight or obese, and having more chronic health conditions are associated with higher CRP, but moderate drinking is associated with lower CRP after a four-year follow-up.

Table 10. Lagged effect linear regression models testing relationship between race x gender x wealth and C-reactive protein over four years (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*B*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.25*	0.11	0.26*	0.11	0.18	0.11	0.12	0.11
Log-CRP at baseline	0.57***	0.01	0.57***	0.01	0.56***	0.01	0.54***	0.01
Race								
Black	-0.03	0.03	-0.01	0.05	-0.01	0.05	-0.01	0.05
Gender								
Woman	0.004	0.01	-0.01	0.03	-0.01	0.04	-0.01	0.03
SES								
Education	-0.004	0.003	-0.004	0.003	-0.002	0.003	-0.002	0.003
Log-income	-0.02	0.02	-0.02	0.02	-0.02	0.02	-0.02	0.02
Log-wealth	-0.01**	0.002	-0.01†	0.005	-0.01	0.01	-0.01†	0.01
Interactions								
Black x woman	0.05	0.04	0.001	0.06	0.003	0.06	-0.001	0.06
Black x log-wealth			-0.01	0.01	-0.01	0.01	-0.01	0.01
Woman x log-wealth			0.003	0.01	0.003	0.01	0.01	0.01
Black x woman x log-wealth			0.01	0.01	0.02	0.01	0.01	0.01
Age	0.0001	0.001	0.0001	0.001	0.0002	0.001	0.0003	0.001
Married	-0.02	0.01	-0.02	0.01	-0.01	0.01	-0.02	0.01
Lifestyle Characteristics								
Smoking								
Former					0.04***	0.01	0.04***	0.01
Current					0.08***	0.02	0.09***	0.02
Drinking								
Moderate					-0.02†	0.01	-0.02	0.01
Heavy					-0.03	0.02	-0.02	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.004	0.02	-0.005	0.02
Frequent					-0.01	0.01	-0.01	0.01
Health Conditions								
Overweight/Obese							0.06***	0.01
CESD							-0.003	0.004
Chronic conditions							0.01†	0.004
R^2	0.3677		0.3680		0.3726		0.3771	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.

Key. † p< .10, **p* < .05, ***p* < .01, ****p* < .001

SUMMARY OF AIM 1 FINDINGS

The purpose of Aim 1 was to determine the ways that race, gender, and socioeconomic status (SES)—as measured by education, income, and wealth—were linked to low-grade CRP levels at baseline and over a four-year follow-up period in a nationally-representative sample of midlife and older adults. Specifically, it tested whether there were independent or interactive effects of social statuses (e.g., race, gender, and SES) on CRP, using the lifespan developmental perspective on relationships between social status and health and intersectionality as guiding frameworks for conceptualizing how social disadvantage due to overlapping lower social statuses might manifest in low-grade inflammation, as measured by C-reactive protein (CRP).

In summary, the findings from Aim 1 indicated that there were significant independent effects of race, gender, education, and wealth on CRP at baseline. In particular, being Black, being a woman, having less education, and having less wealth were associated with higher CRP at baseline. For the most part, these associations persisted after adjustment for sociodemographic, lifestyle, and health characteristics. In the analyses examining the independent of social status variables on CRP at follow-up, only wealth was significantly and negatively associated with elevated CRP at follow-up. The effect of wealth was not attenuated after adjustment for sociodemographic, lifestyle, or health characteristics.

There were no significant race x gender interactions for CRP levels at baseline or at follow-up. Significant three-way interactions were found for: (1) Black x woman x income on CRP at baseline; (2) Black x woman x wealth on CRP at baseline; and (3) Black x woman x education on CRP at follow-up. The significant three-way interactions showed similar relationships: each race-gender group had lower CRP levels with incremental increases in SES, except for Black women, who had higher CRP levels with higher SES. Additionally, Black men

with lower SES had the highest CRP of all groups at any level of SES. With higher SES, Black men had steep declines in CRP levels, suggesting that increases in SES may have particularly strong and protective effects for Black men. The analyses suggested that income and wealth were more significant for shaping race-gender variations in CRP at baseline, rather than change over time, but that education had more importance in producing race-gender variations in CRP at follow-up. Each of the three significant three-way interactions were attenuated after adjustment for lifestyle and health characteristics, suggesting that these might be pathways by which combinations of race, gender, and SES are linked to CRP levels.

RESULTS: AIM 2

This section describes the results from Aim 2, which sought to further explore whether exposure to everyday and lifetime discrimination explains how social statuses (e.g., race, gender, and SES) are linked to low-grade C-reactive protein (CRP) at baseline and over a four-year follow-up. First, the analyses tested whether race, gender, and SES interacted to produce differential exposure to everyday and lifetime discrimination. Then, analyses tested whether everyday or lifetime discrimination were associated with CRP at baseline or at the four-year follow-up. Because the analyses described above did not indicate evidence of mediation, no further tests for mediation were conducted.

Following the results from Aim 2, a summary of the major findings from this chapter is provided.

Social Variation in Exposure to Everyday Discrimination

The following analyses were run to answer research question (RQ) #2, which sought to determine whether there were social variations in exposure to everyday discrimination. Linear regression models were run to determine whether there were independent or interactive effects of race, gender, and measures of SES on everyday discrimination. The results are presented in Table 11. Model 1 tested the independent effects of race, gender, education, income, and wealth on everyday discrimination. Model 2 introduced a race x gender interaction, to examine whether there were differences in everyday discrimination by race and gender. Models 3a-c tested whether there were differences in everyday discrimination by race, gender, and SES by introducing a three-way race x gender x [measure of SES] interaction to the model.

Model 1 shows that being a woman (b = -0.17, SE = 0.03, $p \leq .001$), having less education (b = -0.02, SE = 0.01, $p \leq .05$), having less wealth (b = -0.03, SE = 0.01, $p \leq .001$), being younger (b = -0.02, SE = 0.001, $p \leq .001$), and not being married (b = -0.09, SE = 0.03, $p \leq .001$) were negatively associated with everyday discrimination. Surprisingly, there was not a significant relationship between being Black and everyday discrimination. These results suggest that being Black is not significantly associated with more frequent everyday discrimination, but that being a woman, having more education, and having more wealth are associated with less frequent exposure to everyday discrimination. Being younger and married were also associated with less frequent exposure to everyday discrimination. Model 2, which introduced a Black x woman interaction to the model, revealed a non-significant interaction and no change in the relationships from Model 1. The Black x woman interaction was not statistically significant, which suggests that there are no differences in exposure to everyday discrimination across race-gender groups.

Education. As shown in Model 3a of Table 11, the Black x woman x education interaction was not significantly associated with everyday discrimination. There was a marginally significant and positive woman x education interaction (b = 0.03, SE = 0.01, $p \leq .10$). There were significant and negative effects of being a woman (b = -0.51, SE = 0.19, $p \leq .01$). Education (b = -0.03, SE = 0.01, $p \leq .01$), and wealth (b = -0.03, SE = 0.01, $p \leq .001$) were both negatively associated with everyday discrimination. This suggests that being a woman, having less education, and having less wealth are associated with less frequent everyday discrimination.

Income. As shown in Model 3b, there was not a statistically significant association between Black x woman x income and everyday discrimination. There were significant and negative effects of education (b = -0.02, SE = 0.01, $p \leq .05$) and wealth (b = -0.03, SE = 0.01, $p \leq .001$) on everyday discrimination. This suggests that having more years of education and having more wealth are associated with less frequent everyday discrimination.

Table 11. Linear regression models testing relationship between social status (race, gender, and SES) and everyday discrimination at baseline (n = 5,486).

	Everyday Discrimination									
	Model 1		Model 2		Model 3a		Model 3b		Model 3c	
					Education		Income		Wealth	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	2.36***	0.17	2.36***	0.17	2.53***	0.20	2.28***	0.23	2.31***	0.17
Race										
Black	0.06	0.05	0.08	0.09	-0.48	0.36	0.10	0.54	-0.06	0.15
Gender										
Woman	-0.17***	0.03	-0.17***	0.03	-0.51**	0.19	0.08	0.27	-0.04	0.08
SES										
Education	-0.02*	0.01	-0.02*	0.01	-0.03**	0.01	-0.02*	0.01	-0.02*	0.01
Log-income	0.01	0.03	0.01	0.03	0.01	0.04	0.03	0.05	0.01	0.03
Log-wealth	-0.03***	0.01	-0.03***	0.01	-0.03***	0.01	-0.03***	0.01	-0.02†	0.01
Interactions										
Black x woman			-0.03	0.10	0.83	0.53	-0.42	0.58	-0.02	0.18
Black x education					0.04	0.03				
Woman x education					0.03†	0.01				
Black x woman x education					-0.07	0.04				
Black x log-income							-0.003	0.12		
Woman x log-income							-0.05	0.06		
Black x woman x log-income							0.09	0.12		
Black x log-wealth									0.04	0.02
Woman x log-wealth									-0.03†	0.01
Black x woman x log-wealth									-0.01	0.03
Age	-0.02***	0.001	-0.02***	0.001	-0.02***	0.001	-0.02***	0.001	-0.02***	0.001
Married	-0.09***	0.03	-0.09***	0.03	-0.09***	0.03	-0.09***	0.03	-0.09***	0.03
R^2	0.0777		0.0777		0.0798		0.0781		0.0797	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.

Key. † p< .10, *p < .05, **p < .01, ***p < .001

Wealth. As shown in Model 3c, there was not a statistically significant association between Black x woman x wealth and everyday discrimination. There was a marginally significant woman x wealth (b = -0.03, SE = 0.01, $p \leq .10$) association on everyday discrimination. Education (b = -0.02, SE = 0.01, $p \leq .05$) and wealth (b = -0.02, SE = 0.01, $p \leq .10$) were negatively associated with everyday discrimination. This suggests that race, gender, and wealth do not interact to shape exposure to everyday discrimination, but that having less education and less wealth are associated with more frequent exposure to everyday discrimination.

Social Variation in Exposure to Lifetime Discrimination

In order to determine whether there were social variations in exposure to lifetime discrimination, linear regression models were run to determine whether there were independent or interactive effects of race, gender, and SES on lifetime discrimination. The results are presented in Table 12. Model 1 tested the independent effects of race, gender, education, income, and wealth on lifetime discrimination. Model 2 introduced a race x gender interaction, to examine whether there were differences in lifetime discrimination by race and gender. Models 3a-c tested whether there were differences in lifetime discrimination by race, gender, and SES by introducing a three-way race x gender x [measure of SES] interaction to the model.

As shown in Model 1 on Table 12, there were significant positive relationships between all social status variables, except for income, and lifetime discrimination. Specifically, being Black (b = 0.29, SE = 0.07, $p \leq .001$), being a man (b = -0.22, SE = 0.03, $p \leq .001$), having more education (b = 0.04, SE = 0.01, $p \leq .001$), and having less wealth (b = -0.04, SE = 0.01, $p \leq .001$) were significantly associated with more lifetime discrimination. These results suggest that being Black and having more years of education were associated with more lifetime discrimination,

while being a woman and having more wealth were associated with less exposure to lifetime discrimination.

Education. As shown in Model 3a, the Black x woman x education interaction was not statistically associated with more lifetime discrimination. This suggests that race, gender, and education do not interact to shape exposure to lifetime discrimination. In this model, more education (b = 0.03, SE = 0.01, $p \leq .001$) and less wealth (b = -0.05, SE = 0.01, $p \leq .001$) were associated with more lifetime discrimination. Age (b = -0.01, SE = 0.002, $p \leq .001$) and marriage (b = -0.10, SE = 0.03, $p \leq .01$) were significantly and negatively associated with lifetime discrimination. This suggests that being younger and being unmarried are associated with more lifetime discrimination.

Income. As shown in Model 3b, the Black x woman x income interaction was not statistically associated with lifetime discrimination. This suggests that race, gender, and income do not interact to shape exposure to lifetime discrimination. In this model, more education (b = 0.04, SE = 0.01, $p \leq .001$) and less wealth (b = -0.04, SE = 0.01, $p \leq .001$) were associated with more lifetime discrimination. Older age (b = -0.02, SE = 0.002, $p \leq .001$) and being married (b = -0.10, SE = 0.03, $p \leq .01$) were significantly associated with less lifetime discrimination.

Wealth. As shown in Model 3c, the Black x woman x wealth interaction term was not statistically associated with lifetime discrimination. This suggests that race, gender, and wealth do not interact to shape exposure to lifetime discrimination. In this model, more education (b = 0.03, SE = 0.01, $p \leq .001$) and less wealth (b = -0.07, SE = 0.01, $p \leq .001$) were associated with more lifetime discrimination. In addition, being a woman was significantly associated with less lifetime discrimination (b = -0.40, SE = 0.10, $p \leq .001$). Age (b = -0.01, SE = 0.002, $p \leq .001$) and marriage (b = -0.10, SE = 0.03, $p \leq .01$) were significantly and negatively associated with less lifetime discrimination. This suggests that being younger and being unmarried are associated with more lifetime discrimination.

Table 12. Linear regression models testing relationship between social status (race, gender, and SES) and lifetime discrimination at baseline (n = 5,486).

	Lifetime Discrimination									
	Model 1		Model 2		Model 3a		Model 3b		Model 3c	
					Education		Income		Wealth	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	1.65***	0.28	1.65***	0.28	1.68***	0.30	1.83***	0.37	1.74***	0.29
Race										
Black	0.29***	0.07	0.27*	0.13	-0.15	0.46	-0.99	0.89	0.06	0.28
Gender										
Woman	-0.22***	0.03	-0.22***	0.03	-0.15	0.14	-0.37	0.33	-0.40***	0.10
SES										
Education	0.04***	0.01	0.04***	0.01	0.03***	0.01	0.04***	0.01	0.03***	0.01
Log-income	-0.05	0.05	-0.05	0.05	-0.05	0.05	-0.09	0.07	-0.05	0.05
Log-wealth	-0.04***	0.01	-0.04***	0.01	-0.05***	0.01	-0.04***	0.01	-0.07***	0.01
Interactions										
Black x woman			0.03	0.14	-0.37	0.56	0.94	0.91	0.25	0.32
Black x education					0.03	0.04				
Woman x education					-0.005	0.01				
Black x woman x education					0.03	0.04				
Black x log-income							0.27	0.20		
Woman x log-income							0.03	0.07		
Black x woman x log-income							-0.19	0.20		
Black x log-wealth									0.05	0.06
Woman x log-wealth									0.04	0.02
Black x woman x log-wealth									-0.04	0.07
Age	-0.01***	0.002	-0.01***	0.002	-0.01***	0.002	-0.02***	0.002	-0.01***	0.002
Married	-0.10**	0.03	-0.10**	0.03	-0.10**	0.03	-0.10**	0.03	-0.10**	0.03
R^2	0.0705		0.0705		0.0722		0.0715		0.0720	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, **p* < .05, ***p* < .01, ****p* < .001

Discrimination Exposure and Baseline CRP

The following set of models test research question (RQ) #3, which aimed to assess whether exposure to everyday and lifetime discrimination were significantly associated with CRP at baseline. Model 1 shows the unadjusted relationship between each indicator of discrimination and log-CRP at baseline; Model 2 adjusted for sociodemographic characteristics; Model 3 further adjusted for lifestyle characteristics; and Model 4, the fully adjusted model, included health conditions.

Everyday discrimination

In Model 1 of the OLS regression models, which regressed log-CRP at baseline on everyday discrimination, there was a significant positive association between everyday discrimination and CRP at baseline (b = 0.17, SE = 0.01, $p \leq .001$; Table 3). This suggests that more frequent exposure to everyday discrimination was associated with higher CRP at baseline, and that each unit increase in everyday discrimination was associated with a roughly 48% increase in baseline CRP. More frequent everday discrimination could place a person initially in a low- or moderate- risk level of CRP into a higher risk category. In Model 2, the addition of sociodemographic characteristics attenuated the relationship between everyday discrimination and CRP, but there remained a significant positive association between everyday discrimination and CRP at baseline (b = 0.02, SE = 0.01, $p \leq .05$). This suggests that a unit increase in everyday discrimination (range, 0-5) is associated with approximately 4.71% higher CRP such that , and sociodemographic characteristics may play a role in the relationship between everyday discrimination and CRP, as shown in the attenuation of coefficients by comparing Model 1 to

Model 2. Model 2 shows that being Black (b = 0.07, SE = 0.03, $p \leq .01$), being a woman (b = 0.08, SE = 0.01, $p \leq .001$), having fewer years of education (b = -0.02, SE = 0.003, $p \leq .001$), and having less wealth (b = -0.01, SE = 0.004, $p \leq .01$) were significantly associated with higher CRP at baseline. This suggests that being Black, being a woman, having less education, and having less wealth are all associated with higher CRP at baseline. In particular, the substantive effect of being Black is associated with about 17% higher baseline CRP, being a woman is associated with just over 20% higher baseline CRP, each year of education corresponds to a 4.71% decrease in baseline CRP, and each unit increase in wealth is associated with a 2.3% reduction in the geometric mean of baseline CRP.

Model 3 shows that the addition of lifestyle characteristics to the model attenuated the effect of everyday discrimination on CRP to marginal significance (b = 0.02, SE = 0.01, $p \leq .10$). This suggests that part of the mechanism linking everyday discrimination with higher CRP levels is through the adoption of negative lifestyle factors (e.g., smoking, drinking, and less physical activity). In addition, the effect of wealth was also attenuated but marginally significant (b = -0.01, SE = 0.004, $p \leq .10$). Education remained significantly and negatively associated with CRP (b = -0.02, SE = 0.003, $p \leq .001$). Being a former smoker (b = 0.03, SE = 0.01, $p \leq .05$), and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were significantly and positively associated with CRP at baseline. Moderate drinking (b = -0.05, SE = 0.02, $p \leq .01$), and sometimes (b = -0.04, SE = 0.02, $p \leq .10$) or frequently (b = -0.10, SE = 0.02, $p \leq .001$) engaging in moderate or vigorous physical activity were negatively associated with CRP at baseline.

Table 13. Linear regression models testing relationship between everyday discrimination and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	B	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.02**	0.01	0.57***	0.11	0.49***	0.11	0.10	0.11
Race								
Black			0.07**	0.03	0.06*	0.02	0.05*	0.02
Gender								
Woman			0.08***	0.01	0.09***	0.01	0.11***	0.01
SES								
Education			-0.02***	0.003	-0.02***	0.003	-0.01***	0.003
Log-income			-0.02	0.02	-0.01	0.02	-0.001	0.02
Log-wealth			-0.01**	0.004	-0.01†	0.004	-0.01	0.004
Everyday Discrimination	0.17***	0.01	0.02*	0.01	0.02†	0.01	0.01	0.01
Age			-0.001	0.001	-0.0005	0.001	0.001	0.001
Married			0.003	0.02	0.01	0.02	0.005	0.01
Lifestyle Characteristics								
Smoking								
Former					0.03*	0.01	0.02	0.01
Current					0.09***	0.02	0.12***	0.02
Drinking								
Moderate					-0.05**	0.02	-0.03†	0.02
Heavy					0.01	0.02	0.02	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.04†	0.02	-0.03	0.02
Frequent					-0.10***	0.02	-0.07***	0.02
Health Conditions								
Overweight/Obese							0.25***	0.01
CESD							0.002	0.004
Chronic conditions							0.01	0.01
R^2	0.0018		0.0388		0.0539		0.1213	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

In Model 4, the effect of everyday discrimination on CRP was reduced to non-significance. This suggests that the addition of lifestyle and health characteristics explain part of the relationship between everyday discrimination and baseline CRP. Further, the effect of wealth was reduced to non-significance and the effect of education was halved (b = -0.01, SE = 0.003, $p \leq .001$). This suggests that some of the protective effect of wealth and education work indirectly through health conditions. The effect of being Black was reduced, but remained positively associated with CRP (b = 0.05, SE = 0.02, $p \leq .05$). The effect of being a woman on CRP was slightly stronger (b = 0.11, SE = 0.01, $p \leq .001$). This suggests that, net of sociodemographic, lifestyle, and health characteristics, being Black and being a woman are associated with having higher CRP at baseline. The effects of being a former smoker and sometimes engaging in moderate or vigorous physical activity were reduced to non-significance. This suggests that the effects of being a former smoker and sometimes engaging in moderate or vigorous physical activity may work indirectly through health conditions. The effect of being a current smoker was slightly strengthened (b = 0.12, SE = 0.02, $p \leq .001$) and the protective effects of moderate drinking (b = -0.03, SE = 0.02, $p \leq .10$) and frequent moderate or vigorous physical activity (b = -0.07, SE = 0.02, $p \leq .001$) were also attenuated but remained significant. Overweight/obesity was the only health condition significantly and positively associated with baseline CRP (b = 0.25, SE = 0.01, $p \leq .001$).

Lifetime discrimination

In Model 1 of the OLS regression models, which regressed log-CRP at baseline on everyday discrimination, there was a significant positive association between lifetime discrimination and CRP (b = 0.02, SE = 0.01, $p \leq .05$; Table 14). This suggests that one additional encounter of lifetime discrimination (range, 0-6) is associated with close to 5% higher baseline

CRP. Upon adjustment for sociodemographic characteristics in Model 2, the association between lifetime discrimination (b = 0.03, SE = 0.01, $p \leq .01$) and CRP at baseline was slightly strengthened but remained in the same direction. This suggests suppression effects of the lifetime discrimination-CRP relationship by sociodemographic factors. In addition, controlling for sociodemographic factors strengthens the effect of lifetime discrimination on CRP such that a one additional exposure to indicators of lifetime discrimination is associated with over 7% increase in baseline CRP. Model 2 also shows that being Black (b = 0.06, SE = 0.03, $p \leq .05$), being a woman (b = 0.09, SE = 0.01, $p \leq .001$), having fewer years of education (b = -0.02, SE = 0.003, $p \leq .001$), and less wealth (b = -0.01, SE = 0.004, $p \leq .01$) were significantly associated with CRP at baseline. This suggests that being Black, being a woman, having fewer years of education and less wealth are associated with higher baseline CRP.

In Model 3, which adjusted for lifestyle characteristics, the positive association between lifetime discrimination and higher CRP at baseline was attenuated but significant (b = 0.02, SE = 0.01, $p \leq .01$). This suggests that more lifetime discrimination might be associated with higher CRP partially due to the influence of lifetime discrimination on lifestyle factors. The relationships between being Black (b = 0.05, SE = 0.03, $p \leq .05$) and having less wealth (b = -0.01, SE = 0.004, $p \leq .10$) on CRP were also reduced but remained significant. Being a woman (b = 0.09, SE = 0.01, $p \leq .001$) and having less education (b = -0.02, SE = 0.003, $p \leq .001$) were also associated with CRP. Being a former smoker (b = 0.03, SE = 0.01, $p \leq .05$) and being a current smoker (b = 0.09, SE = 0.02, $p \leq .001$) were positively associated with CRP. There was a negative association between moderate drinking (b = -0.05, SE = 0.02, $p \leq .01$), as well as sometimes (b = -0.04, SE = 0.02, $p \leq .10$) and frequent (b = -0.11, SE = 0.02, $p \leq .001$) moderate or vigorous physical activity and CRP at baseline.

Table 14. Linear regression models testing relationship between lifetime discrimination and C-reactive protein at baseline (n = 5,486).

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*b*	*SE*	*B*	*SE*
Constant	0.18***	0.01	0.58***	0.11	0.50***	0.11	0.10	0.11
Race								
Black			0.06*	0.03	0.05*	0.03	0.04†	0.02
Gender								
Woman			0.09***	0.01	0.09***	0.01	0.12***	0.01
SES								
Education			-0.02***	0.003	-0.02***	0.003	-0.01***	0.003
Log-income			-0.01	0.02	-0.004	0.02	0.0002	0.02
Log-wealth			-0.01**	0.004	-0.01†	0.004	-0.005	0.004
Lifetime Discrimination	0.02*	0.01	0.03**	0.01	0.02**	0.01	0.02*	0.01
Age			-0.001	0.001	-0.001	0.001	0.001	0.001
Married			0.004	0.02	0.01	0.02	0.01	0.02
Lifestyle Characteristics								
Smoking								
Former					0.03*	0.01	0.02	0.01
Current					0.09***	0.02	0.11***	0.02
Drinking								
Moderate					-0.05**	0.02	-0.03	0.02
Heavy					0.004	0.02	0.01	0.02
Moderate/Vigorous Physical Activity								
Sometimes					-0.04†	0.02	-0.03	0.02
Frequent					-0.11***	0.02	-0.07***	0.02
Health Conditions								
Overweight/Obese							0.25***	0.01
CESD							0.002	0.004
Chronic conditions							0.01	0.01
R^2	0.0019		0.0400		0.0550		0.1222	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, **p* < .05, ***p* < .01, ****p* < .001

Model 4, which further adjusted for health conditions, shows that the relationship between lifetime discrimination and CRP remained unchanged ($b = 0.02$, $SE = 0.01$, $p \leq .05$). This suggests that there are other mechanisms, apart from sociodemographic, lifestyle, and health characteristics that may explain the relationship between lifetime discrimination and baseline CRP. Being Black ($b = 0.04$, $SE = 0.02$, $p \leq .10$), being a woman ($b = 0.12$, $SE = 0.01$, $p \leq .001$), having less education ($b = -0.01$, $SE = 0.003$, $p \leq .001$), being a current smoker ($b = 0.11$, $SE = 0.02$, $p \leq .001$), and being overweight/obese ($b = 0.25$, $SE = 0.01$, $p \leq .001$) were positively associated with CRP. This suggests that being Black, being a woman, having less education, being a current smoker, and being overweight/obese are associated with having higher CRP at baseline. Participating in frequent moderate or vigorous physical activity was negatively associated with CRP ($b = -0.07$, $SE = 0.02$, $p \leq .001$).

Discrimination Exposure and CRP at Follow-Up

The following set of models also test RQ #3, which aimed to assess not only whether exposure to everyday and/or lifetime discrimination were significantly associated with CRP at baseline (which was discussed above), but also whether they were associated with CRP at follow-up. Model 1 shows the relationship between each indicator of discrimination and log-CRP at follow-up, adjusted only for CRP at baseline; Model 2 built on Model 1 with the addition of sociodemographic characteristics; Model 3 further adjusted for lifestyle characteristics; and Model 4, the fully adjusted model, added health conditions.

Everyday Discrimination

The results from analyses regressing log-CRP at follow-up on everyday discrimination are presented in Table 15. Model 1 revealed a non-significant association between everyday discrimination and follow-up CRP, after controlling for baseline CRP. With the addition of sociodemographic characteristics in Model 2, the relationship between everyday discrimination and CRP at follow-up remained non-significant. This suggests that there is no association between everyday discrimination and follow-up CRP.

Table 15. Lagged effect linear regression models testing relationship between everyday discrimination and C-reactive protein at follow-up (n = 5,486).

	Model 1		Model 2	
	b	*SE*	*b*	*SE*
Constant	0.04***	0.01	0.25*	0.11
Log-CRP at baseline	0.58***	0.01	0.57***	0.01
Race				
Black			-0.01	0.02
Gender				
Woman			0.01	0.01
SES				
Education			-0.004	0.003
Log-income			-0.02	0.02
Log-wealth			-0.01**	0.002
Everyday Discrimination	0.003	0.01	0.001	0.01
Age			0.0001	0.001
Married			-0.02	0.01
R^2	0.3627		0.3675	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, **p* < .05, ***p* < .01, ****p* < .001

Lifetime Discrimination

The results from analyses regressing log-CRP at follow-up on lifetime discrimination are presented in Table 16. Model 1 revealed a non-significant relationship between lifetime

discrimination and CRP at follow-up, after adjustment for CRP at baseline. Once adjusted for sociodemographic characteristics in Model 2, the relationship between lifetime discrimination and CRP at follow-up remained non-significant. This suggests that lifetime discrimination and CRP at follow-up are not significantly associated.

Table 16. Linear regression models testing relationship between lifetime discrimination and C-reactive protein at follow-up (n = 5,486).

	Model 1		Model 2	
	b	*SE*	*b*	*SE*
Constant	0.04***	0.01	0.25*	0.11
Log-CRP at baseline	0.58***	0.01	0.57***	0.01
Race				
Black			-0.01	0.02
Gender				
Woman			0.01	0.01
SES				
Education			-0.004	0.003
Log-income			-0.02	0.02
Log-wealth			-0.01**	0.002
Lifetime Discrimination	0.002	0.01	0.003	0.01
Age			0.0001	0.001
Married			-0.02	0.01
R^2	0.3627		0.3675	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

SUMMARY OF AIM 2 FINDINGS

The purpose of Aim 2 was to determine whether exposure to everyday and lifetime discrimination were mechanisms linking race, gender, and socioeconomic status (SES) to low-grade CRP levels at baseline and at a four-year follow-up in a nationally-representative sample of midlife and older adults. Specifically, it tested whether there were independent or interactive

effects of social statuses (e.g., race, gender, and SES) on each measure of discrimination exposure.

In summary, the findings from Aim 2 indicated that there were significant independent effects of gender, education, and wealth on everyday discrimination. In particular, being a man, having less education, and having less wealth were associated with more frequent exposure to everyday discrimination. There were no significant race x gender x SES interactions on everyday discrimination, suggesting that the effects of social status on everyday discrimination are independent rather than interactive. In analyses examining the independent effects of social status variables on lifetime discrimination, the following were significantly associated with lifetime discrimination: race, gender, education, and wealth. The findings suggested that being Black, being a man, having more education, and having less wealth were associated with more exposure to lifetime discrimination. There were no significant race x gender x SES interactions on lifetime discrimination, suggesting that the effects of social status on reporting lifetime discrimination are independent rather than interactive, as well.

There was a significant and positive relationship between everyday discrimination and CRP levels at baseline, but not at follow-up. The relationship between everyday discrimination and CRP at baseline were attenuated after adjustment for sociodemographic, lifestyle, and health characteristics, suggesting that these may be potential mechanisms by which everyday discrimination affects CRP levels in the short-term. Similarly, there was a significant and positive relationship between lifetime discrimination and CRP levels at baseline, but not at follow-up. Overall, the relationship between lifetime discrimination and CRP at baseline was relatively unchanged after adjustment for sociodemographic, lifestyle, and health characteristics, suggesting that there might be other mechanisms that link lifetime discrimination to CRP.

In Chapter 6, the findings from this chapter are summarized and discussed in relation to the larger body of literature. In addition, the research gaps this work addressed and limitations are discussed, and future directions are outlined.

CHAPTER 6

FINDINGS, IMPLICATIONS, AND CONCLUSIONS

This chapter provides a review of the findings from the aims guiding this dissertation, within the context of the existing literatures on the dissertation foci. Tables 17 and 18 summarize the original hypotheses and findings by aim. Findings are then discussed in order of RQs, and organized by findings that 1) supported hypothesizes or 2) did not support hypothesizes. Then, I discuss how this work has addressed gaps in the literature is detailed, and provide the major implications of this research. Further, I acknowledge limitations of this work. Finally, I conclude by summarizing the key findings from this dissertation, along with suggestions for future directions of research that will build upon the work from this study and contribute to improving our understanding of the social distribution of health.

REVIEW OF FINDINGS

Aim 1: Examining the Social Distribution of C-Reactive Protein

The goal of Aim 1 was to examine whether social statuses (race, gender, and SES) were independently or interactively associated with CRP levels at baseline and over a four-year follow-up period using data from the nationally-representative Health and Retirement Study. I hypothesized that lower social status (being Black, woman, and having lower SES—measured as education, income, and wealth) would be associated with higher CRP levels measured at baseline and after a four-year follow-up. Next, I hypothesized that these three social statuses would interact to produce differential levels of CRP at baseline and after a four-year follow-up in a

nationally-representative sample of midlife and older adults. In particular, I hypothesized that Black women of lower SES would have the highest CRP due to their overlapping lower social status positions and in contrast, white men of higher SES would have the lowest CRP levels. Support for study Aim 1 is summarized in Table 17 and described in greater detail in subsequent paragraphs.

Table 17. Summary of support for Aim 1 hypotheses.

Hypotheses	Results
1. Non-Hispanic Black adults will have higher CRP than non-Hispanic Whites.	Partially supported—being Black was positively associated with baseline CRP but not follow-up CRP.
2. Women will have higher CRP than men.	Partially supported—being a woman was positively associated with baseline CRP but not follow-up CRP.
3. Those with lower SES (less education, lower income, and less wealth) will have higher CRP than those with higher SES.	Partially supported—education and wealth, but not income, were negatively associated with baseline CRP. Wealth was negatively associated with follow-up CRP, but education and income were not associated with follow-up CRP.
4. Black women will have higher CRP than White women, Black men, and White men.	Partially supported—Descriptives showed that Black women had significantly higher CRP but there was a non-significant association between Black x woman interaction terms with baseline and follow-up CRP. Contrasts showed that Blacks had higher CRP in women but not in men.
5. There will be a significant three-way interaction between race, gender, and SES on CRP levels, where Black women with lower SES will have the highest levels of CRP.	Partially supported—There were significant three-way interactions between race, gender, and SES on CRP levels, but they showed that 1) Black men of low SES had the highest CRP, 2) Black women had a positive SES-CRP association, 3) three-way interactions were not significant across all indicators of SES, and 4) the results from the three-way interactions were different depending on whether they were regressed on baseline CRP or follow-up CRP.

Supported Findings. There were variations in which social statuses were associated with CRP at baseline versus follow-up. This suggests that social statuses may work in divergent ways to produce CRP over the short- and long-term.

Findings partially supported the hypothesis that being Black would be positively associated with CRP. Specifically, being Black was positively associated with CRP at baseline, but not at follow-up. The finding that Blacks have higher CRP than Whites is consistent with the larger body of literature (Kelley-Hedgepeth et al., 2008; Mitchell & Aneshensel, 2016; Nazmi & Victora, 2007). In a review of the literature on racial variations in CRP, Nazmi and Victora (2007) showed that being Black was consistently associated with higher CRP. Subsequent studies have also demonstrated that being Black is associated with higher CRP. For example, a significant positive relationship between being Black and CRP was found in a racially/ethnically diverse sample of women (Kelley-Hedgepeth et al., 2008), a sample of 37 to 55 year old adults (Gruenewald, Cohen, Matthews, Russell, & Seeman, 2009), and a sample of midlife and older adults (McDade, Lindau, & Wroblewski, 2010). One possible explanation for the higher inflammation of Blacks compared to Whites may be greater exposure to stressors and adversity (Alwin & Wray, 2005; Williams & Mohammed, 2009), which results in earlier and more advanced physiological deterioration in the form of allostatic load (Dannefer, 2003; McEwen, 1998).

Findings also partially supported the hypothesis that being a woman would be associated with higher CRP. Being a woman was positively associated with CRP at baseline, not at follow-up. In the literature, women consistently have higher CRP levels than men, particularly in U.S. samples (Khera et al., 2005; Lakoski et al., 2006; McConell et al., 2002). Some explanations for women's higher CRP levels include gender differences in BMI, hormone replacement therapy,

and differences in lifestyle factors, and the accumulation of gendered life experiences, only some of which I was able to test in this study (Khera et al., 2005; Mitchell & Aneshensel, 2017; Rieker & Bird, 2005). However, existing work suggests that while biological (e.g., hormones) and lifestyle factors account for part of the gender-CRP association, they do not fully account for higher CRP in women. This suggests that differences in stress exposure and responses to stress may serve as a potential pathway linking gender to CRP.

The study's findings also partially supported the hypothesis that SES would be negatively associated with CRP—meaning that CRP would be lower for those of higher SES. The findings showed that education and wealth, but not income, were negatively associated with CRP at baseline, only wealth was negatively associated with follow-up CRP. The literature consistently demonstrates that higher SES is associated with lower CRP (Nazmi, Oliveira, Horta, Gigante, & Victora, 2010; Nazmi & Victora, 2007). Most studies examine education as the sole measure of SES, however. There are some studies that measured education and income concurrently (Janicki-Deverts, Cohen, Kalra, & Matthews, 2012), and found that both measures were inversely associated with CRP. In their review, Nazmi & Victora (2007) noted that fewer studies used income as a measure of SES. Of the two studies that examined income, one study found a negative association between income and CRP; the other study found that income was not significantly associated with low- or high-risk CRP, but it was negatively associated with moderate-risk CRP. The present findings for income parallel another study, which found that education, but not income, was negatively associated with CRP (Ranjit et al., 2007). While few studies have examined the relationship between wealth and CRP, one study showed that wealth was negatively associated with CRP (McDade, Lindau & Wroblewski, 2010). Due to the limited literature on the wealth-CRP link, it is unclear how consistent this relationship is, but the findings do support the well-documented SES-health gradient (Glymour, Clark, & Patton, 2014; Link & Phelan, 1995). The relationship between wealth and CRP remained significant in analyses

examining CRP at both baseline and follow-up, indicating that not only is more wealth associated with lower CRP to start out with, but it also reduces the increase in CRP over a four-year span. This suggests that wealth is an important mechanism linked to CRP, partially because it represents the lifetime accumulation of resources. It might also be an important resource particularly for midlife and older adults because it represents economic stability (Boen, 2016; Boen & Yang, 2016; Robert & House, 1996; Shuey & Willson, 2008).

There was alsopartial support for the hypothesis that Black women would have higher CRP than White women, Black men, and White men. The descriptive statistics showed that Black women had significantly higher CRP than White men, White women, and Black men; however, regression analyses showed a non-significant race x gender interaction term on CRP at baseline and follow-up. Much of the literature examining the joint influence of race and gender on CRP find that Black women exhibit the highest CRP levels, followed by White women, Black men, and White men, respectively (Albert et al., 2004; Khera et al., 2005). This was reflected in the descriptive characteristics of this present study; however, a possible explanation for the difference in the findings from this study compared to earlier studies could also lie in the composition of the sample—previous studies have examined the relationship between race-gender on CRP, but in relatively younger samples. Due to selection effects, the combined race-gender effect on CRP may be diminished in older populations.

Finally, the hypothesis that there would be a significant three-way race x gender x SES interaction on CRP at baseline and follow-up was only partially supported by the findings. While there were several significant three-way race x gender x SES interactions with CRP at both baseline and follow-up, there were some differences from the hypothesized associations. The findings indicated that at baseline, there was both a significant Black x woman x income interaction and a significant Black x woman x wealth interaction. However, there was not a significant Black x woman x education interaction on CRP at baseline. At follow-up, there was a

significant association between the Black x woman x education interaction and CRP, but the analyses showed that race and gender did not interact with income or wealth to produce differential levels of CRP at follow-up. My hypothesis was that Black women of lower SES would have the highest levels of CRP, but the results revealed that Black women had a positive SES-CRP relationship instead, and that Black men of lower SES had the highest levels of CRP. This finding will be discussed in more depth in the next section on the unsupported findings for Aim 1.

Unsupported Findings. Contrary to my hypothesis that being Black would be positively associated with CRP at both baseline and follow-up, I found that being Black was not significantly associated with CRP at follow-up. Because the literature linking race to CRP focuses mostly on cross-sectional studies, it is unclear whether racial trajectories of CRP grow, remain stable, or decline. This finding is somewhat inconsistent with the well-documented race-health relationship in the literature suggesting that Blacks generally experience poorer health and more inflammation compared to Whites (Farmer & Ferraro, 2005; Nazmi & Victora, 2007; Williams & Mohammed, 2009; Williams & Sternthal, 2010; Williams, Yu, Jackson, & Anderson, 1997). Some literature regarding the accumulation of disadvantage suggests that Blacks experience even greater health disadvantages over time compared to their White counterparts, but other evidence points to decreasing disparities over time (Brown, Richardson, Hargrove, & Thomas, 2016; Dannefer, 2003; Farmer & Ferraro, 2005). A recent study shows that Blacks have larger increases in measures of inflammation (fibrinogen, CRP, and IL-6) than Whites during a 15-year follow-up (Fuller-Rowell, Curtis, Doan, & Coe, 2015). This suggests that perhaps with a longer follow-up period, there may be an effect of race on CRP over time, but that a four-year follow-up simply was not long enough to capture gradual increases in CRP.

Considering that the current sample consisted of midlife and older adults, another possible explanation for this unsupported finding could be the older age of the sample. The aging-

as-leveler theory suggests that with age, health disparities may disappear, or "level," due to a combination of mortality selection in lower status groups and the later onset of health problems in higher status groups (Brown, Richardson, Hargrove, & Thomas, 2016; Dupre, 2007; Ferraro & Farmer, 1996). Thus, Blacks with the poorest health (and higher CRP levels) might be more likely to die prior to and during the observation period for this study due to selection effects. Consequently, Whites may begin to experience the onset of more health problems later than Blacks do, resulting in leveling of CRP levels over time.

The relationship between gender and CRP was also not fully supported by the findings. In particular, some research (Yang & Kozloski, 2011) suggests that women continue to have higher CRP than men do over time, but that the rate of increase for women slows over time, likely resulting from hormonal factors due to sex differences (e.g., menopause and its effect on estrogen). Some research suggests that with increasing age, the gender gap in CRP converges and eventually crosses over, where older men have higher CRP than older women (Mitchell & Aneshensel, 2016). There is currently limited research examining the longitudinal relationship between gender and CRP, making it difficult to pinpoint how these findings measure up to other studies. This remains an important area for future research.

Regarding the hypothesis that all measures of SES would be negatively associated with CRP both at baseline and at follow-up, education and wealth were associated with CRP at baseline, and only wealth was associated with CRP at follow-up. Much of the existing literature on the SES-CRP relationship is cross-sectional and the majority of existing studies examine one or two measures of SES, such as education and income (Nazmi & Victora, 2007). However, in these studies, there is a robust education-CRP gradient, where higher education is associated with lower CRP. One reason for the difference in findings may again be the age composition of the present sample compared to previous studies: some work suggests that as individuals age, disparities may level off or become less pronounced. Quite a bit of support for these theories

exists. For example, a recent study by Mitchell & Aneshensel (2016) used HRS data to demonstrate that the education-CRP relationship weakens with increasing age, resulting in no significant educational differences at older ages. While baseline analyses showed a negative effect of education on CRP, the findings from follow-up analyses showed a non-significant effect of education on CRP association. I believe that this may indicate some leveling of the education effect on CRP with time, partially due to the cumulative disadvantage of low education. Future work should examine whether there is an education x age interaction to test this hypothesis. Further, it is also possible that education may be associated with CRP but not with the amount of change in CRP. The education-CRP relationship should be further explored with more waves of data for clarification.

Friedman & Herd (2010) used nationally-representative data to cross-sectionally examine the links among education, income, and inflammation and found evidence that contradicts the findings from this study: only income was negatively associated with inflammation. The present work shows that, at least in midlife and older adults, there is not a significant association between income and CRP. Again, this could be due to the age composition of the sample—given that the respondents are older (with many living on pension and Social Security incomes), there may be less variability in income and income may not be as wide and significant compared to populations in which more respondents are working. Wealth was the only measure of SES that was negatively associated with CRP both at baseline and follow-up, which suggests that wealth plays a strong and important role in inflammation, and likely for trajectories of inflammation. There are several explanations for this association: first, wealth represents the lifetime accumulation of resources and provides financial security, particularly during times of financial hardship or distress. Further, wealth is also a reflection of the marriage market and the ability to combine income and additional resources that are components of wealth, which is linked to the accumulation (or lack thereof) of wealth. Given analyses showing significant racial and gender differences in the

probability of being currently married, it is likely that marital status, as well as marital duration, may have important implications for the accumulation of wealth and the effects of wealth on health. Differences in wealth may also capture intergenerational resources, as well as the accumulation of advantage or disadvantage, which can translate to health through allostatic load (Boen, 2016; Boen & Yang, 2016; Dannefer, 2003; McEwen, 1998; Robert & House, 1996; Shuey & Willson, 2008). This accumulation of advantage or disadvantage in resources may be reflected in wealth, and the analyses suggest that there may be a cumulative effect of wealth on CRP over time, but this requires closer examination with more waves of data.

The findings from this study did not provide support for the hypothesis that there would be a significant race x gender interaction term on baseline and follow-up CRP. Instead, the results suggested that the effects of being Black and being a woman were independent of one another, rather than conditional on one another. This finding was particularly interesting given that the literature shows that Black women have significantly higher CRP than White men, White women, and Black men (Albert, Glynn, Buring, & Ridker, 2004; Albert & Ridker, 2006; Khera et al., 2005; Kelley-Hedgepeth et al., 2008). The way that race and gender may interact to produce differential CRP levels is an underexplored area. Despite existing research suggests that the effects of race and gender are compounded to produce higher CRP in Black women (Khera et al., 2005), the findings from this study suggest otherwise, at least in midlife and older ages. Additional research is required to determine whether selection effects might be at work or whether the current study's focus on midlife and older adults may have an effect on the race x gender relationship with CRP. Perhaps race and gender interact to produce differential CRP in younger populations, and given the biological changes associated with age, in addition to differing rates of morbidity and mortality between men and women and Blacks and Whites, there could be some evidence for leveling that may have occurred prior to the observation period due to selection effects.

Concerning the hypothesis that there would be a significant three-way race x gender x SES interaction on CRP at baseline and follow-up, the findings indicated that there were not consistent significant three-way race x gender x SES interactions across all measures of SES, nor were there consistent relationships across both outcomes (CRP at baseline and CRP at follow-up). Further, contrary to my hypotheses, Black women of lower SES were not the group with highest CRP levels—in fact, the analyses examining the association for CRP at baseline suggested that Black women had incremental increases in CRP with more income and more wealth. To date, no studies have examined the simultaneous relationships among race, gender, and SES on CRP. Based on previous theoretical frameworks, I hypothesized that compounding of lower social status would result in greater disadvantages, yielding more stressful experiences such as discrimination, and resulting in higher inflammation. However, this was not the case. While some of the literature suggests that Black women may experience worse CRP profiles relative to Black men, there is also evidence that Black men fare poorest on a number of health indicators (Gilbert et al., 2016).

Figure 5 illustrates the complexity within the Black x woman x income interaction. Interesting and unique relationships among race, gender, and income are evident. First, Black men with low income were actually the worst off in terms of their CRP levels—low-income Black men had higher CRP than any other group, including Black women with higher income. Next, Black women were the only group who had higher CRP with increases in income. At the lowest levels of income, Black women had lower CRP than any other race and gender groups, which was slightly less than half of the logged-CRP levels that White men and women had.

While White men and women did have lower CRP with increasing income, the gains that they experienced with more income were gradual, while Black men experienced drastic reductions in CRP when they had more income. In fact, at the higher end of education, Black men actually had the lowest CRP levels at baseline, suggesting that there are major benefits to Black

men's health when they have more resources, such as income. Regarding wealth, there was a similar story: Black men with the lowest levels of wealth had the highest CRP levels at baseline, relative to all other groups. Again, Black women were the only group whose CRP increased with more wealth, while all other groups had lower CRP when they had more wealth. Of note, at the lowest end of wealth, Black women again had the lowest CRP levels of all groups, though their CRP levels at baseline were much closer to that of White men and women.

At follow-up, there was a significant association between the Black x woman x education interaction and CRP, but the analyses showed that race and gender did not interact with income or wealth to produce differential levels of CRP at follow-up. With fewer years of education, there were less distinguished differences in CRP at follow-up, though Black women had the lowest CRP and Black men had the highest CRP levels. With approximately 12 years of education, nearly all race and gender groups had similar CRP levels at follow-up. However, with more years of education, Black men had the greatest reduction in CRP levels, while Black women had higher CRP levels with more years of education than they did when they had fewer years of education.

These findings lend some support for, but are also inconsistent with, the broader literature on the SES-health relationship, which suggests that improvements in SES benefit health, and that low SES is consequently a "fundamental cause" of poor health outcomes (Link & Phelan, 1995). The results indicated that Black men, White men, and White women had lower baseline CRP levels when they had more income or wealth. This supports the fundamental cause perspective that SES is a valuable resource that can protect health through a variety of indirect and direct mechanisms (Link & Phelan, 1995). However, the finding that Black women with more income and greater wealth have higher CRP than Black women with less income and wealth does not support the hypothesis that higher SES is an advantage to health. In fact, these results suggest the opposite, at least for Black women. Supplementary analyses were conducted (Tables A5-A12 in Appendix) by stratifying regression analyses by race-gender to further investigate the three-way

interactions tested in this study to determine whether there were similar or diverging patterns in the SES-CRP relationship in each of the race-gender groups. In these analyses, the patterns found differed across social groups—in Black women, none of the indicators of SES were significantly associated with CRP at baseline (Table A5) or at follow-up (Table A9). Black men were protected by only income at both baseline (Table A6) and follow-up (Table A10). More consistent with the literature on SES being a fundamental cause of health, the analyses showed that White men were protected by increasing education and wealth at baseline (Table A7) and by more education and higher income at follow-up (Table 11A). White women had lower CRP with more education at baseline (Table A8) and lower CRP with more wealth at follow-up (Table 12A). It is not completely clear why there are no significant relationships between indicators of SES and CRP for Black women, but this supports calls to comprehensively examine within-group variation, and suggests that there are other factors that explain the variation in CRP, particularly for Black women.

There is a growing line of work that demonstrates that SES does not yield the same advantages to the health of Blacks that they do for the health of Whites: it shows that there are diminishing returns to higher SES for Black relative to White adults (Assari, 2018a; Assari, 2018b; Brown, Richardson, Hargrove, & Thomas, 2016; Farmer & Ferraro, 2005; Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009). This means that compared to Whites, Blacks have little to no improvement in health outcomes when they have higher achieved social status. In some cases, the outcomes for Blacks of higher SES are worse than they are for Whites of lower SES. The work examining race and gender in terms of SES variations in health outcomes typically demonstrates that it is Black men, rather than Black women who experience poorer outcomes with increasing SES (Hickson et al., 2012; Karlamangla et al., 2005). The association between SES and poor health for Blacks is referred to as ‘John Henryism’ and more frequently documented for Black men (Bonham, Sellers, & Neighbors, 2004; James, Strogatz, Wing, &

Ramsey, 1987). The John Henryism argument suggests that in order to attain upward mobility, Blacks must engage in high-effort coping which ultimately results in an accumulation of stressors as well as maladaptive coping responses to buffer the effects of stressors in order to move up in their SES, which can then contribute to elevated rates of morbidity and mortality in higher SES Blacks (Hudson, Neighbors, Geronimus, & Jackson, 2016).

There are few studies that have examined the effect that race, gender, and SES simultaneously have on health overall; and none have investigated the intersections of race, gender, and SES on CRP, particularly in midlife and older adults. Although I hypothesized that Black women of low SES would be the most vulnerable to high CRP, Black women's distinctive experiences to other groups might be key to understanding why the SES-CRP relationship is positive rather than negative for them. For instance, there may be additional burdens faced by Black women, particularly because not only do Black women have to face constraints due to their race, but also due to their gender (Brown & Hargrove, 2013; Richardson & Brown, 2016; Brown, Richardson, Hargrove, & Thomas, 2016; Geronimus, 1992; Warner & Brown, 2011). Being a high-SES Black woman may mean having to navigate predominantly White spaces, similar to Black men, but also spaces occupied more by men than women; and, thus, upward social mobility may result in greater stress and poorer health for Black women (Higginbotham & Weber, 1992; Lichter, McLaughlin, Kephart, & Landry, 1992). In conjunction with lower rates of marriage for Black women, the increased likelihood of being in single-parent families, and the greater responsibilities that Black women may face as head of the household, Black women of higher SES, particularly as they age, may face different life experiences that translate to higher inflammatory burden (Higginbotham & Weber, 1992; Krein & Beller, 1988; Lichter, McLaughlin, Kephart, & Landry, 1992).

Aim 2: Testing Whether Discrimination Exposure Accounted for Social Variations in CRP

The goal of Aim 2 was to expand on Aim 1 by examining whether everyday and/or lifetime discrimination exposure were mechanisms linking interactions of race, gender, and SES to CRP at baseline and over a four-year follow-up in a nationally-representative sample of midlife and older adults. I hypothesized that: (1) race, gender, and SES would interact to shape exposure to discrimination, such that being in multiple low status groups would be linked to more exposure to everyday and lifetime discrimination; (2) there would be a positive association between discrimination exposure and CRP; and (3) the social variations in discrimination exposure would account for the significant three-way interactions hypothesized in Aim 1. Support for study Aim 2 is summarized in Table 18 and described in greater detail in subsequent paragraphs.

Supported Findings. The findings suggested that there were social variations in reporting everyday and lifetime discrimination: in descriptive analyses, Blacks reported significantly more everyday and lifetime discrimination than Whites did. This supports the existing literature, which suggests that Blacks are at a disproportionately greater risk of being exposed to discrimination (Kessler, Mickelson, & Williams, 1999; Grollman, 2014; Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009). The findings for this study indicated that Blacks did not report significantly more everyday discrimination than Whites, but they did report significantly more lifetime discrimination than Whites. The link between race and lifetime discrimination is supported by the literature (Kessler, Mickelson, & Williams, 1999; Grollman, 2014; Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009). Historically, Whites viewed Blacks as inferior and because of the pervasive legacy of racism, race is heavily embedded in society and culture, which results in unfair and differential treatment to members of marginalized groups. Racism is used to maintain the status quo, and one of the ways that racism is continually manifested is in Blacks' exposure to discrimination (Williams & Sternthal, 2010).

A breakdown of the social variation in specific items in the lifetime discrimination scale is provided in the Appendix on Table A3. This shows that Blacks reported significantly more lifetime discrimination for each item in the scale. Some of the differences were quite stark: of note, 13.35% of Blacks reported being unfairly stopped, searched, questioned, physically threatened, or abused by the police, compared to just 4.54% of Whites. In addition, almost twice as many Blacks as Whites reported being unfairly denied a promotion, and almost three times as many Blacks reported being unfairly denied a bank loan.

Table 18. Summary of support for Aim 2 hypotheses.

Hypotheses	Results
6. Discrimination exposure (everyday and lifetime) will be significantly higher for Black adults.	Partially supported—Descriptive analyses showed that Blacks reported more everyday and lifetime discrimination than Whites did, but regression analyses showed that there was not a statistically significant relationship between race and either measure of discrimination.
7. Discrimination exposure (everyday and lifetime) will be significantly higher for men.	Supported—Descriptive and regression analyses showed that men reported more exposure to both everyday and lifetime discrimination than women did.
8. Discrimination exposure (everyday and lifetime) will be significantly higher for Black women.	Unsupported—There were no two-way interactions between race and gender on either everyday or lifetime discrimination.
9. Discrimination exposure (everyday and lifetime) will be significantly higher for those with lower SES.	Partially supported—There was evidence that both education and wealth were negatively associated with everyday discrimination; however, education was positively and wealth was negatively associated with lifetime discrimination. There were no significant associations between income and discrimination exposure.
10. Discrimination exposure (everyday and lifetime) will be significantly higher for Black women with lower SES compared to combinations of other social status groups.	Unsupported—There were no three-way interactions among race, gender, and SES on everyday or lifetime discrimination.
11. Everyday discrimination will be positively associated with CRP.	Partially supported—Everyday discrimination was positively associated with baseline CRP, but

	not with follow-up CRP.
12. Lifetime discrimination will be positively associated with CRP.	Partially supported—Lifetime discrimination was positively associated with baseline CRP, but not with follow-up CRP.
13. Everyday discrimination will attenuate the three-way interaction between race, gender, and SES on CRP (as tested in Aim 1).	Unsupported—Analyses showed no significant three-way race x gender x SES interaction on everyday discrimination, suggesting no evidence to test for mediation.
14. Lifetime discrimination will attenuate the three-way interaction between race, gender, and SES on CRP (as tested in Aim 1).	Unsupported—Analyses showed no significant three-way race x gender x SES interaction on lifetime discrimination, suggesting no evidence to test for mediation.

Findings supported the hypothesis that men would report more instances of everyday and lifetime discrimination. In a well-known study on the prevalence of discrimination, men reported more everyday discrimination but there were no gender differences for lifetime discrimination (Kessler, Mickelson, & Williams, 1999). As they note, the gender difference in reporting discrimination could be due to women not recognizing or reporting their experiences (Kessler, Mickelson, & Williams, 1999). Some work also suggests that men may perceive discrimination due to low self-esteem and perceptions of loss of privilege due to movements aimed to decrease disparities between men and women (Kobrynowicz & Branscombe, 1997; Wilkins, Wellman, Babbitt, Toosi, & Schad, 2015). In an attempt to further understand gender differences in everyday discrimination, Appendix A2 shows the distribution of everyday discrimination and individual items in the everyday discrimination scale across race, gender, and gender within race. This shows that men reported significantly more everyday discrimination than women on almost all items in the everyday discrimination scale.

Findings partially supported the hypothesis that lower SES would be associated with more everyday and more lifetime discrimination. The findings from this study show that both education and wealth were negatively associated with everyday discrimination, a finding that

parallels the SES-discrimination relationship found in earlier work (Kessler, Mickelson, & Williams, 1999). Both Turner and Avison (2003) and Hudson and colleagues (2012) similarly found an inverse association between SES and exposure to both everyday discrimination. An explanation for this may be due to their position as a lower status group. In particular, people of lower SES may be treated negatively by people of higher SES due to perceptions of superiority.

The evidence partially supported the hypotheses that both everyday discrimination and lifetime discrimination would be positively associated with CRP. The findings demonstrated that everyday discrimination and lifetime discrimination were positively associated with CRP at baseline, but not at follow-up. The literature on the relationship between discrimination and CRP suggests a positive relationship, especially because CRP is a biomarker that is consistently associated with stress exposure. Evidence suggests that in response to chronic stress, the body's stress response system becomes dysregulated, and evidenced through systemic inflammation (Glaser & Kiecolt-Glaser, 2005; Johnson, Abbasi, & Master, 2013; McEwen, 1998; Pearson et al., 2003). To date, studies have found inconsistent support for the relationship between measures of everyday and lifetime discrimination and CRP, which will be further discussed in the next section on unsupported findings. Similar to earlier work, this study adds to the body of literature supporting that discrimination is a psychosocial stressor that can disrupt biological processes such as inflammation (Lewis et al., 2010; Mitchell, 2014; Stepanikova, Bateman, & Oates, 2017; Van Dyke et al., 2017).

Unsupported Findings. I hypothesized that Blacks would report significantly more everyday and lifetime discrimination than Whites. Descriptive analyses suggested that Blacks report significantly more instances of both everyday and lifetime discrimination, but regression analyses revealed a non-significant association between being Black and everyday discrimination, controlling on age and marital status. This association was unexpected given the large literature documenting that Blacks report more everyday discrimination (Kessler, Mickelson, & Williams,

1999; Grollman, 2014; Sternthal, Slopen, & Williams, 2011; Williams & Mohammed, 2009). Because the analyses were adjusted for other factors, including age, SES, and marital status, it is plausible that they might be mediating and/or moderating factors in the race-everyday discrimination relationship. For instance, some work suggests that race interacts with SES, and that Blacks with higher SES report more frequent exposure to everyday discrimination, because they are more likely to live and work in predominantly White spaces, and may experience more tokenism (Cole & Omari, 2003; Holder & Vaux, 1998; Kessler, Mickelson, & Williams, 1999; Sanchez-Hucles, 1997).

Regarding the relationship between SES and discrimination exposure, I found evidence contrary to my hypotheses that lower SES would be associated with more everyday and lifetime discrimination. While the literature suggests that to lower status groups, including lower SES individuals, report more instances of discrimination (Hutchinson et al., 2012; Kessler, Mickelson, & Williams, 1999; Turner & Avison, 2003), the findings from this study indicated that this is not always the case. Income was not significantly associated with everyday or lifetime exposure to discrimination, and education was positively associated with lifetime discrimination. There is some evidence that having more education, in particular, is associated with less everyday discrimination, and that having more education is associated with more lifetime discrimination (Kessler, Mickelson, & Williams, 1999). However, the findings regarding income are less clear. The null relationship might be due to the age of the sample, who may have relatively similar levels of income, especially if they are retired.

Contrary to my hypothesis that there would be a two-way race x gender interaction on reporting both everyday and lifetime discrimination, the findings revealed non-significant race x gender interactions on both everyday and lifetime discrimination. The literature on the prevalence of discrimination has not focused largely on whether social statuses interact to shape exposure to discrimination. Despite longstanding theoretical interest and frameworks suggesting that Black

women are particularly more vulnerable to experiencing discrimination because they are located in two lower social status groups being that they are racial minorities and women (Geronimus, Hicken, Keene, & Bound, 2006), this is an area in desperate need of empirical investigation. However, one recent study suggests that if there were a significant race x gender interaction, Black men would have reported more discrimination than other groups, but this may occur due to measurement error rather than a true difference in exposure to discrimination between Black men and women (Ifatunji & Harnois, 2015). Using multi-group confirmatory factor analyses, they find that there are no gaps in everyday nor lifetime discrimination exposure between Black men and women, and that measurement error may account for any gender differences in discrimination exposure found within Blacks.

In addition, I found that race, gender, and SES did not interact to shape exposure to either everyday discrimination or lifetime discrimination. My hypotheses suggested that belonging to multiple low status groups (being Black, being a woman, and being of low SES) would interact, and result in disproportionately greater exposure to both everyday and lifetime discrimination. Based on the lifespan developmental perspective on social status and health, intersectionality, and the literature on 'weathering,' this finding was unexpected. The Everyday Discrimination Scale (EDS) was initially designed to capture experiences of day-to-day racial discrimination for Black women (Essed, 1991) and is widely used measures of discrimination. However, although work suggests that the scale can accurately assess day-to-day discrimination across race groups (Lewis, Yang, Jacobs, & Fitchett, 2012), emerging work demonstrates that the EDS may not be generalizable for comparisons of discrimination across social status groups, including race/ethnicity (Reeve et al., 2011), age (Owens, Kristjansson, & Hunte, 2015) gender (Kwate & Goodman, 2015), and SES (Kwate & Goodman, 2015). This work suggests that measures like the EDS may not capture experiences of discrimination at the intersection of social statuses, particularly like race, gender, and SES (Kwate & Goodman, 2015). Recent reviews of the

literature acknowledge these limitations in the EDS and call for additional conceptualizations and measurement of discrimination, including the use of qualitative and quantitative interviews to more precisely measure discrimination (Reeve et al., 2011). Further, others call for assessing other dimensions related to discrimination, such as the distressing nature of the experience (Williams, Oyserman, Sonnega, Mohammed, & Jackson, 2012) and the multifaceted nature of discrimination at multiple levels (Krieger, 2012).

Additionally, some studies (Grollman, 2012; Grollman, 2014) suggest that the specific attributions of discrimination may be more salient, particularly that the cumulative effect of attributions of discrimination has a stronger effect on health outcomes rather than any given attribution (e.g., race-specific discrimination may not be as detrimental to health as experiencing discrimination due to three or more attributions).

An additional explanation for these null results might be due to poorer health of low SES and racial minorities relative to their higher status counterparts. This selection effect could have driven the findings, such that lower status groups who were disproportionately exposed to a lifetime of everyday discrimination as well as more overt experiences of discrimination may have not lived long enough to participate in this study. This is purely speculation, but the literature on 'weathering' suggests advanced physiological deterioration of poor Black women due to more discrimination, so it is possible poor Black women may have not survived to reach midlife or older ages.

There was partial support for hypotheses that there would be positive relationships between everyday and lifetime discrimination with CRP at baseline and follow-up. While the findings from the study showed that there was a significant positive association between everyday discrimination and CRP and lifetime discrimination and CRP at baseline, but these relationships were not significant for CRP at follow-up. This could be due to reverse causality, where higher CRP causes more discrimination, but there is also the possibility that because CRP increases are

gradual, and because more waves of data were not available, it is possible that two waves of data weren't sufficient to draw conclusions regarding the relationship between discrimination and CRP over time.

Finally, because there was not a significant three-way Black x woman x SES interaction on everyday or lifetime discrimination, mediation analyses could not be conducted. Mediation requires a significant association between the predictor and mediator, as well as between the mediator and the outcome (Baron & Kenny, 1986; MacKinnon et al., 2002).

RESEARCH GAPS ADDRESSED & IMPLICATIONS

This study adds to the existing literature in several important ways. First, it provides insight into an underexplored area: the social distribution of inflammation. Its innovation lies in its application of intersectionality in order to better elucidate disparities in CRP, a stress-associated mechanism that underlies major causes of morbidity and mortality. Intersectional models importantly acknowledge that additive models, like those most frequently used in the literature and that typically focus on one dimension of social status (e.g., race, gender, class), are insufficient at capturing the diversity of the experiences of people at the intersection of multiple social status groups. Thus, in utilizing additive, unidimensional models, researchers may overlook important within-group heterogeneity defined by simultaneously being at the top or bottom of multiple systems of social stratification. Intersectionality's strength lies in the fact that it drives scholars towards a multidimensional line of thinking that focuses on the power distribution within and across social status groups based on their ascribed or achieved statuses. It also forces scholars to consider the ways that social statuses interact with one another to create new and different identities, with social experiences that are wholly exclusive to that specific set of identities.

Most importantly, intersectionality can be used to further examine how intersecting social statuses give way to experiences and identities have the possibility to produce within-group heterogeneity in health outcomes, and particularly, it may shed insight on how health disparities may accumulate over time (Brown & Hargrove, 2013; Collins, 2009; McCall, 2005). However, intersectionality is still a relatively new approach that has not fully taken off in health research. Indeed, two recent papers have called on public health researchers to incorporate intersectionality into their research, largely due to the strengths it has to offer, given that it is well-suited for researchers studying disparities, in particular, and may unknowingly be studied (Bauer, 2014; Bowleg, 2012). Intersectionality offers further insights into complex health phenomena above and beyond traditional models, and can aid scholars in establishing a stronger understanding of the pathways linking social status to health.

This work addresses a major gap in the literature on the social patterning of biomarkers, particularly in providing an understanding of how social statuses interact to produce health differentials in CRP, a stress-associated biomarker linked to health conditions where there are large and pervasive disparities. Extant work demonstrates the importance of examining CRP in isolation for assessing future risk for health problems, such as CVD and CV-related mortality (Pearson et al., 2003). In addition, CRP can be added to existing measures of risk assessment, such as the Framingham Risk Score, to gain even more predictive utility for future CVD rsik. These qualities make CRP an important biomarker for understanding how social processes manifest in biological dysregulation. Though an area of less empirical work, some scholars have found that there are important variations in race, gender, and age on the risk categories for CRP (Wener, Daum, & McQuillan, 2000) and that the tertiles of CRP at the aggregate level may not be equivalent across social groups. This work also provides additional insight because these relationships over time are a very underexplored area. As discussed in the literature review, much of the existing literature assumes additive models of social status on CRP, and making the

assumption that race, gender, and SES operate independently of on another on producing inflammation provides a flawed approach—one that masks heterogeneity. This work demonstrates that the benefits of SES are less consistent within Blacks, and it may function in different ways when examining its effects at a single time point versus multiple time points. Much of the extant work on the relationship between social status and biomarkers has utilized additive models of social status, where social statuses are viewed as independent of one another. To date, very few of the available studies have examined intersection of social statuses. The results from this work provide support for incorporating intersectionality into the study of health disparities because, while the findings suggested that being Black and being a woman were both independently associated with higher CRP, when indicators of SES were also considered in this relationship, the relationships among race, gender, and CRP became more complicated. This approach revealed distinct relationships among race, gender, and SES. These relationships would not have been apparent had the study not considered the possibility that social statuses overlap and interact to produce differential levels of CRP.

This work also fills gaps in the literature by clarifying pathways linked to health disparities, specifically by examining the complex relationship underlying combinations of social statuses and how the social construction of their social identity may shape their health through a number of ways, including increased exposure to stressors such as everyday and lifetime discrimination. This was one of the first studies to examine whether social statuses interact to shape exposure to everyday and lifetime discrimination. Existing work has examined discrimination exposure across social status, but typically in one or two domains of social status (typically race, gender, class). To date, only one study has examined whether there were race and gender differences in discrimination exposure, and whether this accounts for variations in CRP in midlife and older adults (Mitchell, 2014). There is some theoretical basis for discrimination varying across combinations of race, gender, and other dimensions of social status, but this

remains an area where the literature is limited. For instance, while intersectionality and 'weathering' frameworks suggest interactive effects of social status on stress exposure (such as discrimination), there are few empirical investigations of how discrimination exposure varies across and within social groups. Even less is known about whether there are differences in midlife and older ages relative to younger ages, or whether there might be variations in perceptions of discrimination across birth cohorts. As Lewis and colleagues (2010) suggest, the experiences of older Black men and women may differ greatly depending on at what point in their lives the Civil Rights movement occurred.

In particular, this work lends support to the stress literature, by showing that more frequent exposure to everyday and lifetime discrimination is associated with CRP, where there are only a handful of studies in existence. These findings suggest that a more in-depth investigation into the discrimination-CRP link is required—existing longitudinal studies show that there are long-term consequences of discrimination exposure on CRP, so a longer follow-up may be necessary for the effects of everyday and lifetime discrimination on CRP over time to become more pronounced.

This study has implications for future studies, as well as for identifying potential intervention points for addressing health disparities. This work is significant in that it provides a novel approach to studying health disparities because it focuses on the biological manifestations of interlocking systems of oppression and the possible psychosocial mechanisms that might be linked to disparities. It provides an in-depth approach to studying health disparities, and focuses on a complex, dynamic process that unfolds over a lifespan to place some individuals at a greater risk for health problems compared to others.

In utilizing a multidimensional and transdisciplinary approach to examining health disparities, additional research can add to the body of literature by identifying specific risk factors that may be particularly important for certain segments of the population who may be more

vulnerable to premature morbidity and mortality, and expand the way that researchers conceptualize health disparities and constructs such as race, gender, and SES. This work may facilitate the development of policy as well as set the stage for more appropriate interventions to address the social determinants of health, such as preventing or reducing exposure to discrimination. In focusing on the way that social structural conditions and the way that social statuses may interact to produce disparate health outcomes, future research can better elucidate health disparities, describe those at greatest risk, and initiate a new line of research that aims to better characterize the heterogeneity of the population, and the risks and protective resources most important to the maintenance of their health and wellbeing.

LIMITATIONS

Despite the potentially noteworthy findings, this study was limited in several ways that must be acknowledged. First, while there were two waves of data, this is not ideal to definitively understand causality among the study variables, particularly because the waves of data were collected over four years, and this may not have been enough time to observe the gradual changes in CRP over time. The HRS has collected subsequent biomarker data from this sample of participants and has already initiated their plans to collect more biomarker data on a wider range of measures, including more measures of inflammation. Therefore, future work can better address the causality and nature of the relationships for everyday discrimination and CRP with additional waves of data. While the findings of this study are instructive, interpretation of them must be taken with caution.

Several respondents were not eligible for the study due to missing data for CRP or had CRP values that were too low to be detected or too high to be classified as low-grade inflammation. This could present several problems with interpreting and generalizing the results

of the study. As some previous work suggests, the very individuals who might be excluded from the study due to these reasons may also be in the population of interest. For instance, the respondents who are excluded are more likely to be racial minorities, which suggests that the estimates derived from analyses on the relationships among race, gender, SES and CRP may be conservative. Despite the HRS's efforts to oversample Blacks, this sample had a small percentage of Blacks relative to the population, therefore making it difficult to assess whether the study's results capture the distribution of discrimination and CRP in the Black population, particularly if those who have experienced discrimination in medical and/or research settings are the same respondents who were less likely to participate (Ofstedal & Weir, 2011). Because the HRS is a nationally representative study of midlife and older adults, the results of the present study may not generalize to younger populations. Finally, with the exception of CRP, the variables in the study were based on self-report which could create problems due to recall bias. Nevertheless, existing work indicates high reliability of the HRS data (Hayward, 2002).

The EDS is one of the most well-known measures of everyday discrimination, but emerging work using differential item functioning suggests that there may be important differences across social status groups (e.g., race/ethnicity, gender, SES, age), and that interpretations for discrimination at the intersection of these groups must also be used with caution due to these differences. Though the items on this scale are given equal weight, as is standard in the literature, it is unclear whether the items on this scale are in fact equally weighted, and this is an area in need of additional research. For these reasons, the EDS may not have reliably captured discrimination at the intersection of race, gender, and SES in this sample of midlife and older adults; and the analyses from this work should be replicated in other samples for clarification. This group may have particularly unique experiences of discrimination apart from younger age counterparts, as well as differences across race within age groups (Owens, Kristjansson, & Hunte, 2015). Though these findings should be interpreted with caution, the EDS

remains one of the most widely used discrimination instruments, with good psychometric properties and the ability to compare EDS scores across race groups (Lewis, Yang, Jacobs, & Fitchett, 2012). As Lewis, Cogburn, and Williams (2015) argue, the EDS has many attractive qualities, but it is limited because it only captures one dimension of discrimination.

Finally, when studying midlife and older adults, particularly disparities within these ages, there is a possibility of bias due to selection effects. Those who are of greatest analytical interest in the study of disparities, such as racial minorities, those of lower SES, are also those who are in the poorest health, and are more likely to die prior to the study. This means that estimates derived from this study may actually be conservative, had these individuals been able to participate in the study.

FUTURE DIRECTIONS AND CONCLUSIONS

A particularly fruitful area for future research would be to examine the relationships among social status indicators and low-grade inflammation longitudinally. Some of the results from this study were suggestive that while education did not interact with race and gender to produce differential levels of CRP at baseline, it did interact with race and gender to produce greater changes in CRP over four years. Given that the analyses were restricted to a four-year follow-up, and because there is also a greater chance of selection effects, I believe that the estimates derived from this work are likely conservative. However, they indicate that there is a need for additional investigation into the long-term benefits (or lack thereof, for Black women) of education on CRP levels. The HRS is collecting additional waves of data, meaning that many of questions regarding whether and how much SES affects CRP over longer durations can readily be answered using growth curve models. While not currently available, the HRS has expanded their biomarker data collection to include more measures of inflammation, but in addition, they have

worked to increase their sample size for biomarker data. Finally, in order to better assess relationships among social status, discrimination, and low-grade inflammation, it would be useful to include more inflammatory biomarkers such as interleukin-6 and fibrinogen to provide clarity to the biological mechanisms that underlie the discrimination-inflammation link.

Future research is warranted and crucial to further address and clarify the psychosocial and biological mechanisms linking low social status to negative health outcomes. In particular, there is a need for longitudinal inquiry into the association between everyday and lifetime discrimination and biological indicators such as low-grade inflammation. The majority of prior work has been cross-sectional, and further examination is needed to make solid conclusions between the causal relationship linking discrimination to CRP. For example, it is unclear what direction the relationship goes in cross-sectional studies: does discrimination cause inflammation or is it possible that inflammation may cause discrimination? Next, multiple measures of both discrimination exposure and CRP should be captured throughout a longer span of time, ideally throughout several stages of the life course. This will be useful in order to more accurately assess the development of age-related pathology, and may provide a clearer picture of the way that discrimination may "get under the skin" to produce racially disparate health outcomes.

There is also a limited literature on the salience of attributions of discrimination on health—some studies report that the attribution of race was not as important as the experience of discrimination itself, but others have found that experiences of discrimination attributed to race were associated with worse outcomes than non-race attributed discriminatory experiences (Lewis et al., 2006; Lewis et al., 2010; Lewis et al., 2012; Troxel, Matthews, Bromberger, & Sutton-Tyrrell, 2003). This remains an important area for future inquiry, particularly regarding the discrimination-CRP relationship.

The present work integrated four well-established theoretical frameworks to gain a more thorough understanding of the social distribution of C-reactive protein (CRP), a stress-related

biological mechanism underlying many chronic diseases that are responsible for excess deaths in the United States. Results from Aim 1 demonstrated that race, gender, and SES did interact to produce differential CRP levels. The results provide several key pieces of insight: first, the relationship between SES and CRP varies across combinations of race and gender. With improvements in SES, Black women do not experience the same benefits to their health that other groups experienced. This suggests that there is something about having higher SES that makes Black women particularly susceptible to low-grade inflammation. Next, Black men have the most to gain from improvements in SES in regard to their health. In each of the significant interactions found in this work, at the higher ends of SES, Black men had the lowest levels of CRP. This also suggests that SES may provide more distinctive benefits for Black men than it does for White men and White women. Results from Aim 2 showed that everyday and lifetime discrimination exposure varies across social status groups. However, there were no significant interactions among race, gender, and SES in predicting discrimination exposure. Further, results showed that both everyday and lifetime exposure to discrimination were significantly associated with CRP levels at baseline, but not with change in CRP over four years. In clarifying the complexity inherent in disparities in low-grade inflammation, as well as potential psychosocial mechanisms responsible for these mechanisms, this work will contribute to a greater understanding of the factors underlying major causes of excess morbidity and mortality in the United States, and may identify potential intervention points for addressing health disparities.

APPENDIX

Table A1. Correlation matrix for variables of interest.

	1	2	3	4	5	6	7
1. CRP, baseline	1.00						
2. CRP, follow-up	0.59***	1.00					
3. Race	0.09***	0.08***	1.00				
4. Gender	0.10***	0.06***	0.05***	1.00			
5. Education	-0.12***	-0.11***	-0.15***	-0.07***	1.00		
6. Income	-0.08***	-0.09***	-0.20***	-0.15***	0.34***	1.00	
7. Wealth	-0.09***	-0.10***	-0.25***	-0.06***	0.21***	0.31***	1.00
8. Everyday discrimination	0.04**	0.03*	0.06***	-0.10***	-0.01	-0.005	-0.10***
9. Major discrimination	0.04**	0.04***	0.14***	-0.10***	0.09***	0.0004	-0.12***
10. Age	-0.01	0.001	-0.06***	0.01	-0.14***	-0.21***	0.10***
11. Marital status	-0.06***	-0.06***	-0.16***	-0.23***	0.11***	0.39***	0.23***
12. Never drinker	0.08***	0.08***	0.11***	0.16***	-0.22***	-0.21***	-0.12***
13. Moderate drinker	-0.09***	-0.09***	-0.10***	-0.07***	0.23***	0.19***	0.14***
14. Heavy drinker	0.01	0.01	-0.02	-0.18***	0.004	0.04**	-0.02
15. Never smoker	-0.05***	-0.08***	-0.03*	0.19***	0.07**	0.04**	0.06***
16. Former smoker	0.01	0.03*	-0.02	-0.20***	-0.02	0.02	0.04**
17. Current smoker	0.06***	0.08***	0.07***	0.002	-0.08**	-0.10***	-0.16***
18. Never moderate/vigorous physical activity	0.10***	0.08***	0.08***	0.10***	-0.14***	-0.11***	-0.15***
19. Some moderate/vigorous physical activity	0.06***	0.05***	0.06***	0.02	-0.08**	-0.05***	-0.02
20. Frequent moderate/vigorous physical activity	-0.12***	-0.09***	-0.10***	-0.09***	0.17***	0.12***	0.12***
21. Overweight/obese	0.25***	0.21***	0.07***	-0.11***	-0.07***	-0.02	-0.07***
22. CESD	0.08***	0.06***	0.08***	0.10***	-0.18***	-0.19***	-0.19***
23. Health Conditions	0.10***	0.11***	0.08***	0.004	-0.19***	-0.18***	-0.11***

	8	9	10	11	12	13	14	15	16
1. CRP, baseline									
2. CRP, follow-up									
3. Race									
4. Gender									
5. Education									
6. Income									
7. Wealth									
8. Everyday discrimination	1.00								
9. Major discrimination	0.28***	1.00							
10. Age	-0.21***	-0.16***	1.00						
11. Marital status	-0.002	-0.03*	-0.13***	1.00					
12. Never drinker	-0.03*	-0.01	0.09***	-0.08***	1.00				
13. Moderate drinker	-0.03*	-0.01	-0.03*	0.08***	-0.85***	1.00			
14. Heavy drinker	0.05***	0.05***	-0.12***	-0.003	-0.36***	-0.19***	1.00		
15. Never smoker	-0.05***	-0.07***	0.02	0.03*	0.14***	-0.08***	-0.12***	1.00	
16. Former smoker	0.02	0.03*	0.07***	0.04**	-0.14***	0.11***	0.06***	-0.81***	1.00
17. Current smoker	0.06***	0.06***	-0.15***	-0.11***	-0.02	-0.04***	0.10***	-0.31***	-0.30***
18. Never moderate/vigorous physical activity	0.02	-0.008	0.10***	-0.07***	0.13***	-0.12***	-0.03	0.01	-0.04***
19. Some moderate/vigorous physical activity	-0.004	0.002	-0.05***	-0.04**	0.03*	-0.05***	0.03**	-0.03*	-0.0004
20. Frequent moderate/vigorous physical activity	-0.01	0.003	-0.02	0.08***	-0.11***	0.13***	-0.01	0.02	0.03*
21. Overweight/obese	0.08***	0.07***	-0.11***	0.03*	0.07***	-0.09***	0.03*	-0.04***	0.09***
22. CESD	0.22***	0.12***	-0.02	-0.17***	0.12***	-0.13***	0.01	-0.05***	-0.01
23. Health Conditions	0.03*	0.02	0.29***	-0.07***	0.15***	-0.13***	-0.05***	-0.08***	0.11***

	17	18	19	20	21	22	23
1. CRP, baseline							
2. CRP, follow-up							
3. Race							
4. Gender							
5. Education							
6. Income							
7. Wealth							
8. Everyday discrimination							
9. Major discrimination							
10. Age							
11. Marital status							
12. Never drinker							
13. Moderate drinker							
14. Heavy drinker							
15. Never smoker							
16. Former smoker							
17. Current smoker	1.00						
18. Never moderate/vigorous physical activity	0.06***	1.00					
19. Some moderate/vigorous physical activity	0.04***	-0.21***	1.00				
20. Frequent moderate/vigorous physical activity	-0.08***	-0.52***	-0.73***	1.00			
21. Overweight/obese	-0.07***	0.06***	0.09***	-0.12***	1.00		
22. CESD	0.11***	0.22***	0.03	-0.18***	0.06***	1.00	
23. Health Conditions	-0.03**	0.18***	0.03*	-0.15***	0.17***	0.19***	1.00

Table A2. Distribution of everyday discrimination across race and gender, Health and Retirement Study, baseline (n=5,486).

		Race		Gender		Race and Gender				
	Total	**Black**	**White**	**Men**	**Women**	**White Men**	**White Women**	**Black Men**	**Black Women**	***p***
Everyday Discrimination	0.67(0.74)	0.83(1.03)	0.66(0.72)	0.76(0.73)	0.59(0.73)	0.75(0.71)	0.58(0.70)*‡	0.93(0.97)*	0.76(1.07)	+†
You are treated with less courtesy or respect than others.	1.12(1.22)	1.36(1.79)	1.10(1.18)	1.23(1.16)	1.02(1.27)	1.21(1.12)	1.01(1.22)*‡	1.57(1.73)*	1.21(1.80)	+†
You receive poorer service than others at restaurants.	0.69(0.92)	0.88(1.27)	0.67(0.90)	0.76(0.89)	0.62(0.94)	0.75(0.87)	0.60(0.91)*‡	0.92(1.17)	0.85(1.34)	+†
People act as if they think you are not smart.	0.80(1.12)	1.09(1.64)	0.78(1.07)	0.82(1.06)	0.78(1.16)	0.79(1.02)	0.76(1.12)	1.20(1.62)*	1.02(1.64)*	+
People act as if they are afraid of you.	0.43(0.87)	0.51(1.25)	0.42(0.85)	0.60(0.94)	0.28(0.74)	0.60(0.92)	0.26(0.70)*‡	0.62(1.21)	0.44(1.26)*	†
You are threatened or harassed.	0.30(0.71)	0.28(0.84)	0.30(0.70)	0.37(0.69)	0.25(0.72)	0.37(0.69)	0.25(0.71)*‡	0.34(0.77)	0.23(0.88)*	†

+ Sig diff $p < .05$ between race groups

† Sig diff $p < .05$ between genders

* Sig diff $p < .05$ from White men

‡ Sig diff $p < .05$ between genders within race

Table A3. Distribution of lifetime discrimination across race and gender, Health and Retirement Study, baseline (n=5,486).

	Race		Gender		Race and Gender				
	Black	**White**	**Men**	**Women**	**White Men**	**White Women**	**Black Men**	**Black Women**	***p***
Lifetime Discrimination	0.88(1.48)	0.50(0.86)	0.64(0.94)	0.43(0.85)	0.62(0.90)	0.40(0.78)*‡	0.95(1.47)*	0.83(1.47)*	+†
At any time in your life, have you ever been unfairly dismissed from a job?	17.50%	20.23%	23.11%	17.33%	23.65%	17.16%*‡	14.87%*	19.26%	†
For unfair reasons, have you ever not been hired for a job?	15.26%	10.16%	12.51%	8.79%	12.39%	8.17%*‡	14.34%	15.87%	+†
Have you ever been unfairly denied a promotion?	19.48%	10.09%	14.35%	7.60%	13.78%	6.78%*‡	23.37%*	16.96%	+†
Have you ever been unfairly prevented from moving into a neighborhood because the landlord or a realtor refused to sell or rent you a house or apartment?	8.58%	1.32%	1.53%	2.11%	1.05%	1.56%	8.95%*	8.35%*	+
Have you ever been unfairly denied a bank loan?	14.77%	4.12%	4.90%	4.87%	4.30%	3.96%	14.06%*	15.24%*	+
Have you ever been unfairly stopped, searched, questioned, physically threatened, or abused by the police?	13.35%	4.54%	7.99%	2.72%	7.18%	2.18%*‡	20.29%*	8.78%‡	+†

+ Sig diff p < .05 between race groups
† Sig diff p < .05 between genders
* Sig diff p < .05 from White men
‡ Sig diff p < .05 between genders within race

Table A4. Logistic regression models testing relationship between study variables and very high (>10 mg/L) C-reactive protein in the HRS (n = 5,486).

	> 10.0 mg/L CRP at baseline (n = 1,029)		> 10.0 mg/L CRP at follow-up (n = 529)		> 10.0 mg/L CRP at baseline and follow-up (n = 200)	
	OR	*95% CI*	*OR*	*95% CI*	*OR*	*95% CI*
CRP (mg/L), baseline	–	–	8.38***	[6.59, 10.65]	153.89***	[84.63, 279.85]
CRP (mg/L), follow-up	12.47***	[9.87, 15.75]	–	–	155.34***	[93.82, 257.22]
Age	1.00	[0.99, 1.01]	1.00	[0.99, 1.01]	0.97***	[0.95, 0.99]
Black	2.03***	[1.64, 2.50]	1.95***	[1.47, 2.60]	2.78***	[1.84, 4.20]
Women	1.38***	[1.17, 1.63]	1.14	[0.91, 1.41]	1.89***	[1.34, 2.66]
Adult Socioeconomic Status						
Education (years)	0.97	[0.91, 1.04]	0.89***	[0.86, 0.92]	0.89***	[0.84, 0.94]
Log-income	0.70***	[0.63, 0.78]	0.71***	[0.59, 0.86]	0.65***	[0.53, 0.79]
Log-wealth	0.89***	[0.85, 0.92]	0.90***	[0.85, 0.95]	0.84***	[0.79, 0.89]
Everyday Discrimination	1.20***	[1.08, 1.33]	1.13	[0.97, 1.32]	1.23	[0.93, 1.64]
Major Discrimination	1.16**	[1.06, 1.28]	1.10	[0.98, 1.23]	1.23*	[1.04, 1.45]
Married	0.64***	[0.54, 0.75]	0.74*	[0.57, 0.97]	0.59**	[0.39, 0.88]
Health Status						
Overweight/Obese	1.87***	[1.47, 2.37]	1.97***	[1.36, 2.87]	3.65***	[2.00, 6.65]
CES-D Score	1.15***	[1.11, 1.19]	1.14***	[1.08, 1.20]	1.22***	[1.13, 1.32]
Health Conditions	1.41***	[1.35, 1.47]	1.43***	[1.32, 1.55]	1.59***	[1.40, 1.80]
Lifestyle Characteristics						
Moderate/Vigorous Activity						
Sometimes	0.48***	[0.40, 0.58]	0.46***	[0.34, 0.62]	0.37***	[0.24, 0.57]
Frequent	0.39***	[0.32, 0.47]	0.38***	[0.29, 0.50]	0.28***	[0.17, 0.46]
Smoking						
Former Smoker	1.22*	[1.01, 1.47]	1.22†	[0.97, 1.53]	0.93	[0.64, 1.35]
Current Smoker	0.90	[0.65, 1.25]	0.67†	[0.45, 1.01]	0.53	[0.23, 1.20]
Alcohol Use						
Moderate	0.46***	[0.36, 0.59]	0.46***	[0.35, 0.60]	0.26***	[0.15, 0.46]
Heavy	0.67**	[0.49, 0.91]	0.65*	[0.41, 1.01]	0.48*	[0.24, 0.94]

Note. HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A5. Linear regression models testing relationship between SES and C-reactive protein in Black women at baseline (n = 379).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.73***	0.20	0.79**	0.24	0.44	0.27
SES						
Education	-0.01	0.01	-0.01	0.01	-0.01	0.01
Log-income	0.04	0.03	0.03	0.03	0.04	0.03
Log-wealth	-0.002	0.01	-0.00001	0.01	0.001	0.01
Age	-0.01†	0.003	-0.01*	0.003	-0.005	0.003
Married	0.10*	0.05	0.09	0.05	0.08†	0.05
Lifestyle Characteristics						
Smoking						
Former			0.01	0.06	-0.01	0.05
Current			-0.001	0.08	0.03	0.07
Drinking						
Moderate			-0.14†	0.08	-0.11	0.07
Heavy			-0.21	0.26	-0.16	0.25
Moderate/Vigorous Physical Activity						
Sometimes			-0.02	0.07	-0.01	0.08
Frequent			-0.11†	0.06	-0.10	0.06
Health Conditions						
Overweight/Obese					0.27***	0.07
CESD					-0.003	0.01
Chronic conditions					-0.01	0.03
R^2	0.0292		0.0608		0.1095	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A6. Linear regression models testing relationship between SES and C-reactive protein in Black men at baseline (n = 203).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	1.31***	0.38	1.46***	0.39	1.31**	0.44
SES						
Education	-0.01	0.01	-0.01	0.01	-0.01	0.01
Log-income	-0.16**	0.05	-0.19***	0.05	-0.20***	0.05
Log-wealth	-0.03*	0.01	-0.03	0.01	-0.03*	0.01
Age	-0.003	0.004	-0.003	0.004	-0.004	0.004
Married	0.08	0.09	0.07	0.08	0.08	0.08
Lifestyle Characteristics						
Smoking						
Former			0.01	0.09	0.01	0.09
Current			-0.18†	0.10	-0.15	0.11
Drinking						
Moderate			0.22***	0.05	0.23***	0.05
Heavy			0.06	0.11	0.08	0.11
Moderate/Vigorous Physical Activity						
Sometimes			0.11	0.17	0.11	0.16
Frequent			0.06	0.15	0.07	0.15
Health Conditions						
Overweight/Obese					0.10	0.06
CESD					-0.002	0.02
Chronic conditions					0.03	0.04
R^2	0.0889		0.1490		0.1664	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A7. Linear regression models testing relationship between SES and C-reactive protein in White men at baseline (n = 2,087).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.47**	0.16	0.27	0.17	-0.02	0.17
SES						
Education	-0.02***	0.004	-0.01***	0.004	-0.01***	0.003
Log-income	-0.01	0.03	0.002	0.02	-0.003	0.03
Log-wealth	-0.02**	0.01	-0.01**	0.01	-0.01	0.01
Age	0.001	0.001	0.003*	0.001	0.004	0.001
Married	-0.01	0.03	0.005	0.03	-0.01	0.03
Lifestyle Characteristics						
Smoking						
Former			0.05*	0.02	0.02	0.02
Current			0.22***	0.03	0.23***	0.03
Drinking						
Moderate			-0.04	0.02	-0.03	0.02
Heavy			0.001	0.03	0.01	0.03
Moderate/Vigorous Physical Activity						
Sometimes			-0.07†	0.04	-0.06	0.04
Frequent			-0.13**	0.04	-0.10**	0.04
Health Conditions						
Overweight/Obese					0.23***	0.03
CESD					0.01	0.01
Chronic conditions					0.005	0.01
R^2	0.0266		0.0617		0.1129	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A8. Linear regression models testing relationship between SES and C-reactive protein in White women at baseline (n = 2,816).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.85***	0.15	0.85***	0.14	0.29**	0.12
SES						
Education	-0.02***	0.004	-0.02***	0.004	-0.01**	0.004
Log-income	-0.02	0.03	-0.01	0.02	0.01	0.02
Log-wealth	-0.01	0.01	-0.01	0.01	-0.002	0.01
Age	-0.003*	0.001	-0.003	0.001	-0.001	0.001
Married	-0.01	0.02	-0.01	0.02	-0.01	0.02
Lifestyle Characteristics						
Smoking						
Former			0.03	0.02	0.02	0.02
Current			-0.003	0.04	0.04	0.03
Drinking						
Moderate			-0.05*	0.02	-0.02	0.02
Heavy			0.03	0.05	0.03	0.04
Moderate/Vigorous Physical Activity						
Sometimes			-0.04	0.03	-0.03	0.03
Frequent			-0.12***	0.03	-0.06*	0.03
Health Conditions						
Overweight/Obese					0.28***	0.02
CESD					0.0003	0.005
Chronic conditions					0.02*	0.01
R^2	0.0227		0.0379		0.1319	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A9. Lagged effect linear regression models testing relationship between SES and C-reactive protein in Black women at follow-up (n = 379).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	-0.09	0.24	-0.05	0.25	0.31	0.28
Log-CRP at baseline	0.59***	0.06	0.58***	0.06	0.57***	0.06
SES						
Education	0.01	0.01	0.01	0.01	0.01	0.01
Log-income	0.04	0.03	0.04	0.04	0.02	0.03
Log-wealth	-0.004	0.01	-0.003	0.01	-0.01	0.01
Age	-0.002	0.003	-0.002	0.003	-0.002	0.003
Married	-0.02	0.05	-0.02	0.04	-0.01	0.04
Lifestyle Characteristics						
Smoking						
Former			0.03	0.05	0.05	0.05
Current			0.02	0.06	0.03	0.06
Drinking						
Moderate			-0.04	0.06	-0.08	0.06
Heavy			0.07	0.13	0.08	0.13
Moderate/Vigorous Physical Activity						
Sometimes			-0.05	0.06	-0.11†	0.06
Frequent			-0.07	0.07	-0.14*	0.07
Health Conditions						
Overweight/Obese					-0.0003	0.06
CESD					-0.03**	0.01
Chronic conditions					-0.04**	0.01
R^2	0.3816		0.3885		0.4215	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A10. Lagged effect linear regression models testing relationship between SES and C-reactive protein in Black men at follow-up (n = 203).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	-0.52	0.59	-0.63	0.56	-0.84	0.61
Log-CRP at baseline	0.64***	0.004	0.66***	0.04	0.64***	0.04
SES						
Education	-0.01	0.01	-0.01	0.01	-0.01	0.01
Log-income	0.07	0.09	0.08	0.09	0.07	0.08
Log-wealth	-0.02*	0.01	-0.02*	0.01	-0.01*	0.01
Age	0.01*	0.004	0.01**	0.003	0.01**	0.004
Married	-0.04	0.06	-0.02	0.06	-0.01	0.06
Lifestyle Characteristics						
Smoking						
Former			0.03	0.06	0.03	0.06
Current			0.10	0.11	0.14	0.10
Drinking						
Moderate			-0.01	0.07	0.01	0.07
Heavy			0.11	0.09	0.13	0.08
Moderate/Vigorous Physical Activity						
Sometimes			-0.19**	0.07	-0.19*	0.09
Frequent			-0.10†	0.06	-0.10	0.06
Health Conditions						
Overweight/Obese					0.16*	0.06
CESD					-0.001	0.02
Chronic conditions					0.005	0.02
R^2	0.4619		0.4893		0.5124	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A11. Lagged effect linear regression models testing relationship between SES and C-reactive protein in White men at follow-up (n = 2,087).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.35*	0.17	0.25	0.17	0.18	0.17
Log-CRP at baseline	0.52***	0.02	0.51***	0.02	0.49***	0.02
SES						
Education	-0.01*	0.003	-0.01	0.003	-0.005	0.003
Log-income	-0.05†	0.03	-0.04	0.03	-0.04†	0.03
Log-wealth	-0.01	0.01	-0.01	0.01	-0.01	0.01
Age	0.002	0.001	0.002	0.001	0.002	0.001
Married	-0.03	0.02	-0.02	0.02	-0.03	0.02
Lifestyle Characteristics						
Smoking						
Former			0.04*	0.02	0.04†	0.02
Current			0.08**	0.03	0.08**	0.03
Drinking						
Moderate			-0.02	0.02	-0.02	0.02
Heavy			-0.03	0.03	-0.02	0.02
Moderate/Vigorous Physical Activity						
Sometimes			0.01	0.03	0.01	0.03
Frequent			-0.01	0.02	-0.005	0.02
Health Conditions						
Overweight/Obese					0.06**	0.02
CESD					0.002	0.01
Chronic conditions					0.01	0.01
R^2	0.3266		0.3317		0.3367	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

Table A12. Lagged effect linear regression models testing relationship between SES and C-reactive protein in White women at follow-up (n = 2,816).

	Model 1		Model 2		Model 3	
	b	*SE*	*b*	*SE*	*b*	*SE*
Constant	0.28*	0.11	0.17	0.12	0.09	0.13
Log-CRP at baseline	0.60***	0.02	0.60***	0.01	0.57***	0.01
SES						
Education	-0.001	0.004	0.001	0.005	0.002	0.005
Log-income	-0.02	0.02	-0.02	0.02	-0.01	0.02
Log-wealth	-0.01*	0.003	-0.004	0.003	-0.003	0.003
Age	-0.001	0.001	-0.001	0.001	-0.001	0.001
Married	-0.02	0.02	-0.01	0.02	-0.01	0.02
Lifestyle Characteristics						
Smoking						
Former			0.04**	0.01	0.04**	0.01
Current			0.10**	0.03	0.11**	0.03
Drinking						
Moderate			-0.03*	0.01	-0.02†	0.01
Heavy			-0.03	0.04	-0.03	0.04
Moderate/Vigorous Physical Activity						
Sometimes			-0.002	0.02	-0.002	0.02
Frequent			-0.001	0.02	0.01	0.02
Health Conditions						
Overweight/Obese					0.06***	0.01
CESD					-0.003	0.004
Chronic conditions					0.01	0.01
R^2	0.3933		0.3991		0.4038	

Note. SES = socioeconomic status; HRS = Health and Retirement Study.
Key. † p< .10, *p < .05, **p < .01, ***p < .001

BIBLIOGRAPHY

Addo, J., Ayerbe, L., Mohan, K. M., Crichton, S., Sheldenkar, A., Chen, R., ... & McKevitt, C. (2012). Socioeconomic status and stroke an updated review. *Stroke*, *43*(4), 1186-1191.

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: the challenge of the gradient. *American Psychologist*, *49*(1), 15.

Adler, N. E., Boyce, W. T., Chesney, M. A., Folkman, S., & Syme, S. L. (1993). Socioeconomic inequalities in health: no easy solution. *JAMA*, *269*(24), 3140-3145.

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: what we know and what we don't. *Annals of the New York Academy of Sciences*, *896*(1), 3-15.

Adler, N. E., & Stewart, J. (2010). Health disparities across the lifespan: meaning, methods, and mechanisms. *Annals of the New York Academy of Sciences*, *1186*(1), 5-23.

Ailshire, J. A., & House, J. S. (2011). The unequal burden of weight gain: an intersectional approach to understanding social disparities in BMI trajectories from 1986 to 2001/2002. *Social Forces*, *90*(2), 397.

Albert, M. A., Glynn, R. J., Buring, J., & Ridker, P. M. (2004). C-reactive protein levels among women of various ethnic groups living in the United States (from the Women's Health Study). *The American Journal of Cardiology, 93*(10), 1238-1242.

Albert, M. A., Ravenell, J., Glynn, R. J., Khera, A., Halevy, N., & de Lemos, J. A. (2008). Cardiovascular risk indicators and perceived race/ethnic discrimination in the Dallas Heart Study. *American Heart Journal*, *156*(6), 1103-1109.

Alwin, D. F., & Wray, L. A. (2005). A life-span developmental perspective on social status and health. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 60*(Special Issue 2), S7-S14.

Aneshensel, C. S., & Mitchell, U. A. (2014). The stress process: its origins, evolution, and future. *Sociology of Mental Health*, 53-74.

Assari, S. (2018). Unequal gain of equal resources across racial groups. *International Journal of Health Policy and Management, 7*(1), 1.

Assari, S. (2018). Health disparities due to diminished return among black Americans: public policy solutions. *Social Issues and Policy Review*, *12*(1), 112-145.

Barnes, L. L., De Leon, C. F. M., Wilson, R. S., Bienias, J. L., Bennett, D. A., & Evans, D. A. (2004). Racial differences in perceived discrimination in a community population of older blacks and whites. *Journal of Aging and Health, 16*(3), 315-337.

Bauer, G. R. (2014). Incorporating intersectionality theory into population health research methodology: Challenges and the potential to advance health equity. *Social Science & Medicine*, *110*, 10-17.

Baum, A., Garofalo, J. P., & YALI, A. (1999). Socioeconomic status and chronic stress: does stress account for SES effects on health? *Annals of the New York Academy of Sciences*, *896*(1), 131-144.

Beatty Moody, D. L., Brown, C., Matthews, K. A., & Bromberger, J. T. (2014). Everyday Discrimination Prospectively Predicts Inflammation across 7-Years in Racially Diverse Midlife Women: Study of Women's Health across the Nation. *Journal of Social Issues*, *70*(2), 298-314.

Bennett, J. M., Fagundes, C. P., & Kiecolt-Glaser, J. K. (2013). The chronic stress of caregiving accelerates the natural aging of the immune system. In *Immunosenescence* (pp. 35-46). Springer New York.

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *International Journal of Epidemiology*, *31*(2), 285-293.

Boen, C. (2016). The role of socioeconomic factors in Black-White health inequities across the life course: Point-in-time measures, long-term exposures, and differential health returns. *Social Science & Medicine*, *170*, 63-76.

Boen, C., & Yang, Y. C. (2016). The physiological impacts of wealth shocks in late life: Evidence from the Great Recession. *Social Science & Medicine*, *150*, 221-230.

Bonham, V. L., Sellers, S. L., & Neighbors, H. W. (2004). John Henryism and self-reported physical health among high–socioeconomic status African American men. *American Journal of Public Health*, *94*(5), 737-738.

Black, P. H. (2003). The inflammatory response is an integral part of the stress response: Implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome X. *Brain, Behavior, and Immunity*, *17*(5), 350-364.

Borrell, L. N., Kiefe, C. I., Williams, D. R., Diez-Roux, A. V., & Gordon-Larsen, P. (2006). Self-reported health, perceived racial discrimination, and skin color in African Americans in the CARDIA study. *Social Science & Medicine*, *63*(6), 1415-1427.

Bowleg, L. (2012). The problem with the phrase women and minorities: intersectionality—an important theoretical framework for public health. *American Journal of Public Health*, *102*(7), 1267-1273.

Braveman, P. A., Cubbin, C., Egerter, S., Williams, D. R., & Pamuk, E. (2010). Socioeconomic disparities in health in the United States: what the patterns tell us. *American Journal of Public Health*, *100*(S1), S186-S196.

Brown, T. H., & Hargrove, T. W. (2013). Multidimensional approaches to examining gender and racial/ethnic stratification in health. *Women, Gender, and Families of Color*, *1*(2), 180-206.

Brown, T. H., Richardson, L. J., Hargrove, T. W., & Thomas, C. S. (2016). Using multiple-hierarchy stratification and life course approaches to understand health inequalities: The intersecting consequences of race, gender, SES, and age. *Journal of Health and Social Behavior, 57*(2), 200-222.

Case, A., & Deaton, A. (2015). Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. *Proceedings of the National Academy of Sciences, 112*(49), 15078-15083.

Chrousos, G. P. (1995). The hypothalamic–pituitary–adrenal axis and immune-mediated inflammation. *New England Journal of Medicine, 332*(20), 1351-1363.

Clarke, P., Fisher, G., House, J., Smith, J., & Weir, D. (2008). *Guide to the content of the HRS psychosocial leave-behind participant lifestyle questionnaires: 2004 & 2006*. Survey Research Center, Institute for Social Research, University of Michigan.

Cole, E. R., & Omari, S. R. (2003). Race, class and the dilemmas of upward mobility for African Americans. *Journal of Social Issues, 59*(4), 785-802.

Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., & Turner, R. B. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences, 109*(16), 5995-5999.

Crenshaw, K. (1989). Demarginalizing the intersection of race and sex: A black feminist critique of antidiscrimination doctrine, feminist theory and antiracist politics. *U. Chi. Legal F.*, 139.

Crenshaw, K. (1991). Mapping the margins: Intersectionality, identity politics, and violence against women of color. *Stanford Law Review*, 1241-1299.

Crimmins, E., Faul, J., Kim, J. K., Guyer, H., Langa, K., Ofstedal, M. B…Weir, D. (2013). *Documentation of biomarkers in the 2006 and 2008 Health and Retirement Study.* Survey Research Center, University of Michigan.

Crimmins, E., Faul, J., Kim, J. K., & Weir, D. (2015). *Documentation of biomarkers in the 2010 and 2012 Health and Retirement Study.* Survey Research Center, University of Michigan.

Crimmins, E., Kim, J. K., McCreath, H., Faul, J., Weir, D., & Seeman, T. (2014). Validation of blood-based assays using dried blood spots for use in large population studies. *Biodemography and Social Biology*, *60*(1), 38-48.

Cunningham, T. J., Seeman, T. E., Kawachi, I., Gortmaker, S. L., Jacobs, D. R., Kiefe, C. I., & Berkman, L. F. (2012). Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation in the CARDIA cohort of 4 US communities. *Social Science & Medicine*, *75*(5), 922-931.

Dailey, A. B., Kasl, S. V., Holford, T. R., Lewis, T. T., & Jones, B. A. (2010). Neighborhood-and individual-level socioeconomic variation in perceptions of racial discrimination. *Ethnicity & Health*, *15*(2), 145-163.

Dannefer, D. (2003). Cumulative advantage/disadvantage and the life course: Cross-fertilizing age and social science theory. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *58*(6), S327-S337.

Dill, B. T. & Zambrana, R. E. (2009). Critical Thinking about Inequality: An Emerging Lens. Pp. 1-21 in *Emerging Intersections: Race, Class, and Gender in Theory, Policy, and Practice*, edited by Bonnie Thornton Dill and Ruth. E. Zambrana. Piscataway, NJ: Rutgers University Press.

Dinwiddie, G. Y., Zambrana, R. E., Doamekpor, L. A., & Lopez, L. (2015). The Impact of Educational Attainment on Observed Race/Ethnic Disparities in Inflammatory Risk in the 2001–2008 National Health and Nutrition Examination Survey. *International Journal of Environmental Research and Public Health*, *13*(1), 42.

Do, D. P., Frank, R., & Finch, B. K. (2012). Does SES explain more of the black/white health gap than we thought? Revisiting our approach toward understanding racial disparities in health. *Social Science & Medicine*, *74*(9), 1385-1393.

Dowd, J. B., Simanek, A. M., & Aiello, A. E. (2009). Socio-economic status, cortisol and allostatic load: a review of the literature. *International Journal of Epidemiology*, 1-13.

Du Bois, W. E. B., & Eaton, I. (1899). *The Philadelphia Negro: a social study* (No. 14). Published for the University.

Dupre, M. E. (2007). Educational differences in age-related patterns of disease: Reconsidering the cumulative disadvantage and age-as-leveler hypotheses. *Journal of Health and Social Behavior*, *48*(1), 1-15.

Farmer, M. M., & Ferraro, K. F. (2005). Are racial disparities in health conditional on socioeconomic status? *Social Science & Medicine*, *60*(1), 191-204.

Ferraro, K. F., & Farmer, M. M. (1996). Double jeopardy, aging as leveler, or persistent health inequality? A longitudinal analysis of white and black Americans. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *51*(6), S319-S328.

Friedman, E. M., & Herd, P. (2010). Income, education, and inflammation: differential associations in a national probability sample (the MIDUS study). *Psychosomatic Medicine*, *72*(3), 290.

Gabay, C., & Kushner, I. (1999). Acute-phase proteins and other systemic responses to inflammation. *New England Journal of Medicine*, *340*(6), 448-454.

Geronimus, A. T. (1992). The weathering hypothesis and the relationship of maternal age to birth outcome: evidence and speculations. *Ethnicity & Disease*, *2*, 207-221.

Geronimus, A. T., Bound, J., Keene, D., & Hicken, M. (2007). Black-white differences in age trajectories of hypertension prevalence among adult women and men, 1999-2002. *Ethnicity & Disease*, *17*(1), 40-49.

Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *American Journal of Public Health*, *96*(5), 826-833.

Gilbert, K. L., Ray, R., Siddiqi, A., Shetty, S., Baker, E. A., Elder, K., & Griffith, D. M. (2016). Visible and invisible trends in black men's health: pitfalls and promises for addressing racial, ethnic, and gender inequities in health. *Annual Review of Public Health*, *37*, 295-311.

Glaser, R., & Kiecolt-Glaser, J. K. (2005). Stress-induced immune dysfunction: implications for health. *Nature Reviews Immunology*, *5*(3), 243-251.

Glymour, M. M., Clark, C. R., & Patton, K. K. (2014). Socioeconomic determinants of cardiovascular disease: recent findings and future directions. *Current Epidemiology Reports*, *1*(2), 89-97.

Goosby, B. J., Malone, S., Richardson, E. A., Cheadle, J. E., & Williams, D. T. (2015). Perceived discrimination and markers of cardiovascular risk among low-income African American youth. *American Journal of Human Biology*, *27*(4), 546-552.

Graham, G. (2015). Disparities in cardiovascular disease risk in the United States. *Current Cardiology Reviews*, *11*(3), 238-245.

Grollman, E. A. (2012). Multiple forms of perceived discrimination and health among adolescents and young adults. *Journal of Health and Social Behavior*, *53*(2), 199-214.

Grollman, E. A. (2014). Multiple disadvantaged statuses and health the role of multiple forms of discrimination. *Journal of Health and Social Behavior*, *55*(1), 3-19.

Gruenewald, T. L., Cohen, S., Matthews, K. A., Tracy, R., & Seeman, T. E. (2009). Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Social Science & Medicine*, *69*(3), 451-459.

Hänsel, A., Hong, S., Cámara, R. J., & Von Kaenel, R. (2010). Inflammation as a psychophysiological biomarker in chronic psychosocial stress. *Neuroscience & Biobehavioral Reviews*, *35*(1), 115-121.

Hatch, S. L., & Dohrenwend, B. P. (2007). Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *American Journal of Community Psychology*, *40*(3-4), 313-332.

Hayward, M. D. (2002). Using the Health and Retirement Survey to investigate health disparities. *Unpublished manuscript*.

Hayward, M. D., Miles, T. P., Crimmins, E. M., & Yang, Y. (2000). The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *American Sociological Review*, 910-930.

Heidenreich, P. A., Trogdon, J. G., Khavjou, O. A., Butler, J., Dracup, K., Ezekowitz, M. D., ... & Lloyd-Jones, D. M. (2011). Forecasting the future of cardiovascular disease in the United States. *Circulation*, *123*(8), 933-944.

Herd, P., Karraker, A., & Friedman, E. (2012). The social patterns of a biological risk factor for disease: race, gender, socioeconomic position, and C-reactive protein. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *67*(4), 503-513.

Heron, M. (2015). Deaths: Leading causes for 2011. *National Vital Statistics Reports*, *64*(7), 1-96.

Hickson, D. A., Diez Roux, A. V., Gebreab, S. Y., Wyatt, S. B., Dubbert, P. M., Sarpong, D. F., ... & Taylor, H. A. (2012). Social patterning of cumulative biological risk by education and income among African Americans. *American Journal of Public Health*, *102*(7), 1362-1369.

Higginbotham, E., & Weber, L. (1992). Moving up with kin and community: Upward social mobility for black and white women. *Gender & Society*, *6*(3), 416-440.

Hinze, S. W., Lin, J., & Andersson, T. E. (2012). Can we capture the intersections? Older Black women, education, and health. *Women's Health Issues*, *22*(1), e91-e98.

Holder, J. C., & Vaux, A. (1998). African American professionals: Coping with occupational stress in predominantly white work environments. *Journal of Vocational Behavior*, *53*(3), 315-333.

Holmes, C. J., & Zajacova, A. (2014). Education as “the great equalizer”: health benefits for black and white adults. *Social Science Quarterly*, *95*(4), 1064-1085.

HRS Staff (2017). *Sample Sizes and Response Rates*. Ann Arbor, MI: Survey Research Center, Institute for Social Research, University of Michigan.

Hudson, D. L., Bullard, K. M., Neighbors, H. W., Geronimus, A. T., Yang, J., & Jackson, J. S. (2012). Are benefits conferred with greater socioeconomic position undermined by racial discrimination among African American men?. *Journal of Men's Health*, *9*(2), 127-136.

Hudson, D. L., Neighbors, H. W., Geronimus, A. T., & Jackson, J. S. (2016). Racial discrimination, John Henryism, and depression among African Americans. *Journal of Black Psychology*, *42*(3), 221-243.

Hunt, M. O., Wise, L. A., Jipguep, M. C., Cozier, Y. C., & Rosenberg, L. (2007). Neighborhood racial composition and perceptions of racial discrimination: evidence from the Black Women's Health Study. *Social Psychology Quarterly*, *70*(3), 272-289.

Ifatunji, M. A., & Harnois, C. E. (2016). An explanation for the gender gap in perceptions of discrimination among african americans: Considering the role of gender bias in measurement. *Sociology of Race and Ethnicity*, *2*(3), 263-288.

Jackson, C. L., Szklo, M., Yeh, H. C., Wang, N. Y., Dray-Spira, R., Thorpe, R., & Brancati, F. L. (2013). Black-white disparities in overweight and obesity trends by educational attainment in the United States, 1997–2008. *Journal of Obesity*, 2013, 140743.

James, S. A., Strogatz, D. S., Wing, S. B., & Ramsey, D. L. (1987). Socioeconomic status, John Henryism, and hypertension in blacks and whites. *American Journal of Epidemiology*, *126*(4), 664-673.

Jemal, A., Ward, E., Anderson, R. N., Murray, T., & Thun, M. J. (2008). Widening of socioeconomic inequalities in US death rates, 1993–2001. *PloS One*, *3*(5), e2181.

Johnson, T. V., Abbasi, A., & Master, V. A. (2013). Systematic review of the evidence of a relationship between chronic psychosocial stress and C-reactive protein. *Molecular Diagnosis & Therapy*, *17*(3), 147-164.

Juster, F. T., & Suzman, R. 1995. An overview of the health and retirement study. *Journal of Human Resources*, *30*, S7-S56.

Kanjilal, S., Gregg, E. W., Cheng, Y. J., Zhang, P., Nelson, D. E., Mensah, G., & Beckles, G. L. (2006). Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971-2002. *Archives of Internal Medicine*, *166*(21), 2348-2355.

Karlamangla, A. S., Merkin, S. S., Crimmins, E. M., & Seeman, T. E. (2010). Socioeconomic and ethnic disparities in cardiovascular risk in the United States, 2001–2006. *Annals of Epidemiology*, *20*(8), 617-628.

Kelley-Moore, J. A., & Ferraro, K. F. (2004). The black/white disability gap: persistent inequality in later life?. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *59*(1), S34-S43.

Kelley-Hedgepeth, A., Lloyd-Jones, D. M., Colvin, A., Matthews, K. A., Johnston, J., Sowers, M. R., ... & SWAN Investigators. (2008). Ethnic differences in C-reactive protein concentrations. *Clinical Chemistry*, *54*(6), 1027-1037.

Kershaw, K. N., Lewis, T. T., Roux, A. V. D., Jenny, N. S., Liu, K., Penedo, F. J., & Carnethon, M. R. (2016). Self-reported experiences of discrimination and inflammation among men and women: The multi-ethnic study of atherosclerosis. *Health Psychology*, *35*(4), 343-350.

Kessler, R. C., Mickelson, K. D., & Williams, D. R. (1999). The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. *Journal of Health and Social Behavior*, *40*(3), 208-230.

Khera, A., McGuire, D. K., Murphy, S. A., Stanek, H. G., Das, S. R., Vongpatanasin, W., ... & de Lemos, J. A. (2005). Race and gender differences in C-reactive protein levels. *Journal of the American College of Cardiology*, *46*(3), 464-469.

Kobrynowicz, D., & Branscombe, N. R. (1997). Who considers themselves victims of discrimination?. *Psychology of Women Quarterly*, *21*(3), 347-363.

Krein, S. F., & Beller, A. H. (1988). Educational attainment of children from single-parent families: Differences by exposure, gender, and race. *Demography*, *25*(2), 221-234.

Krieger, N. (2012). Methods for the scientific study of discrimination and health: an ecosocial approach. *American Journal of Public Health*, *102*(5), 936-944.

Krieger, N., Rowley, D. L., Herman, A. A., & Avery, B. (1993). Racism, sexism, and social class: implications for studies of health, disease, and well-being. *American Journal of Preventive Medicine*, *9*(6 Suppl), 83-122.

Krieger, N., Waterman, P. D., Kosheleva, A., Chen, J. T., Carney, D. R., Smith, K. W., ... & Thornhill, G. (2011). Exposing racial discrimination: implicit & explicit measures–the My Body, My Story study of 1005 US-born black & white community health center members. *PloS One*, *6*(11), e27636.

Kristenson, M., Eriksen, H. R., Sluiter, J. K., Starke, D., & Ursin, H. (2004). Psychobiological mechanisms of socioeconomic differences in health. *Social Science & Medicine*, *58*(8), 1511-1522.

Kung, H. C., Hoyert, D. L., Xu, J., & Murphy, S. L. (2008). Deaths: final data for 2005. *National Vital Statistics Report*, *56*(10), 1-120.

Kurian, A. K., & Cardarelli, K. M. (2007). Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. *Ethnicity & Disease*, *17*(1), 143.

Kwate, N. O. A., & Goodman, M. S. (2015). Racism at the intersections: Gender and socioeconomic differences in the experience of racism among African Americans. *American Journal of Orthopsychiatry*, *85*(5), 397.

Lakoski, S. G., Cushman, M., Criqui, M., Rundek, T., Blumenthal, R. S., D'Agostino, R. B., & Herrington, D. M. (2006). Gender and C-reactive protein: data from the Multiethnic Study of Atherosclerosis (MESA) cohort. *American Heart Journal*, *152*(3), 593-598.

Lantz, P. M., House, J. S., Lepkowski, J. M., Williams, D. R., Mero, R. P., & Chen, J. (1998). Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. *JAMA*, *279*(21), 1703-1708.

Lantz, P. M., Lynch, J. W., House, J. S., Lepkowski, J. M., Mero, R. P., Musick, M. A., & Williams, D. R. (2001). Socioeconomic disparities in health change in a longitudinal study of US adults: the role of health-risk behaviors. *Social Science & Medicine*, *53*(1), 29-40.

Lewis, T. T., Aiello, A. E., Leurgans, S., Kelly, J., & Barnes, L. L. (2010). Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. *Brain, Behavior, and Immunity*, *24*(3), 438-443.

Lewis, T. T., Cogburn, C. D., & Williams, D. R. (2015). Self-reported experiences of discrimination and health: scientific advances, ongoing controversies, and emerging issues. *Annual Review of Clinical Psychology*, *11*, 407-440.

Lewis, T. T., Everson-Rose, S. A., Powell, L. H., Matthews, K. A., Brown, C., Karavolos, K., ... & Wesley, D. (2006). Chronic exposure to everyday discrimination and coronary artery calcification in African-American women: the SWAN Heart Study. *Psychosomatic Medicine*, *68*(3), 362-368.

Lewis, T. T., Williams, D. R., Tamene, M., & Clark, C. R. (2014). Self-reported experiences of discrimination and cardiovascular disease. *Current Cardiovascular Risk Reports*, *8*(1), 1-15.

Lewis, T. T., Yang, F. M., Jacobs, E. A., & Fitchett, G. (2012). Racial/ethnic differences in responses to the everyday discrimination scale: A differential item functioning analysis. *American Journal of Epidemiology*, *175*(5), 391-401.

Lloyd-Jones, D. M., Liu, K., Tian, L., & Greenland, P. (2006). Narrative review: assessment of C-reactive protein in risk prediction for cardiovascular disease. *Annals of Internal Medicine*, *145*(1), 35-42.

Lichter, D. T., McLaughlin, D. K., Kephart, G., & Landry, D. J. (1992). Race and the retreat from marriage: A shortage of marriageable men?. *American Sociological Review*, 781-799.

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior*, 80-94.

Lu, Y., Ezzati, M., Rimm, E. B., Hajifathalian, K., Ueda, P., & Danaei, G. (2016). Sick Populations and Sick Subpopulations: Reducing Disparities in Cardiovascular Disease Between Blacks and Whites in the United States. *Circulation*, *134*(6), 472-485.

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G. & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7*, 83-104.

Marmot, M. (2005). Social determinants of health inequalities. *The Lancet*, *365*(9464), 1099-1104.

Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences, 37*(7), 1401-1415.

McCall, L. (2005). The complexity of intersectionality. *Signs*, *30*(3), 1771-1800.

McConnell, J. P., Branum, E. L., Ballman, K. V., Lagerstedt, S. A., Katzmann, J. A., & Jaffe, A. S. (2002). Gender differences in C-reactive protein concentrations—confirmation with two sensitive methods. *Clinical Chemistry Lab Medicine, 40*, 56-59.

McDade, T. W., Lindau, S. T., & Wroblewski, K. (2010). Predictors of C-reactive protein in the national social life, health, and aging project. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *66*(1), 129-136.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179.

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *896*(1), 30-47.

Mensah, G. A., & Brown, D. W. (2007). An overview of cardiovascular disease burden in the United States. *Health Affairs*, *26*(1), 38-48.

Mensah, G. A., Mokdad, A. H., Ford, E. S., Greenlund, K. J., & Croft, J. B. (2005). State of disparities in cardiovascular health in the United States. *Circulation*, *111*(10), 1233-1241.

Meyer, I. H., Schwartz, S., & Frost, D. M. (2008). Social patterning of stress and coping: Does disadvantaged social statuses confer more stress and fewer coping resources? *Social Science & Medicine*, *67*(3), 368-379.

Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: a glucocorticoid-resistance model. *Health Psychology*, *21*(6), 531-541.

Mitchell, U. A. (2014). *Mechanisms of health disparities in inflammation: a test of the differential stress exposure and differential stress vulnerability hypotheses* (Unpublished doctoral dissertation). University of California, Los Angeles, Los Angeles, CA.

Mitchell, U. A., & Aneshensel, C. S. (2016). Social Inequalities in Inflammation Age Variations in Older Persons. *Journal of Aging and Health*, 0898264316645546.

Mulder, B. C., de Bruin, M., Schreurs, H., van Ameijden, E. J., & van Woerkum, C. M. (2011). Stressors and resources mediate the association of socioeconomic position with health behaviours. *BMC Public Health*, *11*(1), 798.

Nazmi, A., Oliveira, I. O., Horta, B. L., Gigante, D. P., & Victora, C. G. (2010). Lifecourse socioeconomic trajectories and C-reactive protein levels in young adults: findings from a Brazilian birth cohort. *Social Science & Medicine*, *70*(8), 1229-1236.

Nazmi, A., & Victora, C. G. (2007). Socioeconomic and racial/ethnic differentials of C-reactive protein levels: a systematic review of population-based studies. *BMC Public Health*, *7*(1), 212.

Ofstedal, M. B., & Weir, D. R. (2011). Recruitment and retention of minority participants in the health and retirement study. *The Gerontologist*, *51*(suppl_1), S8-S20.

Ore, T. E. (2003). Constructing differences. In T. E. Ore (Ed.), *The Social Construction of Difference and Inequality: Race, Class, Gender, and Sexuality* (pp. 11-17). Boston: McGraw Hill.

Ortman, J. M., Velkoff, V. A., & Hogan, H. (2014). An aging nation: the older population in the United States. *Washington, DC: US Census Bureau*, 25-1140.

Owens, S., Kristjansson, A. L., & Hunte, H. E. (2015). A differential item functional analysis by age of perceived interpersonal discrimination in a multi-racial/ethnic sample of adults. *Ethnicity & Disease*, *25*(4), 479.

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: a meta-analytic review. *Psychological Bulletin*, *135*(4), 531-554.

Pearlin, L. I. (1989). The sociological study of stress. *Journal of Health and Social Behavior*, *30*(3), 241-256.

Pearlin, L. I., & Bierman, A. (2013). Current issues and future directions in research into the stress process. *Handbook of the Sociology of Mental Health*, 325-340.

Pearlin, L. I., Menaghan, E. G., Lieberman, M. A., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior*, *22*(4), 337-356.

Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon, R. O., Criqui, M., ... & Rifai, N. (2003). Markers of inflammation and cardiovascular disease application to clinical and public health practice: a statement for healthcare professionals from the centers for disease control and prevention and the American Heart Association. *Circulation*, *107*(3), 499-511.

Pepys, M. B., & Hirschfield, G. M. (2003). C-reactive protein: a critical update. *The Journal of Clinical Investigation*, *111*(12), 1805-1812.

Puhl, R. M., Andreyeva, T., & Brownell, K. D. (2008). Perceptions of weight discrimination: prevalence and comparison to race and gender discrimination in America. *International Journal of Obesity*, *32*(6), 992-1000.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement, 1(3), 385-401.

RAND HRS Data, Version P. Produced by the RAND Center for the Study of Aging, with funding from the National Institute on Aging and the Social Security Administration. Santa Monica, CA (August 2016).

Ranjit, N., Diez-Roux, A. V., Shea, S., Cushman, M., Ni, H., & Seeman, T. (2007). Socioeconomic position, race/ethnicity, and inflammation in the multi-ethnic study of atherosclerosis. *Circulation*, *116*(21), 2383-2390.

Reeve, B. B., Willis, G., Shariff-Marco, S. N., Breen, N., Williams, D. R., Gee, G. C., ... & Levin, K. Y. (2011). Comparing cognitive interviewing and psychometric methods to evaluate a racial/ethnic discrimination scale. *Field Methods*, *23*(4), 397-419.

Richardson, L. J., & Brown, T. H. (2016). (En) gendering racial disparities in health trajectories: A life course and intersectional analysis. *SSM-Population Health*, *2*, 425-435.

Rieker, P. P., & Bird, C. E. (2005). Rethinking gender differences in health: why we need to integrate social and biological perspectives. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *60*(Special Issue 2), S40-S47.

Robert, S., & House, J. S. (1996). SES differentials in health by age and alternative indicators of SES. *Journal of Aging and Health*, *8*(3), 359-388.

Romero, C. X., Romero, T. E., Shlay, J. C., Ogden, L. G., & Dabelea, D. (2012). Changing trends in the prevalence and disparities of obesity and other cardiovascular disease risk factors in three racial/ethnic groups of USA adults. *Advances In Preventive Medicine*, *2012*.

Sanchez-Hucles, J. V. (1997). Jeopardy not bonus status for African American women in the work force: Why does the myth of advantage persist?. *American Journal of Community Psychology*, *25*(5), 565-580.

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science*, *308*(5722), 648-652.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions 1. *Endocrine Reviews*, *21*(1), 55-89.

Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Annals of the New York Academy of Sciences*, *1186*(1), 223-239.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, *130*(4), 601-630.

Servais M. *Overview of HRS Public Data Files for Cross-sectional and Longitudinal Analysis*. Ann Arbor, Michigan: Institute for Social Research, University of Michigan; 2010.

Shuey, K. M., & Willson, A. E. (2008). Cumulative disadvantage and black-white disparities in life-course health trajectories. *Research on Aging*, *30*(2), 200-225.

Singh-Manoux, A., Adler, N. E., & Marmot, M. G. (2003). Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Social Science & Medicine*, *56*(6), 1321-1333.

Ski, C. F., King-Shier, K. M., & Thompson, D. R. (2014). Gender, socioeconomic and ethnic/racial disparities in cardiovascular disease: a time for change. *International Journal of Cardiology*, *170*(3), 255-257.

Smith, J., Fisher, G., Ryan, L., Clarke, P., House, J., & Weir, D. (2013). *Psychosocial and lifestyle questionnaire, 2006-2010.* Survey Research Center, Institute for Social Research, University of Michigan.

Stepanikova, I., Bateman, L. B., & Oates, G. R. (2017). Systemic inflammation in midlife: race, socioeconomic status, and perceived discrimination. *American Journal of Preventive Medicine*, *52*(1), S63-S76.

Steptoe, A., & Marmot, M. (2002). The role of psychobiological pathways in socio-economic inequalities in cardiovascular disease risk. *European Heart Journal*, *23*(1), 13-25.

Sternthal, M. J., Slopen, N., & Williams, D. R. (2011). Racial disparities in health. *Du Bois Review: Social Science Research on Race*, *8*(01), 95-113.

Survey Data Analysis with Stata 14. UCLA: Statistical Consulting Group. https://stats.idre.ucla.edu/stata/seminars/survey-data-analysis-with-stata-14/

Taylor, T. R., Kamarck, T. W., & Shiffman, S. (2004). Validation of the Detroit Area Study Discrimination Scale in a community sample of older African American adults: the Pittsburgh healthy heart project. *International Journal of Behavioral Medicine, 11*(2), 88-94.

Troxel, W. M., Matthews, K. A., Bromberger, J. T., & Sutton-Tyrrell, K. (2003). Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. *Health Psychology, 22*(3), 300-309.

Turner, R. J. (2013). Understanding health disparities: the relevance of the stress process model. *Society and Mental Health, 3*(3), 170-186.

Turner, R. J., & Avison, W. R. (2003). Status variations in stress exposure: Implications for the interpretation of research on race, socioeconomic status, and gender. *Journal of Health and Social Behavior,* 488-505.

Turner, R. J., & Lloyd, D. A. (1999). The stress process and the social distribution of depression. *Journal of Health and Social Behavior, 40*(4), 374-404.

Turner, R. J., & Lloyd, D. A. (2004). Stress burden and the lifetime incidence of psychiatric disorder in young adults: racial and ethnic contrasts. *Archives of General Psychiatry, 61*(5), 481-488.

Van Dyke, M. E., Vaccarino, V., Dunbar, S. B., Pemu, P., Gibbons, G. H., Quyyumi, A. A., & Lewis, T. T. (2017). Socioeconomic status discrimination and C-reactive protein in African-American and White adults. *Psychoneuroendocrinology, 82*, 9-16.

Warner, D. F., & Brown, T. H. (2011). Understanding how race/ethnicity and gender define age-trajectories of disability: An intersectionality approach. *Social Science & Medicine, 72*(8), 1236-1248.

Weir, D. R. (2017). *HRS Institutional Review Board Information*. Ann Arbor, MI: Survey Research Center, Institute for Social Research, University of Michigan.

Wener, M. H., Daum, P. R., & McQuillan, G. M. (2000). The influence of age, sex, and race on the upper reference limit of serum C-reactive protein concentration. *The Journal of Rheumatology, 27*(10), 2351-2359.

Wilkins, C. L., Wellman, J. D., Babbitt, L. G., Toosi, N. R., & Schad, K. D. (2015). You can win but I can't lose: Bias against high-status groups increases their zero-sum beliefs about discrimination. *Journal of Experimental Social Psychology, 57*, 1-14.

Williams, D. R. (2012). Miles to go before we sleep racial inequities in health. *Journal of Health and Social Behavior, 53*(3), 279-295.

Williams, D. R. (1999). Race, socioeconomic status, and health the added effects of racism and discrimination. *Annals of the New York Academy of Sciences, 896*(1), 173-188.

Williams, D. R., & Collins, C. (1995). US socioeconomic and racial differences in health: patterns and explanations. *Annual Review of Sociology*, 349-386.

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

Williams, D. R., John, D. A., Oyserman, D., Sonnega, J., Mohammed, S. A., & Jackson, J. S. (2012). Research on discrimination and health: an exploratory study of unresolved conceptual and measurement issues. *American Journal of Public Health*, *102*(5), 975-978.

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

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

Williams, D. R., John, D. A., Oyserman, D., Sonnega, J., Mohammed, S. A., & Jackson, J. S. (2012). Research on discrimination and health: an exploratory study of unresolved conceptual and measurement issues. *American Journal of Public Health*, *102*(5), 975-978.

Williams, D. R., Priest, N., & Anderson, N. B. (2016). Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health Psychology*, *35*(4), 407-411.

Williams, D. R., & Sternthal, M. (2010). Understanding racial-ethnic disparities in health sociological contributions. *Journal of Health and Social Behavior*, *51*(1 suppl), S15-S27.

Williams, D. R., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health socio-economic status, stress and discrimination. *Journal of Health Psychology*, *2*(3), 335-351.

Wray, L. A., Alwin, D. F., & McCammon, R. J. (2005). Social status and risky health behaviors: results from the health and retirement study. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *60*(Special Issue 2), S85-S92.

Yang, Y., & Kozloski, M. (2011). Sex differences in age trajectories of physiological dysregulation: inflammation, metabolic syndrome, and allostatic load. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, *66*(5), 493-500.

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EDUCATION

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B.A. in Gerontology; B.S. in Public Health — *August 2005-August 2012*

FELLOWSHIPS AND AWARDS

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Alfred P. Sloan University Center for Exemplary Mentoring Scholarship (2015-2018)
Eugene W. and Lucy Kemmerer Lederer Memorial Scholarship (2015-2018)
Graduate Student Travel Award, Department of Biobehavioral Health (2013-2015)
Bunton-Waller Award (2013)
Healthy People Penn State Conference and Expo Scholarship (2012)
Bright Futures Florida Medallion Scholarship (2005-2006, 2010-2012)
Federal Pell Grant (2006)

MANUSCRIPTS

Baker, T.A., Roker, R., **Collins-Farmer, H.R.**, Johnson-Lawrence, V., Thorpe, R. J., Mingo, C., & Vasquez, E. (2016). Beyond race and gender: Measuring behavioral and social indicators of pain treatment satisfaction in older Black and White cancer patients. *Gerontology & Geriatric Medicine 2*, 1-8.

Farmer, H. R., Wray, L. A., & Thomas, J. R. Race and everyday discrimination on mortality risk in the Health and Retirement Study. Submitted for review at *Gerontology & Geriatric Medicine* for a special issue on aging in diverse populations.

PROFESSIONAL PRESENTATIONS

Collins-Farmer, H. R., Wray, L. A., & Alwin, D. F. (2017). Socioeconomic factors and the Black-White difference in C-reactive protein: evidence from the Health and Retirement Study. Paper presented at the Lives of Color: Race-ethnicity and the Life Course conference, University Park, PA, June 2017.

Collins-Farmer, H. R., Wray, L. A., & Thomas, J. R. (2017). Everyday discrimination is associated with all-cause mortality: evidence from the Health and Retirement Study. Paper accepted for the American Sociological Association Annual Meeting, Montreal, Canada, August 2017.