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Socioeconomic Status Mobility and Lifetime Exposure to Discrimination on Cardiovascular Disease Events

Nkenge H. Jones-Jack
Walden University

Walden University

College of Health Sciences

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Nkenge Jones-Jack

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Review Committee
Dr. Angela Prehn, Committee Chairperson, Public Health Faculty
Dr. JaMuir Robinson, Committee Member, Public Health Faculty
Dr. John Oswald, University Reviewer, Public Health Faculty

Chief Academic Officer
Eric Riedel, Ph.D.

Walden University
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Abstract

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Nkenge Hawanya Jones-Jack

MPH, University of Alabama, Birmingham, 1997

BS, University of Alabama, Birmingham, 1994

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health

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Abstract

Blacks in the United States have the highest rates of hypertension in the world, and their cardiovascular disease mortality rates are higher than for any other population group as a result of traditional risk factors such as obesity and stronger family history. However, additional underlying factors, such as social determinants of health (e.g., socioeconomic status [SES]) and macrosocial factors (e.g., racism), also correlate with adverse health outcomes. This study investigated whether the interaction between SES mobility over the lifecourse and lifetime racial discrimination influenced the extent to which hypertension contributed to the cardiovascular disease health disparities observed among Blacks in the Jackson Heart Study (JHS). Using a socioecological framework, cross-sectional data collected from the baseline period on a cohort of 5,302 JHS participants were analyzed with multiple regression techniques. The study findings indicated that SES mobility, as measured by education, predicted both the racial discrimination exposure and the burden that individuals experience. However, neither SES mobility nor racial discrimination had any effect in moderating the relationship between hypertension and cardiovascular disease when examined individually or collectively. This study examined a new approach for measuring the influence of racial discrimination on health outcomes. Multidisciplinary public health and research partners should continue to advance understanding of the complex health impact of such experiences on individuals and the dynamics that create racial factors in order to effect social change.

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Dedication

First and foremost, I give all the praise to my Lord and Savior because it is only through Him that all things are possible. This dissertation is dedicated to my incredible parents, Stanley and Catherine Jones, who taught me the importance of knowledge and hard work at an early age. You instilled the philosophy that knowing and doing "just enough to get by" was not enough; hence, my continued quest to learn. Thank you for always being my rock, knowing the heights to which I could soar, and giving me the greatest of opportunities. I especially dedicate my dissertation to my loving husband, Leonard Jack, Jr., and amazing children, Yahzarah and Koben Jack. You all have been on this journey with me daily, sacrificing countless hours of family time, and continuing to love, support, and tolerate me through all of my highs and lows along the way. To my husband, thank you for your tremendous patience and insightful understanding of what this process required. Your confidence in my ability and celebration of every milestone along the way reignited excitement to continue moving forward. To Yahzarah and Koben, the amount of love, encouragement, and patience you have given me has been incredible. From sitting with me while I work to making me smile, laugh, and feel light-hearted, in spite of myself, your love never fails. You have a very special way of giving me focus and purpose in all that I do. I hope that being a part of this journey with me has inspired you to know that you too can persevere through challenging tasks and accomplish marvelous things. Last, but certainly not least, I want to thank my brothers, Jawanza and Koro Jones, aunts, uncles, nieces, cousins, in-laws, church family, friends, and colleagues for their unwavering prayers, encouragement, and support.

Acknowledgments

As I reach this incredible milestone in my life, I would like to acknowledge the support of some incredible people in my life. First, I would like to first like to thank my dissertation committee Chair, Dr. Angela Witt Prehn, for her knowledge, time, support, and expertise in guiding me through this process. The impact that you have had on my life is surely to be realized for many years to come. Thank you to my remaining committee members, Dr. JaMuir Robinson and Dr. John Weiland Oswald, for your comments and support in further refining my research.

I would also like to thank the Jackson Heart Study (JHS) for use of their expansive dataset. Thank you to Dr. Mario Sims and Dr. DeMarc Hickson for their time in answering questions regarding JHS methodology and the development of an analysis plan that was best suited for my study. I would acknowledge Stanford Mwasongwe for creating my JHS data set.

A special thank you to my high school Advanced Placement English teacher, Linda Patterson, for teaching me the art of writing thoughtful, well-constructed, and grammatically correct papers. All of your red pen corrections were not in vain.

Finally, I would like to thank some special friends and colleagues who have continued to support me in various ways over the years, including: Kelli Pack Smith, Belinda Gaston, Dr. Tekeda Ferguson, Dr. Machell Town, Dr. Laurie Elam-Evans, Dr. Chinaro Kennedy Chukwu, Kartena Hall, Walter Sykes, Dr. Andrea Morris, and Dr. Sandra Hayes.

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Chapter 1: Introduction to the Study

Introduction

Blacks in the United States are documented to have hypertension rates higher that any other population group (Flack, Ferdinand, & Nasser, 2003; Go et al., 2012; Kurian & Cardarelli, 2007; Quinones, Liang, & Ye, 2012; Williams, 2009). While there are numerous risk factors that have been examined and found to be correlated to hypertension, researchers have not been able to consistently justify why adverse CVD health outcomes occur among Blacks across the spectrum of many risk factors. A fundamental risk factor for overall health is socioeconomic status (SES); yet, Blacks in the United States at all levels of SES experience higher rates of CVD compared to their White counterparts. However, research investigating the role of macrosocial factors, such as racism, as an underlying cause of health disparities is immature (Gee & Ford, 2011; Gee, Walsemann, & Brondolo, 2012; Krieger, 2000; Shuey & Willson, 2008; Sims et al., 2012; Williams & Jackson, 2005; Williams & Mohammed, 2013).

This study was designed to assess the relationship between exposure to lifetime racial discrimination and changes in SES over the lifecourse, so as to provide new insight on why Blacks are more likely than Whites to have higher rates of hypertension and poor CVD outcomes. This chapter offers some background on the relevance of this study, explains the public health problem, states the research questions that the study will answer, the conceptual frameworks used to justify the study, and the how this study contributes to the field of public health.

Background

Socioeconomic status (SES) has been well-documented as a strong predictor of adverse cardiovascular health outcomes (Banks, Marmot, Oldfield, & Smith, 2006; Do & Finch, 2008; Lynch & Kaplan, 2000). SES is frequently based on several parameters beyond just income and education, and interacts with complex demographic, environmental, and social attributes which further contribute to adverse health outcomes (Wamala, Lynch, & Kaplan, 2001). Studies have historically found that lower SES during childhood typically remains consistent into adulthood (Corcoran, 1995; Hardaway & McLoyd, 2008; Johnson-Lawrence, Kaplan, & Galea, 2013; Kearney, 2006), and SES mobility strongly impacts health status in adulthood (Hardaway & McLoyd, 2008; Johnson-Lawrence et al., 2013). A recent study comparing the SES trajectories of adults from Alameda County, CA for nearly 30 years found that as SES improved, CVD mortality risk decreased, even after adjusting for age, race/ethnicity, marital status, and gender (Johnson-Lawrence et al., 2013). However, Hardaway et al. (2008) argued that SES mobility studies have not adequately considered the significance of race. Furthermore, Mays et al. (2007) argued that even when adjusting for SES, Blacks suffer from excess overall death at a rate equivalent to 1.1 million years of life lost, or roughly 38,000 deaths per year. Hence, race should not be used as a proxy for SES (Jones, 2002; Kawachi et al., 2005).

Racism occurs at multiple levels and contributes to inequities in the allocation of services, goods, resources, and health outcomes (Jones, 2000). Some researchers argue that although social stressors (e.g., racism) are not responsible for lower SES, it has been

strongly suggested that the stressors associated with lower SES often directly or indirect influence health and well-being (Schulz et al., 2001; Thoits, 2010). Although all individuals' experiences are impacted on some level by stress, it could be argued that Blacks generally experience more stress and are more greatly impacted due to racial discrimination. It is important to note that Black-White differences in cardiovascular disease exist among Blacks across SES groups (Krieger et al., 2013; Williams & Jackson, 2005; Wyatt, Williams, et al., 2003).

Researchers have explored various approaches of how the stress associated with perceived racism may transcend multiple aspects of an individual's life. These aspects include:

- residentially segregated communities;
- stereotypical or derogatory media portrayals;
- level of control or flexibility at work;
- availability, quality, and affordability of resources and services; and
- understanding of cultural differences (Brondolo, Gallo, et al., 2009; Myers, 2008; Williams, Mohammed, Leavell, & Collins, 2010).

Of greater concern are the multiple pathways through which racism affects health (Brondolo, Gallo, et al., 2009). While several studies exploring SES mobility have similar findings, Hardaway et al. (2008) acknowledges the failure to understanding the consequences of racism on social mobility creates unique challenges for Blacks.

Problem Statement

Hypertension is considered to be the most important risk factor for CVD (Ferdinand & Sounders, 2006; Williams, 2009), and Blacks have the highest rates of hypertension in the world (Flack et al., 2003; Kurian & Cardarelli, 2007; Roger et al., 2010; Thomas, Thomas, Pearson, Klag, & Mead, 1997; Watson, 2008; Williams, 2009). Watson (2008) estimates that hypertension among Blacks may be correlated to CVD mortality rates that are 3-5 times greater than Whites. Blacks experience more CVD risk and burden as a result of traditional risk factors (e.g., higher rates of obesity, stronger family history); however, there are also additional underlying factors that contribute to this overwhelming disparity. Racial health disparities have long been suggested to be the result of differences in socioeconomic status (SES) indicators (e.g., education, income) (Farmer & Ferraro, 2005; Laveist, Thorpe, Galarraga, Bower, & Gary-Webb, 2009; Wang & Chen, 2011; Williams & Jackson, 2005; Williams, 2012). More specifically, life-long changes in SES such as SES mobility have been identified as a pathway linking lifecourse SES with CVD outcomes (Hogberg, Cnattingius, Lundholm, Sparen, & Iliadou, 2011; James et al., 2006; Johnson-Lawrence et al., 2013; Pensola, 2003; Pollitt, Rose, & Kaufman, 2005). Race combined with income gradient is a strong predictor in determining housing conditions, neighborhood characteristics, quality of education, purchasing power, social class, and political influence (Dupre, 2008; Jones, 2000; PolicyLink, 2007; Subramanian et al., 2005; Weden et al., 2008).

Sentinel research also suggests that the negative impact of social stressors (i.e., racism) that Blacks have experienced over generations is associated with higher levels of

resting blood pressure (Clark, Anderson, Clark, & Williams, 1999; Harrell, Hall, & Taliaferro, 2003; James et al., 2006; Mays et al., 2007). However, there is limited research that explores the pathway by which the combination of SES and racism impact CVD outcomes.

In order to address the burden of CVD among Blacks, it is important to further investigate the underlying causes. This study examined data from a cohort of more than 5,000 Blacks enrolled in the Jackson Heart Study (JHS). It was specifically designed to explore potential interactions between the levels of SES and levels of racism (i.e., SES-Racism Effect), and how the multiple effects of these interactions moderate the relationship between hypertension and CVD outcomes among a population of Blacks in Jackson, MS. Mississippi has the highest prevalence of CVD in the nation (CDC, 2013a), and overall CVD mortality rates that far exceed the U.S. rates of CVD mortality (Taylor, 2005). In addition, Mississippi has the largest proportion (36%) of Blacks in the United States (Taylor, 2003).

Purpose of the Study

The purpose of this study was to demonstrate whether or not racism contributes to the CVD health disparities observed among Blacks in the Jackson Heart Study (JHS), and to what extent. More specifically, this study investigated how Blacks in the JHS cohort experience racism at different levels of SES mobility, and how the interaction between SES mobility and racism (SES-Racism Effect) influences the extent to which hypertension leads to CVD outcomes observed among participants in the JHS. Quantitative analysis of this secondary dataset was conducted to first determine whether a

relationship between levels of perceived racial discrimination over the lifecourse and SES mobility exists. Using secondary data from the Jackson Heart Study provided a unique opportunity to examine the interaction between social, racial, psychological, and environmental factors in combination with traditional and nontraditional biological data. The methods used in this study emphasize that social influences on health and environmental context are unavoidably linked to individual health risk.

Research Questions and Hypotheses

The research questions in this study examined the relationship between SES mobility and discrimination attributed to race, and if these constructs moderated the relationship between hypertension and CVD. Hypotheses were identified to empirically test each research questions as follows:

Research Question 1

What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of lifetime racial discrimination, as measured by the occurrence of cumulative perceived lifetime discrimination exposure attributed to race?

- Hypothesis 1: Increasing levels of SES mobility are associated with decreasing levels of perceived lifetime discrimination exposure attributed to race after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol

consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

- Null Hypothesis 1: The association between levels of SES mobility and levels of perceived lifetime discrimination exposure was attributed to race after adjusting for identified covariates.

If an association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was identified, the following subhypotheses were also tested (Figure 1):

Hypothesis 1b: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was inversely moderated by age.

Null Hypothesis 1b: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by age.

Hypothesis 1c: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was more strongly moderated by males than females.

Null Hypothesis 1c: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by males than females.

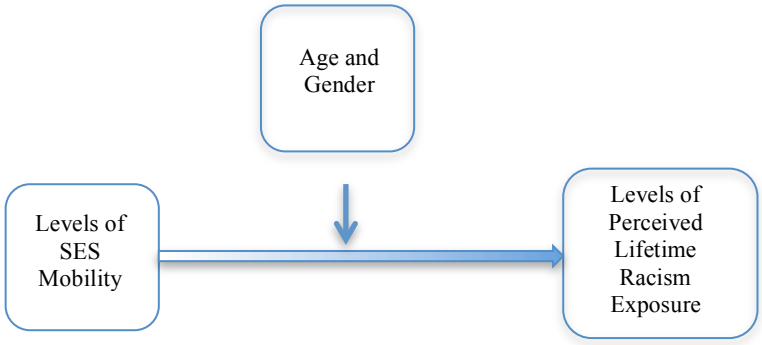

Figure 1. Causal pathway between Levels of SES Mobility and Levels of Perceived Lifetime Racial Discrimination moderated by Age and Gender.

Research Question 2

What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of burden attributed to perceived lifetime racial discrimination, as measured by the extent of life stressfulness, difficulty, and productivity as a result of perceived lifetime discrimination attributed to race?

- Hypothesis 2: Increasing levels of SES mobility are associated with decreasing levels of burden attributed to perceived lifetime racial discrimination after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

- Null Hypothesis 2: There are no statistically significant associations between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination after adjusting for identified covariates.

If an association between levels of SES mobility and levels burden attributed to perceived lifetime racial discrimination was identified, the following subhypotheses were also tested (Figure 2):

- Hypothesis 2b: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was inversely moderated by age.
- Null Hypothesis 2b: There are no statistically significant differences in the association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination when moderated by age.
- Hypothesis 2c: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was higher in males than females.
- Null Hypothesis 2c: The association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination was moderated by gender.

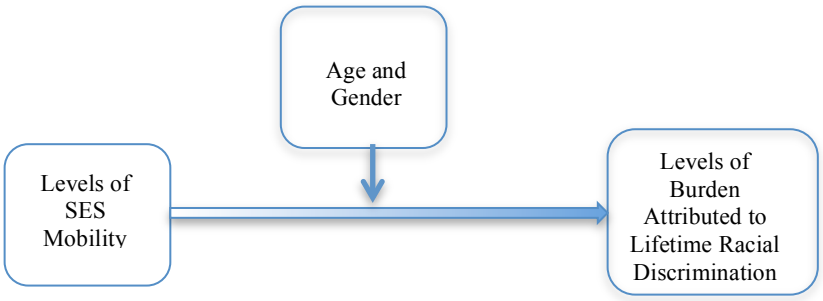

Figure 2. Causal pathway between Levels of SES Mobility and Levels of Burden Attributed to Lifetime Racial Discrimination moderated by Age and Gender.

Research Question 3

Do the levels of SES mobility, perceived lifetime racial discrimination exposure, or burden moderate the relationship between hypertension and cardiovascular disease (Figure 3)?

- Hypothesis 3: The relationship between hypertension and CVD was inversely moderated by increasing levels of SES mobility.
- Null Hypothesis 3: The relationship between hypertension and CVD was not moderated by increasing levels of SES mobility.
- Hypothesis 3b: The relationship between hypertension and CVD was positively moderated by increasing levels of perceived lifetime discrimination attributed to race.
- Null Hypothesis 3b: The relationship between hypertension and CVD was not moderated by increasing levels of perceived lifetime discrimination attributed to race.
- Hypothesis 3c: The relationship between hypertension and CVD was positively moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.

- Null Hypothesis 3c: The relationship between hypertension and CVD was not moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.

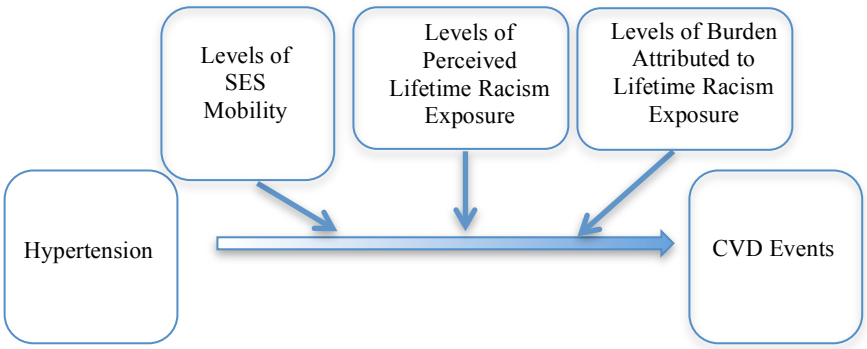

Figure 3. Causal pathway between hypertension and CVD outcomes moderated by levels of the SES mobility, perceived lifetime racial discrimination exposure, or burden.

Research Question 4

If a relationship between levels of SES mobility and levels of perceived lifetime discrimination exposure attributed to race is found (i.e., SES-Racism Effect), does the SES-Racism Effect moderate the relationship between hypertension and cardiovascular disease?

- Hypothesis 4: The relationship between hypertension and CVD was positively moderated by the SES-Racism Effect.
- Null Hypothesis 4: The relationship between hypertension and CVD was not moderated by the SES-Racism Effect.

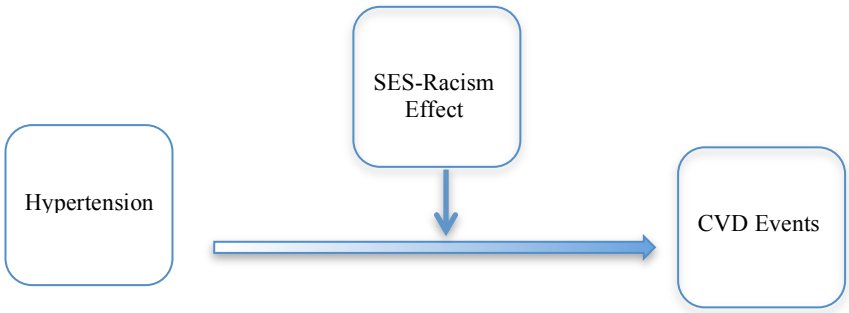

Figure 4. Causal pathway between hypertension and CVD outcomes moderated by the SES-Racism Effect.

Conceptual Framework

The investigation of social determinants (e.g., SES) encourages more in-depth understanding of how and why some individual-level risk factors (e.g., obesity, smoking, physical inactivity) affect some populations in greater proportion. By definition, SES is an influential determinant as it impacts the context of one's surroundings and availability of resources (Johnson-Lawrence et al., 2013). Changes in SES over the lifecourse can either facilitate or inhibit health-promoting practices during critical life periods, such as during childhood or later life (Hardaway & McLoyd, 2008; Johnson-Lawrence et al., 2013; Pollitt et al., 2005). Moreover, macrosocial factors contribute to the establishment of policies, practices, and social norms that have been directly and indirectly associated with adverse health outcomes (Ahmed, Mohammed, & Williams, 2007; Brondolo, Gallo, & Myers, 2009; Thoits, 2010; Wise, Jhally, Young, Rabinovitz, & Media Education Foundation, 2008). For example, racism is hypothesized to be an underlying cause of health disparities because it is associated with the unequal distribution of privileges, resources, and power (Brondolo, Gallo, & Myers, 2009; Jones, 2002). A host of

theoretical frameworks have been proposed to explore the racism-health dynamic (Ahmed et al., 2007; Clark, Anderson, Clark, & Williams, 1999; Myers, 2008; Williams & Mohammed, 2013); however, research that actually demonstrates the processes for how racism and health are related is still in its infancy (Brondolo, Gallo, et al., 2009).

To further examine the relationship between racial discrimination and poor health, this research study was guided by the combination of two theoretical frameworks that illustrate multiple pathways by which social determinants, specifically exposure to racism, lead to adverse health outcomes. Figure 5 examines the interconnected pathway by which macrosocial factors, such as racial discrimination, influence both socioeconomic position and risk factors to determine CVD outcomes inclusive of life course, historical, and geographic context (Harper, Lynch, & Smith, 2011). Figure 6 illustrates racism as a basic cause of health outcomes, which manifest through a pathway of proximal causes over time (Williams & Mohammed, 2013). This framework also acknowledges that there are social inequities occurring at each stage of the process that determine an individual's health response. The first framework implies that socioeconomic position determines the prevalence of risk factors, and therefore extent of CVD in a population; yet, the second framework expands this concept by illustrating the steps by which a macrosocial factor (i.e., racism) influences the multiple risk factors that cause adverse health outcomes over time. The rationale for the selected frameworks and a more in depth examination of these constructs is presented in the next chapter.

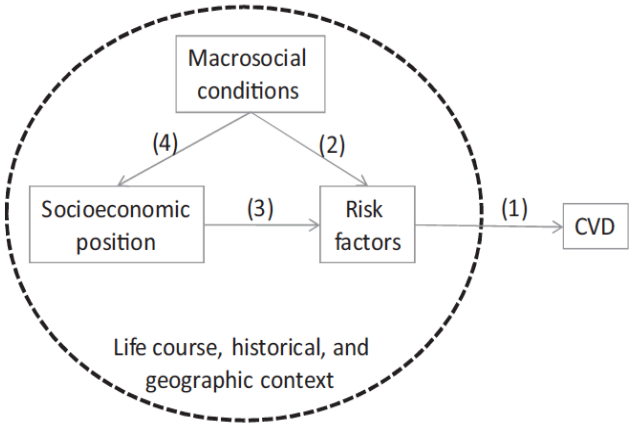

Figure 5. Macrosocial conditions, socioeconomic position, risk factors and CVD risk historical, geographic, and life course context. From "Social Determinants and the Decline of Cardiovascular Diseases: Understanding the Links," by Harper et al., 2011, *Annual Review of Public Health*, 32(1), p. 40. Reprinted with permission requested from *Annual Reviews of Public Health*. The model implies that the prevalence of risk factors establishes the level of CVD in a population (arrow 1), these risk factors are influenced by both the extent of macrosocial factors (arrow 2) and socioeconomic position (arrow 3), socioeconomic position is determined by macrosocial conditions (arrow 4), and all of these constructs are dynamically connected and embedded in multiple environments.

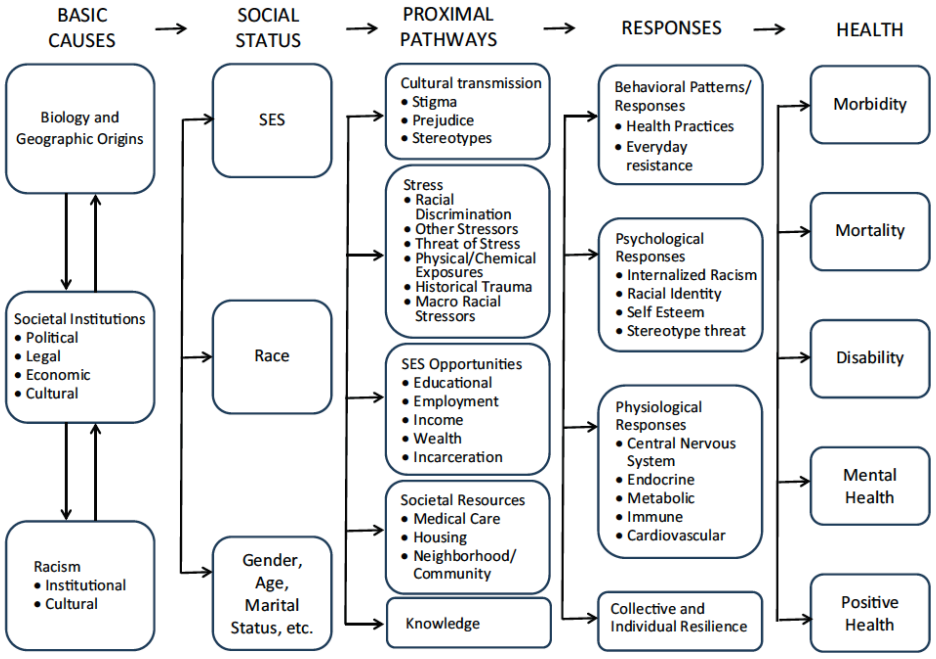

Figure 6. A framework for the study of racism and health. From "Racism and Health I:

Pathways and Scientific Evidence," by Williams and Mohammed, 2013, *American Behavioral Scientist*, 57(8), p. 1157. Reprinted with permission from Sage Journals.

Nature of the Study

This research study was based on a cross-sectional analysis of data extracted from the Exam 1 period (2004) of the Jackson Heart Study (JHS) to examine the causal pathway by which racial discrimination impacts CVD. The JHS has collected data on constructs used to measure racial discrimination in the domains of everyday experiences, major life events (lifetime), burden of discrimination, and the effect of skin color (Payne et al., 2005; Sims et al., 2012). SES mobility was calculated using variables to define the change in SES from childhood to adulthood. To understand this pathway, the presence of a SES-Racism Effect was examined by understanding the relationship between SES mobility and perceived lifetime racism; examining how levels of SES mobility and levels of perceived lifetime racism independently affect the association between hypertension and CVD; and understanding whether the SES-Racism Effect (e.g., low, high) modified the association between hypertension and CVD outcomes.

Access to JHS data was granted based on a research proposal that I submitted and which was approved by JHS faculty. The JHS research proposal described the study and the variables needed for analysis, from which a study specific dataset was created.

Operational Definitions of Key Terms

The key terms used throughout this study were based on variables derived from JHS data collection forms. Some variables are calculated variables that were defined by

JHS researchers, while others were recoded for the purpose of this research based on cutpoints identified in the literature.

Body mass index (BMI): A calculated measure using an individual's height and weight to assess overweight/obesity status and health risk (CDC, 2011a).

Burden of lifetime racial discrimination: A calculated variable based on three JHS measures of stressfulness due to discrimination attributed to race (Sims, et al., 2012).

Cardiovascular Disease: A term used to represent a wide range of conditions categorized by the International Classification of Diseases, Tenth Revision [ICD-10] codes, including heart attack, stroke, and coronary heart disease (CHD; Go et al., 2012).

Cumulative discrimination: A calculated measure that combines the frequency of discrimination exposure and burden to determine its overall impact.

Exposure to lifetime racial discrimination: A factor measured by JHS as a composite of exposure to discrimination attributed to race occurring across different domains throughout an individual's lifetime (Sims et al., 2012).

Health disparities: Described as both inequality and inequity in access to, utilization of, and quality of care and/or services, as well as the environments, that affect the health status or health outcomes of individuals and populations (Carter-Pokras & Baquet, 2002). CDC (2014) defines health disparities as a health differences among populations groups that experience greater systematic social and economic disadvantages as a result of historical and discriminatory barriers.

Hypertension (HTN): This study used JHS' definition of a systolic blood pressure of 140mmHg or greater, and diastolic blood pressure was 90mmHg or greater (Sims et al,

2012). JHS participants taking antihypertensive medications were also identified as hypertensive.

John Henryism: An individual with a strong behavioral tendency or drive to meet environmental or occupational demands through hard work and determination as a strategy for coping with difficult social and economic stress (Payne et al., 2005; Subramanyam et al., 2013).

Parental education attainment: Assessed in the JHS as a self-reported measurement of the highest level of school completed by each parent (Parental Socioeconomic Status Form, 2001).

Perceived racial discrimination: The perception that certain racial/ethnic populations experience differential or negative attitudes, judgment, or unfair treatment compared to a other racial/ethnic groups (Clark et al., 1999; Pascoe & Smart Richman, 2009; Williams, Neighbors, & Jackson, 2003).

Physical Activity: In the context of this study, measured as any physical movement (e.g., walking, biking, gardening, dancing) performed on most days during a week over an extended period of time (NIH, 2011).

Racism: A systematic or institutional belief that members of a certain racial/ethnic population have abilities, characteristics, or qualities that are inferior to other racial groups, which may be used to oppress or maintain power over that population (Hoyt, 2012; Jones, 2000).

Risk Factors: Individual characteristics (such as age, gender, and race) or the behaviors (poor dietary practices, tobacco use, and physical inactivity) that may contribute to adverse health outcomes (CDC, 2007).

Socioeconomic status: A demographic variable that is a composite of social (i.e., education), economic (i.e., income), and work status (i.e., employment) indicators; indicators which are independent of one another, but often related (CDC, 2014).

SES mobility: Changes in the upward or downward trajectory of an individual's socioeconomic status measured between childhood and adulthood (Pollitt et al., 2005).

Social determinants of health: The social, economic, political conditions that shape an individual's health, as well as the systems available to prevent and manage health outcomes (CDC, 2014; Wilcox, 2007).

Assumptions

As in any research study, certain assumptions about the population, the data, or other aspects of the study are essential. I assumed that JHS participants are comfortable self-reporting data related to sensitive topics, such as racial discrimination, without bias. The JHS is the largest single site study focusing on the CVD outcomes and associated risk factors among Blacks (Taylor, 2003, 2005a). Moreover, the JHS was methodologically modeled after the larger multisite Atherosclerosis Risk in Communities (ARIC) study, and roughly one-third of its original cohort has participated in the JHS (Wyatt, Diekelmann, et al., 2003). The ARIC study, initiated in 1987, was instrumental in providing extensive data that observed CVD differences between Whites and Blacks regarding physical, behavioral, and environmental indicators. The large number of Blacks

that participated in the ARIC, including the entirely Black Jackson, MS cohort, served as a springboard for continued study of CVD outcomes. Therefore, the JHS is a uniquely stable population from which health data has been gathered for an extended time period.

For the purposes of this study, I assumed that all individuals living in Jackson, MS have comparable contextual beliefs, as well as generational and historical context, about how discrimination attributed to race is defined and perceived. Discrimination attributed to race was presumed to be synonymous with racism. Because all participants are confined to a single geographic area, I assumed that all participants have had the same opportunities over their lifecourse to be exposed to racial discrimination. I also assumed that the change in SES from childhood to adulthood is an accurate measure of SES mobility; and there are no significant fluctuations in between these two measurement periods, particularly given that only measure of SES can be captured from each time period.

Limitations and Delimitations

As with any research study, there are limitations in the strength of the study and its findings. First, this study is based on the analysis of secondary data; therefore, the use of fixed survey questions limits the specificity of the data. The data ascertained are related to discrimination attributed to race, and do not entirely encompass the definition of racism. Secondly, a cause-effect relationship cannot be demonstrated using this correlational study to assess how the relationship between hypertension and CVD over the lifecourse is moderated by racial discrimination and SES. Finally, the study measures only Blacks located in the metro Jackson, MS area, and any findings are not generalizable

to geographic areas or other racial/ethnic populations that may experience racial discrimination. These limitations provide justification for further research to be conducted in a wider population, additional geographic locations, and using more specific methodologies.

Significance of the Study

The central goal of public health is the prevention of disease and improvement of overall health. Some of the milestones by which public health success was initially measured included the institution of sanitation services, the identification of penicillin, and the development of vaccines to eliminate many common infectious diseases (CDC, 2013b). In more recent years, key public health improvements have also included policies to reduce tobacco exposure and the reduction of heart disease and stroke deaths (CDC, 2013b). While all of these improvements have culminated in longer life expectancies and improved quality of life, macrosocial factors (e.g., SES mobility, racial discrimination) have historically diminished the potential for optimal health outcomes among Blacks. For example, the consequence of poor SES mobility among Blacks has been extensive multilevel deprivation that inhibits ability to adequately practice healthy behaviors. In addition, the social trauma of racial discrimination that Blacks experience over their lifecourse is multilevel deprivation in and of itself.

This study offers an opportunity to increase awareness concerning the long-term effects of perceived racial discrimination, even in subtle forms. Improving understanding of how lifetime racial discrimination may be directly or indirectly related to adverse CVD outcomes can serve as a platform to diminish or alleviate the environmental and social

injustices experienced by Blacks in Jackson, MS. While racial discrimination is often based on the vantage point of the individual(s) often discrimination against, improvements in health disparities will depend upon all individuals being amendable to changing social norms. Creating equity regarding macrosocial issues may be a catalyst for eliminating CVD health disparities.

Conclusions

CVD is a major cause of morbidity and mortality among Blacks. Furthermore, the rates of hypertension in Mississippi have consistently been among the highest in the nation. This study explored how Blacks perceived their exposure to racial discrimination and the burden it has on them over their lifecourse, whether exposure and burden differ based on levels of SES mobility, and the association that both have the rates of hypertension and CVD outcomes among Blacks in Jackson, MS. In Chapter 2, the impact of social and economic well-being over the lifecourse (i.e., SES mobility) and racial discrimination on health outcomes was examined; thereby, identifying research gaps in understanding Black-White differences in CVD health outcomes and evidence to support continued investigation. Findings from this study provide further understanding to how racial discrimination contributes to poor health outcomes, and provide evidence for needed changes in policies, practices, infrastructure, and/or social norms in order to improve the racial disparities that exist for CVD and other diseases.

Chapter 2: Literature Review

Introduction

Blacks experience hypertension at higher rates than any other racial/ethnic population (Go et al., 2014; Howard et al., 2011; Mozaffarian et al., 2015; Quinones et al., 2012). Several studies have been conducted to investigate the role of traditional risk factors on hypertension prevalence among Blacks, such as:

- SES (Allen, McNeely, Waldstein, Evans, & Zonderman, 2014; Conroy, Sandel, & Zuckerman, 2010; Phelan, Link, & Tehranifar, 2010; Quinones et al., 2012),
- level of education (Non, Gravlee, & Mulligan, 2012; Subramanyam et al., 2013), and
- physical activity patterns (Bell, Lutsey, Windham, & Folsom, 2013; Bostean et al., 2013; Howard et al., 2011; Sallis, Floyd, Rodriguez, & Saelens, 2012) .

Although these risk factors generally support a positive association with hypertension among Blacks when compared to White populations, the study findings have often failed to produce consistent, straightforward results that explain why health disparities exist between the two groups. Researchers have begun to speculate that socially-mediated factors, such as racism, may be the root cause of health disparities (Gee & Ford, 2011; Gee et al., 2012; Sims et al., 2012; Williams & Mohammed, 2013; Williams & Sternthal, 2010).

This chapter presents a review of the literature focusing on the potential connections between race, racism, socioeconomic status mobility (SES mobility), and hypertension by examining these variables, as well as the larger issues related to health

disparities between races, what is known about cardiovascular disease, and how the Jackson Heart Study was used as a basis for this research. Prior knowledge of seminal research in the field of social determinants of health and health disparities provided a list of keywords that were used to initiate the research found within this chapter. Synonyms and alternative terms were also used to thoroughly assess the literature. Electronic databases, including ProQuest, PubMed, Google Scholar, and EBSCO Host, were used to identify published research based on the following terms or phases: *cardiovascular disease (CVD) among Blacks/African Americans*, *differences in hypertension prevalence between Blacks and Whites*, *CVD risk factors and health disparities*, *social determinants of health and CVD*, *SES and hypertension*, *racism and hypertension*, *racism and cardiovascular disease*, *racism and health disparities*. *SES mobility and health*. *SES mobility and CVD*, *SES mobility and racism*, *racism as a chronic stressor on health*, and the *Jackson Heart Study*.

The preliminary literature review was limited to peer review journal articles, books, internet-based resources, and presentations published between 2010 and 2016. However, publications from earlier time periods that were regularly found in the reference list were also reviewed for inclusion as historical context to justify the research. The search outcomes were carefully evaluated for incorporation into the literature review based on their relevance to the research project, ability to support the importance of the research, or identify gaps for continued investigation.

This literature review presents relevant contextual information to guide a study on the relationship between levels of SES mobility and levels of perceived lifetime racism,

and whether or not the interaction of these two constructs impact the relationship between hypertension and CVD. In this chapter, I provide an overview of research on the burden of cardiovascular disease in the United States, the Black-White differences in CVD health outcomes and risk factors, and the rationale for the inclusion of social constructs (i.e., racism and SES mobility) as contributing to health disparities. A review of prior research was conducted to assess the multiple factors that influence racial disparities in CVD outcomes, as well as justification to support racial discrimination and SES mobility as having an important role in the health outcomes of Blacks.

This section includes a discussion of how racism is defined and provides evidence for the presence and magnitude of racial discrimination across multiple domains. Research also reflects the relevance of SES mobility as a function of CVD risk, and how the trajectory of SES measures may be correlated to sociocultural norms (e.g., racism). This chapter concludes with a summary of studies that have been conducted using data from the Jackson Heart Study to investigate the prevalence of racism, and its impact on hypertension, among Blacks in the Jackson metro area of Mississippi.

Overview of CVD Burden in the United States

CVD is a term used to represent a wide range of conditions categorized by the International Classification of Diseases, Tenth Revision [ICD-10] codes. For the purposes of this research, CVD was defined primarily as heart attack, stroke, and coronary heart disease (CHD). With the exception of the influenza pandemic of 1918, CVD has consistently prevailed as the leading cause of death since 1900 (Go et al., 2014; Mozaffarian et al., 2015; Roger et al., 2012). CVD has had consistently high prevalence

in the United States; almost one in three Americans will be diagnosed with cardiovascular disease in their lifetime (Go et al., 2014; Mozaffarian et al., 2015; Roger et al., 2012). Despite the high prevalence, since 1950, CVage-adjusted mortality rates in the United States have declined approximately 60%, a public health accomplishment acclaimed as one of the most notable of the 20th century (Centers for Disease Control and Prevention (CDC), 1999; Kramer, Valderrama, & Casper, 2015). According to the most recent estimates, the 2013 overall death rate from CVD was 222.9 per 100,000, which is a decline of 28.8% since 2003 (Mozaffarian et al., 2015).

Despite these gains, CVD continues to rank as the leading cause of death in the United States. In 2013, more than 2,200 deaths per day were attributed to CVD in the United States alone, approximately one death every 40 seconds (Mozaffarian et al., 2015). However, this estimate varies significantly by demographic factors. The 2013 mortality data documented more CVD deaths among males (269.8 per 100,000) compared to females (184.8 per 100,000) for the first time since 1983 (Mozaffarian et al., 2015). Furthermore, an estimated 43.9% of all Americans will have at least one type of CVD by 2030 (Mozaffarian et al., 2015), which indicates the possibility that the prevalence of CVD is rising. This is due largely to lifestyle factors such as poor nutrition and inadequate physical activity which increases individuals' risk at younger ages (Go et al., 2014; Mozaffarian et al., 2015).

Although CVD may affect individuals of all age, racial/ethnic, sociodemographic, and geographic populations, researchers agree that age is the most influential predictor of CVD risk (Kramer et al., 2015; Quinones et al., 2012; Roger et al., 2011). A cross-section

of the 2007-2010 NHANES population indicated that the prevalence of CVD increased exponentially with age. Adults between the ages of 20-39 had prevalence rates below 15%, with more than a two-fold higher prevalence (nearly 40%) observed for adults between the ages of 40-59 (Go et al., 2012). Go et al. (2012) further documented that this pattern continues for adults 60-79, and at least 80 years of age (more than 70% and 80%, respectively).

Data gathered by the National Heart Lung and Blood Institute (NHLBI) from 1980 to 2003 also shows an increasing CVD incidence and prevalence across the life course. NHLBI documented that men experience their first cardiovascular event at an average rate of 3 per 1000 among men aged 35 to 44, which escalates to a rate of 74 per 1000 among men aged 85 to 94 (Go et al., 2014). Whereas the first cardiovascular event for women typically occurs 7-10 years later than males of comparable age groups (Go et al., 2014; Maas et al., 2011), the CVD incidence rates of males and females are more similar in later life (Go et al., 2014; Kramer et al., 2015; Maas et al., 2011). However, Maas et al. (2011) argued that although men and women share similar risk factors, there are gender-specific differences in the attention given to these risk factors that may attribute to an underestimate of CVD rates in women.

A comparison of NHANES data (1988-1994 vs. 1999-2004) showed that the prevalence of CVD among women aged 35-54 has increased as men of similar age have decreased (Maas et al., 2011). The correlation between age and risk of CVD is particularly concerning given that *Baby Boomers* (adults born between 1946 and 1964) are reaching the age of high risk and comprise roughly one-fourth of the U.S. population

(King, Matheson, Chirina, Shankar, & Broman-Fulks, 2013). In an analysis comparing NHANES data from 1988-1994 to 2007 and 2010 (i.e., previous generation to baby boomers, respectively), King et al. (2013) found the Baby Boomer generation to be less healthy than the previous generation, largely due to increased rates of hypertension, diabetes, obesity, and elevated cholesterol levels. This large segment of the U.S. population may strain the health care system and its resources as they access care for a host of conditions, including CVD (King et al., 2013). In addition, the baby boomer generation itself is likely to have disparities in CVD outcomes when the group's racial differences are explored similar to other population subgroups.

Although attention has been paid to the correlation between age and CVD, less exploration has been conducted into the disproportionate rate of CVD burden repeatedly documented among specific racial/ethnic populations (e.g., Blacks); this difference warrants immediate attention (Go et al., 2012; Jolly, Vittinghoff, Chattopadhyay, & Bibbins-Domingo, 2010). The disparity between races has implications for both prevention and treatment of CVD, but first, the potential sources of the differences must be explored.

CVD Differences Between Blacks and Whites

Since the mid-1980s, efforts have been made to address the sizeable gaps and persistent inequalities in health status and life expectancy that exist between Whites and other racial/ethnic populations in the United States (Centers for Disease Control and Prevention (CDC), 2012; Kochanek et al., 2013; Kramer et al., 2015; Safford et al, 2012). A 1986 study released by the U.S. Department of Health and Human Services noted that

CVD accounted for 24% of excess deaths among Black males and 41% among females; these rates were 30% and 18% higher than their White counterparts, respectively (Wyatt, Williams, et al., 2003). In 2010, over two decades later, more than one-fourth of the racial gap in life expectancy due to CVD mortality remained (Kramer et al., 2015).

Despite the substantial decline in CVD mortality rates during the last several decades in the overall population and national efforts to improve racial/ethnic health disparities, significant disparities in CVD mortality rates between Blacks and Whites remain (Bostean et al., 2013; Kramer et al., 2015; Safford et al., 2012). In 1950, the difference in the age-adjusted death rate between Blacks and Whites was 1.9 (586.7 compared to 584.8, respectively; Williams & Jackson, 2005). The stark black-white gap of 71.4 (324.8 compared to 253.4, respectively) has decreased since 2000 (Williams & Jackson, 2005) to 23.6 in 2009 (141.3 compared to 117.7, respectively; Centers for Disease Control and Prevention, 2013). While prevention efforts are reaching their intended target audiences, the magnitude of the reach continues to not uniformly be observed.

Researchers agree that racial disparities are better illustrated by race-sex differences (Go et al., 2012, 2014; Kochanek et al., 2013; Kramer et al., 2015; Mozaffarian et al., 2015). The National Health and Nutrition Education Survey (NHANES) estimated 2010 prevalence rates for CVD at 36.6% and 32.4% for White males and females, respectively, compared to 44.4% and 48.9% for their Black counterparts (Go et al., 2012). In 2013, the overall mortality rates from CVD for White males were reported at 270.6 per 100,000, but 356.7 per 100,000 among Black males

(Mozaffarian et al., 2015). Although CVD death rates are lower among women, a wide gap still exists between races. In 2013, White females had an annual CVD death rate of 183.8 per 100,000 persons compared to Black females at 246.6 (Mozaffarian et al., 2015). The disparities Blacks experience are displayed not only as higher CVD mortality rates, but also as higher incidence of first cardiovascular event occurring at younger ages.

While CVD mortality rates are higher among Blacks than any other population group, some have suggested that the age of disease onset creates black-white differences in CVD prevalence and mortality that are even more alarming (Jolly et al., 2010; Kramer et al., 2015). In a cross-sectional study of NHANES survey data between 1996 and 2006, Jolly et al. (2010) observed significant differences between Blacks and Whites when prevalence ratios are stratified by age group. Blacks under the age of 44 were twice as likely to have a cardiovascular event compared to their White counterparts, with differences gradually diminishing as age increased (Jolly et al., 2010). Jolly et al. (2010) found similar patterns for black-white differences for all cardiovascular-related disease conditions (e.g., heart failure, stroke, myocardial infarction)., Jolly et al. (2010) posited that differences in prevalence ratios by age group remained even after controlling for CVD risk factors, comorbidities, socioeconomic factors, and access to health care.

Kramer et al. (2015) observed similar differences in age-specific heart disease mortality rates from 2008-2010 comparing race-sex groups across the lifestage. Black males and females aged 35-39 were more than twice as likely to experience premature death due to heart disease than their White counterparts (50.3 and 24.5 per 100,000 vs. 22.5 and 9.5 per 100,000, respectively), a disportionate trend was found to continue

across the lifestage until 65 to 69 years of age (Kramer et al., 2015). In fact, blacks males are reported to have the lowest life expectancy and highest rates of mortality when compared to other race-sex groups across the United States (Chae, Lincoln, Adler, & Syme, 2010; Kochanek et al., 2013; Kramer et al., 2015). Hence, there are possibly environmental factors (e.g. community-level inequities) or social norms (e.g. racism) that strongly influence the observed differences between these racial groups.

Researchers have also investigated differences in the mortality rates of specific CVD-related conditions (i.e., coronary heart disease (CHD), stroke, heart attack). In 2011, the stroke death rate among Black males and females (55.3 and 47.0 per 100,000, respectively) far exceed the overall rate of 37.9 per 100,000 (Mozaffarian et al., 2015). The excess burden of death from stroke has been observed in Blacks 45 to 74 years of age, indicating a relative risk 47% greater than that of Whites at comparable ages (Centers for Disease Control and Prevention, 2013). Ford (2011) investigated trends in CVD mortality rates among people with and without hypertension. Data from NHANES I (1971-1992) and NHANES III (1988-2006) indicated continuing disparities between Blacks and Whites (Ford, 2011). Data show that the CVD mortality rates among Blacks exceeded Whites in both cohorts among individuals with (NHANES I: 13.3 versus 9.3 per 1000 person-years; NHANES III: 8.1 versus 6.2 per 1000 person-years) and without hypertension (NHANES I: 7.2 versus 5.5 per 1000 person-years; NHANES III: 4.5 versus 3.0 per 1000 person-years; (Ford, 2011). The mortality rate over the course of the two cohort periods reduced by 3.1 per 1000 among Whites, and 5.2 per 1000 among Blacks; therefore, the mortality gap between the population groups was estimated to have

shrunk from 4.0 per 1000 person-years to 1.9 per 1000 person-years (Ford, 2011). Despite this shrinking gap, CVD remains a major public health concern among Blacks for numerous reasons. Although Blacks (excluding recent immigrants) comprise substantially less of the population compared to Whites (13.1% vs. 77.9%, respectively; U.S. Census Bureau, 2014), they experience disproportionately higher rates of chronic disease risk factors, inadequate access to health care and resources, and have a life expectancy approximately three years less than their White counterparts (Mozaffarian et al., 2015; Office of Minority Health & Health Equity (OMHHE), 2014).

Risk Factors Contributing to CVD Prevalence Differences by Race

CVD is a complex disease with multiple risk factors, both traditional and nontraditional. Nontraditional risk factors often reflect social phenomena, which are outside of an individual's control. Researchers agree that traditional risk factors are commonly observed as contributors to elevated CVD risk include obesity, physical inactivity, family history, hypertension, dyslipidemia, and diabetes (Bauer, Briss, Goodman, & Bowman, 2014; Centers for Disease Control and Prevention, 2013; Djoussé et al., 2015; Mozaffarian et al., 2015; Sallis et al., 2012). The racial differences in the prevalence of these risk factors have been widely explored, and research suggests that some risk factors occur more frequently among Black populations (Djoussé et al., 2015; Holmes, Hossain, Ward, & Opara, 2013; Quinones et al., 2012; Thacker et al., 2014).

Obesity and physical inactivity. Multiple studies have been conducted to assess racial differences in CVD risk factors, such as obesity and physical inactivity. Overall,

Blacks are more likely to experience a higher prevalence of obesity and physical inactivity than their White counterparts.

Ogden, Carroll, Kit, & Flegal (2014) reported NHANES prevalence estimates for 2011-2012 indicating that Blacks were more likely to be obese (47.8%) compared to Whites (32.6%). Researchers generally agree that Black women have a higher rates of corpulence than their male counterparts (Flegal, 2012; National Center for Health Statistics (NCHS), 2012; Ogden et al., 2014, 2014; Romero, Romero, Shlay, Ogden, & Dabelea, 2012; Schiller, Lucas, Ward, & Peregoy, 2012). Data reported from NHANES 2011-2012 show age-adjusted obesity prevalence estimates at 29.2% for Black women and 15.9% for Black men, compared to 15.3% for White women and 11.2% for White men. Fakhouri, Ogden, Carroll, Kit, & Flegal (2012) found the highest rates of obesity occur among Black women aged 65 and older. Data analyzed from the NHANES, 2007-2010, indicated that 53.9% (aged 65-74) and 49.4% (aged 75 and older) of Black women were obese, compared to 38.9% and 27.5% of White women, respectively; however, obesity rates among men were lower and no significant differences were observed between racial groups. Obesity rates by race and sex seem to follow similar trends as the two risk factors are often strongly correlated (Burke & Heiland, 2011; National Center for Health Statistics (NCHS), 2012; Schiller et al., 2012). The elevated obesity rates observed among Blacks are proposed to account for at least 30% of the black-white gap in life expectancy (Krueger & Reither, 2015); however, data from the Southern Community Cohort Study (2002-2009) found BMI >40 to be more strongly associated with excess CVD mortality among White males and females, HR=2.10, 95% CI [1.15,

3.83] and HR=2.62, 95% CI [1.41, 4.87], respectively, than their Black counterparts, HR=1.40, 95% CI [0.92, 2.14] and HR=1.17, 95% CI [0.78, 1.75] (Cohen et al., 2012). This suggests that the impact of the Black-White difference in obesity observed across studies is inconsistent; therefore, other factors (e.g. age, education, SES, community-level factors, social norms, and public policies) may need to be considered in conjunction with obesity to understand its impact on CVD health disparities.

Additionally, researchers contend that the higher overall prevalence of obesity among Blacks may be linked to environmental and social factors more than level of education or income (Bower et al., 2015; Kirby, Liang, Chen, & Wang, 2012; Thorpe, Bell, et al., 2015; Thorpe, Kelley, et al., 2015). Specifically, residential environment has been found to be positively correlated with obesity risk. Bower et. al (2015) found Black women to have a 1.06 times greater risk of obesity for every one-point increase in the degree to which Blacks are isolated from Whites. While disparities in obesity rates between Black and White men are not consistently exhibited (Burke & Heiland, 2011), Thorpe et al. (2015) demonstrated that environmental and social residential conditions may contribute to the differences that do occur. Data from the 2003 National Health Interview Survey found Black men to have 1.29 greater odds of obesity compared to White men; whereas the Exploring Health Disparities in Integrated Communities Study, a cross-sectional study of Black and White adults of comparable median incomes living in contiguous census tracks in Southwest Baltimore, Maryland, illustrated similar risk (OR=1.06; Thorpe, et al., 2015). Hence, chronic disease risk factors, such as obesity, should not be solely address on an individual level.

Physical activity is customarily measured only as leisure time activity. Few studies have measured the differences in physical activity by race/ethnicity, and consistently reported Blacks to be less active than Whites (Bell et al., 2013; Buchowski et al., 2010; Burke & Heiland, 2011; Marquez, Neighbors, & Bustamante, 2010; Sallis et al., 2012; Schiller et al., 2012; Wang & Chen, 2011; Wilson-Frederick et al., 2014). In 2010, data from NHIS showed that 30.8% of Whites were physically inactive compared to 41.3% of Blacks, based on the Federal 2008 Physical Activity Guidelines for Americans (Schiller et al., 2012). In the Continuing Survey of Food Intake by Individuals (CSFII) conducted from 1994 to 1996, controlling for education and income had minimal impact on the differences in participation in physical active among Blacks adults (20 and older) compared to Whites, OR=0.69, 95% CI [0.49, 0.96] vs. OR=0.63, 95% CI [0.45, 0.89] (Wang & Chen, 2011). Moreover, Bell et al. (2013) compared of Blacks and Whites in the Atherosclerosis Risk in Communities Study and found that although physical activity patterns between Blacks and Whites were similar to other studies, physical activity and CVD incidence were inversely related in both racial groups, after adjusting for potential confounders (such as age, sex, education, smoking status, alcohol usage, etc.).

The racial differences in physical activity are further separated by gender and age groups. Black women engage in work-related and leisure time physical activity less frequently than White women (Burke & Heiland, 2011; Schiller et al., 2012). Schiller et al. (2012) found that among adult women (aged 18 and older) who participated in the 2010 NHIS, Black women were more physically inactive than White women at 46.8%

and 30.9%, respectively, OR=2.15, 95% CI [2.06, 2.24]. However, Buchowski et al.(2010) found that White women who were likely to experience moderate to severe obesity as sedentary behavior increased. White women in the highest quartiles of sedentary behavior (>12 hrs/day) had a OR=4.03, 95% CI [3.08, 5.28] of severe obesity compared to OR=1.56, 95% CI [1.35, 1.81] among Black women in the same sedentary behavior quartile (Buchowski et al., 2010).

The gap in physical inactivity among racial groups has been noted to increase as the population increases in age. Data analyzed from NHANES III, 1988-1994, indicated that 48.4% of Black women 65-84 were physically inactive, compared to 30.8% of White women, OR=2.62, 95% CI [1.82, 3.76], after adjusting for age and education; physical inactivity rates among men were much lower at 27.7% and 17.7%, respectively, OR=1.88, 95% CI [1.19, 2.97] (Sundquist, Winkleby, & Pudaric, 2001). It is important to acknowledge, however, that physical activity among Blacks may be underreported due to how it is generally measured.

Although many studies measure physical activity based on leisure activities, Sallis et al. (2012) stated that there are actually four categories of physical activity: leisure/recreational/exercise, occupation/school, transportation, and household. Hence, Blacks may actually be more physically active than routinely documented when considering the other less frequently measured categories (He & Baker, 2005; Kurian & Cardarelli, 2007; Marquez et al., 2010; Sallis et al., 2012). Although Blacks are more likely to never engage in leisure-time physical activity compared to their White counterparts, Black men and women were more likely to have jobs that require strenuous

activities at least most of the time (34.1% and 31.1%, respectively) compared to Whites (30.7% and 21.8%, respectively; (He & Baker, 2005). Interestingly, leisure-time physical activity among Whites decreased as education decreased, and work-related physical activity among Blacks decreased as education increased (He & Baker, 2005). Several other studies support the finding that Blacks are more likely to engage in occupational physical activity (Centers for Disease Control and Prevention (CDC), 2011; Marquez et al., 2010; Marshall et al., 2007), and leisure physical inactivity is strongly correlated to social class (Marshall et al., 2007). In fact, neighborhoods with high racial/ethnic (95%) and low-SES populations (≤5% without college education) are 46% less likely to have at least one park or recreational facility (Sallis et al., 2012). Although few studies have investigated the relationship between physical activity and CVD risk in Blacks (Shiroma & Lee, 2010), the link between physical activity and obesity is well established. Diet also has significant consequences for health, and cholesterol is a key metric for CVD risk.

Cholesterol. Individuals with increased prevalence of obesity and physical inactivity are also more likely to be at increased risk for other CVD risk factors (Abell et al., 2008). Interestingly, there appears to be no significant difference in the prevalence of elevated total cholesterol between Blacks and Whites (Hurley, Dickinson, Estacio, Steiner, & Havranek, 2010). In fact, researchers posit that Blacks appear to have lower age-adjusted prevalence of elevated cholesterol than Whites (Fryar, Hirsch, Eberhardt, Yoon, & Wright, 2010) despite greater consumption of high fat, high cholesterol foods (Williams, 2009). Furthermore, Fryar et al. (2010) found that Whites exhibit higher age-adjusted prevalence of a diagnosed and undiagnosed comorbid combination of elevated

cholesterol and hypertension compared to Blacks (9.3% vs. 8.9%, respectively). Researchers argue that Blacks historically have had physically active occupations that protected their cholesterol levels and CVD risk; however, this finding is likely waning as adults aged 35-44 report a 56% prevalence of sedentary occupations lifestyles (Harman et al., 2011).

Diabetes. The presence of comorbid conditions, such as diabetes only serves to exacerbate the prevalence of CVD morbidity and mortality in the affected population. In 2010, the Centers for Disease Control and Prevention (2014) reported that adults (aged ≥ 20 years of age) were 1.8 and 1.5 times more likely to be hospitalized for a heart attack and stroke, respectively; hence, diabetes may be considered to be potentially the most influential risk factor to CVD. Individuals with diabetes are consistently reported to be between two to four times more likely to experience a cardiovascular event (e.g., heart attack or stroke) (American Heart Assoication, 2012; World Heart Federation, 2013). Furthermore, approximately 65% of people with diabetes die as a result of heart disease or a stroke (American Heart Assoication, 2012). The overall prevalence of diabetes has accelerated rapidly in the last two decades; however, racial/ethnic and socioeconomically disadvantaged populations are affected more substantially than their White counterparts (Centers for Disease Control and Prevention, 2014). Data from multiple sources clearly reflects that Blacks have diabetes rates significantly higher than Whites (Centers for Disease Control and Prevention, 2013; Chatterjee et al., 2013; Fryar et al., 2010; Gaskin et al., 2013). The Centers for Disease Control and Prevention (CDC) (2011) indicated that the age-adjusted prevalence of Blacks diagnosed with diabetes rose from 4.5% to

9.0%, compared 2.6% to 5.8% among Whites, from 1980 to 2009. Fryar et al. (2010) suggests that the risk factors commonly found among individuals at greater risk for CVD, such as obesity, hypertension, and dyslipidemia, are also more common among Blacks than Whites with diabetes. In addition, researchers posit that elevated diabetes prevalence among Blacks is often patterned by socioeconomic factors (Gaskin et al., 2013; Sims et al., 2011). Sims et al. (2011) found that low-incomes Blacks have greater risk of diabetes than high-income Blacks, RP=1.94, 95% CI [1.28, 2.92] and RP=1.35, 95% CI [1.04, 1.74], respectively). The increased health risk associated with lower SES is not isolated to Blacks alone, as Gaskin et al. (2013) found that the odds of having diabetes was similar for Blacks and Whites who experience the disadvantage of living in impoverished neighborhoods. However, poverty in Black communities is more prevalent (Gaskin et al., 2013); thereby promoting negative CVD-related health outcomes and further magnifying the diabetes-CVD mortality association. Another risk factor that has been documented to have significantly disproportionate prevalence rates in Blacks is hypertension.

Hypertension. Hypertension has been considered the most important CVD risk factor (Centers for Disease Control and Prevention, 2013; Cuffee, Hargraves, & Allison, 2012; Gillespie & Hurvitz, 2013; Holmes et al., 2013; Mozaffarian et al., 2015; Yoon et al., 2015), and at 44.9% and 46.1% for males and females, respectively, Blacks in the United States have the highest rates of hypertension in the world (Cuffee et al., 2012; Dolezsar, McGrath, Herzig, & Miller, 2014; Fuchs, 2011; Hicken, Lee, Morenoff, House, & Williams, 2014; Holmes et al., 2013; Mozaffarian et al., 2015; Quinones et al., 2012). Hypertension is more likely to be undiagnosed or uncontrolled among Blacks, and

therefore is associated with a significantly higher CVD mortality rates . Watson (2008) estimated that hypertension among Blacks may be correlated with CVD mortality rates that are three to five times greater than Whites.

Go (2014) noted that among Blacks, hypertension contributes to higher rates of nonfatal strokes, fatal strokes, heart disease mortality, and end-stage renal disease (OR= 1.3, 1.8, 1.5, and 4.2, respectively) compared to the general population. Comparative analysis of race-sex group data indicated 2010 mortality rates for hypertension per 100,000 to be 50.2 for Black males and 37.1 for Black females compared to 17.2 and 15.0, respectively, for their White counterparts (Go et al., 2014). Similar Black-White differences are observed in prevalence rates of hypertension.

Empirical evidence suggests that the prevalence rate of hypertension is roughly 40% among Blacks, compared to approximately 27% among Whites (Go et al., 2014; Hicken et al., 2014), Rates of hypertension are higher among men in both racial groups, <45 years of age; however, adults aged 45 to 54 have similar rates of hypertension, which become higher in women after age 55 (Go et al., 2014). NHANES data reported over three separate time periods indicate age-adjusted prevalence rates of hypertension in Black women as slightly higher (38.2-42.9%) than Black men (37.5-40.1%; (Mozaffarian et al., 2015). Moreover, data indicated that even after controlling for known hypertension risk factors (i.e., age, education, household income, martial status, gender, BMI, physical activity, smoking, and alcohol use), a statistically greater risk for hypertension among Blacks persisted compared to Hispanics, OR=2.12, 99% CI [1.90, 2.35] (Holmes et al., 2013) and Whites, OR=2.74, 95% CI [2.32, 3.25] (Kershaw et al., 2011).

Raising further concern, researchers have begun to report hypertension in children as a mounting public health concern (Assadi, 2012; Brady, Fivush, Parekh, & Flynn, 2010; Freedman et al., 2012; Rosner, Cook, Daniels, & Falkner, 2013). In 1999, researchers analyzed of NHANES III data (1988-1994) to find that the mean systolic BP for Black girls age 6-9 was 96.4 mm Hg compared to 95.4 mm Hg among White girls (Winkleby, Robinson, Sundquist, & Kraemer, 1999). A similar pattern of black-white differences is observed for boys across age groups, but initiates during age 10-13 (Winkleby et al., 1999). However, Rosner et al. (2009) discovered that the prevalence of hypertension among normal weight children (age 1-17) was significantly higher among Black boys compared to White boys, OR=1.14, 95% CI [1.03, 1.27], $p < 0.01$; yet, the rate of prehypertension was higher among Black girls of normal weight compared to White girls, OR=1.32; 95% CI [1.17, 1.49], $p < 0.001$. More recent analysis comparing NHANES III to NHANES data (1999-2008) revealed that the increase in childhood obesity has increased the odds of elevated blood pressure in children (OR=1.27, P=0.069; Rosner, Cook, Daniels, & Falkner, 2013). Brady et al. (2010) revealed Black-White differences in blood pressure (BP) are observed in children <13 and ≥13 years of age. A cross-sectional analysis of children (aged 3-20) referred for nephrology and hypertension care across three different facilities (i.e., University of Michigan, Johns Hopkins University, and Children's Hospital at Monteflore) found that Black children aged 13 and older observed higher rates of elevated blood pressure compared to their White counterparts (Brady et al., 2010). Although the mean systolic BP gradually increases as age increases for both racial groups, the Black-White differences remain and gradually

widen (Brady et al., 2010). Given that Blacks are more likely than Whites to display onset of hypertension approximately five years earlier, have a hypertensive family history, are significantly less likely to have adequate BP control (even with the use of medications), and experience more severe hypertension increasing the implications for greater CVD morbidity and mortality in adulthood (Fuchs, 2011; Gillespie & Hurvitz, 2013; Kramer et al., 2015).

Family history. Historic evidence from epidemiologic studies supports the association of family history with increased predisposition to CVD risk (Kurian & Cardarelli, 2007; Lloyd-Jones et al., 2004; Valdez, Greenlund, Khoury, & Yoon, 2007). Valdez et al. (2007) explained that in the case of multidimensional diseases such as CVD, differentiating genetic causes from environmental causes is often difficult. However, determining whether or not a first or second-degree relative has also been affected by CVD can be a useful assessment strategy (Mozaffarian et al., 2015).

Results from a longitudinal study that followed a dual cohort of male Black students from Meharry Medical College and White students from Johns Hopkins University School of Medicine for a median period of 29 years support the claim that family history plays a significant role in Blacks' risk for hypertension and CVD (Thomas, Thomas, Pearson, Klag, & Mead, 1997). Thomas et al. (1997) found that the presence of parental hypertension was a strong influence in predicting disease manifestation in Blacks during adulthood. Thomas et al. (1997) noted that the prevalence rates of hypertension among the Black cohort significantly increased as parental hypertension changed from both parents being negative (39%), mother only (41.4%), father only

(60.5%), to both parents positive (73.7%). Thomas et al. (1997) found parental history of hypertension to positively associated with race; the Black physicians from Meharry having a relative risk of 2.53 higher than the White physicians from Johns Hopkins, 95% CI [1.55-4.13], P<0.001.

In a population-based epidemiologic study, the offspring of Framingham Study participants were studied as a cohort to determine whether or not parental CVD could be used to positively predict CVD among the offspring. The participants in this study were at least 30 years of age, had no CVD at the onset of the study, and both parents were members of the original Framingham Study cohort (Lloyd-Jones et al., 2004). Lloyd-Jones et al. (2004) found that parental CVD increased the strength of association for CVD among both men and women (age-adjusted OR=2.6 and 2.3 times, respectively) when at least one parent had early onset of CVD (defined as father <55 years of age or mother <65 years of age). Mozaffarian et al. (2015) agrees that individual are predisposed to genetic factors, which are strongly influenced by the clustering of environmental, lifestyle, and other risk factors within families. No only does the extent of familial association increase CVD, but so does the age of the parental event. For example, a heart attack in one parent over the age of 50 increases an individuals odds of having a heart attack by 1.67; however, if both parents have heart attacks and are over the age of 50, the odds of a heart attack increase to 2.90 (Mozaffarian et al., 2015). Comparatively, Mozaffarian et al. (2015) reported that heart attacks experienced by younger parents (<50 of age) exponentially increases risk (OR=2.36 for one parent vs. OR=6.56 for both parents). Researchers also found that adjustment for traditional risk factors did not

diminsh the association of parental CVD with CVD incidence in their offspring (Lloyd-Jones et al., 2004). Because the evidence of family history has been so compelling, researchers now advocate screening of all children and youth who have at least one first degree relative with CVD or diabetes as a prevention strategy to identify families at increased risk of obesity, hypertension, dyslipidemia, metabolic syndrome, and CVD (Valdez et al., 2007).

Moreover, Reis et al. (2006) analyzed data from the Family Strategies Concentrating on Risk Evaluation (SCORE), a community-based cohort of children and their parents in Pittsburgh, PA, to discover that children may serve as the index case for families at elevated risk for CVD. There were a total of 141 children who participated in this study, with demographics consisting of an average age of 10.5 ± 3.4 years, predominately Black (69%), and male (60%), and 108 parents with a mean age of 38.5 ± 7.5 years, mostly Black (60%), and female (83%) (Reis et al., 2006). Researchers found that a strong correlation between children with CVD risk factors and the presence of the same risk factors among their parents (Reis et al., 2006). In fact, children who were obese ($\geq 95^{th}$ percentile) or had a waist circumference greater than 85^{th} percentile were almost 6 times more likely to have parents who were obese (BMI $\geq$ 30) or had abnormally large waist circumference (adjusted for age, race, and gender of the parent and age of the child OR=5.97 and 5.65, respectively) (Reis et al., 2006).

Effectiveness of hypertension control. The American Heart Association defines normal blood pressure as a systolic blood pressure (SBP) less than 120 mm Hg, and a diastolic blood pressure (DBP) less than 80 mm Hg (Mozaffarian et al., 2015). However,

there are differences not only in the prevalence of diagnosed hypertension between Blacks and Whites, but also the prevalence of controlled hypertension.

When determining the percentage of patients who achieve target blood pressure (BP) by race, the Black-White differences in BP control become more evident. From 2011-2012, Blacks were more likely to than Whites to take antihypertensive medication (77.4% vs. 76.7%, respectively; Mozaffarian et al., 2015), yet Blacks to have 40% greater odds of have uncontrolled BP (Delgado, Jacobs, Lackland, Evans, & Mendes de Leon, 2012). Sehgal (2004) noted that the Black-White difference in reaching target BP is only about 8% (68% versus 77%, respectively) when both groups have comparable baseline BPs (e.g., 6 mm Hg above a target DBP). However, the Black-White difference in reaching target BP significantly increased from 19% (58% versus 77%, respectively) to 30% (47% versus 77%, respectively) as baseline blood pressures for Blacks increased (8 mm Hg and 10 mm Hg above a target DBP, respectively), while that of Whites remained constant (e.g., 6 mm Hg above a target DBP; (Sehgal, 2004). These findings support the rationale for why Blacks often require at least two or more antihypertensive medications to achieve BP control (Delgado et al., 2012; Ferdinand & Sounders, 2006; Fernandez et al., 2011; Watson, 2008). The inability of Blacks to achieve adequate BP control contributes even further to complexities of the CVD epidemic.

In a cohort of more than 1000 low-income, hypertensive Blacks, Fernandez et al. (2011) investigated the effectiveness of combined provider and patient-level interventions. Findings indicated that gaps in blood pressure control are typically not the result of lack of awareness or poor health literacy among Blacks, but rather numerous

patient-level barriers (e.g., behavioral, psychosocial, environmental) to achieving and maintaining BP control (Fernandez et al., 2011). In other words, there are multilevel factors and socioenvironmental conditions that may lie outside of an individual's domain but influence one's ability to adequately manage hypertension, which warrant further investigation.

Environmental influence. Recent studies have addressed the importance of evaluating the role that the environment plays in significantly shifting the continuum of health outcomes based on SES, noting significant difference between Black and White populations (Conroy et al., 2010; Gaskin et al., 2013; Kershaw et al., 2011; Thorpe, Bell, et al., 2015). Whites of low socioeconomic status are more likely to have better health care, job opportunities, access to resources and services, and living conditions than their Black counterparts of comparable financial means (Kennedy, Paeratakul, Ryan, & Bray, 2007; Subramanian, Acevedo-Garcia, & Osypuk, 2005; Williams & Jackson, 2005). Blacks of lower SES are disportionately exposed to deleterious neighborhoods charactersitics, which exert negative effects on multiple aspects of their health and well-being, unlike their White counterparts (Johnson, 2011; Jones-Jack, Jack, Jr., Jones, & Scribner, 2010). Evidence reflects that Black neighborhoods, often also low-income neighborhoods, have multiple inequities (e.g., depleted community resources, limited employment opportunities, increased crime rates) that facilitate greater potential for adverse health conditions for the residents of those communities (Schootman, Andresen, Wolinsky, Malmstrom, Miller, & Yan, 2007; Weden, Carpiano, & Robert, 2008). Jones-Jack et al. (2010) maintains that poor neighborhood characteristics are strongly correlated

to limited access to healthy food options, quality health care facilities, reliable transportation, and other environmental factors that derail an individual's ability to achieve good health or effectively manage chronic health conditions. For example, the physician-to-patient ratio in Black neighborhoods is substantially lower (ranging from 1:10,000 to 1:15,000) compared to White neighborhoods (1:300) (Jones-Jack et al., 2010); and predominantly Black neighborhoods have a higher concentration of fast food restaurants per square mile compared to predominantly White neighborhoods (2.4 versus 1.5, respectively; Block, Scribner, & DeSalvo, 2004). Hence, place is also an important consideration in understanding disparities in CVD outcomes.

Cumulative disadvantage. Blacks experience more disease risk and burden as a result of traditional CVD risk factors (e.g., higher rates of obesity, stronger family history); however, there are additional underlying factors that contribute to this overwhelming disparity. Research suggests that Blacks are more likely to have exposure to multiple risk factors simultaneously, which creates a synergistic effect culminating in even more negative consequences over time (Flack et al., 2003; Kurian & Cardarelli, 2007; Watson, 2008; Williams, 2009). Although genetic factors (e.g., elevated cholesterol and blood pressure) may predispose certain individuals to CVD, it is the combination of those genes with lifestyle (e.g., physical inactivity and poor diet) and environmental (e.g., neighborhood characteristics and social injustices) factors that create the excess burden of CVD morbidity and mortality (Cubbin et al., 2006; Sundquist et al., 2006). In a study of urban neighborhoods in Sweden, Sundquist et al. (2006) found that in neighborhoods with high rates of violent crimes and unemployment, men, OR= 1.75, 95% CI [1.37,

2.22] and OR= 2.05, 95% CI [1.62, 2.59], respectively and women, OR= 1.39, 95% CI [1.19, 1.63] and OR= 1.50, 95% CI [1.28, 1.75], respectively) were more likely to experienced CHD. Adding to the multifaceted nature of this disease, lifestyle factors and environmental conditions are strongly linked to not only families, but also SES (Johnson, 2011; Johnson-Lawrence et al., 2013; Jones-Jack et al., 2010).

Researchers agree that lower SES is directly and indirectly associated with cumulative disadvantage because individuals lack the skills and resources necessary to adequately maintain health-promoting lifestyles (Gaskin et al., 2014; Dupre, 2008; Kim & Richardson, 2011; Shuey & , 2008). More importantly, the experiences and risk factors that result from deprivation build upon one another over the life course (Hertzman, 2004; James et al., 2006); hence, elderly individuals are more likely to have poorer health due to accumulated health risks (Dupre, 2008).

However, the results of cumulative disadvantage studies across the life-course are somewhat conflicting. After acknowledging that Blacks have poorer health outcomes in later life than Whites, Kim and Richardson (2011) indicated that loss of income and assets in later life attributed to substantial reduction in physical performance, particularly among women; however, the rate of this decline was comparable for Blacks and Whites after controlling for SES. Conversely, other researchers contend that Blacks at higher levels of education experience greater disparity in health outcomes than Blacks at lower levels of education (Farmer & Ferraro, 2005; Shuey & Willson, 2008), but in older ages, Blacks continue to have a faster rate of physical decline compared to Whites, regardless of education (Delgado et al., 2012; Fakhouri et al., 2012; Shuey & Willson, 2008).

Although Shuey and Willson (2008) maintained that increased income and wealth is equally advantageous for both Blacks and Whites in later years, the inability of Blacks to translate income into wealth only further supports the cumulative disadvantage (Jones-Jack et al., 2010). Finally, Dupre (2008) found that the impact of educational differences appears to diminish across age because individuals of lower educational levels have higher mortality rates at younger ages, leaving only the strongest survivors of the disparity group for comparison in later life. It is important to note that the observed differences in cumulative disadvantage across the life-course are possibly linked to how the constructs used to measure SES (e.g., income, education, employment status, wealth) differ across studies (Shuey & Willson, 2008). Therefore, researchers need to cast a wider net in understanding the totality of these risks and linking them to health outcomes.

Social Determinants of Health and CVD Risk

Berkman and Kawachi (2014) provided significant evidence to indicate that as early as the seventeenth century, researchers have understood that social conditions play an integral role in health and well-being. Social epidemiology is defined as a branch of epidemiology that encompasses numerous disciplines (e.g., sociology, anthropology, politics, and psychology), to comprehensively investigate "nontraditional" factors, or social determinants, that directly or indirectly influence health (Berkman & Kawachi, 2014). It also encourages more in-depth understanding of how and why some individual-level risk factors (e.g., obesity, smoking, physical inactivity) affect some populations in greater proportion by allowing social conditions that either facilitate or inhibit health-promoting practices to be examined as a correlate of health.

Wilcox (2007) theorized that social determinants of health assist researchers in understanding how factors considered to be "upstream" affect factors "downstream" (p. 1). As social epidemiology is intended to deepen our understanding of causation, Berkman & Kawachi (2014) argued that multifaceted philosophical questions must be addressed (e.g., addressing issues of accountability and determining where responsibility for the patterns of disease and social inequality lie). Social determinants of health enable researchers to explore multiple mechanisms that may account for differences in health disparities between Black and White populations.

The Influence of SES on Health Outcomes

SES is a multidimensional construct that researchers have commonly implicated as an influential determinant in the health disparities observed between racial groups (Harper et al., 2011; Johnson-Lawrence et al., 2013; Jolly et al., 2010; Laveist et al., 2009; Subramanyam et al., 2013; Williams, 2012). The CDC (2011d) defined SES as an integrated measurement of an individual's economic (e.g. income), social (e.g. education), and work status (e.g. occupation). However, studies that investigate the impact of SES on health outcomes inconsistently use these indicators to measure SES (Farmer & Ferraro, 2005; Glymour, Avendano, & Kseschi, 2014; Kennedy et al., 2007; Laveist et al., 2009).

Though education and income are commonly used markers to determine SES (Glymour et al., 2014; Kennedy et al., 2007), SES may be defined solely based on educational attainment. Using this single construct, researchers have found strong evidence correlating poor educational attainment with poor health outcomes (Banks et al.,

2006; Conroy et al., 2010). For example, prevalence rates were 14.3% for diabetes, 46.3% for hypertension, and 17.1% for heart disease among individuals with low years of schooling, compared with 9.5%, 37.0%, and 12.0% among individuals with high years of schooling, respectively. Conroy, Sandel, and Zuckerman (2010) maintained that despite this evidence, SES based on education does not determine other social determinants, such as income; nor are the result of difference in health outcomes necessarily the result of the interaction of income and education (Kennedy et al., 2007). Meaning, it is possible for individuals of lower educational attainment to have a high socioeconomic position, and vice versa. Yet, other researchers argued the contrary (Do & Finch, 2008; Finch et al., 2010; Iton, 2005; Jones-Jack et al., 2010; Schulz et al., 2008). Finch et al. (2010) specifically addressed this issue by describing not only the difference in educational attainment by poverty level based on U.S. Census data, but also suggested that the data underestimate the true differences in education across neighborhoods of varying SES. In addition, researchers (Finch et al., 2010; Kennedy et al., 2007) argued that there is ambiguity about the interaction between income and education when demographic factors (e.g. age, race, and sex) are considered.

Regardless of the construct used to measure SES, researchers have validated that the extent of one's financial means impacts the context of their surroundings and availability of resources, directly relating to their health outcomes (Harper et al., 2011; Johnson-Lawrence et al., 2013; Laveist et al., 2009; Williams, 2012). Individuals of higher SES are far more likely to experience positive health outcomes, while those of lower SES experience negative health outcomes (Galea, Tracy, Hoggatt, DiMaggio, &

Karpati, 2011; Harper et al., 2011; Johnson-Lawrence et al., 2013; Williams, 2012). Though the SES phenonmenon is not distinctive to any one country, the strength of the association seems to be more profound in the United States than elsewhere (Iton, 2005; Kawachi & Subramanuan, 2014). For example, a study was conducted to compare the association of SES and health behavior practices (i.e., diet, physical activity, smoking, and alcohol consumption) in China compared to the United States (Kim, 2004). Researchers found that individuals at the highest SES levels in China showed an inverse relationship to healthy behavior (OR=0.19), whereas in the United States, there was a direct correlation between highest levels of SES and increased healthy behaviors (OR=3.81; (Kim, 2004). Banks, Marmot, Oldfield, and Smith (2006) found that although differences in SES gradients and health status in England paralleled the United States, the prevalence rates of all diseases studied were significantly higher in the United States at each SES level. Prevalence rates of hypertension, for example, were documented at 36.7%, 34.6%, and 30.3% for England and 46.3%, 43.6%, and 37.1% for the United States at low, medium, and high-income gradients, respectively (Banks et al., 2006). Within the United States, the economic divide between the wealthy and the poor continue to widen (Kawachi & Subramanuan, 2014).

Economic divide. Unfortunately, a recent study determined that the gap between the wealthy and the poor in the United States has increased more than four-fold in the past 20 years, and the economic divide distinctly lies along racial lines (Kochhar, Taylor, & Fry, 2011; Shapiro, Meschede, & Sullivan, 2010). A prospective study that followed a cohort of families from 1984 to 2007 revealed the Black-White gap in wealth increased

from $20K to $95K during this time period (Shapiro et al., 2010); however, Domhoff (2011) estimated the wealth gap between average White and Black families to be 15-fold during 2007. If home equity is excluded from calculations to determine wealth, the income and wealth ratio by race escalates to 100:1 (Domhoff, 2011). Domhoff (2011) posited that while those with the top 20% of income control approximately 85% of the wealth in the United States, those at the bottom 40% of income hold a mere 0.3% of the wealth.

Although the concentration of wealth distribution has historical context that dates back to the 19th century (Domhoff, 2011), the wealthy have continued to gained more resources over time as the poor have retained less of what they had (Domhoff, 2011; Rigney, 2010; Shapiro et al., 2010). These observed gaps in wealth may be hypothesized to impact, either directly or indirectly, the social and environmental factors observed in Black and White communities, which are known to have strong linkages to health outcomes.

SES mobility. Researchers have historically noted that one's health is strongly linked to the SES characteristics of the community in which they reside, even after contolling for education and income (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010).

> Early in the foundation of social epidemiology, Krieger et al. (1997) stated,
>
> No single 'factor' accounts for links between socioeconomic position and health. Instead, numerous investigators have delineated myriad interconnected pathways, preceding conception and ending at death, whereby people's health is harmed or

> helped by their standard of living, workplace conditions, and social and psychological interactions with others at home, work, and other public settings. (p. 343)

Researchers have determined that an environment of life-long poverty or socioeconomic disadvantage strongly impacts an individual's health status in adulthood (Conroy et al., 2010; Galobardes, Lynch, & Smith, 2007; Johnson-Lawrence et al., 2013). There are multiple indicators of socioeconomic status (SES), such as education, income, wealth, employment status, occupation, and home ownership, which may have a different periods of validity or impact on health outcomes at different stages throughout the lifecourse. There are four primary causal pathways used to explore SES across the lifecourse, including: 1) *latent effects* (early life adversities increasing later life risk); 2) *pathway effects* (early life experiences creating a life trajectory that effects that impact adult health); 3) *social mobility* (changes in SES from early life to adulthood determining adult health); and 4) *cumulative effects* (the accrual of early and later life experiences to influence health) (Adler & Stewart, 2010; Pollitt et al., 2005). Each hypothesis is based on the premise that measures of SES, socioeconomic advantage or disadvantage, during childhood has some bearing on one or more periods during adulthood, and culminate in impacting adult health outcomes (Conroy et al., 2010; Galobardes et al., 2007).

Recent studies support an inverse association between lifecourse SES and CVD-related health outcomes (Adler & Stewart, 2010; Berry et al., 2012; Hogberg et al., 2011; Johnson-Lawrence et al., 2013; Roberts et al., 2010; Wamala et al., 2001). Gebreab et al. (2015) identified adult socioeconomic positioning as a strong predictor of cardiovascular

events in women. However, life-long (early and later life combined) socioeconomic disadvantage increased CHD risk by 4.2-fold compare to women who had not experienced any socioeconomic disadvantage (Johnson-Lawrence et al., 2013; Wamala et al., 2001).

Researchers have identified SES mobility as a pathway linking lifecourse SES with CVD outcomes (Hogberg et al., 2011; James et al., 2006; Johnson-Lawrence et al., 2013). Pollitt et al. (2005) argues that previous studies typically compare only unwavering low and high SES trajectories; little empirical evidence was found to assess how improved or diminished SES mobility is associated with CVD risk factors or CVD morbidity and mortality.

Studies that have examined the link between SES mobility on CVD outcomes (Johnson-Lawrence et al., 2013; Pensola, 2003) or hypertension (Hogberg et al., 2011; James et al., 2006), which included upward and downward mobility, agreed that individuals of high and increasing SES had lower risk. Johnson-Lawrence et al. (2013) found an inverse relationship in the proportional hazard ratios for CVD mortality across progressive income trajectory categories in the Alameda County study. Findings indicated a higher hazard ratio among individuals of stable low SES compared to moderately low SES, HR=2.52, 95% CI [1.77, 3.59], as well as compared to upwardly mobile individuals, HR=12.92, 95% CI [4.05, 41.21], with results persisting after controlling for age, race, marital status, and gender (Johnson-Lawrence et al., 2013). In a cohort of men in Finland, Pensola (2003) discovered that the CVD mortality rate was

higher among those who experienced consistently low or upward SES mobility (3.55 and 2.29, respectively) compared to men who experienced downward SES mobility (1.27).

Similar results were observed in studies examining the association of SES mobility on hypertension risk. Among participants of the Swedish Twin Registry, Hogberg et al. (2011) found that individuals whose SES mobility remained low throughout their lifecourse had a higher prevalence rate of hypertension (15.4%), compared to those who experienced upward mobility (12.5%), downward mobility (10.8%), or stable high SES (8.0%). James et al. (2006) found increased risk of hypertension among a stable low SES group compared to a stable high SES reference group, OR= 7.27; 95% CI [1.91, 27.51], in a study of Black men in Pitt County, North Carolina. However in this study, downward mobility had greater association with hypertension risk, OR= 5.87; 95% CI [1.25, 27.49], than upward mobility, OR=3.85, 95% CI [0.91, 16.13] (James et al., 2006). These studies support SES during childhood as having a strong influence on CVD outcomes during adulthood. Considering that Blacks experience greater CVD mortality rates and socioeconomic disadvantage throughout the lifecourse than whites (Williams & Collins, 1995), exploring the role of lifecourse SES on the racial disparities in CVD outcomes may provide useful insight.

Data from the Atherosclerosis Risk in Communities (ARIC) study was analyzed to evaluate the multiple indicators of SES (e.g., parental or adulthood education, occupation, and home ownership) at three time points during an individual's life (i.e., early-life, young adulthood, and mid-to-older adulthood), as well as a summary score representing the lifecourse SES, and the impact of SES on heart failure incidence

(Roberts et al., 2010). Roberts et al. (2010) found that not only were Blacks more likely to be exposed to low SES during early life, but they were also more likely to experience a higher prevalence of CVD risk factors regardless of early life SES than their White counterparts. Consequently, Blacks had a greater overall age-adjusted incidence rate of heart failure than Whites (5.23 events per 1,000 person-years vs. 3.18 events per 1,000 person-years, respectively; Roberts et al., 2010). Moreover, even Blacks of high summary SES experienced greater incidence of heart failure than Whites of low summary SES (4.38 events per 1,000 person-years vs. 3.99 events per 1,000 person-years, respectively; Roberts et al., 2010). Researchers noted that parental education was a strong predictor of heart failure for both racial groups; however unlike Whites, Blacks were also adversely impacted by the lack of parental home ownership (Roberts et al., 2010). Although this study suggests that early life SES shapes exposure to risk factors that increase health outcomes in adulthood among both Blacks and Whites, questions remain about why disparities among Blacks continue to exist.

In addition to increased CVD morbidity, childhood SES disadvantage also increases risk for CVD mortality among Blacks. Researchers discovered a 1.32-fold increased risk of CVD mortality among men who experience socioeconomic disadvantage during childhood, which remained after adjusting for behavioral risk factors and socioeconomic position in adulthood (Kauhanen et al., 2006). Mays et al. (2007) argued that even when adjusting for SES, Blacks suffer from excess death at a rate equivalent to 1.1 million years of life lost, or roughly 38,000 deaths per year.

Role of place. Recent studies have addressed the importance of evaluating the role that place plays in significantly shifting the continuum of health outcomes based on SES (Chen & Paterson, 2006; Conroy et al., 2010; Do & Finch, 2008; LaVeist, Gaskin, & Trujillo, 2011). LaVeist et al. (2011) recently investigated three models to determine the whether or not living in a predominantly minority neighborhood negatively impacts health, living in neighborhoods with high rates of poverty impact health, or if it is some combination of both.

Using a sample of over 17,000 White, Black, and Hispanic adults, LaViest et al. (2011) assessed five measures of health status (i.e., self-reported general health status, mental health status, diabetes, hypertension, and/or stroke). The researchers found that a high poverty community (i.e. place) was a stronger predictor of poorer health than a highly minority-based community (i.e. race) for general health (OR= 1.386 vs. 1.058, $p < 0.001$), mental health (OR=1.304 vs. 348, $p < 0.05$), and diabetes (OR= 1.202 vs. 1.052, $p < 0.05$). However, being Black was associated with twice the hypertension risk than living in a high poverty community (LaVeist et al., 2011). Overall, researchers found that minority communities were more likely to have greater health risk when they were poor communities. However, poor communities had greater health risks due to the neighborhood characteristics that were strongly influenced by high poverty (e.g., limited access to resources, higher crime rates, higher unemployment rates, poorer quality of education); these were also more likely to be Black communities.

Blacks are more frequently exposed to deleterious neighborhoods charactersitics, which exert negative effects on multiple aspects of their health and well-being, than their

White counterparts (Diez Roux & Mair, 2010). The U.S. Census Bureau (2010) recently cited that although the overall poverty rate was 15.1%, it was approximately substantially lower for Whites (9.9%) and almost double for Blacks (27.4%). Other researchers concur that Black neighborhoods are often also low-income neighborhoods, and have multiple inequities that facilitate greater potential for adverse health conditions for the residents of those communities (Schootman, Andresen, Wolinsky, Malmstrom, Miller, & Yan, 2007; Weden, Carpiano, & Robert, 2008).

Researchers noted an extensive list of objective and subjective characteristics for which community equality can be assessed (Gary, Stark, & LaVeist, 2007; Jones-Jack et al., 2010; Schootman et al., 2007; Weden, Carpiano, & Robert, 2008). Objective characteristics included the availability or quality of housing and neighborhood conditions, such as pollution, noise emission from traffic or industries, garbage collection, street lighting, banking services, recreational facilities, public transportation, number of grocery stores, and the condition or presence of sidewalks and yards surrounding homes (Mackenbach et al., 2014; Schootman et al., 2007; Weden et al., 2008). Subjective characteristics related more to how individuals feel about their neighborhood in terms of factors such as drug and/or gang activity, safety of roads, crime activity, frequency of fast food restaurants, quality of available resources and services, billboards and signage, and graffiti (Schootman et al., 2007; Weden et al., 2008).

Interestingly, early researchers associate the depravity of low SES conditions as imposing psychological risks on the resident population (Gary et al., 2007; Kauhanen et al., 2006). However, Gary et al. (2007) suggested that Blacks were less likely to view

their neighborhood conditions as imposing severe problems, and more likely to indicate a sense of neighborhood cohesion, even in the absence of the availability of a community leader. This cohesive community experienced by Blacks was found to be associated with lower concentrations of anxiety, stress, and depression compared to their White counterparts (Kauhanen et al., 2006). Some data suggest that the more positive mental health status noted by Gary et al. (2007) may be attributed to subjective neighborhood assessments, and have a stronger association to health outcomes than objective neighborhood conditions (Ludwig et al., 2012). Ludwig and colleagues (2012) recommended that the measurement of both neighborhood aspects is needed to determine the impact of health because race and neighborhood disadvantage are explicitly intertwined.

It is important to again note that SES is based on several parameters beyond just income and education, and interacts with complex demographic, environmental, and social atrributes. Several researchers argue that race combined with income gradient is a strong predictor in determining housing conditions, neighborhood characteristics, quality of education, purchasing power, social class, and political influence (Gary et al., 2007; Gee & Ford, 2011; Schootman et al., 2007; Weden et al., 2008). Moreover, Blacks isolated to low income neighborhoods, devoid of access to adequate resources and services, are typically burdened by unfair societal practices, such as higher rates of crime, drug activity, and exposure to trash (Dinwiddie, Gaskin, Chan, Norrington, & McCleary, 2013). Adding to the complex pathways by which SES mediates health outcomes is the effect that SES has on overall well-being.

The Meharry-Hopkins Cohort Study: Are Affluent Blacks Protected?

Although lower SES influences a plethora of mechanisms that underlie poor health outcomes, it should not be assumed that higher SES offers protective health benefits (Weden et al., 2008). Williams et al. (2010) stated that despite the greater risk for living in poverty and greater risk of mortality among Blacks, elevated mortality rates for all causes persist among Blacks even after controlling for SES and education. Williams et al. (2010) also argued that although Black populations are more likely to live in poverty, the outcomes routinely resulting from these circumstances do not completely explain the disparities in health. The Meherry-Hopkins Cohort Study is a prime example of these inconsistencies (Thomas et al., 1997).

A cohort of Black medical students from Meherry Medical College and White students from Johns Hopkins University were followed over a 23-35 year period. At follow-up, Thomas et al. (1997) found that Black physicians were more likely to develop hypertension, RR=2.00, 95% CI [1.6, 2.6], $p \leq 0.001$, CVD incidence, RR=1.65, 95% CI [1.13, 2.41], and coronary heart disease, RR=1.18, 95% CI [0.36, 3.84] than their White counterparts, after multivariate adjustment for factors such as age, cigarette smoking, BMI, and systolic blood pressure. The author argued that certain confounders found in several other studies, such as education and SES, are eliminated because all participants are physicians (Thomas et al., 1997). Yet other socially-mediated confounders remain (e.g., differences in stressful experiences and exposure to racism/discrimination); understanding of their role in health outcomes is essential.

The John Henry (JH) scale was used in the Meherry-Hopkins cohort study to measure one's ability to manage environmental psychosocial stress. While the results of this study did not indicate an association between JH and hypertension for either population, differences in how Black and White cultural experiences influence psychosocial stressors (e.g., racism) were not assessed (Thomas et al., 1997). This study suggests that despite education and affluence, Blacks of higher SES have the potential for health outcomes more similar to Black of lower SES than Whites of high SES. Interestingly, the hypertension prevalence rates in the Meharry cohort exceeded that of the at-large Black male population (Thomas et al., 1997), suggesting that this sample may not be reflective of the overall population of Whites or Blacks (Baum, Garofalo, & Yali, 1999). The contradictions found between studies of education and affluence indicate that there is a need for additional research to establish a better understanding of differences in psychosocial stress exposures and coping mechanisms by race; additional research may assist in explaining why disparities in CVD occur regardless of education and income.

Thomas et al. (1997) did, however, explore the circumstances pertaining to perceived stress that allowed the Hopkins cohort to have more job control than the Meharry cohort. Although both cohorts had extensive debt after completing medical school, the environmental culture of Meharry Medical College led the Meharry cohort to be more likely (75%) to pursue opportunities to work in medically underserved areas and receive debt forgiveness; while the Hopkins cohort was more likely to opt for more lucrative medical specialties (Thomas et al., 1997). Furthermore, rural environments may have positioned the Black cohort to be less likely to have a regular health care provider,

availability to adequate health care facilities or services, or the flexibility to take time away from work than their White counterparts (Thomas et al., 1997).

Although the reason for the gaps in access and resources were not provided, one may speculate that choosing to work in an underserved area is likely devoid of supportive resources (e.g, network of physicians to distribute responsibility). In addition, it is important to note that more than 90% of the Black study participants were male (Thomas et al., 1997), and there may be other characteristics specific to Blacks males (e.g., lack of trust, lack of peer respect, fear) that limit health seeking practices (Thomas et al., 1997).

The Importance of Race

Historically, race has been a socially-defined construct that frequently reflects a ideologic, economic, and sociopolitical hierachy, which in turn serves to mediate the perpetuation of health disparities (Jones, 2000; Jones et al., 2008; Kawachi et al., 2005; McFayden, 2009; Mersha & Abebe, 2015; Thomas et al., 1997). Although the term *race* is solely based on the physical and cultural characteristics of a group (Jones, 2000; Kawachi et al., 2005; Thomas et al., 1997), it has historical context dating back to the institution of slavery. Kawachi et al. (2005) attributed the development of the term to the "pre-Civil War debate" used to provide rationalization for why Blacks could acceptably be used as slaves because they were inferior to Whites (e.g., differences in cranial size existed between the races; p. 344).

While the basis for these claims are completely without merit, biologically-based research (i.e., the human genome project) has continued to posit that genetic differences in disease susceptibility are based on racial differences (Kawachi et al., 2005). However,

the explanation that these genetic differences are based on "race" should give pause to those who advocate this belief because other African-originating populations display significantly different health outcomes (Kawachi et al., 2005). For example, Kawachi et al. (2005) argued that although Blacks suffer from excessive rates of hypertension and diabetes, these rates are documented to be approximately two to five times greater than that of non-United States-based populations who share the same genetic makeup (e.g.,West Africans). Hence, researchers have commonly using race as a proxy for other indicators such as SES, class, and culture (Jones, 2002; Kawachi et al., 2005; Williams & Sternthal, 2010). Both race and social class play a vital role in health status; however, researchers are challenged in defining the effects of them as independent, yet interactive, constructs largely because race and class are highly correlated (Isaacs & Schroeder, 2004; Kawachi et al., 2005).

Kawachi et al. (2005), Jones (2002), and Williams and associates have all argued that if health status was truly based on SES, then low income Blacks and low income Whites would have similar outcomes. As such, researchers (Lynch & Kaplan, 2000; Matthews & Gallo, 2011) suggested that sociology-based perspectives recommend measurement strategies based on social class. For example, the Weberian tradition imposes that society should be stratified by class, status, and political power so that groups share in "life chances" (Lynch & Kaplan, 2000). The Weberian tradition also recognizes that class systems emphasizes the concept of "working class, who were at a competitive disadvantage in the marketplace because they had fewer goods, abilities, and skills that they might exchange for income" (Lynch & Kaplan, 2000, p. 15).

Acceptance of this philosophy, however, would require that low income Whites be considered socially in the same fashion as individuals that have been historically promoted as being inferior. While Americans have been conditioned to be acutely aware of race, the notion of class gives pause to the consideration that upward mobility in "the land of opportunity" is limited (Isaacs & Schroeder, 2004; Williams & Sternthal, 2010). Not accepting the philosophy that poor Blacks and Whites be viewed equally has been the basis for creating and continuing to fuel the multiple disparities in the lives of Blacks, which lead to disparities in health outcomes. LeBron and colleagues (2015) and Kawachi et al. (2005) contended that both race and class be considered as codeterminants of health disparities, mediated by racism as a pathway.

To effectively address issues of health disparities, some researchers argue that differences in social class must be examined in combination with race (Isaacs & Schroeder, 2004; Jones, 2002; Kawachi et al., 2005; LeBrón et al., 2015). Diemer et al. (2013) defined social class as "denoting power, prestige, and control over resources and focus on the two most prominent ways that psychologists have conceptu-alized and measured aspects of social class. The first approach, socioeconomic status (SES), indexes one's position within a power hierarchy via relatively objective indicators of power, prestige, and control over resources, such as income, wealth, education level, and occupational prestige" (p. 3).

White privilege. The history of inequality in this country is based on racism and discrimination (Hudson, Puterman, Bibbins-Domingo, Matthews, & Adler, 2013). Poor SES Whites have often supported policies, practices, and social norms that counter the

political or economic interests that best align with their SES, in order to have certain (i.e., better) rights and privileges common to others who share their racial identity (Hughey, Embrick, & Doane, 2015; Kawachi et al., 2005; Wise et al., 2008). In the 1960s, President Lyndon Johnson stated, "If you can convince the lowest white man that he's better off than the best colored man, he won't notice you picking his pocket. Hell, give him somebody to look down on, and he'll empty his pockets for you" (Kawachi et al., 2005, p. 349). Hence, the structuring of housing policies, educational systems, employment opportunities, and civil rights have created the system of racism that gave unfair and unjust advantages primarily to White population groups, regardless of SES. This system of racism that created unfair advantage for all White people (e.g., power, resources, prestige), regardless of their socioeconomic position, is also known as *White privilege* (Hughey et al., 2015; Rigney, 2010; Wise et al., 2008).

Not only does White privilege create opportunities or advantages for Whites that other racial/ethnic groups do not have, but it also defines how Whites view the world, as well as how the world views them (Holladay, 2000; McIntosh, 1989; Wise et al., 2008). McIntosh summed up the concept in stating, that White privilege is being "taught to see racism only as individual acts of meanness, not in invisible systems conferring dominance on my group" (pg. 1). Social systems have conditioned Whites to think that they are entitled to such treatment (Holladay, 2000; Hughey et al., 2015; Wise et al., 2008); therefore, Whites may potentially never encounter the reversed experience that many Blacks routinely endure, which begets a sense of violation, anger, worthlessness, and unrelenting stress. Moreover, White privilege serves as an underlining cause of the

health disparities that Blacks experience, which results in unequal access to health-promoting resources (e.g., close proximity to healthy food choices, availability of quality health care) and opportunities for the development of sustainable behaviors (e.g., availability of nutritious foods, safe environments for regular exercise) (Jones-Jack et al., 2010).

Whites are afforded the luxury to remain oblivious to how the same institutional systems that are inherently protective for them create barriers for others. How an individual is viewed by the institutional systems they are required to navigate, and the inroads or roadblocks that those systems impose upon them, is essential in understanding the behavioral choices available to them, and hence their ability to thrive, manage stress, and have an overall sense of well-being. Addressing health disparities requires not only the elimination of unmerited favor that gives power and privilege to Whites, but also raising awareness to the spoke and unspoken biases that influence social systems and norms.

Inequity among racial groups. Shaw-Ridley and Ridley (2010) contended that efforts to understand and address the fundamental causes of health disparities are misguided, because the interplay of humans, their environments, and social conditions are vastly complex, and the examination of the health disparities phenonmenon does not embrace a historical perspective rooted in power, politics, and racism. Dating as far back as the early 1900s, W.E.B. Du Bois (1906) documented the need to address poorer health among Blacks through social reform. Since then, researchers have continually cited the persistent nature of health disparities as a benchmark for Black health, and the necessity

to change the laws and practices that perpetuate social and structural inequalities (Jones-Jack et al., 2010; Taylor, 2015). However, many researchers have been known to use the term "disparity" interchangeably with "inequality" and "inequity."

Carter-Pokras and Baquet (2010) discussed how the interchanging of these terms has created disagreement and confusion regarding whether or not either term includes any judgement of unfair treatment or centers around avoidable decisions. More importantly, policy makers allocate resources and impose political agendas based on the interpretation of "*who* is deciding what is avoidable and unjust, and *how* it is decided" (DuBois, 1906). For example, improvements in health disparities cannot occur if Blacks are simply provided equal access to healthy food choices in low income communities, but there is no equity in the quality or cost (Shaw-Ridley & Ridley, 2010). Hence, there is a strong argument to shift from a focus of population groups having equal access, resources, opportunities, etc. to efforts that create equity among these groups. For example, Shapiro et al. (2010) highlighted that even when Blacks and Whites have income equality, Blacks are still twice as likely to experience discriminatory high-cost lending practices, which leads to greater risk for foreclosure. Wise (2007) further added that it was these inequities in lending practices that facilitated the wealth gap by allowing even low income Whites the advantage of owning their own home. Over time, generational advantages were created as the wealth from these assets were passed down. More clearly stated, White privilege allowed low income Whites to incur greater wealth than Blacks of higher earning potential (Carter-Pokras & Baquet, 2002).

Though income provides individuals with access to resources, Domhoff (2011) argued that wealth is a resource in achieving power and better health outcomes. Given the impact of poverty on Blacks, Wyatt et al. (2003) contended that Blacks have little or no opportunity to achieve wealth because they are twice as likely to live in deprived commuities and be unemployed; therefore, Blacks are less likely to have resources (e.g., health insurance) and access healthcare in a timely manner (Mays, Cochran, & Barnes, 2007; McFayden, 2009; Whitfield et al., 2014). Furthermore, researchers have begun to suspect that the negative impact of social stressors that Blacks have experienced as a result of generations of repeated exposure to racism is associated with higher levels of resting SBP (Harrell, Hall, & Taliaferro, 2003; James et al., 2006; Mays et al., 2007; Muennig & Murphy, 2011).

To this end, there is more than one ideology used to permeate the mechanisms that allow racism to affect hypertension. Williams and Neighbors (2003) described these ideologies as: (a) restricting socioeconomic achievement (i.e., opportunities for education, employment, and income) as a means of ultimately affecting health; (b) depriving access to goods, services, and resources (e.g., medical facilities, standards of treatment and care, built environments) that promote health; and (c) tolerance or adoption of stereotypes, characterizations, or beliefs that potentially encourage adverse health outcomes. Wyatt et al. (2003) suggested that although attitudes about racism have dramatically changed, discriminatory attitudes "continue to coexist with a desire to maintain at least some social distance from blacks" (p. 316). This school of thought displays little commitment to change or prevent the implementation of policies that

would rectify the practice of White privilege (Williams & Neighbors, 2001; Wise et al., 2008). In order for improvements in health disparities to occur, racism needs to be more widely observed as fundmental contributor to adverse health outcomes, and greater commitment to preventing the permeation of racism within societal institutions is essential (Williams & Wyatt, 2015).

The Connection between Racism and Health Disparities

There are social, political, and cultural norms that support many of the multilevel injustices that drive health disparities (Jones, 2000; Krieger, 2012; Rigney, 2010; Williams & Mohammed, 2013; Williams, 2012). Racism is hypothesized to be an underlying cause of health disparities. *Racism* has been defined as a system of practices, policies, beliefs, attitudes, and institutional norms that tends to assert opportunity and worth to some individuals, but disparage and create disadvantage for others due to the observed physical characteristics of populations or communities (Blank, Dabady, & Citro, 2004; Oakes & Kaufman, 2006; Williams & Chung, 2004). More generically, the term itself has long been associated with unequal distributions of privileges, resources, and power (Brondolo, Brady ver Halen, Libby, & Pencille, 2009; Jones, 2002); however, clarity on how racism may be consistently defined and objectively measured is needed (Atkins, 2014). Furthermore, Williams and Mohammed (2013) contended that the strong association observed between SES and race in the United States is perpetuated by the historical injustices of racism. Like White privilege, the Matthew Effect is the presence of structural and cultural barriers that have historically created inequalities observed between individuals or communities, with Blacks having experienced generations of

disadvantage and inequality at much greater proportion than their White counterparts. The term *Matthew Effect*, coined by sociologist Robert Merton (2000), originates from the Bible's Book of Matthew and is based on the concept that "the rich get richer while the poor get poorer" (Rigney, 2010, p. 1).

Williams and Mohammed (2013) and others (Brondolo, Gallo, & Myers, 2009; Jones, 2002) define racism as occurring at multiple levels (e.g., institutional, personally-mediated/interpersonal, internalized, cultural), and frameworks have been developed to depict the pathways through which racism mediates health outcomes (Paradies, 2006). While the health outcomes that may result from exposure to racism are not specifically related to the type of racism exposure, Table 1 describes the levels of racism and provides examples of how each type may manifests itself in society.

Table 1

Description of the Multilevels of Racism

Types of Racism	Description	Manifestations	Example
Institutionalized Racism	Differential or restricted access to material goods, services, opportunities, and power between racial groups	• Unearned privileges • Unfair societal norms • Structural barriers	• Access to quality education • Safe, clean environments • Voting rights • Representation in government offices
Personally-mediated/ Interpersonal Racism	"assumptions about the abilities, motives, and intentions" (Jones, 2000. p. 1212) or actions toward an individual or group based on race that is prejudicial or discriminatory	• Intentional • Unintentional • Lack of respect • Unwarranted suspicion • Devaluing • Dehumanization	• Poor or no service • Lowering of standards • Omitting or limiting information • Hate crimes • Subtle or blatant messages of rejection or exclusion
Internalized Racism	"acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth" (Jones, 2000, p. 1213)	• Embracing "whiteness" or societal opinions • Eroded self value • Helplessness/ Hopelessness	• Preference for lighter skin tone • Use of racial nicknames • Giving up on personal dream or talents
Cultural Racism	Explicit and implicit communication	• Media portraying of minorities in a stereotypical or derogatory manner	• Drug dealers or nannies in movies • Absentee or unfit parents

Defining Racism

Racism continues to be a manifestation of social systems and institutions, varying in degree from blatant actions (e.g., hate crimes) to more common subtle expressions (e.g., stereotypical references, interpersonal discrimination, individual biases) (Williams & Neighbors, 2001). For example, Shaver and Shavers (2006) stated that almost half of the nearly 7,500 hate crimes reported in the United States during 2002 were motivated by race. At the opposite end of the spectrum, the hiring of Blacks has been documented to decline when the race of the hiring manager is not of the same racial group as the applicants.

In a 30-month study of a large nationwide retailer, more than 1,500 managers were assessed to understand the hiring practices of approximately 100,000 entry-level employees at more than 700 stores across the United States (Giuliano, Levine, & Leonard, 2009). Researchers found that when Blacks managers were replaced with White managers, the hiring rate of Black employees dropped from 21% to 17%; while the rate of hiring for White employees increased from 60% to 64% (Giuliano et al., 2009). This pattern is even more prominent in southern states; the hiring rate for Black employees fell from approximately 29% to 21% (Giuliano, Levine, & Leonard, 2009). Regardless of whether it occurs in the social norms of organizational policies and practices or interpersonal interactions, the more subtle perceptions of racism documented in empirical research are often self-reported subjective measurements (Giuliano et al., 2009).

Review of the literature indicates that the degree to which racism occurs (or is interpreted) may have broad variation, even among individuals of the same racial

population, based on numerous factors, such as personal experiences, historical events, attitudes and beliefs, demographics, and coping abilities (Das, 2013; Myers, 2008; Paradies, 2006), that impact how individuals interpret social interactions. Moreover, Myers (Giuliano et al., 2009; 2008) suggested that Blacks have been conditioned to have "more sensitive racial filters and lower response thresholds that may predispose them to interpret a wider range of experiences and events as 'racially meaningful'; and have a more intense reaction to them" (p. 14), particularly when the experience is construed as ambiguous.

Racism: The Chronic Stressor

The extent to which an individual experiences stress (e.g., harm, loss, threat, or other challenges) is often subjectively appraised (Brondolo, Brady ver Halen, Libby, & Pencille, 2009; Hicken et al., 2014; Vines et al., 2006; Williams, 2012). Chronic stress may contribute to an array of negative psychosocial characteristics, such as feelings of depression, hopelessness, life dissatisfaction, vulernability, deprivation, dependence, and helplessness versus feelings of happiness, security, stability, power, self-confidence, and self-motivation (Das, 2013). Some researchers argue that although lower SES is not definitively responsible for chronic stress, it has been strongly suggested that the stressors associated with lower SES often directly or indirect influence health and well-being (Subramanyam et al., 2013; Thoits, 2010).

Thoits (2010) also explained how the impact of a single stressor can proliferate into other areas or domains of an individual's life. For example, the responsibilities of caregiving to an elderly parent can lead to financial challenges, marital problems,

interference with work performance, depression or other personal health issues. Furthermore, stressors can proliferate over an individual's life course, as well as across generations. Individuals who experience the stress of childhood poverty report a greater frequency of poverty in adulthood (Baum et al., 1999; Conroy et al., 2010; Harper et al., 2011; Hogberg et al., 2011; Johnson-Lawrence et al., 2013; Pollitt et al., 2005; Ratcliffe, McKernan, & Institute, 2010). In acknowledging the potential processes of cumulative advantage/disadvantage (i.e., stress proliferation vs. the Matthew Effect), the difference in the two processes is that one occurs at the individual level over a period of time, whereas the other is the result of structural and/or cultural forces impacting the individuals at the aggregate level (Baum et al., 1999; Schulz, Parker, Israel, & Fisher, 2001; Thoits, 2010). The effects of racism occur in both.

Thoits (2010) argued that there is unequal distribution of stress by age, sex, SES, and race, which parallels disparities in both physcial and mental health issues; specifically, higher rates of stress are reported among adolescence/young adult and older age groups, women, persons of lower SES, education, or "occupational prestige," and Blacks. Although all individuals experience and are impacted on some level by stress, it could be argued that Blacks generally experience more stress and are more greatly impacted. While it may be hypothesized that Blacks of lower SES are exposed to greater doses of chronic stress, it is important to note that Black-White differences in cardiovasuclar disease exist among Blacks across SES groups (Hicken et al., 2014; Jolly et al., 2010; Williams & Mohammed, 2013; Williams, 2012). Therefore, the unanswered question is whether there are chronic stressors commonly experienced by Blacks,

regardless of SES, that have greater impact or are more difficult to recover from (e.g., racism, discrimination).

Williams and Mohammed (2013) contended that race-related stress often imposes an additional layer of chronic stressors, typically not associated with White populations, which likely exacerbate the significance of many other life stressors. Conversely, Brondolo et al. (2009) argued that acts of racism not only impact those who are targeted, but also those who observe and enact the behavior, because it influences one's own self-perception, their perception of others, and the social environment around them. Reactions to racially-motivated experiences may generate self-perceptions that are minimalizing or threatening for the targeted population, but empowering or self-promoting for non-targeted populations.

Racism has routinely been viewed as a vehicle for Whites to maintain privileges, resources, and influences (Dolezsar, McGrath, Herzig, & Miller, 2014; Thoits, 2010; Williams & Mohammed, 2013; Williams, 2012; Wise et al., 2008); these perspectives have contributed to the establishment of policies, practices, and social norms that have directly and indirectly been associated with adverse health outcomes (Ahmed, Mohammed, & Williams, 2007; Brondolo, Gallo, et al., 2009; Thoits, 2010; Wise et al., 2008). For example, Executive Order 12898 is a government policy established in 1994 by President Clinton that required the Environmental Protection Agency to defined the parameters of environmental justice for minority and low SES populations (Adler & Newman, 2002). Despite the establishment of this policy, lack of enforcement in poor minority communities continues to translate to greater exposure to environmental risk

factors (e.g., landfills, noise, crowding, deteriorated housing). As such, it is estimated that the relative risk for mortality in poor minority communities is 1.9 to more than 5 times greater than in communities without environmental risks, which are typically predominantly White communities (Adler & Newman, 2002).

Researchers have explored various dimensions of how stress associated with perceived racism may transcend multiple aspects of an individual's life (e.g., residentially segregated communities; stereotypical or derogatory media portrayals; level of control or flexibility at work; availability, quality, and affordability of resources and services; understanding of cultural differences; (Ahmed et al., 2007; Brondolo et al., 2009; Din-Dzietham et al., 2004; Wise, 2010). Of greater concern are the multiple pathways through which racism affects health (Brondolo et al., 2009).

The Relevance of Racism to Health

Jones (2002) stated that "'Racial health disparities are produced on at least three levels: Differential care within the health care system, differential access to health care, and differences in exposures and life opportunities that create different levels of health and disease" (p. 8). While it is important to specifically address each of these aspects of racism, it can be argued that the difference in exposure and life opportunities is a precursor for the previous two levels. A host of theoretical frameworks have been proposed to explore the racism-health dynamic (Williams & Mohammed, 2013; Williams, Mohammed, Leavell, & Collins, 2010); however, research that actually demonstrates the processes for how racism and health are related is still emerging (Williams & Mohammed, 2013).

While there has been a growing interest in efforts to understand the relationship between racial discrimination and poor health outcomes, publications centered around this topic are more frequently conceptual than empirical. Empirical research has often had results that are widely inconsistent from one study to the next. The foundation of this research study is that there are obvious differences in quality, frequency, or availability of exposures and life opportunities, which commonly occur along color lines; these differences are significant enough to drastically skew health outcomes. Hence, it is important that conceptual frameworks used to assess racial discrimination, as a pertinent risk factor for hypertension and CVD among Blacks, clearly articulate the interconnectedness of the constructs previously identified and the implication of their collective measurement.

Association between racism and CVD outcomes. The literature review indicated mixed results regarding whether or not the perceived racism that Blacks experience predisposes them to greater risk for adverse health outcomes (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Chae et al., 2010; Cuffee, Hargraves, & Allison, 2012; Dolezsar et al., 2014; Guyll, Matthews, & Bromberger, 2001; Hicken et al., 2014; Krieger et al., 2013; Sims et al., 2012). Some empirical studies have documented a positive association between exposure to racism and increased risk for hypertension and CVD related outcomes (Roberts, Vines, Kaufman, & James, 2008; Sims et al., 2012). Conversely, earlier studies indicate little or no association between racial discrimination and adverse health outcomes (Barksdale et al., 2009; Brown, 2004; Krieger, 1990; Peters, 2006).

These earlier studies have inconsistent results that may be attributed to significant variations in measurement strategies, study quality, population, setting, sample size, perceptions of racism, and frequency of exposure. In a U.S. study of 109 Black and 225 White women, Troxel et al. (2003) observed a linear increase between combined stress (i.e., life events, ongoing stressors, economic hardships, and unfair treatment) and thickening of the carotid wall in the heart among Blacks; however, no association was observed among Whites.

Studies that do link chronic stressors experienced by Blacks (many of which may be rooted in racially motivated discriminatory practices) to increased CVD risk (e.g., hypertension), often possible physiological explanations. Researchers attribute the carotid wall thickening to increased exposure to chronic stressors begins earlier in life for Blacks, thereby resulting in an accelerated CVD incidence trajectory (Troxel et al., 2003). Cozier et al. (2006) discovered an association between racial discrimination and hypertension among some participants of from the Black Women's Health Study cohort. Most of the women in the study had experienced some form of racial discrimination. At least one experience of personally-mediated racism per month was reported by 48% of women, and was more common among obese women (BMI of 30 kg/m^2 or greater) (Cozier et al., 2006). In addition, 70% of women reported at least one situation in which they experience institutionalized racism, which was more common among highly educated women (Cozier et al., 2006). Furthermore, both forms of racism were more likely to occur among women who were born in the United States and raised in predominantly White neighborhoods (Cozier et al., 2006). However, the association between

hypertension and racism was only observed within subgroups of women. Women born primarily in the Caribbean and Central and South American had incidence rate ratios of 1.6 and 1.8 among those who experienced personally-mediated and institutional racism, respectively; and women who grew up in primarily White neighborhoods and experienced personally-mediated racism had an IRR of 1.7, but no association for institutionalized racism (Cozier et al., 2006).

Secondary analysis of the "Everyday Life for Black American Adults" Study (Brown, 2004) examined the correlation of racial discrimination and blood pressure among more than 200 Blacks (147 women, 64 men) between the ages of 25 and 79, with 86% of participants being <60 years of age. While this study explained 27% of the variance in systolic blood pressure and 17% of the variance in diastolic blood pressure, overall findings indicated that chronic stress does not have negative effects on the blood pressures of highly educated, middle income Black adults

Some researchers argue that an inverse relationship between racial discrimination and hypertension exists (Roberts et al., 2008; Singleton et al., 2008). Blacks, who do not express their feelings about being exposed to racial discrimination, may be internalizing their feelings. The lack of acknowledgement of racial discrimination, or an inability to identify it, seems to generate similar findings as those among individuals who accept or keep quiet about unfair treatment (Barksdale et al., 2009; Roberts et al., 2008; Singleton et al., 2008). For example, the Coronary Artery Risk Development in Young Adults (CARDIA) study was conducted among a sample of 831 Black men, 1134 Black women, 1006 White men, and 1106 White women between the ages of 25-37 over a 7-year period

(Krieger & Sidney, 1996). From this large prospective study, Krieger and Sidney (1996) found that there was a positive relationship between racial discrimination and elevated blood pressure among professional Blacks, but it was a U-shaped association among working-class Blacks. In fact, working-class Black women were more likely to have an increased risk difference (RD) for elevated systolic blood pressure if they accepted exposure to racial discrimination and kept it to themselves, compared to women of comparable SES who vocalized their concerns, RD=4.3, 95% CI [-0.3, 8.9]; this risk was observed to a lesser extent among for professional Black women, RD=1.3, 95% CI [-4.9, 7.6] (Krieger & Sidney, 1996). Among Black men, the risk of elevated systolic blood pressures among working-class occurred in those who accepted racial discrimination, but discussed it with others (3.6) compared to professional Black men (-0.9; Barksdale et al., 2009). Furthermore, lower blood pressures (7 mmHg and 9-10 mmHg) were observed among both working-class and professional workers, respectively, who challenge racially discriminatory treatment (Chae et al., 2010; Din-Dzietham et al., 2004; Krieger & Sidney, 1996; Roberts et al., 2008).

These findings are similar to Din-Dzietham et al. (2004), who found that among Black women in the Metro Atlanta Heart Disease Study, those who reported having zero exposure to race-based discrimination at work had an age-adjusted odd ratio of 1.4 for hypertension. Chae et al. (2010) and Roberts et al. (2008) agreed that Black men who deny having exposure to racial discrimination were at the greatest risk for hypertension. Specifically, Chae et al. (2010) found a positive association between perceived racial discrimination and increased CVD risk among Black men who had less internalization of

negative racial attitudes, particularly among Black men reporting two and three or more exposures (OR=4.93 and 4.37, respectively). Even more striking is that Black men found to have the worst CVD outcomes were those who had high internalized racism, but reported no racial discrimination exposure (Chae et al., 2010).

Conversely, Paradies' (2006) systematic review of the literature evaluated 171 empirical studies, and 36% of the studies (n=61) found a significant association between negative physical health outcomes and self-reported racism. Only 1% of the studies indicated a negative association, with the remaining 63% reflecting no association at all. Of those studies where a positive association was found, the majority of the negative physical health outcomes were associated with hypertension (n=19; Paradies, 2006). One explanation for the inconsistencies found across studies is that a consensus or standard measurement strategy to be used across studies has yet to be determined.

The ability of Blacks to cope with experiences of racism has also been shown to adversely impact health outcomes. The social history of the United States triggers guarded tendencies (e.g., suspiciousness, mistrust) among Blacks when engaging in interracial interactions, and learned coping strategies are based on continued anticipation or expectation of racially discriminatory occurrences (Myers, 2008; Singleton et al., 2008). Individual experiences interpreted as social rejection (i.e., racism) become embedded, as memories are used to cognitively appraise potentially harmful future encounters and avoid them (Mays et al., 2007). Gardner, Pickett, and Brewer (as cited in Mayes et al., 2007, pp. 213-214) conducted an experiment and found that individuals remembered experiences of social rejection more frequently than those who experienced

social acceptance. Hence, researchers essentially have identified racism as pyschosocial stressor that causes many Blacks to be in a constant state of guarded awareness of any perceived differences that may be attributed to race; yet an individual's ability to cope with this constant stressful state varys not only by individual but also by situation or circumstance (Brondolo et al., 2007; Brondolo, Brady ver Halen, et al., 2009 Singleton et al., 2008).

In laboratory-based studies, the chronic stress imposed by racism and the ability to cope with the ongoing appraisal of environmental and interpersonal situations has been suggested to explain the differences in rates of hypertension between Blacks and Whites (Gee et al., 2012; Singleton et al., 2008). Guyll et al. (2001) contended that although Black women displayed higher diastolic blood pressure (DBP) than White women after experiencing subtle forms of discrimination (e.g., not being treated with respect or courtesy, treated as though others are better than you, receiving poorer quality customer service), DBP was even higher among Blacks when they perceived the discrimination was racially motivated.

Furthermore, researchers (Hicken et al., 2014; Singleton et al., 2008; Vines et al., 2006) agree that the extent to which Black's blood pressure levels are influenced by their exposure to racism depends on the the strategies they use to cope with the the stressful experience. For example, a random survey of Black women aged 20-80 found that women who utilized passive coping strategies (e.g., accepting or internalizing experiences of racial discrimination) were 4.4 times more likely to have hypertension than women who responded with active coping (e.g., addressing their anger, talking to

someone; Krieger, 1990). Singleton et al. (2008) found that Blacks who chose to avoid or passively cope with racism had higher systolic blood pressure (SBP) and DBP (124.18 and 78.18 mmHg, respectively) compared to those who addressed their feelings (117.59 and 74.15 mmHg, respectively). However, other researchers challenge the correlation of BP to exposure to racism and chronic stress, because laboratory-based studies may not accurately depict the impact of racism on BP under "real-world" conditions (Brondolo et al., 2007; Hill et al., 2007).

Measurement of racial discrimination. In measuring racial discrimination, it is important to delineate the factors that contribute to the challenges in clearly determining the impact of racism. Today's expressions of racial discrimination may occur as subtle, perhaps unintentional, behaviors (e.g., interrupting an individual attempting to share information or asking fewer questions vital to health), which are sometimes difficult to recognize and even more difficult to prove (Williams & Mohammed, 2013). Furthermore, racial discrimination may occur across multiple domains (e.g., neighborhood demographics, educational quality, employment opportunities, access to resources, health outcomes) and have cumulative effects on an individual or population (Jones, 2000; Krieger, 2012; Sims et al., 2012; Tarman & Sears, 2005; Williams & Mohammed, 2013; Williams, 2012).

There are several different instruments that have been used to measure racial discrimination. While some of these scales are commonly used and have provided reasonable support for racial discrimination as a contributor to poorer health outcomes, there are some important hindrances to the consistency and reliability of these

instruments (Atkins, 2014; Blank et al., 2004). Researchers have frequently used race, racism, and discrimination interchangeably; therefore, there is no consistent terminology to assess the construct of racial discrimination (Bastos, Celeste, Faerstein, & Barros, 2010; Sellers, Bonham, Neighbors, & Amell, 2009; Sweet, McDade, Kiefe, & Liu, 2007; Williams et al., 2012). In addition, the gauge for intensity, duration, and frequency of exposure to racial discrimination varies across scales (Atkins, 2014; Bastos et al., 2010; Blank et al., 2004; Sims, Wyatt, Gutierrez, Taylor, & Williams, 2009). Although racism is a multidimensional construct, researchers typically only utilize instruments that measure a single aspect of how racism impacts a population (Sims et al., 2012, 2009). There are three scales commonly used to capture these singular measurements of discrimination:

- The Discrimination Scale (Krieger & Sidney, 1996; Krieger, 1990) measured individuals' perceptions of racial discrimination, as well as their responses to perceived unfair treatment, to examine its association to elevated blood pressure. Results from the CARDIA Study were based on approximately 4,000 Black and White total adults in a prospective multisite study across multiple life domains during the 7th year of the study (Krieger & Sidney, 1996). Although there were slightly more Whites, the racial composition was relative comparable. Krieger and Sidney (1996) found that 77% and 84% Black women and men, respectively, experienced racial discrimination in one of the seven domains assessed, with at least 50% of them having experienced racial discrimination in three or more settings. Furthermore, Blacks (particularly women) were almost 20% more likely

than Whites not to respond or act on the unfair treatment they experienced (Krieger & Sidney, 1996). This instrument was also found to have high reliability for both Blacks and Whites (0.81 and 0.77, respectively; (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005).

- Everyday Racial Discrimination Questionnaire (Williams et al., 1997) is among the most widely used discrimination scales in epidemiologic and public health research. It measures perceptions of discrimination based on socioeconomic position and the impact of day-to-day exposures to perceived race-related stress on health outcomes. Data from this study consisted of approximately 1100 adults from the Detroit metro area during 1995, with relatively equal representation of Blacks and Whites (Williams et al., 1997). An assessment of racial differences in indicators of SES, social class, and stress indicated that Blacks has significantly higher values for all indicators, except chronic stress. For example, Williams et al. (1997) found that Blacks were 1.6 times more likely to have less than high school education, four times more likely to have income less than $10K, and almost twice as likely to not have supervisory/managerial type jobs. Furthermore, reported Blacks being twice as likely to experience financial and stress and life-events, as well as 2-7 times more likely to experience multiple discriminatory events (Williams et al., 1997). Although Blacks had poorer overall health status, the psychological well-being was poorer among Whites, suggesting that Blacks have better coping mechanisms. Although this scale has been found to have good internal reliability (0.88; Williams et al., 1997), the validity of the instrument has

been questioned due to the neutral terminology used to define/describe racial discrimination (Bastos et al., 2010).

- Perceived Racism Scale (McNeilly et al., 1996) was developed to measure the frequency of exposure to both individual and institutional racial discrimination across multiple domains (i.e., job-related, within academic settings, and in general public domain). This scale also measures exposure to racist statements, as well as individual's emotional and behavioral coping responses. The sample size of this study was considerably smaller (< 200 adults), more unevenly distributed by gender (almost twice as many women than men), and younger (predominately college students) than the previous studies (McNeilly et al., 1996). In addition, there was no White comparison population. This scale displayed the greatest amount of internal reliability, with a score of 0.96 for lifetime discrimination and 0.92 for behavioral coping (McNeilly et al., 1996).

Researchers (Bastos et al., 2010; Williams et al., 2012) agree that the effect that exposure to racial discrimination potentially has on pathways of disease and health outcomes may vary depending on how racism is manifested (e.g., job-related discrimination, exclusion/rejection, threats/aggressive behaviors or acts). Little or no evidence is available regarding the effect of internalized racism, whether dose-response from exposure exists, how racism exposure interacts with other social factors to influence health outcomes, and how racial discrimination can be comprehensively assessed over the lifecourse (Chae et al., 2010; Williams & Mohammed, 2013). Hence, there is a need to address some of these conceptual and measurement issues.

Racial discrimination and health care. Exposure to racism has also been speculated to impede trust in the healthcare system, specifically the development of beneficial patient-provider relationships (Brondolo, Brady ver Halen, et al., 2009; Hausmann, Kressin, Hanusa, & Ibrahim, 2010; Klonoff, 2009; Musa et al., 2009). For example, a study of a diverse cohort of approximately 55,000 California residents revealed that compared to Whites, all other race/ethnic populations were less likely to receive preventive care services (Trivedi & Ayanian, 2006). Study participants who reported discrimination exposure were statistically less likely to receive four of the six preventive care services (i.e., diabetic foot exams, HbA1c testing, cholesterol testing, and influenza vaccination), even after adjusting for demographics, perceived health status, and frequency of doctor visits (Trivedi & Ayanian, 2006). Interestingly, Blacks were also less likely than Whites to receive four of the six services even after adjusting for perceived discrimination (Trivedi & Ayanian, 2006). However, Hausmann, Jeong, Bost, and Ibrahim (2008) argued that although persons who perceive negative discrimination had lower healthcare utilization rates, those rates were not statistically significant after controlling for demographic variables (e.g., race, education, income, health coverage), and other factors that guide patient behaviors should be considered. For example, Blacks may be less likely to utilize or receive healthcare services in part due to limited trust in the healthcare system.

Researchers posit mistrust of the healthcare system and healthcare providers, largely initiated by the Tuskegee Syphilis study's legacy, but also including factors such as other systematic efforts to 'treat' minority groups without adequate informed consent,

conspiracy theories, physician biases, and disparities in access to health care services (Klonoff, 2009; Musa et al., 2009). In a study of Medicare enrollees aged 65 years and older in Pennsylvania, Blacks were found to have less trust in their healthcare provider, and more trust in social networks that may provide health information (e.g., family, friends, church leaders) than Whites (Musa et al., 2009). Musa et al. (2009) further postulated that the level of trust observed by older Blacks in this population is likely to be higher than that of Blacks in younger age groups, due to an increase need for continuity of care and more frequent provider-patient interactions in later life. These findings support the need for standard tools to measure aspects of racism (e.g., discrimination in healthcare). Improved techniques for the measurement of racial discrimination may provide parameters by which healthcare providers can improve or repair the generational distrust of Black patients. The elevation of racial discrimination in healthcare may potentially constitute significant improvements in health disparities.

SES associated stress. In earlier sections of this chapter, the relationship of SES to health outcomes was addressed. The social benefits afforded to individuals of higher SES may create assumptions that they experience less overall chronic stress than individuals of lower SES. Myers (2008) argued that being of lower SES and person of color is a dual social burden. As such, Blacks are commonly and disproportionally burdened by complex macrosocial issues (e.g., poverty, residential segregation, resource deprivation), which demands overutilization of already deprived community resources and significantly increases one's exposure to chronic stress.

Some reseachers agree that individuals of lower SES are more likely to experience repetitive exposure to economic hardships, other stressful life events, or even some combination of the two over their lifecourse or within a short period of time (e.g., 1-2 years; Libman, Fields, & Saegert, 2012; Kapuku, Treiber, & Davis, 2002). Some studies indicate that Blacks with more education or middle-class income are more likely to report racially-motivated experiences, and suffer more emotional distress as a result of such experiences than lower income Blacks (Sellers et al., 2009; Sellers, Neighbors, & Bonham, 2011); whereas, other research depicts lower income Blacks as more likely to be subjected to racism due to limited decision-making ability over work-related demands (e.g., number of tasks to complete in an allotted period of time, length of a workday, flexibility for time off (Myers, 2014; Sellers et al., 2009).

Skin color. The skin color of Blacks varies widely from very fair to very dark complexions. Some researchers contend that skin color, as opposed to race, predisposes Blacks to hypertension due to a more graduated risk of racism exposure (Monk, 2015; Hall, 2007; Sweet et al., 2007). An empirical test to determine whether or not skin color was correlated with differences in exposure to racial discrimination revealed that dark-skinned Blacks were approximately 11 times more likely to have frequent exposures to racial discrimination than light-skinned Blacks (Klonoff & Landrine, 2000).

Klonoff and Landrine (2000) also found that men were three times more likely than women to have a high frequency of exposure to racial discrimination. Hall (2007) maintained that dark-skinned Black males are more likely than light-skinned Black males to be vilified by Whites, the media, and law enforcement as having committed heinous

crimes, simply because the contrast of their skin color represents nonconformity with mainstream American norms. Black males, particularly those of darker skin, are acutely aware that "given the power of the media to impose and to monitor norms, such victimization may keep those who are otherwise ordinary law abiding citizens under constant emotional and psychological stress" (Hall, 2007, p. 207).

Analysis of CARDIA study data indicated that Blacks of darker skin tone had higher systolic blood pressures (118.6 mmHg vs. 115.7 mmHg) and were more likely to take antihypertensive mediation (14.37% v. 9.34%) compared to Blacks of lighter skin tone, respectively (Sweet et al., 2007). These findings were attributed to darker skin tone Blacks experiencing more chronic stress associated with racial discrimination than lighter skin tone Blacks (Sweet et al., 2007), suggesting that the American society generally views darker skin color as more threatening.

Skin color has also been considered a potential confounder of SES (Klag et al., 1991; Sweet et al., 2007). For example, researchers have postulated that dark-skinned Blacks are more likely to have lower income and less prestigous jobs than light-skinned Blacks (Sweet et al., 2007). Researchers disagreed about whether or not skin color and SES interact to affect blood pressure (Hall, 2007; Klag et al., 1991; Sweet et al., 2007). Klag et al. (1991) observed a 2-point increase in SBP and DBP for each measured increase in skin darkness, with dark-skinned Blacks having the highest blood pressures.

Conversely, Sweet et al.'s (2007) findings illustrated that as SES increased, blood pressure decreased among light-skinned Blacks in a protective pattern similar to that observed among Whites; however, increasing SES had no effect on blood pressure

among dark-skinned Blacks. Although Klag et al. (1991) suggested that dark-skinned Blacks suffer greater burden of psychosocial stressors (i.e., racism) and hypertension attributable to the combination of darker skin color and SES, the association of factors may actually be more complex. However, Sweet et al. (2007) supported Hall's (2007) argument that it is the social experiences, more so than SES, of dark-skinned Blacks that contribute to higher blood pressures.

There does not appear to be consensus about whether or not specific groups of Blacks have greater exposure to racism or discriminatory practices than others. The lack of consensus regarding perceived racism within the Black population results from inconsistencies in the focus of the study participants (e.g., age, SES, skin color), differences in how racism is measured, whether or not coping ability is assessed, and how the process used to connect racism to health outcomes (Sellers et al., 2009; Sweet et al., 2007). In addition, the perceptions of exposure to racism and ability to cope with occurrences vary greatly across individuals and events. Further investigation is necessary to better understand the association racism has to hypertension, and identify opportunities for social changes that may improve this health disparity. Empirical studies have demonstrated that racism strongly influences SES, and SES is a known indicator of health risk. Therefore, there is a convincing argument to investigate SES and racism concurrently.

Conceptual Framework

There are multiple models that have been developed to explore the relationship of racial discrimination and health outcomes, and each model focuses on different aspects of

their connection. Some frameworks proved to be less of an appropriate fit for this study than others due to the approach for measuring the racism-health relationship. For instance, Pascoe and Smart Richman (2009) developed a model that measured different pathways by which experiences of perceived racial discrimination may mentally and physically affect health outcomes. The pathways illustrated in this model are mediated by physiology, behavioral, and physical stress responses (Pascoe & Smart Richman, 2009), and do not consider the role of institutional, socioeconomic, or cultural exposures. In addition, the impact of racism exposure over the life course is not measured. Cuffee et al. (2012) developed a conceptual model adapted the Model of Perceived Discrimination and Health Outcomes to identify intervening opportunities between perceived discrimination and hypertension. However, the basis of this model was to hypothesize that the pathway racial discrimination to adverse health outcomes was influenced by aspects of the patient-provider relationship (i.e., trust in providers, communication, patient self-efficacy), along with genetic and sociodemographic factors (Cuffee et al., 2012).

Krieger (2012a) recently crafted a complex ecosocial model to illustrate how exposure to racism occurs simultaneously in occupational, environmental, and social domains, along with historical and generational context, over the life course in a multi-level ecosystem (e.g., area, regional, national). Krieger's study (2012a) found that while more than 85% of participants reported high exposure to at least 1 of the 3 domains for racism, Blacks experienced the most racism exposure (with 20 to 30% experiencing high exposure in all three domains). Moreover, individuals experience the impact of exposure, susceptibility, and resistance to injustice practices simultaneously because their race,

SES, gender, and other social characteristics are embodied simultaneously. Therefore, an ecosocial approach is needed in order to fully understand the impact of both individual and collective harm on the health of Blacks. Although this framework has a comprehensive approach, it considers indicators of social and economic depravity that are more associated with the domains (e.g., exposure to toxins and pathogens, marketing of harmful products to targeted audiences, and resistance or injustice to discriminatory behaviors and practices) than the individual. Therefore, the pathways to assess the impact of racism in this framework do not align with the focus of this study.

Gee et al. (2012) developed a conceptual model describing how racism affects the life course trajectory to create disparities in life expectancy. Researchers compared a typical life course trajectory with a life course trajectory influenced by racism to illustrate that individuals who do not have exposure to racism experience longer prenatal, education, work, and retirement periods, which culminates in a longer life trajectory (Gee et al., 2012). Conversely, individuals in a life course trajectory shaped by racism experience greater mid-life periods of poor health, incarceration, and unemployment, thereby creating a shorter life trajectory (Gee et al., 2012). However, this framework does not include indicators of SES, which overlooks a key element of the study.

There are two theoretical frameworks identified that most closely align with the constructs in this literature review and delineate the relationship between them. First, Harper et al. (2011) developed a model to examine how the role of social determinants in CVD outcomes is embodied by life course, historical, and geographic context. As shown in Figure 5, the model implies that the level of CVD in a population is contingent upon

socioeconomic position determining the prevalence of risk factors; however, both the distribution of socioeconomic position and risk factors are dynamically influenced by macrosocial conditions across time and place (Harper et al., 2011). This study was used for a global examination of the interconnected pathway for investigating CVD, and imparts empirical evidence to show that socioeconomic depravity can occur without causing increases in CVD risk (Harper et al., 2011). Hence, Harper et al. (2011) urges that "their effect [social determinants] are conditional on the strength of the links between macrosocial changes and more proximal causes of disease" (p. 54). Although this study did not address issues of racism or racial discrimination as a macrosocial condition, and no publications were identified which applied this model specifically to a study of racial discrimination, racial discrimination has previously been defined as a social determinant of health. Therefore, in addition to Harper's framework (2011), another framework was be used to systematically outline the proximal pathways by which exposure to racism leads to adverse health outcomes.

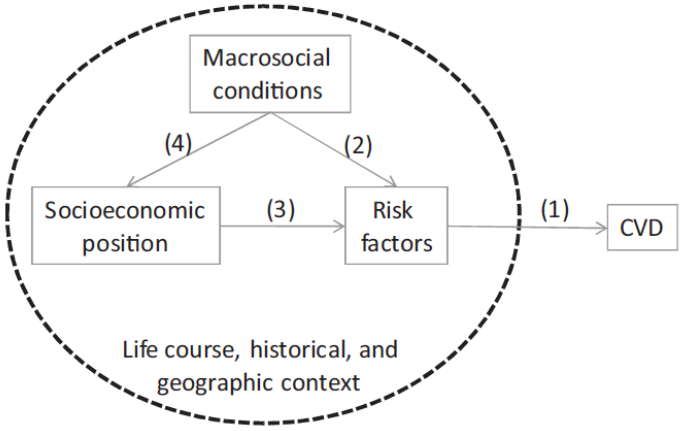

Figure 5. Macrosocial conditions, socioeconomic position, risk factors and CVD risk historical, geographic, and life course context. From "Social Determinants and the Decline of Cardiovascular Diseases: Understanding the Links," by Harper et al., 2011,

Annual Review of Public Health, 32(1), p. 40. Reprinted with permission requested from *Annual Reviews of Public Health*. The model implies that the prevalence of risk factors establishes the level of CVD in a population (arrow 1), these risk factors are influenced by both the extent of macrosocial factors (arrow 2) and socioeconomic position (arrow 3), socioeconomic position is determined by macrosocial conditions (arrow 4), and all of these constructs are dynamically connected and embedded in multiple environments.

Williams and Mohammed (2013) recently created a framework for examining racism and health (Figure 6). This model illustrates that racism and social status create the proximal pathways that link risk factors to adverse health outcomes. Specifically, Williams and Mohammed (2013) contend that "Racism is not only the determinant of intervening mechanisms, but its presence as a fundamental cause in a society can alter and transform the other social factors and can exacerbate the negative effects of other risk factors for health" (p. 1158). Because inequities occur at each stage of the process, it is often difficult to fully grasp the multi-layer, and likely cumulative, impact that racism has on health. Although not captured in the framework itself, researchers do recommend that exposure to racial discrimination be measured over the life course (Williams & Mohammed, 2013). Hence, although the entire framework was not be investigated in this study, it supports the constructs previously discussed in this chapter, and improves understanding of their relationship.

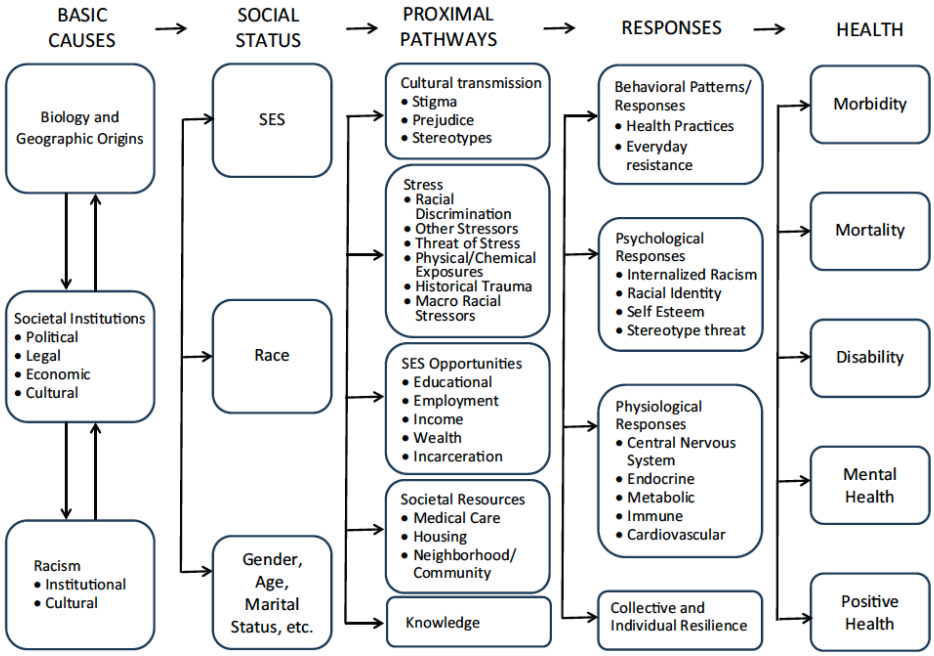

Figure 6. A framework for the study of racism and health. From "Racism and Health I: Pathways and Scientific Evidence," by Williams and Mohammed, 2013, *American Behavioral Scientist*, 57(8), p. 1157. Reprinted with permission from Sage Journals.

CVD Burden in Mississippi

Mississippi has the highest prevalence of CVD in the nation (CDC, 2013a), which is estimated to be approximately 30% higher than the overall U.S. prevalence rate ("The 2005 Mississippi State of the Heart Report," 2005). The state of Mississippi reported, in 2002, that the overall CVD mortality rate was 420.7/100,000 compared to 319.0/100,000 for the U.S. (Taylor et al., 2005; "The 2005 Mississippi State of the Heart Report," 2005). Moreover, data show elevated trends for CVD mortality rates among Blacks in Mississippi, particularly men, have remained since 1979, compared to rates for their White counterparts (Jones et al., 2000; "The 2005 Mississippi State of the Heart Report,"

2005). Specifically, the Black-White gap reached as high as 46% higher in 1995 (Jones et al., 2000), but was reported at 18% in 2004 ("The 2005 Mississippi State of the Heart Report," 2005). Of greater concern, Mississippi investigators agreed that Black men experienced alarming rates of premature CVD mortality (under the age 65), which increased from 27% in 1979 to 45% in 2004; yet, rates among White men hovered between approximately 25% to 30% during the same time period (Jones et al., 2000; "The 2005 Mississippi State of the Heart Report," 2005). It is likely that a combination of factors (e.g., elevated rates of hypertension, increased exposure to lower SES, and stress induced by racism) may explain the differences in black-white mortality distribution.

Not only is Mississippi a state where Blacks have some of the highest rates of hypertension in the country, but it is also a state with high poverty and a complex racial history. There were 10 states in 2009 that had hypertension prevalence rates greater than or equal to 30.6%; however, the prevalence of hypertension in Mississippi was the highest in the country (U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion, 2010). Similar to CVD mortality rates in Mississippi, Jones et al. (2000) estimated that Black men experience hypertension rates roughly four-fold greater than White men.

The U.S. Census Bureau ("Mississippi QuickFacts from the U.S. Census Bureau," 2011) indicated that Mississippi has a higher population of Blacks and individuals living below the poverty compared to the United States overall (37.0% versus 12.6% and 21.8% versus 14.3%, respectively). Finally, Mississippi has a long, and sometime highly

publicized, history of racism. Even as recently as August 2011, blatant hate crimes were committed against a Black male for no other reason than the color of his skin (CNN, 2011). Though some may consider this an isolated incident, Blacks in Mississippi may believe that they experience a greater degree of racial assaults on a regular basis, and therefore may face higher levels of race-based stress.

Review of the Jackson Health Study

The Jackson Heart Study (JHS) cohort is a viable population to investigate the correlation of psychosocial factors (e.g., racism) and SES on hypertension risk among Blacks. JHS was a collaborative longitudinal study primarily focused on understanding and preventing the causes of CVD among Blacks. Since 2000, JHS has been the largest single-site epidemiologic study to understand CVD among Blacks (Taylor, 2003), thereby creating the opportunity to investigate strategies to prevent adverse CVD outcomes among Black in manner that is likely more relevant and comprehensive than other studies.

JHS grew out of findings from the Atherosclerosis Risk in Communities (ARIC) Study, which indicated that Blacks who had a myocardial infarction were not only more likely to die before being admitted to the hospital and less likely to receive cardiac process, but also more likely to the be hypertensive and of lower SES (Taylor et al., 2005). Although there were four ARIC study sites, the Jackson, Mississippi location was the only one that was predominantly Blacks, and researchers initiated the JHS to further understand CVD preventative strategies that may be more impactful among Blacks. In addition, Mississippi has the largest proportion (36%) of Blacks in the United States

(Taylor, 2003). A cohort of 5,302 Blacks was recruited from the three counties that comprise the Jackson metropolitan area, through a combination of recruitment strategies: random (17 %), volunteer (22%), current enrollment in the ARIC Study (30%), and family members of enrollees (31%). All participants were required to be noninstitutionalized adults between the ages of 35-84; however, adult family member aged 21-34 were also included the study. Furthermore, retention of study participants for long-term follow up is high due to the limited migration of this population outside of the metropolitan area (Taylor, 2003).

To date, JHS has collected data at three different intervals, including Exam 1 (2004), Exam 2 (2005-2008), and Exam 3 (2009-2012). Data collection includes a mixture of biomedical sampling (i.e., obtained from medical history, physical exam, and blood and urine samples) and personal interviews of each participant; hence, researchers have been able to compile an wide array of information about not only traditional CVD risk factors, but also aspects of psychosocial functioning, spirituality, stress, racism and discrimination exposure, socioeconomic position, and access to health resources, which may be compared to medical records over time (Taylor et al., 2005). The extensive data collected by the JHS furthers the opportunity to provide evidence related to the ongoing debate on the effect that racism may have on health outcomes (e.g., hypertension) among Blacks, and whether or not measures of SES contributes to the effect observed.

Overview of JHS study findings

As of 2012, three studies have been conducted to explore racism in the JHS cohort. One study was conducted to test the multidimensional Jackson Heart Study

Discrimination (JHSDIS) Instrument (Sims et al., 2009). Another investigated the association of perceived discrimination with hypertension among cohort participants (Sims et al., 2012). Finally, Hickson et al. (2012) examined the association between discrimination and abdominal fat among JHS men and women.

Preliminary findings from the testing of the JHSDIS instrument (JHS data collected from 2000-2004) revealed that racial discrimination is a cause for concern among Blacks in Jackson, MS. Roughly all JHS participants (n=5200) completed the JHSDIS instrument. Most Blacks indicated having experienced some type of discrimination, and nearly half of the discrimination documented was attributed to race (Sims et al., 2009). Each construct was also analyzed to determine differences by age group (21-34, 35-44, 45-64, and 65+) and sex. There was a higher occurrence of everyday discrimination compared to other types, particularly among those ages 21-44. Among the nine domains of everyday discrimination assessed, individuals 21-44 perceived discrimination at higher rates (68-74%) than individuals of all other age groups (22-68%) for five of the nine domains (Sims et al., 2009), but were also higher for the remaining domains. However, the population subgroup found to be most impacted by discrimination depended on the construct of discrimination being measured.

For major life events, racial discrimination occurred more frequently among individuals aged 35-64 compared to all other age groups, and was reported most frequently at work (68-70% vs. 54-64%, respectively) or when trying to get a job (49-52% vs. 31-45%, respectively; Sims et al., 2009). As age groups increased, individuals seem to have been more impacted by the burden that discrimination had on their life.

Among individuals age 21-34, 14% reported that discrimination made life stressful, 20% reported that it interfered with the fullness of their life, and approximately 24% reported it made life hard (Sims et al., 2009). However, rates for the same measures were 24%, 38%, and 42%, respectively among those 45-64 (Sims et al., 2009). Minimal difference was reported by age for how skin color affected treatment by Blacks and Whites. Across all constructs and for the majority of the domains, males reported higher rates of perceived discrimination even though there are almost twice as many females in the study. In addition, discrimination rates were lower overall in the 65 and older age group, which may be attributed to females living longer, and therefore comprising a larger proportion of the respondents in this age category.

Sims et al. (2012) analyzed JHS data to ascertain if health behaviors (e.g., unhealthy eating, sedentary lifestyle, tobacco, and alcohol use) partially mediated a positive association between perceived discrimination and hypertension among Blacks, and if effect modification by gender existed. The dichotomous responses for domains of each discrimination category were combined to generate a score, and then stratified into low or high quartiles based on race as an attributor. As such, the association between perceived discrimination and hypertension was estimated before and after controlling for age, gender, health behaviors, and SES (i.e., education, income, and occupation). Data were also stratified by gender, but no interaction was found (Sims et al., 2012).

Sims et al. (2012) found that although the prevalence of hypertension was slightly higher among women (64%) compared to men (60%), men reported higher levels of everyday, lifetime, and burden of discrimination attributed to race (27.1%, 45.8%, and

40.3%, respectively) compared to women (18.4%, 33.7%, 29.8%, respectively). However, women were more likely than men to indicate that life was very stressful as a result of lifetime discrimination (27.2% vs. 19.6%, respectively; (Sims et al., 2012).

Researchers found a positive association between high levels of lifetime discrimination and measures of high SES; however, health behaviors varied in terms of the category of discrimination which were associated as depicted in Table 3 (Sims et al., 2012). Higher BMI was associated with everyday discrimination; physical activity with both everyday and lifetime discrimination; and smoking, dietary fiber, and sodium intake with everyday and burden from discrimination (Sims et al., 2012). Alcohol intake was not found to have an association to discrimination.

Furthermore, Sims et al. (2012) found that the association of hypertension and discrimination was largely influenced by the measures used to define discrimination. Hypertension was not associated with everyday discrimination; however, the participants in the JHS had a 4% increase in hypertension prevalence for each increase in standard deviation for lifetime discrimination, PR=1.04; 95% CI [1.01,1.06] (Sims et al., 2012). Even after controlling for health behaviors, the association persisted. Additionally, individual who reported lifetime discrimination in at least one domain and had high burden of discrimination were also found to have a 9% higher prevalence of hypertension, even after controlling for demographic factors, such as age, gender, SES, PR for Q4 vs. Q1= 1.09, 95% CI [1.02,1.16], P for trend=0.01 (Sims et al., 2012). There was a 2% increase in hypertension prevalence for each increase in standard deviation in burden of discrimination; however, the association loses its statistical significance after

controlling for health behaviors, PR=1.02, 95% CI [1.00,1.05] (Sims et al., 2012). Interestingly, high levels of lifetime discrimination were associated with hypertension regardless of the attributing factor (racial or nonracial), PR=1.07, 95% CI [1.01,1.14] compared to PR=1.08, 95% CI [1.00,1.16], respectively); however, elevated hypertension rates were associated with an increased burden of discrimination only when it was attributed to racial factors, PR attributed to race=1.08, 95% CI [1.02, 1.14] compared to PR attributed to nonracial factors=1.03, 95% CI [0.96,1.10] (Sims et al., 2012).

The study conducted by Sims et al. (2012) is among the first to examine multiple measures of discrimination and their association with hypertension, and revealed that high levels of lifetime discrimination and burden from discrimination were most impactful on blood pressure. This finding provides weight for understanding how the stress of discriminatory experiences, particular those that impact the necessary functions of one's life (e.g., the ability to work, live in a safe environment, having adequate housing) and the extent of exposure (e.g., over the course of one's lifetime), contribute to adverse health outcomes. Sims et al. (2012) posit that the lack of association between everyday discrimination and hypertension is because this measure of discrimination may only reflect brief changes in blood pressure; whereas high levels of lifetime discrimination or high burden from discrimination reflect more sustained blood pressure changes (Sims et al., 2012). Although the JHS is the largest CVD study among Black conducted to date, its results are not generalizable to Blacks nationwide. The history of racial tension in Mississippi may increase the potential for lifetime discrimination or

greater burden, even from a single instance, resulting either from personal experiences, attitudes, or beliefs regarding what is fair.

Research Gaps within JHS

The JHS data provides significant insights into the interactions between race and other factors related to health status. However, continued investigations of these data to examine the interaction of other factors that may explain the CVD disparities observed among Blacks are needed. Use of different study designs, better discrimination measurements, and effect modification of coping strategies are some of the recommended research methods endorsed by Sims et al. (2012) to improve understanding of how discrimination affects hypertension.

It is important to note that JHS data has not been examined to assess the association between discrimination and hypertension across all exam periods for which data was collected. In addition, measures of SES only reflected the participant's education, income, and occupation; however, JHS also collected data pertaining to parental SES and neighborhood SES. Perhaps exploration of these measures of SES, either individually or collectively, can further guide our understanding of how CVD health disparities manifest.

Conclusions (Impact for Social Change)

The purpose of this study is to provide additional support to the argument of whether or not racism contributes to the health inequalities between Blacks and Whites, and how. The literature cited throughout this chapter provides insight on how an individual's SES, beginning in childhood and continuing over their lifecourse, strongly

influences multiple aspects of their social and economic well-being. In addition, the cited literature described mixed results about whether or not racial discrimination contributes to the health disparities experienced by Blacks. The lack of standardized measurement strategies used to assess racism are likely a major reason for those inconsistencies. Hence, further research should seek to develop methods to define the impact of racism exposure over the lifecourse. Research gaps also exist in understanding whether or not the racial discrimination that Blacks experience over their life course are moderated by an individual's level of SES.

The study aims to advance Sims' research on the influence of perceived lifecourse racial discrimination and levels of SES, measured over the lifecourse, on increased rates of hypertension and CVD outcomes among Blacks in Jackson, MS. Chapter 3 identifies research questions and define the methods that were used to address these research gaps. The findings contribute greater understanding to the racial dynamics that influence poor health outcomes, and provide evidence for needed changes in policies, practices, infrastructure, and/or social norms in order to improve the racial disparities that exist for CVD and other diseases.

Chapter 3: Research Method

Introduction

The purpose of this study was to demonstrate whether or not racism contributes to the cardiovascular disease (CVD) health disparities observed among Blacks in the Jackson Heart Study (JHS), and to what extent. More specifically, this study investigated how Blacks in the JHS cohort experience racism at different SES levels, and how the interaction between SES mobility and racism influences the extent to which hypertension leads to CVD outcomes.

This research project was conducted using secondary data from Exam 1 of the Jackson Heart Study (JHS). The JHS is a large, single-site cohort study that has prospectively examined genetic and psychosocial factors that influence hypertension, heart disease, stroke, and other health outcomes among the Black population of more than 5,300 men and women in the Jackson, MS metro area (Fuqua et al., 2005; Taylor, 2003, 2005b; Taylor et al., 2005). During Exams 1 (2004) and 3 (2009-2012), JHS collected data on constructs used to measure discrimination in the domains of everyday experiences, major life events (lifetime), burden of discrimination, and the effect of skin color. For the purposes of this study, only a cross-sectional analysis of data collected from the baseline period (Exam 1) was used to explore the interaction between SES mobility over the lifecourse and lifetime racial discrimination (i.e., SES-Racism Effect).

Because the Exam 3 were not available for analysis at the time of this study (M. Sims, personal communication, August 29, 2013), this study provides some baseline information about whether Blacks who report higher burden of the SES-Racism Effect

are more likely to experience a higher prevalence of hypertension and CVD compared to Blacks who report lower burden of the SES-Racism Effect. The results of this study may provide further opportunity to compare the impact of the SES-Racism Effect at different time periods.

In this chapter, the methodology used to conduct this research study is described in detail. This chapter begins with the identification of the research questions to be answered. Next, a description of the research design, study population, instruments, variables, and the analytic plan used to guide this investigation are illustrated. The chapter concludes with a discussion of the methodological concerns that pose potential threats to the study's validity.

Research Design and Rationale

This study was based on a quantitative cross-sectional analysis of secondary data to examine the causal pathway by which racial discrimination impacts CVD. To understand this pathway, the presence of a SES-Racism Effect was examined by understanding the relationship between SES mobility and perceived lifetime racism. Additional analysis was conducted to explore whether age, gender, or both influence the strength or direction of this relationship. Figure 1 below illustrates the causal path using a moderated effect; gradient shading is used to denote the change in the strength or direction between the independent variable (i.e., levels of SES mobility) and the dependent variable (i.e., levels of perceived lifetime racial discrimination exposure). This model (Figure 2) was repeated with a different dependent variable (i.e., levels of burden attributed to lifetime racial discrimination). Next, I examined how SES mobility and

perceived lifetime racial discrimination independently effect the association between hypertension and CVD. Figure 3 provides a visual illustration for how SES mobility, perceived lifetime racial discrimination exposure, and burden may independently moderate the pathway between hypertension and CVD. The previously described analyses investigated perceived lifetime racism using constructs to independently measure both racism exposure and burden. Based on the findings of these analyses, the racism construct determined to have the strongest association was used to define the SES-Racism Effect. Finally, I explored whether the SES-Racism Effect (e.g., low, high) modifies the association between hypertension and CVD outcomes. The relationship between hypertension and CVD has been well-established in the literature (Flack, Ferdinand, & Nasser, 2003; Go et al., 2012; Williams, 2009; Wyatt et al., 2008). Figure 4 illustrates how the SES-Racism Effect may moderate the causal pathway between hypertension and CVD. Hence, this study focused on four main research questions. These research questions are listed below, along with the related hypotheses and statistical tests. In addition, all hypotheses were adjusted for the following covariates: age, gender, BMI ($kg/m^{2)}$, smoking status (current, former, never), physical activity score, diabetes status, alcohol consumption, diet (% fat consumption), total cholesterol (mg.dL), LDL (mg/dL), HDL (mg/dL), and John Henryism.

Research Questions

Research Question 1 (RQ1): What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of

lifetime racial discrimination, as measured by the occurrence of cumulative perceived lifetime discrimination exposure attributed to race?

- Hypothesis 1: Increasing levels of SES mobility are associated with decreasing levels of perceived lifetime discrimination exposure attributed to race after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.
- Null Hypothesis 1: The association between levels of SES mobility and levels of perceived lifetime discrimination exposure was attributed to race after adjusting for identified covariates.

If an association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was identified, the following subhypotheses were also tested (Figure 1):

Hypothesis 1b: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was inversely moderated by age.

Null Hypothesis 1b: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by age.

Hypothesis 1c: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was more strongly moderated by males than females.

Null Hypothesis 1c: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by males than females.

- Independent variables: Levels of SES mobility (4 categorical variables), Age (5 interval variables), and Gender (2 categorical variables)
- Dependent variable: Levels of Lifetime Racial Discrimination Exposure (3 categorical variables)
- Statistical Test: Polytomous Logistic Regression

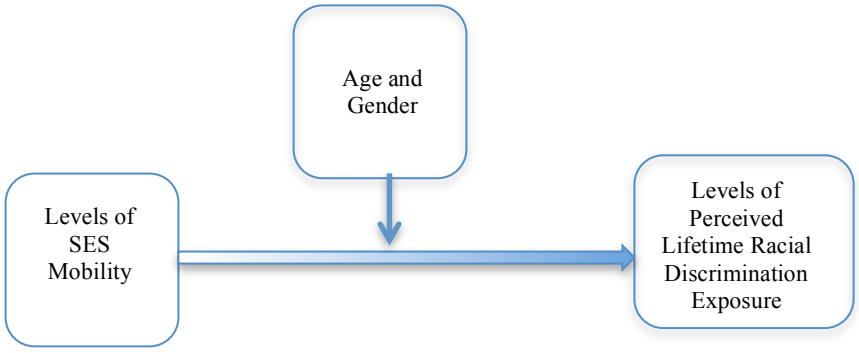

Figure 1. Causal pathway between Levels of SES Mobility and Levels of Perceived Lifetime Racial Discrimination moderated by Age and Gender.

Research Question (RQ2): What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of burden attributed to perceived lifetime racial discrimination, as measured by the extent of

life stressfulness, difficulty, and productivity as a result of perceived lifetime discrimination attributed to race?

- Hypothesis 2: Increasing levels of SES mobility are associated with decreasing levels of burden attributed to perceived lifetime racial discrimination after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.
- Null Hypothesis 2: There are no statistically significant associations between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination after adjusting for identified covariates.

If an association between levels of SES mobility and levels burden attributed to perceived lifetime racial discrimination was identified, the following subhypotheses were also tested (Figure 2):

- Hypothesis 2b: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was inversely moderated by age.
- Null Hypothesis 2b: There are no statistically significant differences in the association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination when moderated by age.

- Hypothesis 2c: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was higher in males than females.
- Null Hypothesis 2c: The association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination was moderated by gender.
    - Independent variables: Levels of SES mobility (4 categorical variables), Age (5 interval variables), and Gender (2 categorical variables)
    - Dependent variable: Levels of Burden Attributed to Lifetime Racial Discrimination (3 categorical variables)
    - Statistical Test: Polytomous Logistic Regression

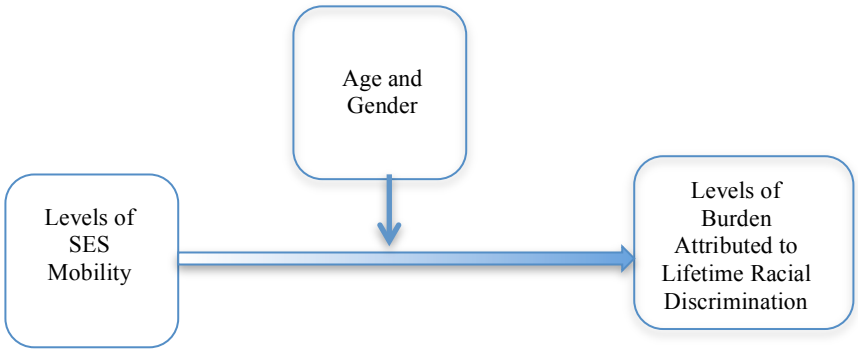

Figure 2. Causal pathway between Levels of SES Mobility and Levels of Burden Attributed to Lifetime Racial Discrimination moderated by Age and Gender.

Research Question 3 (RQ3): Do the levels of SES mobility, perceived lifetime racial discrimination exposure, or burden moderate the relationship between hypertension and cardiovascular disease (Figure 3)?

- Hypothesis 3: The relationship between hypertension and CVD was inversely moderated by increasing levels of SES mobility.
- Null Hypothesis 3: The relationship between hypertension and CVD was not moderated by increasing levels of SES mobility.
- Hypothesis 3b: The relationship between hypertension and CVD was positively moderated by increasing levels of perceived lifetime discrimination attributed to race.
- Null Hypothesis 3b: The relationship between hypertension and CVD was not moderated by increasing levels of perceived lifetime discrimination attributed to race.
- Hypothesis 3c: The relationship between hypertension and CVD was positively moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.
- Null Hypothesis 3c: The relationship between hypertension and CVD was not moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.
    - Independent variables: Hypertension (dichotomous variable), Levels of SES mobility (4 categorical variables), Levels of perceived racism exposure (3 categorical variables), and Levels of perceived racism burden (3 categorical variables)
    - Dependent variable: Cardiovascular Disease (dichotomous variable)
    - Statistical Test: Multivariate logistic regression

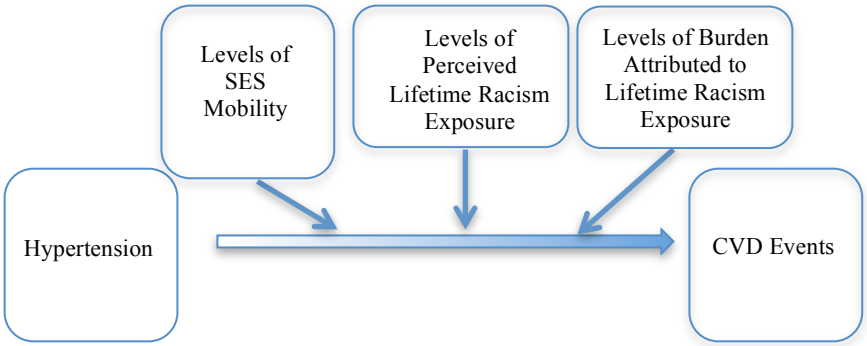

Figure 3. Causal pathway between hypertension and CVD outcomes moderated by levels of the SES mobility, perceived lifetime racial discrimination exposure, or burden.

Research Question 4 (RQ4): If a relationship between levels of SES mobility and levels of perceived lifetime discrimination exposure attributed to race is found (i.e., SES-Racism Effect), does the SES-Racism Effect moderate the relationship between hypertension and cardiovascular disease?

- Hypothesis 4: The relationship between hypertension and CVD was positively moderated by the SES-Racism Effect.
- Null Hypothesis 4: The relationship between hypertension and CVD was not moderated by the SES-Racism Effect.
    - Independent variable: Hypertension (dichotomous variable) and SES-Racism Effect (categorical variable)
    - Dependent variable: Cardiovascular disease (dichotomous variable)
    - Statistical analysis: Multivariate logistic regression

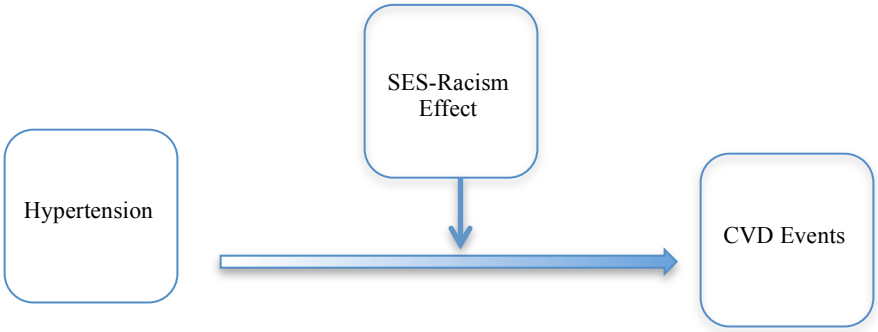

Figure 4. Causal pathway between hypertension and CVD outcomes moderated by the SES-Racism Effect.

Methodology

Although there are a variety of recruitment techniques typically used by researchers to recruit participants into a study (e.g., social marketing, researcher-researched contextual matching, participatory action), JHS researchers recommended that the recruitment techniques used for the JHS tackle the long-standing issues for Black populations, such as distrust of research motives and practices, negative stereotypes, and fear of abuse (Sims et al., 2009). Researchers for the JHS recognized that mistrust and cultural insensitivity were barriers for the recruitment and retention of Blacks participating in the Jackson cohort of the Atherosclerosis Risk in Communities (ARIC) study (Fuqua et al., 2005; Sims et al., 2009; Wyatt, Diekelmann, et al., 2003). Consequently, the population used for the JHS has been uniquely defined and developed through the culmination of lessons learned from previous research conducted within the Jackson, MS community.

Researchers have utilized the JHS Participant Recruitment and Retention Survey

(PPRS) as the initial basis for determining specific factors and experiences that either promoted or inhibited participation in the ARIC study or the intended protocol for the JHS. This is especially true among individuals at the younger and older ends of the projected age range (Wyatt, Diekelmann, et al., 2003). PRRS was a pilot project to identify effective strategies to engage Black's ongoing participation in a research study (Wyatt, Diekelmann, et al., 2003). Specifically, the aims of the PRRS included improved understanding of:

1. factors that facilitated and inhibited participation among Jackson Atherosclerosis Risk in Communities Study (ARIC) cohort participants and dropouts,
2. enabling or hindering factors for participation, particularly among younger and older Black adults in the Jackson community, and
3. how typical experiences related to participation in a research study may influence the JHS research protocol (Wyatt, Diekelmann, et al., 2003).

PRRS provided quantitative and qualitative results, which were culturally appropriate and community specific, as the theoretical basis for the Community-Driven Model used to recruit and retain the cohort of participants for the JHS.

Although the JHS is similar to the Framingham Heart Study, one of the first prospective cohort studies to examine the physical and lifestyle patterns related to CVD development (Arruda, 2013), the JHS focused solely on the Black community and used a Community-Driven Model (Wyatt, Diekelmann, et al., 2003). Wyatt et al. (2003) described community-based approaches to research as "a step toward resolving the

potential conflicts between science and community needs and addressing the problems of implementation…" in a manner that is "particularly effective in studies with health screenings, illness identification, referrals and assistance with behavioral changes" (p. 444).

Community members offer the wisdom of community strengths and assets, as well as social/political challenges and barriers, providing a bridge to trusted community networks. To this end, the development of the Community-Driven Model based on PRRS positioned the Jackson community members to be an integral part of process for developing the JHS research protocol, serving as coinvestigators in the study, and contributing to the dissemination process of study findings (Wyatt et al., 2003). Researchers and community members were aware of the need to maintain ongoing reciprocal trust and respect, as well as balanced distribution of power, to prevent undermining the overall study results. Therefore, recruitment for the JHS was based on a community participatory strategy, which demonstrated respect for individuals in Jackson, MS communities; a model that has successfully used for more than a decade.

Sampling Procedures

JHS participants were recruited based on a combination of four sampling frames. First, a sample of individuals from the participant pool of the Jackson, MS site of the ARIC study was recruited. When the JHS began, a total of 3371 ARIC participants were still alive, with ages ranging from 57 to 76. However, death of ARIC cohort participants caused reduction of the eligible sample to 3027. Second, individuals were chosen randomly from the Accudata America commercial listing, which provided a list of the

majority of households in the Jackson metro area with individuals at least 35 years of age (n=123,403). The listing of Accudata household was connected to Census data to identify neighborhoods with the greatest prevalence of Blacks (neighborhoods with less than 30% Black residents were deleted from the list). Third, volunteers were accepted if they met the Census-match for age, sex, and socioeconomic status (SES) criteria for the Jackson metropolitan statistical area (MSA). Finally, family members of participants recruited from the ARIC, random, and volunteer samples of the JHS study were recruited if they had at least two full siblings and four first degree relatives who resided in the Jackson MSA who were also willing to participate in the study. Unlike the samples recruited from the other sampling frames, participants of the family component were eligible to participate at the age of 21 (with no upper age limit), but continued to be matched to the distribution of the Jackson MSA population for age, sex, and SES (Fuqua et al., 2005).

Study Population

To be eligible to participate in the JHS, participants were required to be Blacks who resided within three counties (i.e., Hinds, Madison, and Rankin) that comprise the Jackson, MS metropolitan statistical area (MSA), were noninstitutionalized, and were between 35 to 84 years of age as of September 1, 2000 (n=76,420; Fuqua et al., 2005; Jackson Heart Study, 2001). In an effort to identify a sample representative of the Jackson metro area, demographic factors (i.e., age, sex, and socioeconomic status) were matched to the distribution of the geographic population (Fuqua et al., 2005). While there is a vast age range of participants that were eligible for participation, Fuqua et al. (2005) noted that the majority of study participants aged 35 to 54 and more likely to be of

middle to high SES. In addition, JHS made further allowances to include specific population groups, including women in their final trimester of pregnancy, women postpartum less than three months, individuals with language problems who had someone to serve as an interpreter, and individuals who were temporarily outside of the study area during the time of recruitment (Jackson Heart Study, 2001). Based on the inclusion criteria, the individuals who were subsequently excluded included individuals who resided outside the tri-county study area, were identified by the trained recruiters to be physically or mentally incapable, or indicated that relocation would occur within 12 month of the study's initiation (Fuqua et al., 2005; Jackson Heart Study, 2001). In addition, any individuals who resided within group settings containing 10 or more adults, in which nine or more were not related to one another, were not eligible to participate in this study. Examples of these group settings include prisons, dormitories, military quarters, and nursing and mental facilities (Jackson Heart Study, 2001). For the purposes of this research study, additional exclusion criteria were imposed. Participants who were identified during analysis to have incomplete or missing discrimination, hypertension, CVD outcomes, or demographic data will be excluded from this study analysis. In addition, participants that were identified by Sims et al. (2012) to attribute their lifetime discrimination exposure to nonracial factors were also excluded from analysis to align with the research questions, which specifically center around factors related to racial based discrimination.

Sample Size

The JHS has prospectively monitored 5301 Black adults at Exam 1. Fuqua et al.

(2005) described that while the original sample size for the study was 6500, power analysis proved that all study questions could be adequately measured with a sample of 5500. The sample was divided as follows: 30.7% consisted of ARIC Study participants (n=1,626), 17.4% comprised the random selection (n=921), 29.6% represented the volunteer sample (n=1570), and 22.4% constituted the family sample (n=1185) (Fuqua et al., 2005). The sample is a representation of adults between the ages of 35 and 84 who reside in the Jackson, MS tri-county area. While the entire sample was used for this study, participants with incomplete data were dropped from the final sample used for analysis. Similar to a previously conducted study, participants were excluded from analysis if all discrimination data (n=283), education (n=20), or hypertension (n=59) are missing, providing a final sample size of 4939 participants (Sims et al., 2012). To align with the research questions, participants that were identified by Sims et al. (2012) to attribute their lifetime discrimination exposure to nonracial factors were also excluded from analysis (n=1626). Although the size of the existing sample is known, an additional power analysis was conducted to ensure that the final sample size was sufficient enough to answer the research questions.

Preliminary "posteriori" power analysis was conducted using G*Power 3.1.7 (Erdfelder, Faul, & Buchner, 1996; Faul, Erdfelder, Lang, & Buchner, 2007) to determine the feasibility of the JHS sample in addressing the research questions. Given that the sample size for the study is known (n=3313), a posteriori power analysis was used to determine whether or not the sample provides adequate power for the study. A multiple regression design was selected to solve for power based on a sample size of 3300, and

using a two-sized t-test with an alpha significance level of 0.05 (JHS Coordinating Center, 2008). The analysis controlled for SES and racism as independent variables, and accounted for the adjustment of the 12 identified covariates. The analysis revealed that this study has more than adequate statistical power (80%) to detect a small effect (0.10) of SES mobility and racial discrimination on the relationship between hypertension and CVD (Research Question 3).

Study Instrument

To assess the interaction of racism and biological factors that affect CVD health outcomes, JHS devised the JHS Discrimination Instrument (JHSDIS), which is an adaptation of multiple previously developed and tested racism measurement instruments (Sims et al., 2009). While there are some discrimination scales that are highly regarded and widely used by other researchers, none of them comprehensively measure the multidimensional construct; therefore, " no gold standard measure of discrimination exists" (Sims et al., 2009, p. 56). Wyatt et al. (2003) advised that to appropriately "tease out the complex additive and interactive relationships that are likely to account for the relationship of various dimensions of racism and cardiovascular disease in African Americans" a multidimensional discrimination scale would be needed. Hence, the JHS used a combination of the Discrimination Scale (Krieger, 1990), Everyday Racial Discrimination Questionnaire (Williams, Yu, Jackson, & Anderson, 1997), and Perceived Racism Scale (McNeilly et al., 1996) to more comprehensively measure how participants identified their experiences and reactions to institutional and personally mediated racism (Payne et al., 2005; Sims et al., 2012, 2009). The combined effect of these instruments

provided a unique opportunity to examine the usefulness of a multidimensional instrument, better understand the complex factors associated with racial discrimination, and how they may contribute either directly or indirectly to hypertension among Blacks.

This revised instrument stratifies discrimination into two categories (i.e., everyday and major life events). In addition, JHSDIS measures the frequency of exposure to discrimination, the physical or personal attributes for which the discrimination is targeted, the individual's coping strategy, and perceptions of how one's skin color effects their treatment by Whites or Blacks. Overall, these various domains measure perceived/personally mediated racism. These measures are the classified as four major constructs (see Table 4).

Table 2

Description of JHSDIS Constructs

Discrimination Construct	Conceptual Indicator
Everyday Discrimination	• Occurrence and frequency (Number of times, number of years ago, number of months ago, number of months) • Targeted attribute (age, sex, race, height or weight, other) • Coping strategy (speak up, accept it, ignore, try to change, keep to self, work harder, pray, avoid, violence, forget, blame self, other)
Major Life Events	• Occurrence (At school, getting a job, at work, getting housing, getting resources/money, getting medical care, in public, other) • Overall lifetime frequency (Number of times, number of years ago, number of months ago) • Targeted attribute (age, sex, race, height or weight, other) • Coping strategy (speak up, accept it, ignore, try to change, keep to self, work harder, pray, avoid, violence, forget, blame self, other)
Burden	• Lifetime frequency • Stressful life • Life made hard due to discrimination • Less productive life
Skin Color	• Treatment by Whites • Treatment by Blacks

The JHSDIS instrument has high overall reliability, with the internal consistency of everyday (α=0.88) and lifetime discrimination (α=0.78) being similar to values as the commonly used scales previously mentioned (Sims et al., 2009); however, the internal reliability of emotional and behavioral coping was lower (0.66; Sims et al., 2009). JHS attributes the lower reliability score for coping to differences in the measurement of

coping (individual in JHS vs. global in previous studies) and sample population (men and women in JHS vs. women only in previous study) (Sims et al., 2009).

Study Variables

All of the variables used in this study originated from the JHS. The dataset was drawn from a variety of JHS data collections forms, further described below. Additional variables were created to represent cumulative scores that were used in the study. All data collection forms from which variables for this study were drawn are included as appendices. All data collection forms are available on the JHS website, and communication with a JHS researcher revealed that formal approval for the usage of these forms was not required (M. Sims, personal communication, August 29, 2013).

Demographic Variables

The following variables to assess demographic information regarding study participants were drawn from the JHS Eligibility Form (Appendix A):

- ID Number – the ID number is a unique identifier given to each participant, which is used consistently across all data collection forms as a method of correlating an individual participant's responses.
- Date of Interview – The date that the interview was conducted is a numeric variable that includes a two-digit month, two-digit, day, and four-digit year to capture the date that the information for each participant was collected.
- Gender – The participant's gender was recorded as a dichotomous variable for which participants self-report as either "Male" or "Female."

- Age – The participant's age was a numeric variable based on a two-digit month, two-digit, day, and four-digit year that identifies the participant's date of birth. Each participant's age at the time of Exam 1 was calculated based on the date of the interview. Age was then be categorized into an interval variable that was used for analysis, which includes 5 age groupings: 35-44, 45-54, 55-64, 65-74, and 75-84.

SES Variables

For the purposes of this study, SES mobility represented a calculated indicator derived from variables used to measure childhood and adult SES. The variables used to measure the childhood SES construct were taken from the Parental Socioeconomic Status Form (Appendix B); whereas, adult SES were taken from the Personal Data/Socioeconomic Status data collection form (Appendix C). All variables for childhood SES are listed first. Childhood SES was based on a cumulative score (ranging from 0 to 28) derived from not only from parental employment and education variables, but also access to resources during early life experiences suggested to be related to health outcomes. The coding mechanism used for the childhood SES variables was consistent with previous JHS research (Subramanyam et al., 2013). Adult SES also represented a cumulative score (ranging from 0 to 19) constructed from variables used to measure education, income, and occupation, with coding mechanism consistent with previous JHS research (Sims et al., 2012; Subramanyam et al., 2013). A cumulative score for both childhood and adulthood SES was calculated based on the sum of the respective

variables, and each summary score was divided into lower and upper strata based on the median value.

Childhood SES was tracked using a variety of factors:

- ID Number – The ID number is a unique identifier given to each participant, which is used consistently across all data collection forms as a method of correlating an individual participant's responses.
- Father's employment – The father's level of employment was given a score of 0 to 4 based on a combination of three variables. First, the status of the participant's father, or male custodian, being gainfully employment during the time the participant was growing up was measured using the responses: yes, no, there was no father/male guardian was present, or don't know. Responses of no were scored as 0 (unemployed); no father/male guardian was present or don't know will be coded as missing. All responses of yes were used to categorize employment based on two text variables that relate to the description of the father's primary job and the father's primary work duties during the participant's childhood. The father's employment was divided into four discrete categories with scores of 1 through 4 (1=production/construction, 2=service, 3=sales, 4=professional/managerial).
- Father's education – The father's level of education was given a score of 0 to 5 based on a combination of two variables. First, the father's highest level of education was measured using a categorical responses, including number of years in school up to grade 12, some vocational/trade school with no certificate,

vocational/trade school with certificate, some college with no degree, associate degree, bachelor's degree, and graduate degree. Responses captured as the completion of grades 1 through 11 were scored as 0 (no high school diploma). The completion of grade 12 was scored as 1 (high school diploma). In addition, vocational/trade school with no certificate and some college with no degree are both scored as a 2; associate degree is a 3, bachelor's degree is a 4, and graduate degree is a 5. If the father had less that a 12^{th} grade education, they were asked if they had received a GED. A response of yes (GED received) was scored as 1, and no GED was scored as 0.

- Mother's employment – The mother's level of employment was given a score of 0 to 4 based on a combination of three variables. First, the status of the participant's mother, or female custodian, being gainfully employment during the time the participant was growing up was measured using the responses: yes, no, there was no mother/female guardian was present, or don't know. Responses of no were scored as 0 (unemployed); no mother/female guardian was present or don't know was coded as missing. All responses of yes were used to categorize employment based on two text variables that relate to the description of the mother's primary job and the mother's primary work duties during the participant's childhood. The mother's employment was defined into four discrete categories with scores of 1 through 4 (1=production/construction, 2=service, 3=sales, 4=professional/managerial).

- Mother's education – The mother's level of education was given a score of 0 to 5 based on a combination of two variables. First, the mother's highest level of education was measured using a categorical responses, including number of years in school up to grade 12, some vocational/trade school with no certificate, vocational/trade school with certificate, some college with no degree, associate degree, bachelor's degree, and graduate degree. Responses captured as the completion of grades 1 through 11 was scored as 0 (no high school diploma). The completion of grade 12 was scored as 1 (high school diploma). In addition, vocational/trade school with no certificate and some college with no degree are both scored as a 2; associate degree is a 3, bachelor's degree is a 4, and graduate degree is a 5. If the mother had less that a 12^{th} grade education, they were asked if they had received a GED. A response of yes (GED received) was scored as 1, and no GED was scored as 0.
- Parent's residence – Whether the parents/guardians owned or were buying, renting, or had other living arrangements during the participant's childhood (until the age of 10) was measured as a nominal variable, which was reverse scored as 2, 1, and 0, respectively.
- Quality of residence – The following indicators measure housing quality during childhood. The response value for each indicator was totaled to create an overall value for quality of residence. For all indicators, yes responses were scored as 1, and no responses were scored as 0.

  - Indoor Plumbing – The availability of indoor plumbing at the place of residence during childhood (up to age 10) was measured as a dichotomous variable with a response of "Yes" or "No."
  - Electricity – The presence of electricity at the place of residence during childhood (up to age 10) was measured as a dichotomous variable with a response of "Yes" or "No."

- Household possessions – The following indicators measure the availability of various household possessions during childhood. The response value for each indicator was totaled to create an overall value for household possessions. For all indicators, yes responses were scored as 1, and no responses were scored as 0.
  - Refrigerator – The presence of a refrigerator at the place of residence during childhood (up to age 10) was measured dichotomous variable with a response of "Yes" or "No."
  - Car – The existence of a family car during childhood (up to age 10) was measured as a dichotomous variable with a response of "Yes" or "No."
  - Telephone – The availability of a telephone at the place of residence during childhood (up to age 10) was measured using a dichotomous variable with a response of "Yes" or "No."
  - TV – The presence of a television at the place of residence during childhood (up to age 10) was measured using a dichotomous variable with a response of "Yes" or "No."

- AC - Air conditioning at the place of residence during childhood (up to age 10) was measure using a dichotomous variable with a response of "Yes" or "No."

Adult SES: The variable categories defined below are derived from

- ID Number – the ID number is a unique identifier given to each participant, which is used consistently across all data collection forms as a method of correlating an individual participant's responses.
- Education – The participant's level of education was given a score of 0 to 5 based on a combination of two variables. First, the participant's highest level of education was measured using a categorical responses, including number of years in school up to grade 12, some vocational/trade school with no certificate, vocational/trade school with certificate, some college with no degree, associate degree, bachelor's degree, and graduate degree. Responses captured as the completion of grades 1 through 11 were scored as 0 (no high school diploma). The completion of grade 12 was scored as 1 (high school diploma). In addition, vocational/trade school with no certificate and some college with no degree are both scored as a 2; associate degree is a 3, bachelor's degree is a 4, and graduate degree is a 5. If the participant had less that a 12^{th} grade education, they were asked if they had received a GED. A response of yes (GED received) was scored as 1, and no GED was scored as 0.
- Employment – The participant's current employment status was measured using 9 categorizes of employment, including full-time work, part-time work,

temporarily laid off work, sick/health leave from work, unemployed but looking for work, unemployed and not looking for work, homemaker, retired not working, retired but working for pay.

- Occupation – Regardless of employment status, participants were asked about their occupation. Two text variables that relate to the description of the participant's primary job and the participant's primary work duties were used to defined the participant's occupation into four discrete categories with scores of 1 through 4 (1=production/construction, 2=service, 3=sales, 4=professional/managerial).
- Income – Income is accessed both as the income that the participant contributes, as well as total family/household income. Similar to how individuals benefit from the combined income of the household during childhood, the total family/household income during adulthood was used to determine SES mobility for the purposes of this study. Income was collected using 13 categorical responses (less than $5K, $5K-$7,999, $8K-$11,999, $12K-$15,999, $16K-$19,999, $20K-$24,999, $25K-$34,999, $35K-$49,999, $50K-$74,999, $75K-$99,999, $100K or more, don't know, or refuse). In accordance with previous JHS research that has utilizes this construct (Hickson et al., 2011, 2012; Sims et al., 2012), income was dichotomized into four nonoverlapping categories to represent poor (less than the poverty level), lower-middle (between 1 to 1.5 times the poverty level), upper-middle (greater than 1.5 but less than 3.5 times the poverty level), and affluent (at least 3.5 times the poverty level). Categorical

levels are based on a combination of family size, U.S. Census poverty levels, and year of baseline data (Sims et al., 2012).

SES Mobility

In an unpublished JHS manuscript proposal, Diez-Rouz et al. (in press) proposes that SES mobility is defined based on two separate cumulative scores for childhood SES and adult SES. The median value for each of these summative scores was used as the cutpoint to dichotomize childhood and adult SES into lower and upper strata. The lower and upper strata for childhood SES were matched with the lower and upper strata for adult SES to create four distinct, nonoverlapping groupings that illustrate the potential SES mobility pathways between childhood and adulthood. The Diez-Rouz manuscript proposal represents the first attempt to measure SES mobility in the JHS; hence, this methodology was used. These categories are as follows:

1. Stable High (HH) (Childhood high, Adult high)
2. Diminishing (HL) (Childhood High, Adult low)
3. Increasing (LH) (Childhood low, Adult high)
4. Stable Low (LL) (Childhood low, Adult low)

The Stable High group was expected to have the lowest risk, followed by Increasing, and Diminishing. The Stable Low group is expected to have the greatest risk. In studies that assessed trends of upward and downward mobility, researchers agreed that individuals of high and increasing SES had lower health risk (Johnson-Lawrence et al., 2013; Pensola, 2003; Hogberg et al., 2011; James et al., 2006). If review of the data indicated that inadequate sample sizes were available for testing each of these subgroups, categories

were collapsed into High (including Stable High and Increasing) and Low (Stable Low and Diminishing).

Discrimination Variables

In previous JHS studies conducted by Sims et al. (2012) and Hickson et al. (2012), a methodology for scoring perceived discrimination was defined. For the purposes of this study, this construct was derived from multiple indicators used to measure perceived/personally mediated discrimination as previously established. Perceived discrimination was assessed using the JHS Discrimination Form (Appendix F) using indicators to document everyday and lifetime exposure to racial discrimination across nine domains, and whether or not experiences were attributed to race, skin color, or some other attribute. Participants responded "yes" or "no" (scored as 1 and 0, respectively) regarding whether or not unfair treatment was experienced in each of the following nine environments at any time during their lifetime: school, getting a job, at work, getting housing, getting money or resources, getting medical care, in a public place, getting services, or in some other environment. A composite score, ranging from 0 to 9, for each participant was used to capture lifetime exposure to perceived discrimination. In addition to exposure to racial discrimination, the burden that discrimination imposes on an individual was also assessed.

Previous JHS research has also examined the burden of discrimination within the JHS population. Sims et al. (2012) calculated the overall burden imposed by discrimination based on the cumulative scoring of three variables (i.e., stressfulness of experiences, interference in life, and life difficulty) that measure the stressfulness of

exposure. The Likert responses for each of the variables were reverse coded; responses ranged from 1 to 4 for each variable. To understand lifetime burden of racial discrimination, an additional variable to capture the frequency of experience was added to this construct. A composite score, ranging from 4 to 16, for each participant was used to capture lifetime exposure to perceived discrimination. All variables to measure burden imposed by discrimination exposure are described below.

- Stress experienced – The stressful experiences of unfair treatment have been over the participant's lifetime was measure based on 3 categorical responses (very stressful, moderately stressful, or not stressful), reverse scored as 4, 2.5, and 1, respectively.
- Inference in life – The amount of inference discrimination had on the participant's life productivity was based on 4 categorical responses (a lot, some, a little, not at all), reverse scored as 4 through 1, respectively.
- Life difficulty – The amount of difficulty created in the participant's life as the result of exposure to discrimination was measured based on 4 categorical responses (a lot, some, a little, not at all), and was reverse scored as 4 through 1, respectively.
- Frequency of experiences – The current frequency of discrimination experiences is compared to when the participant was younger using 3 categorical responses (more frequent, about the same, and less frequent), which was reverse scored as 4, 2.5, and 1, respectively.

- Reason for treatment – The main reason for the unfair treatment was measured using 5 categories (age, gender, race, height/weight, other). However, for the purposes of this study, these responses were dichotomized as racial and nonracial.

Cumulative Discrimination Exposure and Burden

Cumulative discrimination values were determined based on the frequency of discrimination exposures, as a measure of the number of times discrimination was perceived, was summed across nine domains (i.e., school, getting a job, at work, getting housing, getting money or resources, getting medical care, in a public place, getting services, or in some other environment). Sims et al. (2012) derived exposure to lifetime discrimination by determining the median value for the discrimination exposure to create five distinct categories: no exposure to discrimination (score=0), low exposure (score ranging from 1 to 4), and high exposure (score ranging from 5 to 9), which were stratified by racial and nonracial attributed causes. For the purposes of this study, racism attributed to nonracial causes was excluded from the sample. However to create the cumulative lifetime racial discrimination exposure score, the sum of the frequency of exposure to racial discrimination across all domains was summarized and the median value was used to stratify exposure as follows:

1. No exposure to discrimination
2. Low racially attributed exposure to discrimination
3. High racially attributed exposure to discrimination

In addition, the a burden score (cumulative burden attributed to lifetime racial discrimination) was determined by combining scores for perceived burden to racial

discrimination for the three domains: stress experienced, interfered with having full life, and made life difficult. The cumulative value for burden due to racial discrimination, ranging between 4 and 16 was dichotomized into lower and upper strata (based on the median). These strata for discrimination burden represent the potential overall impact of racism burden (low vs. high) on JHS participants.

Hypertension Variables

In this study, hypertension status was determined in the same manner as previously established JHS manuscripts. Hypertension was derived from the average of two blood pressure measurements taken 1 minute apart from the right arm of the participant who had been seated for at least 5 minutes (Harman et al., 2013; Sims et al., 2012; Wyatt et al., 2008). An individual was identified as hypertensive if the average systolic blood pressure was 140mmHg or greater, and diastolic blood pressure was 90mmHg or greater (Sims et al, 2012). Additionally, participants taking antihypertensive medications were also identified as hypertensive (Harman et al., 2013; Sims et al., 2012; Wyatt et al., 2008). Blood pressure measurement data was recorded on the JHS Sitting Blood Pressure Form (Appendix D), and usage of antihypertensive mediations was recorded on the Medication Survey Form (Appendix E). All medications, including dosage and frequency of administration, were precisely recorded and participants were asked if their medications were associated with a list of health conditions.

CVD Variables

Any participants of the JHS who reported experiencing myocardial infarction, angina, coronary revascularization, CHD, cerebrovascular disease, and stroke were

considered to have CVD (USDHHS, 2008). Discussion with a JHS researcher revealed that the Exam 1 dataset contains a dichotomous variable that accounts for the presence of any CVD-related conditions previously identified versus no CVD (M. Sims, personal communication, November 6, 2013). This variable was used in all study analyses exploring CVD outcomes among JHS participants.

Covariates

The following variables were used as covariates in this study: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism. Most of these variables were selected based on their previous use in similar JHS studies (Sims et al., 2012). Each covariate (with the exception of diabetes status) was based on the American Heart Association's (AHA) guidelines, which define Life's Simple Seven (LSS) using three derived levels of health status (i.e., poor, intermediate, and ideal). Life's Simple 7 (LSS) is a new health metric devised by the American Heart Association (AHA) to promote improvements in cardiovascular health by tracking modifiable risk factors (Lloyd-Jones et al., 2010; Thacker et. al., 2014). This new concept of prevention takes into consideration that the most effective strategies for avoiding clinical events over the lifecourse is to avoid adverse risk factors, empirical evidence that CVD risk factors frequently begin developing early in life, and the need for an appropriate balance between population and individual level approaches to health promotion and disease prevention (Lloyd-Jones et al, 2010). This construct is composed of four modifiable health behaviors (i.e., BMI, physical activity, healthy diet, and smoking status) and three modifiable biological

factors (i.e., blood pressure, total cholesterol, and fasting glucose; Lloyd-Jones et al, 2010; Thacker et al, 2014; Djousse et al, 2015).

Although individuals who adhere to ideal health practices are less likely to experience adverse health outcomes (Djousse et al., 2015; Lloyd-Jones et al., 2010), fewer than 1% of U.S. adults meet the standard of practicing all seven ideal metrics (Shay et al., 2012), Americans were least likely to met the ideal standard for a healthy diet (Shay et al., 2012), and variations in overall ideal LSS seem to be inversely observed by age group (Fang et al., 2012). The prevalence of data assessing LSS in African American populations is limited. Data from 2003-2008 NHANES found that none of the African Americans met all seven of the ideal (Shay et al., 2012), and prevalence estimates of meeting all seven ideal health practices was similar among non-Hispanic White populations as well (Alman et al., 2014; Oikonen et al., 2013).

BMI (body mass index) is defined using a standardized measurement of weight in kilograms divided by height squared in meters (Sims, 2012). The physical activity is derived from the JHS physical activity instrument (Dubbert et al., 2005) and based on the sum of active living, occupational, home life, and sports-related index scores (Sims, 2012). Alcohol consumption defined into four categories based on the number of drinks per week: none, 1 to 7 drinks, 8 to 14 drinks, and more than 14 drinks per week (Sims, 2012). Sims et al. (2012) also examined the percentage of dietary fat calories, sodium, potassium, calcium, and fiber related to the Dietary Approaches to Stop Hypertesnion (DASH) diet. Smoking status was defined as JHS participants who were current, former, or never cigarette smokers (Sims, 2012). Diabetes status is defined by the presence or

absence of diabetes (Diez-Rouz, in press). In addition, total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) are measured as mg/dL (Diez-Rouz, in press). Finally, the presence or absence of John Henryism was also be measured (Clark & Adams, 2004; Pascoe & Smart Richman, 2009; Subramanyam et al., 2013).

The LSS health factors and health behaviors were identified as being risk factors known to adversely influence health outcomes, and therefore critical to achieving ideal cardiovascular health. JHS determined each of these LSS covariates used categorical definitions that varied depending on the AHA recommendations (Djoussé et al., 2015). The dietary components were adapted based on JHS data available; fasting glucose was the only LSS variable not included as a covariate in this study. Body mass index (BMI) was defined as ideal (normal weight = <25 kg/m^2), intermediate (overweight = 25 to 29.9 kg/m^2), and poor (obese = ≥ 30 kg/m^2). Physical activity was defined as ideal (≥150 min/wk of moderate or ≥75 min/wk of vigorous activity), intermediate (1-149 min/wk of moderate or 1-74 min/wk of vigorous or 1-149 min/wk of moderate and vigorous activity), and poor (0 min/wk of physical activity). Smoking was defined as ideal (never smoked or former smoker who quit >12 months prior to data collection), intermediate (former smoker who quit within the past 12 months prior to data collection), and poor (current smoker).

The dietary LSS categories were based on individuals meeting a set of criteria, including: ≥4.5 cups/day of fruits and vegetables, ≥ two 3.5 ounce servings/wk of fish, ≥3 one ounce servings/day of whole grains, <1.5g/day of sodium, and <36 fluid ounces of sugar-sweetened beverages. An individual was given one point for each criteria met, and

ideal (4-5 points), intermediate (2-3 points), and poor (0-1 point) was based on the total number of criteria meet. Blood pressure was defined as ideal (untreated systolic BP <120 and diastolic BP < 80mm Hg), intermediate (untreated systolic BP ≥120 and <140 or diastolic BP ≥80 and <90mm Hg, or treated systolic BP <120 and diastolic BP <80mm Hg), and poor (systolic BP ≥140 or diastolic BP ≥90 mm Hg). Finally, cholesterol was defined as ideal (<200 mg/dL untreated), intermediate (≥200 and <240 mg/dL untreated, or <200 mg/dL treated), and poor (≥240 mg/dL). JHS defined the diabetes status categories prior to the adoption of the LSS definitions.

Data Access

The mechanism for noninvestigative researchers to acquire access to JHS data includes the submission of a manuscript proposal, which describes the intended study, variables needed, and analysis strategies. A JHS Manuscript Proposal was developed and submitted for reviewed by the JHS Publications and Presentations Subcommittee (Appendix G). A JHS investigator is required to participate in the development of manuscripts involving any noninvestigative researchers. For the purposes of this study, three JHS investigators were included as coauthors of that manuscript, including a lead mentor, a biostatistician, and the study's principal investigator. Additional coauthors include all Walden faculty serving on the dissertation committee.

All coauthors were required to provide a statement of agreement in support of the manuscript proposal submitted prior to the review process. Upon approval, a JHS Data and Material Distribution Agreement must be completed and approved to obtain access to

the requested data. The review of the data request may include email or phone follow-up questions to clarify request details. Data received was deidentified to protect study participants' personal information, and provided using a password electronic media. Use of the data requires adherence to the JHS Data and Materials Sharing Agreement. Failure to comply with the terms of this agreement may result in not only terminated access to JHS data, but also legal action initiated by multiple parties (e.g., JHS participants, their families, the federal government) (JHS, 2012).

Data Analysis

The dataset provided by the JHS was specifically created based on the variables included in the JHS Study Proposal. Upon approval of the proposal, the dataset was generated from secondary data that has already undergone an extensive data cleaning process. Some additional observations may be dropped from analysis based on missing data for calculated variables. SPSS version 21 statistical software was used to perform descriptive, trend, and moderated multiple regression analysis.

Measures of central tendency (e.g., frequency, median, standard deviations) were calculated for the univariate distribution for all JHS variables used in this study. Distributions were also be stratified by age and gender. In the process of defining calculated variables (e.g., levels of SES mobility and levels of perceived lifetime racism), cross-tabulation tables were generated to illustrate the frequency distribution across the strata (i.e., LL, LH, HL, HH). Contingency tables were evaluated to ensure that cell sizes are appropriate. In the event that cell sizes are too small (<50), calculated variables were

redefined into broader categories (M. Sims, personal communication, June 5, 2014). In addition, covariates were independently be tested for collinearity. Variables determined to have high multicollinearity may be either eliminated or combined to create a composite index variable, depending on empirical justification. Covariates were added last to each model to determine the presence of confounding.

Analysis for Research Questions 1 and 2

RQ1 aimed to explore the relationship between levels of SES mobility and levels of perceived lifetime racial discrimination exposure, and factors (i.e., age and gender) that may moderate the relationship. Similarly, the aim of RQ2 was to explore the relationship between levels of SES mobility and levels of perceived lifetime racial discrimination burden. As such, the following analysis plan was applied to both questions. Multinomial logistic regression was used to measure the linear relationship between the levels of perceived lifetime racism and levels of SES mobility, and how the relationship was influenced by age and gender. First, a chi-square test was applied to the categorical variables, based on the appropriate degrees of freedom (df), to determine whether or not the distributions of SES mobility levels and racial discrimination patterns were statistically independent, with *p*-values (0.05) included to illustrate significance. This strategy was applied to each moderator and covariate to evaluate the contribution in the overall relationship. Only covariates with a bivariate association with the dependent variable at $p<0.20$ will be included in the multivariate model, suggested to be a standard practice (Greenland, 2007). Moderators and covariates were fit to the logistic model in a stepwise fashion. Model 1 for both research questions includes the independent and

dependent variables (i.e., SES mobility and perceived lifetime racial discrimination exposure/burden, respectively). The evaluated covariates will be introduced as blocks in successive models; the stepwise addition will begin with demographics followed by adjustment for lifestyle behaviors, risk factors, and then other social stressors. Included in the output will be a parameter estimates table, which generates the B coefficient and p-value, and a classification table, which determines the accuracy of the model. If the p-value is less than the significance level of $p<0.05$, the $H1a_{Null}$ hypothesis will be rejected; it will be concluded that a relationship between levels of SES mobility and levels of perceived lifetime racism exists.

Finally, if the regression analysis produces a large standard error or B coefficient, additional analysis will be conducted to investigate problems that may not be detected by SPSS version 21.0 (e.g., multicollinearity). A scatterplot will be used to detect whether or not the relationship between the independent and dependent variables monotonically increases or decreases (i.e., in a manner may or may not be linear), and to identify possible outliers.

Analysis for Research Questions 3 and 4

RQ3 aimed to explore if the relationship between hypertension and CVD end points is moderated by levels of SES mobility, perceived lifetime racial discrimination, or burden. Each of these moderators will be modeled separately. In addition, RQ4 investigated whether the relationship between hypertension and CVD cumulative incidence was moderated by the SES-Racism Effect. Since the hypotheses for both research questions have the same independent and dependent variables, the overall plan

of analysis (Cox regression) was the same. Cox regression of CVD cumulative incidence observed during the period of risk was used to explore the influence of multiple variables on survival time. Estimating the potential impact of social constructs over the lifecourse (i.e., levels of SES mobility, levels of perceived lifetime racial discrimination, burden, and SES-Racism Effect) provided increased understanding of for whom or under what conditions the relationship between hypertension and CVD outcomes may change.

For RQ3, Cox regression models were used to analyze the association between all independent variables (levels of SES mobility, levels of perceived lifetime discrimination attributed to race, and burden due to racial discrimination) to determine which of these factors had the most robust relation to risk for CVD events, adjusting for covariate factors. Additional Cox regression models examined the extent to which SES-Racism Effect determines the occurrence of CVD events. Bivariate analysis was conducted to describe the direction and extent of each association, statistical significance, and intercorrelations among independent and dependent variables. Only covariates with a bivariate association with the dependent variable at $p<0.20$ were included in the model.

Moderators and covariates were fit to the cox regression model in a stepwise fashion. Three primary models were analyzed including the independent and dependent variables (i.e., hypertension and CVD, respectively), with each model examining the independent interaction of each moderator (i.e., levels of SES mobility, levels of perceived lifetime racial discrimination exposure, and burden attributed to lifetime racial discrimination). In addition, the evaluated covariates were introduced as blocks in successive models; the stepwise addition began with demographics followed by

adjustment for lifestyle behaviors, risk factors, and then other social stressors. The hazard ratios for risk of CVD were used to illustrate differences across models. Consideration for time-dependent effects will be made. If the p-value is less than the significance level of $p<0.05$, the $H1a_{Null}$ hypothesis was rejected; it was concluded that a relationship between respective independent and dependent variables exists.

Threats to Validity

Numerous factors can threaten the validity of inferences that may be drawn from a study. The ability of the study to answer hypothesized questions (i.e., internal validity), and the extent to which the results of the study may be generalized to other population groups or settings (i.e., external validity), both gauge how well the study may be perceived (Rothman, 2008; Woodward, 2005). This study utilized cross-sectional analysis of a survey conducted among a cohort of Blacks who reside in the Jackson, MS metro area. While the JHS's multilevel recruitment strategy illustrates careful consideration for the population demographics of Blacks in the Jackson metro area (Fuqua et al., 2005), no study is flawless.

Issues that affect internal validity are inherent to observational studies (Rothman, 2008); selection bias, confounding, and interaction effect may be potential causes for concern in this study. The individuals recruited for the JHS study consisted of a combination of previous study participants, volunteers, randomly selected individuals, and participant's family members (Fuqua et al., 2005; Wyatt et al., 2003). Study participants who decided to participate in JHS may have more proactive health behaviors

or some level of interest in CVD that creates selection bias when compared to the general population of the Jackson, MS metro area.

Understanding the frequency and extent of racism exposure that a participant may have experienced over their lifetime was subjectively monitored based on the participant's ability to recall these events. Some events may be easily recalled due to the extent to which the event impacted the individual; however, other events (e.g., subtle discriminatory experiences) may be dismissed, overlooked, or forgotten over time and not accurately captured in the data collected. Furthermore, how an individual perceives interracial interactions and the extent to which those interactions are acknowledged varies widely from one individual to the next. Finally, individuals who actively express interest in improving their health outcomes may be more attune to how stressors, such as racism, impact their health than the overall population. Sims et al. (2009) suggests the need to test the JHSDIS instrument not only among other racial/ethnic populations and geographic settings, but also exploring the effect on other health outcomes.

While the JHS is the largest study to explore CVD health issues solely among Blacks, the single-site study has a specific pool of study participants. The historical, cultural, and social dynamics that exist for Blacks in Mississippi, both previously and currently, may influence how participants perceive interracial interactions; dynamics that may be different in other states or geographic areas. Hence, the results of this investigation may only be generalizable to the population of Black adults within the Jackson, MS geographic area. It would be useful to replicate the JHS in another southern state with similar historical, cultural, and/or social dynamics, or other geographic areas,

to determine which findings can be reproduced. In addition, only Exam 1 data will be analyzed for this study. Analysis of cross-sectional data collected at the onset of the study (i.e., Exam 1) may have similar or different outcomes from data collected at a later time period (i.e., Exam 3). At the onset of a study, participants may be more likely respond to survey questions based on perceived expectations or anxiety about the study outcomes; however at a later time period, participants may be more comfortable with the data collection process. Further investigation upon the availability of Exam 3 data will serve as an opportunity to compare the consistency of data over time.

Ethical Considerations

The data utilized for this study was received and analyzed in accordance with JHS guidelines. Acceptance of the data requires that a signed Data and Materials Distribution Agreement remain on file with JHS (Appendix F). JHS guidelines mandate that all data analyses are limited to the scope of work identified in the research proposal shared with JHS. JHS does not distribute the file linking participant name and demographic information to subject ID, therefore confidentiality of the study participants was maintained. Data was stored on my personal computer's external drive, which is password protected. Although the results of this study may be published, data will not be transferred to any other researchers. At least one JHS investigator served as a collaborator on this research project; therefore, any resulting publications must be reviewed for consistency and data interpretation prior to dissemination.

Given that this study employed secondary data analysis, it posed minimal risk to the JHS participants. Each participant provided informed consent when signing up for the original study, with the ability to withdraw from the study at any time. However, this study requires no further contact with study participants, use of incentives, or conflict of interest. An Institutional Review Board approved an application (IRB# 09-10-14-0138785) to conduct research through Walden University. Any identifying information contained within the dataset was included for analysis in an effort to maintain the anonymity of each participant.

Summary

This chapter provided detailed information on how this research project was conducted. The hypotheses used to answer each research questions, as well as a description of the study population, instruments, variables, and methodology used to guide this investigation have been illustrated. While this study included constructs and variable definitions based on previous JHS research, it also sought to define new constructs based on identify research gaps. Chapter 4 tests the relationship between levels of SES mobility and levels of perceived lifetime racism and factors that potentially impact the strength and direction of that association. It also tests the extent to which these constructs impact the relationship between hypertension and CVD outcomes. The results of these analyses provide insight into the possible impact of SES-Racism Effect and whether or not additional investigation is needed.

Chapter 4: Results

Introduction

This chapter presents the findings from analysis conducted with the Jackson Heart Study (JHS) data and illustrates how the data support the study research questions. First, modifications and rationale to the research plan are described, followed by descriptive data characterizing JHS variables such as the number of study participants, gender, age groups, income status, education levels, and occupation. A description and results of the statistical analysis to address the four research questions follow, which include detailed information about the independent and dependent variables, as well as covariates, used to support each research question. Finally, Chapter 4 concludes with a summary of the results.

Research Questions

Before describing the data that were analyzed, below is a review of the research questions identified in the previous chapter.

RQ1: What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of lifetime racial discrimination, as measured by the occurrence of cumulative perceived lifetime discrimination exposure attributed to race?

- Hypothesis 1: Increasing levels of SES mobility are associated with decreasing levels of perceived lifetime discrimination exposure attributed to race after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI

($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

- Null Hypothesis 1: The association between levels of SES mobility and levels of perceived lifetime discrimination exposure was attributed to race after adjusting for identified covariates.

If an association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was identified, the following subhypotheses were also tested (Figure 1):

Hypothesis 1b: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was inversely moderated by age.

Null Hypothesis 1b: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by age.

Hypothesis 1c: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race was more strongly moderated by males than females.

Null Hypothesis 1c: There are no statistically significant differences in the association between levels of SES mobility and levels of perceived lifetime exposure attributed to race when moderated by males than females.

RQ2: What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of burden attributed to perceived

lifetime racial discrimination, as measured by the extent of life stressfulness, difficulty, and productivity as a result of perceived lifetime discrimination attributed to race?

- Hypothesis 2: Increasing levels of SES mobility are associated with decreasing levels of burden attributed to perceived lifetime racial discrimination after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.
- Null Hypothesis 2: There are no statistically significant associations between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination after adjusting for identified covariates.

If an association between levels of SES mobility and levels burden attributed to perceived lifetime racial discrimination was identified, the following subhypotheses were also tested (Figure 2):

- Hypothesis 2b: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was inversely moderated by age.
- Null Hypothesis 2b: There are no statistically significant differences in the association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination when moderated by age.

- Hypothesis 2c: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race was higher in males than females.
- Null Hypothesis 2c: The association between levels of SES mobility and levels of burden attributed to perceived lifetime racial discrimination was moderated by gender.

RQ3: Do the levels of the SES mobility, cumulative perceived lifetime racial discrimination exposure, or burden moderate the relationship between hypertension and cardiovascular disease?

Hypothesis 3: The relationship between hypertension and CVD is moderated by increasing levels of SES mobility.

Null Hypothesis 3: The relationship between hypertension and CVD is not moderated by levels of SES mobility.

Hypothesis 3b: The relationship between hypertension and CVD is moderated by increasing levels of perceived lifetime discrimination attributed to race.

Null Hypothesis 3b: The relationship between hypertension and CVD is not moderated by levels of perceived lifetime discrimination attributed to race.

Hypothesis 3c: The relationship between hypertension and CVD is moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.

Null Hypothesis 3c: The relationship between hypertension and CVD is not moderated by levels of burden attributed to perceived lifetime racial discrimination.

RQ4: If a relationship between levels of SES mobility and levels of perceived lifetime discrimination exposure attributed to race is found (i.e., SES-Racism Effect), does the SES-Racism Effect moderate the relationship between hypertension and cardiovascular disease?

- Hypothesis 4: The relationship between hypertension and CVD was positively moderated by the SES-Racism Effect.
- Null Hypothesis 4: The relationship between hypertension and CVD was not moderated by the SES-Racism Effect.

Data Retrieval

To acquire access to the secondary data used for this study, I developed a JHS Manuscript Proposal and submitted it for approval to the JHS Publications and Presentations Subcommittee (Appendix G). Upon approval, I also completed a JHS Data and Material Distribution Agreement and submitted it for approval prior to obtaining access to the requested data. De-identified data were downloaded from a password-protected link provided by JHS. During the process of reviewing and cleaning the dataset, I identified multiple problems. Several follow-up communications with JHS' coordinating center were required to request and understand the derived variables and variable formats not included in any of the variable lists or codebooks used to originally generate the data request, as well as appropriately differentiate multiple variables that represented the same indicator.

After receiving the derived variables, I determined that analysis of occupational data would need to be excluded from analysis. A derived variable was available to

categorize adult occupations; however, a comparable derived variable was not available for the parental occupation variable. An attempt to recode the text values for parental occupation into categories comparable to the adult occupation categories revealed incompatibility between the codes. Occupation was not used as a measure of SES mobility because a comparison of the adult and parental individual participants' responses for occupation indicated nonparallel values; therefore, no categorical variable for parental occupation was created for comparative analysis. Thus, SES mobility for occupation was not measured in this study.

In addition, adjustments in the original SES mobility construct were made. The original SES mobility construct was defined based on a manuscript proposal submitted to JHS, by a researcher previously associated with JHS, roughly a year prior to this study. However, the manuscript, and subsequent SES mobility construct, was never developed as planned. Thus, for this analysis the SES mobility construct definition was modified to measure the change in childhood to adult income and education independently, rather than as a cumulative measure. The terms *parental* and *childhood* are used interchangeably.

Descriptive Statistics for Analysis Variables

Participants

The dataset originally acquired from JHS contained data from 5,301 participants, from which participants were excluded if education (n = 289), hypertension status (n = 274), cardiovascular disease status (n = 77), and lifetime discrimination (n = 1110) data were missing; a total of 4,117 participants remained (566 participants had multiple

missing variables). Additional participants were excluded if their lifetime discrimination exposure was attributed to nonracial factors (i.e, age, gender, height, or some other factor, n = 1527). Data analysis were based on a final sample size of 2,590 participants who attributed lifetime discrimination exposure to race.

Table 3 provides descriptive data on the demographic characteristics of the study participants. The JHS dataset consists of 1,505 female respondents (58.1%) and 1085 male respondents (41.9%). The majority of respondents were equally distributed between the 45-54 and 55-64 age groups (28.6% and 27.6%, respectively), with a mean age of 56 years. Thirty-seven percent of participants had at least a college degree or greater education, while 15.9% had less than a high school diploma. The majority of respondents were employed in a managerial-professional job (40.4%). Occupational status reflected occupation over the adult lifetime, not current employment status. JHS used participant responses to derive two groups of categorical values for occupation (i.e., a three-category classification and a 12-category classification) based on the U.S. Census standard for job codes (Sims et al., 2011, 2012).

Table 3 also shows the distribution of income across 11 categories, with the majority of participants possessing higher income categories. However for the purpose of further analysis, a derived income variable provided by JHS was used to describe participants' socioeconomic status. It was based on the U.S. Census poverty estimates, which took into consideration total family income and number of household residents. The distribution of this derived income variable indicated that 11.6% were poor, 22.2%

and 30.0% were lower-middle and upper-middle income respectively, and 36.2% of respondents were classified as affluent (Table 3).

Table 3

Frequencies: Demographics of Study Participants

Indicator	*n*	%
Gender		
Male	1085	41.9
Female	1505	58.1
Age Group		
35-44	515	19.9
45-54	741	28.6
55-64	714	27.6
65-74	485	18.7
75-84	135	5.2
Education Level		
Less than high school	413	15.9
High school graduate or equivalent	424	16.4
Some college, vocational, or trade	585	22.6
Associates degree	198	7.6
Bachelor's degree	468	18.1
Graduate degree (Master's or Ph.D.)	502	19.4
Employment Status		
Managerial-Professional	1047	40.4
Service	545	21.0

Indicator	*n*	%
Sales	480	18.5
Farming/ Construction/Production	518	20.0
Income Level		
< $5,000	60	2.7
$5,000 - $7,999	91	4.1
$8,000 - $11,999	108	4.8
$12,000 - $15,999	137	6.1
$16,000 - $19,999	111	4.9
$20,000 - $24,999	193	8.6
$25,000 - $34,999	251	11.2
$35,000 - $49,999	351	15.7
$50,000 - $74,999	458	20.5
$75,000 - $99,999	234	10.5
$100,000 or more	240	10.7
Socioeconomic Status		
Poor	257	11.6
Lower-Middle	494	22.2
Upper-Middle	667	30.0
Affluent	805	36.2

Table 4 illustrates data related to childhood demographics that are used to measure SES mobility based on education and income. The data show that fathers are more likely to have less than a high school education than mothers (76% vs. 64%); whereas mothers are more likely to have a college education or greater than fathers (7.8% vs. 4.4%). Indicators of childhood residential quality and material resources were used as a proxy for measuring the participants' parental income. Mississippi is known to be a highly rural and historically impoverished state. Data show that while it was common for participants to experience poor residential quality or access to material resources during childhood, participants were most likely to have electricity (76%), a refrigerator (69%), and a car (67%) during childhood and least likely to have air conditioning (21%).

Table 4

Frequencies: Childhood Demographics for Education and Income (N = 2590)

Indicator	*n*	%
Father's Education Level		
Less than high school	1847	76.4
High school graduate or equivalent	301	12.4
Some college, vocational, or trade	115	4.8
Associates degree	51	2.1
Bachelor's degree	67	2.8
Graduate degree (Master's or Ph.D.)	38	1.6
Mother's Education Level		
Less than high school	1580	64.0
High school graduate or equivalent	477	19.3
Some college, vocational, or trade	150	6.1
Associates degree	68	2.8
Bachelor's degree	112	4.5
Graduate degree (Master's or Ph.D.)	81	3.3
Childhood Residential Quality and Material Resources		
Indoor plumbing	1847	53.1
Electricity	1843	76.4
Refrigerator	1666	69.0

Indicator	*n*	%
Car	1612	66.8
Telephone	1158	48.1
Television	1050	50.4
Air conditioning	493	20.5

Each covariate (with the exception of diabetes status) was based on the American Heart Association's (AHA) guidelines, which define Life's Simple Seven (LSS) using three derived levels of health status (i.e., poor, intermediate, and ideal) (Thacker et al., 2014).

Table 5 provides descriptive statistics for the covariates used in the study. Among the variables identified as LSS for ideal cardiovascular health, smoking was the only variable with the majority of respondents having ideal health (85.2%). JHS participants most commonly experienced poor health in the areas of BMI (51.9%) and dietary intake (55.0%), followed by physical activity (46.9%). Further analysis found that the number of ideal LSS health factors or behaviors that participants practiced ranged from zero (6.8%) to five (0.23%), with participants being most likely to practice one (40.3%) or two (37.5) ideal LSS health factors or behaviors. Similar to previously cited studies (Djoussé et al., 2015; Thacker et al., 2014), none of the participants in this sample practiced all seven LSS components. Participants were mostly likely to be non-diabetic (44.8%).

Categorical responses for a total of 12 variables, used to defined the behavioral characteristics of John Henryism (JH), were reverse coded with values ranging from 0

(completely false) to 3 (completely true) and summed. Overall JH scores ranged from 7 to 36 (M = 29.64, SD = 4.33). More than half of participants reported a completely true response for 9 of the 12 behavioral characteristics of JH; the remaining characteristics showed participants with responses split between those behavioral characteristics being completely true and somewhat true. Hence, participants reported an elevated prevalence of JH overall. When exposure was divided into high and low exposure to JH across the median (Table 5), respondents more frequently experienced low JH status (57.8%). Finally, the prevalence of financial adversity experienced by participants was measured. A participant was categorized as having experienced financial adversity if the participant responded affirmatively that he/she or someone in their household had lost a job within the past 12 months or the participant's derived variable for unemployment was "Yes." About 8% of the JHS participants were identified as having experienced financial adversity (Table 5). The number of alcoholic drinks per week and per month were not included in Table 5 due to fewer than half of participants responding to this question. However, of those who answered the question, participants consumed between 0 to 30.4 alcoholic drinks per month (M = 4.96, SD = 7.27) and 0 to 7 per week (M = 1.24, SD = 1.81).

Table 5

Frequencies: Covariates Used in the Analysis

Indicator	*n*	%
BMI		
Poor Health	1343	51.9
Intermediate Health	880	34.0
Ideal Health	363	14.0
Physical Activity		
Poor Health	1217	46.9
Intermediate Health	822	31.7
Ideal Health	551	21.3
Smoking Status		
Poor Health	342	13.4
Intermediate Health	37	1.5
Ideal Health	2176	85.2
Nutrition		
Poor Health	1299	55.0
Intermediate Health	1034	43.8
Ideal Health	28	1.2
Blood Pressure		
Poor Health	381	15.6
Intermediate Health	1781	72.8

Ideal Health	284	11.6
Total Cholesterol		
Poor Health	362	16.8
Intermediate Health	922	42.8
Ideal Health	871	40.4
Diabetes Status		
Diabetic	545	21.2
Pre-diabetic	872	33.9
Non-diabetic	1152	44.8
John Henryism		
Low exposure	1497	57.8
High exposure	1093	42.2
Financial Adversity		
No	2377	91.8
Yes	213	8.2

Preliminary Analysis Procedures

Preliminary Comparative Analyses

The independent variables used to examine the research questions are described in Table 6 below. Analysis indicated that 49.7% of the original JHS study population (n = 5301) attributed their lifetime discrimination exposure to race; however, 62.9% of participants, who met the study criteria of not having missing responses for education,

hypertension, and CVD (n = 4117), attributed their lifetime discrimination to race. Further analysis was conducted to examine the frequency of exposure to discrimination across nine domains (Table 6), as well as the perceived burden that racial discrimination may have contributed to one's lifetime experiences.

Discrimination descriptive data. Study participants experienced the fewest encounters of racial discrimination in the domains of getting housing, receiving medical care, or some "other" area. Participants did not respond to each of the discrimination domains as mutually exclusive settings. Therefore, nearly half of the study participants indicated experiences of racial discrimination in 6 out of the 9 domains, with the highest frequency of experience being in a work environment (75.4%). A total lifetime racial discrimination exposure score was created by recoding the dichotomous responses for each domain to 0 (No) and 1 (Yes) in order to calculate a cumulative value. Total lifetime racial discrimination exposure scores ranged from 0 to 9 (M = 3.67, SD = 1.92). The mean score was used as the cutpoint for determining that participants were more likely to have a low lifetime exposure to racial discrimination (66.8%), compared to no or high exposure (1.1% and 32.1%, respectively). Hence, lifetime discrimination was defined as a categorical dependent variable that contains three categories, no discrimination (0 on the discrimination scale), low discrimination (1 to 4 on the discrimination scale), and high discrimination (5 to 9 on the discrimination scale). While study participants must have attributed discrimination experiences to race, this response assessed the primary perception for discrimination experiences overall. Therefore, it may be possible for

individuals to attribute everyday discrimination to race, yet experience no lifetime racial discrimination exposure.

Chi-square tests for independence were conducted to determine whether relationships between lifetime discrimination and gender or age existed. Males were more likely than females (71.9% vs. 57.7%, respectively) to experience lifetime discrimination attributed to race, $\chi^2(1, n = 4117) = 84.13$, $p = 0.000$, $phi = 0.143$. While men more frequently attributed the lifetime discrimination that they experienced to race, women reported greater occurrence of lifetime racial discrimination across all domains (Table 6). When lifetime racial discrimination was stratified across age groups, older participants (age 75-84) were more likely than participants in the 34-44, 45-54, 55-64, and 65-74 age cohorts to attribute their lifetime discrimination to race (68.9% vs. 59.3%, 64.5%, 62.1%, 64.2%, respectively; $\chi^2(4, n = 4117) = 9.85$, $p = 0.0431$, $phi = 0.0489$). However, adults aged 45-54 and 55-64 experienced a greater number of occurrences across domains (28.6% and 27.6%, respectively) compared to all other age groups.

Similarly, the results showed that all participants experienced some level of burden across each characteristic associated with lifetime racial discrimination. Overall burden attributed to racial discrimination was calculated based in the recoding and summation of three indicators as described in Chapter 3 (Table 6), with overall burden scores ranging from 2 to 12 (M=7.23, SD=2.38). The mean burden score showed that participants were equally likely to have a low and high burden from lifetime racial discrimination (49.9% vs. 50.1%, respectively). Overall, women experienced more burden than men (OR = 1.23, p <0.05).

SES mobility descriptive data. SES mobility was measured using two independent constructs based on the change in parental to adult income and education. Hence, education and income were each used as a proxy for measuring SES mobility. SES mobility is a categorical variable where a stable low SES mobility represents participants who consistently experienced a lower SES from childhood through adulthood; increasing includes participants who experienced an increase in SES from childhood to adulthood; diminishing includes participants who experienced a decrease in SES from childhood to adulthood; and stable high includes participants who consistently experienced a higher SES from childhood through adulthood. These SES mobility categories were measured identically for both income and educational indicators of this construct.

Table 6 also examines the childhood-adult education and income status as measures of SES mobility as previously defined. Contingency tables were used to illustrate four SES mobility trajectories used for each SES mobility indicator: 1) stable high (high childhood and high adult measure), 2) increasing (low childhood and high adult measure), 3) diminishing (high childhood and low adult measure), and 4) stable low (low childhood and low adult measure). The mean value of education was used as the cutpoint for determining high and low categories for adult education (M = 3.54, SD =1.75), as well as both father (M = 1.47, SD = 1.06) and mother's education (M = 1.74, SD = 1.29). A previous study comparing the educational status of adults with their parents used different measures of low educational attainment for each population due to the decline in high school dropout rates over time (Salsberry & Reagan, 2009). The

distribution of education during adulthood (Table 3) compared to childhood (Table 4) demonstrated that adults tend to be more educated than their parents. Therefore, a higher threshold for low educational status was used for adulthood (i.e., less than a college degree) compared the parental education (i.e., high school diploma or less). Furthermore, an assumption was made that just as a household benefits from dual incomes, it would also benefit from one parent having greater education than the other. Data from Table 4 illustrates that mothers were more highly educated than fathers; mothers had a lower prevalence estimate for less than a high school education (64.0% vs. 76.4%, respectively) and high prevalence of having at least a college degree (7.8% vs. 4.3%, respectively). Therefore, because parental education data was available for both parents, parental education was stratified as low only if both parents had low educational status. When the educational status of both parents was combined, 79.0% of parental education was considered low compared to 62.5% of adult education. As such, analysis showed that more than half of participants (54.2%) had a stable low SES mobility status.

Measures of SES mobility using income were also analyzed. The derived JHS income variable was used as the measure of adult household income. The derived income status indicator consisted of four categorical measurements (i.e., poor, lower-middle, upper-middle, and affluent), which were collapsed to create high (upper-middle and affluent) and low (poor and lower-middle) strata. A proxy measure for childhood household income was calculated based on the collective indicators of home ownership status, residential quality, and household possessions. More than half (54.8%) of participants' parents were homeowners, while another 31.0% were renters and 14.2% had

some other living arrangement. The quality of childhood residence was determined based on a combination of participants having access to indoor plumbing (53.3%) and electricity (76.5%) during childhood, each with a dichotomous variable recoded as 0 ("No") and 1 ("Yes"), and the number of rooms within the residence (ranging from 1 to 17, $M = 5.56$, $SD = 1.97$). Finally, the household possessions (i.e., refrigerator, car, telephone, television, air conditioning) that participants had during childhood were calculated from a list of dichotomous variables in the same manner as residential quality. About half of all households had a telephone and a television during childhood, but were most likely to have a refrigerator (69.0%) and least likely to have air conditioning (20.5%). The average score for cumulative childhood SES was 10.65 ($SD = 4.36$), which appears to follow a normal distribution ranging from 0 to 26 (data not shown). Therefore, the high and low categories for childhood income were based upon the mean using a cutpoint of 11. The high and low childhood and adult values were combined to create the categories to measure SES mobility for income based on the previously defined categorical values.

Table 6

Frequencies: Descriptive and Comparative Analysis of Lifetime Racial Discrimination

Indicator	Overall		Males		Females	
	N	%	*N*	%	*N*	%
Lifetime Discrimination Attributed to Race (*n*=4117)**						
No	1527	37.1	422	28.0	1105	42.3
Yes	2590	62.9	1085	72.0	1505	57.7
Domains of discrimination exposure (*n*=2590)						
At school/training	1374	53.1	578	42.1	796	57.9
Getting a job**	1462	56.4	671	45.9	791	54.1
At work	1954	75.4	816	41.8	1138	58.2
Getting housing*	441	17.0	214	48.5	221	51.5
Getting resources/money**	1211	46.8	576	47.6	635	52.4
Getting medical care**	428	16.5	140	32.7	288	67.3
In public places**	1224	47.3	583	47.6	641	52.4
Getting services	1251	48.3	527	42.1	724	57.9
In other ways	160	6.2	73	45.6	87	54.4
Burden of stressfulness due to lifetime discrimination*						
Very stressful	548	21.2	237	21.9	311	20.7
Moderately stressful	1411	54.6	615	56.8	796	53.1
Not stressful	625	24.2	232	21.9	393	26.2
Burden of discrimination made life harder**						
At lot	696	26.9	247	22.8	449	30.0
Some	777	30.1	332	30.6	445	29.7
A little	777	30.1	336	31.0	441	29.4
Not at all	333	12.9	169	15.6	164	10.9
Burden due to interference in life**						
At lot	551	21.4	197	18.2	354	23.6
Some	795	30.8	345	31.9	450	30.0
A little	865	33.5	347	32.1	518	34.6
Not at all	370	14.3	193	17.8	177	11.8

SES mobility based on education*						
Stable high	327	12.6	139	12.8	188	12.5
Increasing	643	24.8	259	23.9	3840	25.5
Diminishing	216	8.3	114	10.5	102	6.8
Stable low	1404	54.2	573	52.8	831	55.2
SES mobility based on income*						
Stable high	1281	49.5	562	51.8	719	47.8
Increasing	191	7.4	45	8.8	96	6.4
Diminishing	798	30.8	301	27.7	497	33.0
Stable low	320	12.4	127	11.7	193	12.8

Note. p-value comparisons for gender*: *p=value <0.05, **p-value <0.01*

Hypertension and CVD both represent dependent variables that were measured in this study. The prevalence of hypertension and CVD in the study population were 62.7% and 10.2%, respectively. Figure 7 shows that the prevalence of hypertension was higher among females (64.1%) than males (60.7%), but a chi-square test for independence determined that differences by gender were not statistically significant, $\chi^2(1, n = 2590) = 3.64$, $p = 0.564$, $phi = -0.0375$. However, significant differences in the relationship between age and hypertension were observed as the prevalence of hypertension increases with age, $\chi^2(4, n = 2590) = 291.16$, $p < 0.001$, $phi = 0.335$. Figure 8 shows that the prevalence of CVD was higher among males (10.9%) than females (9.6%), and the prevalence of CVD increased with age. No significant differences were observed for prevalence of CVD between males and females (10.9% vs. 9.7%, respectively, $\chi^2(1, n = 2590) = 1.06$, $p = 0.3023$, $phi = 0.020$; yet, significant differences were again observed by age, $\chi^2(4, n = 2590) = 16.58$, $p = 0.002$, $phi = 0.081$.

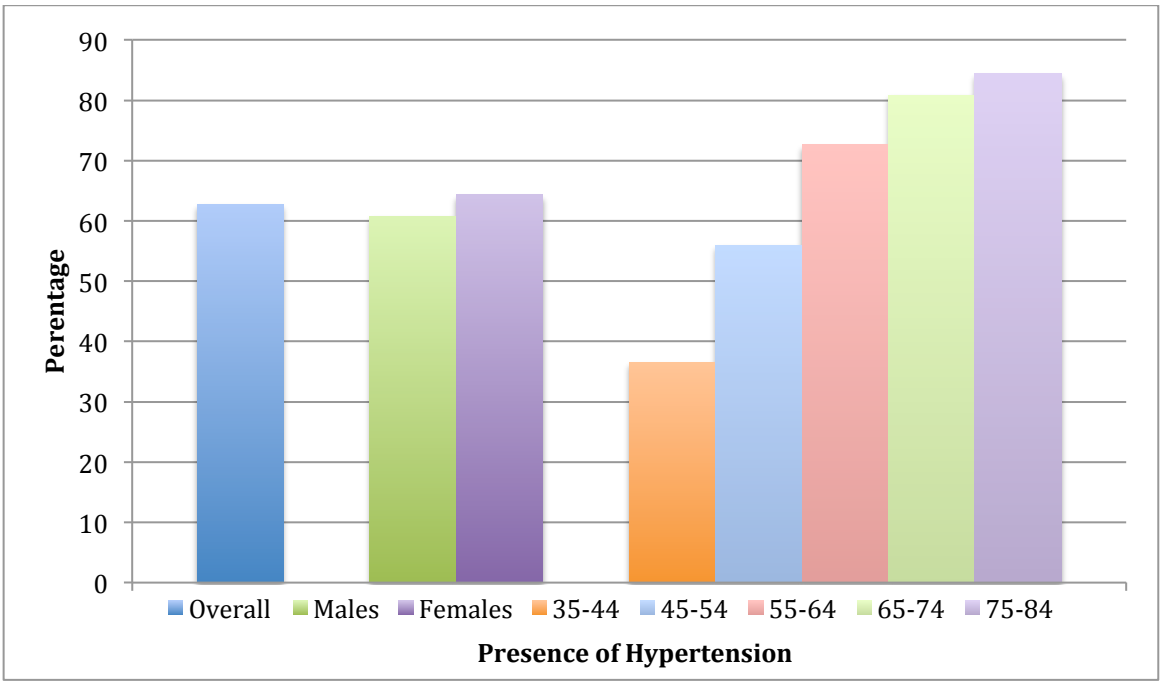

Figure 7. A bar graph showing the distribution of hypertension by gender and age.

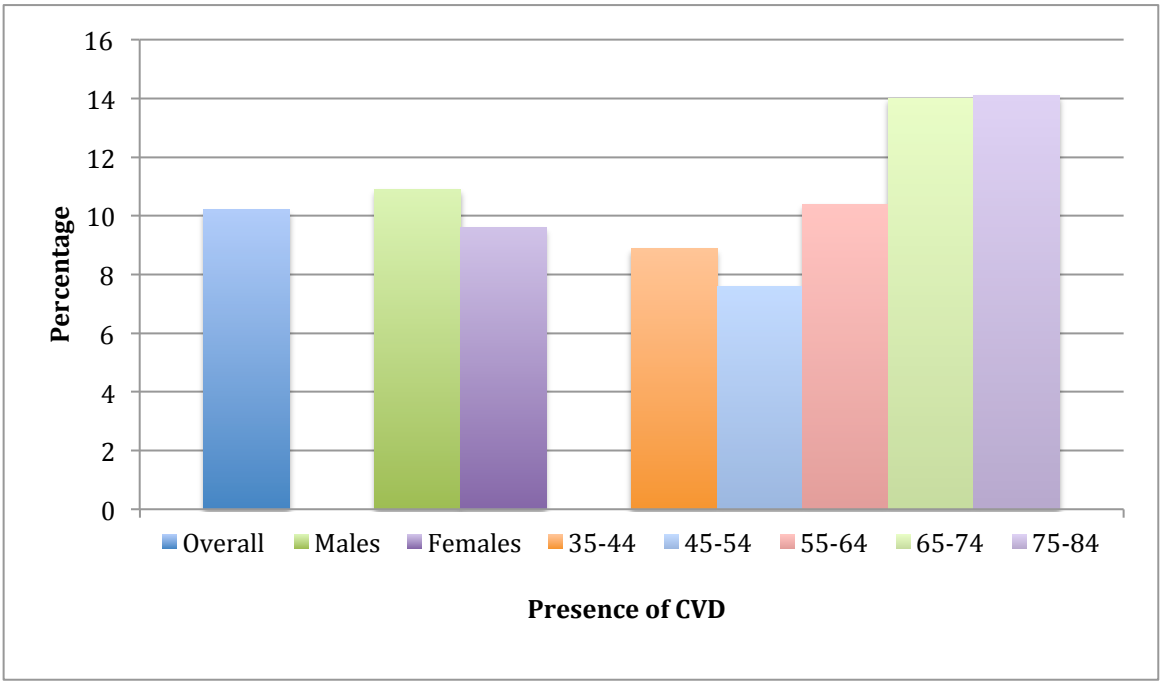

Figure 8. A bar graph showing the distribution of CVD by gender and age.

Coding of Variables

Prior to answering the research questions, the covariate, independent, and dependent variables were examined; the number of responses for each variable and the frequencies for each category were assessed. Two of the covariates (i.e., number of drinks per week and number of drinks per month) only had responses for half of the sample; therefore, these variables were not included in subsequent procedures. Instead, the binary variable measuring alcohol consumption in the past 12 months was used.

The categories of the following variables were collapsed either due to very low frequencies or to lack of convergence in the regression procedure: levels of lifetime exposure to racial discrimination, levels of burden attributed to a lifetime of racial discrimination, age group, nutrition, and smoking. First, only 29 participants had no lifetime exposure to racial discrimination (compared to 1731 with low and 830 with high lifetime racial discrimination); thus, the no lifetime racial discrimination category was collapsed with the low lifetime racial discrimination. Lifetime exposure to racial discrimination then became a binary variable: low vs. high discrimination exposure. Second, there were no participants who experienced no burden attributed to lifetime racial discrimination; therefore, this variable was measured using only two categories (i.e., low vs. high burden).

Third, due to lack of convergence in the logistic regression procedure, the five-category age group variable was collapsed into a three-category variable; the first two categories were combined into a single group (i.e., 35 to 54 years), the fourth and fifth categories were combined into a single group (i.e., 65 to 84 years), and the third category

remained as previously defined (i.e., 55 to 64 years). Fourth, the third category of nutrition health only had 28 responses (in contrast to 1200 and 1034 for the first and second categories); therefore, this category was collapsed with the second category. Thus, this variable became a binary variable: poor vs. intermediate health. Last, the second category of the smoking health variable only had 37 responses (in contrast to 342 and 2176 for the first and third categories, respectively). Accordingly, the second category was integrated into the third category; the new binary variable has now been defined in terms of poor vs. ideal health.

Assessing the Relationship Between Independent Variables and Dependent Measures

An initial effort was made to include all the independent variables in the logistic regression models, but their models yielded nonconvergence. Hence, separate regression procedures were conducted, one for each dependent measure, to determine which independent variables significantly predicted the dependent variable. Only the independent variables that marginally ($p < .10$) or significantly ($p < .05$) predicted the dependent variable were included in the main logistic regression models.

Regression model for exposure to racial discrimination. As shown in Table 7, the following independent variables marginally or significantly predicted exposure to lifetime racial discrimination: physical activity, alcohol use, financial adversity, and the composite John Henryism measure of social stress. Therefore, only these independent variables were included in a single logistic regression model testing the relationship between SES mobility, age, and gender on exposure to racial discrimination.

Table 7

Bivariate Regression Results for Lifestyle Behaviors, Risk Factors, Social Stressors, and Exposure to Discrimination (N = 1963)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Nutrition: poor vs. intermediate health[1]	.06	.535	1.06	.87	1.30
Smoking: poor vs. ideal health[1]	.23	.169	1.26	.91	1.74
Physical activity		.031			
Poor vs. intermediate health[1]	.29	.010	1.34	1.07	1.67
Poor vs. ideal health[1]	.21	.107	1.23	.96	1.60
No alcohol vs. alcohol in past year[1]	.38	.000	1.46	1.20	1.78
BMI	.00	.571	1.00	.99	1.02
HDL levels	-.15	.257	.86	.66	1.12
LDL levels	-.06	.260	.94	.84	1.05
No financial stress vs. financial stress[1]	.68	.000	1.97	1.43	2.72
John Henryism	-.02	.076	.98	.96	1.00

Note. OR = odds ratio. CI = confidence interval.

[1] Reference categories appear first in the group comparison.

Regression model for level of burden. The findings in Table 8 revealed that smoking and financial adversity significantly predicted level of burden due to lifetime exposure to racial discrimination in the model. Similar to the previous procedure, only these independent variables were included in a single logistic regression model testing the

relationship between SES mobility, age, and gender on level of burden due to lifetime racial discrimination exposure.

Table 8

Logistic Regression Results for Lifestyle Behaviors, Risk Factors, Social Stressors, and Level of Burden Due to Lifetime Exposure to Racial Discrimination (N = 1959)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Nutrition: poor vs. intermediate health[1]	-.06	.529	.94	.79	1.13
Smoking: poor vs. ideal health[1]	-.36	.018	.70	.52	.94
Physical activity		.547			
Poor vs. intermediate health[1]	.12	.272	1.12	.91	1.38
Poor vs. ideal health[1]	.06	.643	1.06	.84	1.34
No alcohol vs. alcohol in past year[1]	.02	.849	1.02	.85	1.22
BMI	.00	.700	1.00	.99	1.02
HDL levels	.02	.902	1.02	.80	1.29
LDL levels	-.03	.612	.98	.88	1.08
No financial stress vs. financial stress[1]	.33	.045	1.39	1.01	1.91
John Henryism total score	-.00	.705	1.00	.98	1.02

Note. OR = odds ratio. CI = confidence interval.

[1] Reference categories appear first in the group comparison.

Regression model for cardiovascular disease. The findings in Table 9 show that the following independent variables significantly predicted likelihood of having cardiovascular disease in the model: age, nutrition, HDL, and LDL levels. Thus, only these variables were included in the logistic regression procedure testing the relationship

between SES mobility, and hypertension on the likelihood of having cardiovascular disease.

Table 9

Logistic Regression Results for Demographic Factors, Lifestyle Behaviors, Risk Factors, Social Stressors, and Likelihood of Having Cardiovascular Disease (N = 1963)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.006			
34 to 54 vs. 55 to 64	.44	.018	1.55	1.08	2.24
34 to 54 vs. 65 to 84	.59	.003	1.81	1.22	2.67
Gender: male vs. female[1]	.13	.437	1.13	.83	1.56
Nutrition: poor vs. intermediate health[1]	-.35	.028	.70	.51	.96
Smoking: poor vs. ideal health[1]	.08	.762	1.08	.66	1.78
Physical activity		.338			
Poor vs. intermediate health[1]	.05	.770	1.05	.75	1.48
Poor vs. ideal health[1]	-.28	.210	.76	.49	1.17
No alcohol vs. alcohol in past year[1]	-.12	.477	.89	.65	1.23
BMI	.01	.526	1.01	.99	1.03
HDL levels	-.51	.021	.60	.39	.93
LDL levels	-.19	.029	.83	.70	.98
No financial stress vs. financial stress[1]	.20	.434	1.23	.74	2.04
John Henryism total score	-.03	.105	.97	.94	1.01

Note. OR = odds ratio. CI = confidence interval. Overall model $\chi^2(13) = 29.91$, $p < .01$.

[1] Reference categories appear first in the group comparison.

The Relationship Between SES Mobility and Exposure to Discrimination (Research Question 1)

The first research question sought to determine the relationship between levels of SES mobility and levels of lifetime discrimination attributed to race, and determine whether or not the relationship was moderated by age or gender. The first logistic regression was conducted with lifetime discrimination exposure as the dependent variable and SES mobility income and SES mobility education as the independent variables. To answer this first question, a hierarchical logistic regression procedure was conducted. In the first step, the demographic variables were entered into the equation; in the second step, the lifestyle variables were entered; in the third step, the social stressors were entered; in the fourth step, the independent variables were entered. Age and gender were also evaluated to assess their affect on the direction and/or strength of the relationship between lifetime racial discrimination exposure and SES mobility based on education or income. None of the interaction terms were found to be statistically significant, indicating that neither age nor gender moderated this relationship; therefore, these findings are not presented. Further analysis was conducted to assess the relationship between lifetime racial discrimination exposure and SES mobility based on education and income after controlling for gender and age. These results are presented in Table 10 below.

Table 10 displays the *p*-values, the exponentiated B values (Exp(B), odds ratios (OR), and the confidence intervals (CI) of the OR. The high discrimination group is the reference category. The findings in Table 10 indicate that after controlling for age,

gender, physical activity, alcohol, and social stressors, SES mobility in terms of education significantly predicted exposure to racial discrimination ($p < .001$). Specifically, respondents categorized as having stable low SES mobility based on education were less likely to experience a high level of discrimination than respondents who were categorized as having increasing SES mobility, *OR* = 1.63, 95% CI [1.31, 2.02], and respondents who were categorized as having stable high SES mobility, *OR* = 1.38, 95% CI [1.05, 1.81]. However, SES mobility based on income did not significantly predict exposure to racial discrimination ($p = .633$). To ensure that the two measures of SES mobility were not closely interrelated, the relationship between education and income mobility were tested for collinearity. SES education mobility was not significantly related to SES income mobility $\chi^2(9) = 12.52$, $p = .186$. Moreover, this model did not show SES mobility to account for the variance (5.6%) in racial discrimination exposure.

Table 10

Logistic Regression Results for SES Mobility and Lifetime Discrimination Exposure (N = 2254)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.000			
55 to 64 vs. 34 to 54	-.10	.374	.91	.73	1.13
55 to 64 vs. 65 to 84	-.53	.000	.59	.46	.76
Gender: male vs. female[1]	.28	.003	1.32	1.10	1.60
Physical activity		.127			
Poor vs. intermediate health[1]	.21	.047	1.24	1.00	1.52
Poor vs. ideal health[1]	.05	.688	1.05	.83	1.34
No alcohol vs. alcohol in past year[1]	.19	.059	1.21	.99	1.48
No financial stress vs. financial stress[1]	.64	.000	1.90	1.40	2.58
John Henryism total score	-.01	.217	.99	.97	1.01
SES mobility in education[1]		.000			
Stable low vs. diminishing	.11	.520	1.12	.80	1.56
Stable low vs. increasing	.49	.000	1.63	1.31	2.02
Stable low vs. stable high	.32	.023	1.38	1.05	1.81
SES mobility in income[1]		.633			
Stable low vs. diminishing	.15	.345	1.17	.85	1.60
Stable low vs. increasing	-.06	.790	.94	.62	1.44
Stable low vs. stable high	.07	.638	1.08	.79	1.46

Note. Overall fit for the fourth and final step, $\chi^2(14) = 92.83$, $p < .001$, Nagelkerke $R^2 = .056$.

[1] Reference categories appear first in the group comparison.

The Relationship Between SES Mobility and Levels of Burden Due to Exposure to Discrimination (Research Question 2)

The second research question sought to determine the relationship between levels of SES mobility and levels of burden attributed to lifetime racial discrimination, and determine whether or not the relationship was moderated by age or gender. A binomial logistic regression was conducted with lifetime discrimination burden as the dependent variable and SES mobility income and SES mobility education as the independent variables. With the binomial logistic regression, there are only two categories of the dependent variable. So, the reference category was the low discrimination group. Therefore, all significant Exp(B) that were greater than 1, were more likely to be in the high discrimination group versus the low discrimination group. Conversely, all significant Exp(B) that were less than 1, were less likely to be in the high discrimination group versus the low discrimination group. As with the multinomial logistic regression, the independent variable categories are compared to their corresponding stable-low category (ex. stable-low income vs. stable-high income, increasing income, and diminishing income).

To answer this second question, a hierarchical logistic regression procedure was conducted as in the previous model. In the first step, the demographic variables were entered into the equation; in the second step, the lifestyle variables were entered; in the third step, the social stressors were entered; in the fourth step, the independent variables were entered. Similar to the previous research question, age and gender were also

evaluated to determine the presence of effect modification. None of the interaction terms were found to be statistically significant, indicating that neither age nor gender moderated this relationship; therefore, these findings are not presented. Further analysis was conducted to assess the relationship between lifetime racial discrimination burden and SES mobility based on education and income after controlling for gender and age. These results are presented in Table 11 below.

As shown in Table 11, after controlling for age, gender, smoking activity, and social stressors, SES mobility based on education significantly predicted lifetime burden due to exposure to racial discrimination, $p < .05$. Specifically, respondents categorized as having stable low SES mobility in terms of education were more likely to experience a high burden due to racial discrimination than respondents who were categorized as having diminishing SES mobility, $OR = .71$, 95% CI [.53, .96]. Yet, SES mobility based on income did not significantly predict lifetime burden due to exposure to racial discrimination ($p = .821$). Moreover, this model did not show SES mobility to account for the variance (1.6%) in racial discrimination burden.

Table 11

Logistic Regression Results for SES Mobility and Burden Levels (N = 2551)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.145			
55 to 64 vs. 34 to 54	.15	.113	1.16	.97	1.40
55 to 64 vs. 65 to 84	.17	.098	1.18	.97	1.45
Gender: male vs. female[1]	.19	.018	1.21	1.03	1.42
Smoking: poor vs. ideal health[1]	-.34	.005	.71	.56	.90
No financial stress vs. financial stress[1]	.33	.026	1.39	1.04	1.85
SES mobility in education[1]		.038			
Stable low vs. diminishing	-.34	.025	.71	.53	.96
Stable low vs. increasing	.13	.186	1.14	.94	1.38
Stable low vs. stable high	-.02	.860	.98	.77	1.25
SES mobility in income[1]		.821			
Stable low vs. diminishing	-.01	.935	.99	.76	1.29
Stable low vs. increasing	-.10	.586	.90	.63	1.30
Stable low vs. stable high	-.08	.522	.92	.72	1.18

Note. Overall fit for the fourth and final step, $\chi^2(11) = 31.38$, $p < .001$, Nagelkerke $R^2 = .016$

[1] Reference categories appear first in the group comparison.

The Relationship Between Hypertension, SES Mobility, Levels of Discrimination, Levels of Burden Due to Exposure to Discrimination, and Cardiovascular Disease (Research Question 3)

The third research question sought to determine whether SES mobility, cumulative perceived lifetime racial discrimination exposure, and burden due to racial discrimination exposure would moderate the relationship between hypertension and cardiovascular disease. To address this research question, first a binomial logistic regression was performed to establish if there was a significant association between hypertension, the independent variable, and cardiovascular disease, the dependent variable. Hypertension was a dichotomous variable where 0 was no hypertension and 1 represented a hypertension diagnosis. Cardiovascular disease was also a dichotomous variable where 0 was no cardiovascular disease, and 1 indicted a cardiovascular disease diagnosis. Results of the binomial logistic regression indicated that there was a significant relationship between hypertension and cardiovascular disease, $\chi^2(1) = 177.779$, $p < .001$, where the explained variability in cardiovascular disease status ranged from 3.3% (Cox and Snell R squared) to 6.9% (Nagelkerke R squared). The results also indicated that respondents with hypertension were 4.3 times more likely to have cardiovascular disease than those who did not have hypertension, $Exp(B) = 4.369$, $p < .001$ (Table 12).

Table 12

Binomial Logistic Regression - Cardiovascular Disease and Hypertension

	B	*SE*	Wald	*df*	*p*	Exp(B)
Hypertension	1.474	.127	134.439	1	.000	4.369
Constant	-3.239	.117	767.344	1	.000	.039

To answer this third question, a hierarchical logistic regression procedure was conducted as in previous models. In the first step, the demographic variables were entered into the equation; in the second step, the lifestyle variables were entered; in the third step, the risk factors were entered; in the fourth step, the independent variables were entered; in the final step, the interaction terms were entered. The main terms (i.e., HTN and SES-E, SES-I, discrimination, and burden) were entered into the model separately before adding the interaction terms (results not shown). The findings in Table 13 indicate that SES mobility, cumulative perceived lifetime racial discrimination exposure, and burden due to racial discrimination exposure did not significantly moderate the relationship between hypertension and likelihood of cardiovascular disease.

Table 13

Logistic Regression Results for SES Mobility, Discrimination Exposure, Burden Levels, Hypertension, and Cardiovascular Disease (N = 2590)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.001			
34 to 54 vs. 55 to 64 (Age 1)	.39	.031	1.48	1.04	2.10
34 to 54 vs. 65 to 84 (Age 2)	.69	.000	1.99	1.39	2.86
Nutrition: poor vs. intermediate health	-.33	.024	.72	.54	.96
HDL	-.45	.024	.64	.43	.94
LDL	-.27	.001	.76	.65	.90
Hypertension: no vs. yes (HTN)[1]	.40	.494	1.49	.48	4.61
SES mobility in education[1]		.056			
Stable low vs. diminishing	.09	.814	1.10	.50	2.40
Stable low vs. increasing	-.93	.012	.40	.19	.81
Stable low vs. stable high	-.46	.231	.63	.30	1.34
SES mobility in income[1]		.640			
Stable low vs. diminishing	.48	.354	1.62	.59	4.45
Stable low vs. increasing	.77	.201	2.16	.66	7.04
Stable low vs. stable high	.42	.406	1.52	.57	4.03
Burden: low vs. high[1]	.07	.792	1.07	.64	1.80
Discrimination: low vs. high[1]	.34	.211	1.41	.82	2.40

HTN x SES mobility in education[1]		.400			
HTN x SES-E 1	.07	.888	1.08	.39	2.95
HTN x SES-E 2	.72	.090	2.06	.89	4.76
HTN x SES-E 3	.05	.921	1.05	.39	2.81
HTN x SES mobility in income[1]		.963			
HTN x SES-I 1	-.22	.719	.81	.25	2.61
HTN x SES-I 2	-.33	.655	.72	.17	3.01
HTN x SES-I 3	-.12	.841	.89	.29	2.76
HTN x discrimination	-.21	.504	.81	.43	1.51
HTN x burden	-.34	.308	.71	.37	1.37

Note. Overall fit for the fifth and final step, $\chi^2(22) = 54.94$, $p < .001$.

[1] Reference categories appear first in the group comparison.

The Relationship Between Hypertension, SES-Racism, and Cardiovascular Disease (Research Question 4)

The fourth research question sought to determine whether the SES-racism effect would moderate the relationship between hypertension and cardiovascular disease. A SES-racism effect variable was created by combining SES mobility (of both education and income independently) and lifetime racial discrimination exposure to examine its interaction with hypertension. To answer this fourth question, a hierarchical logistic regression procedure was conducted as in previous models. In the first step, the

demographic variables were entered into the equation; in the second step, the lifestyle variables were entered; in the third step, the risk factors were entered; in the fourth step, the independent variables were entered; in the final step, the interaction terms were entered. Neither the interaction nor the main effect terms were found to be significant when examining them independently. Hence, interaction terms are not reported. Furthermore, the inclusion of both SES-Racism variables (for education and income) in the model yielded missing output. As a result, the main effect terms are presented separately in Tables 14 and 15.

Table 14

Logistic Regression Results for SES-Racism Effect (Education), Hypertension, and Cardiovascular Disease (N = 2289)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.001			
34 to 54 vs. 55 to 64 (Age 1)	.37	.040	1.44	1.02	2.05
34 to 54 vs. 65 to 84 (Age 2)	.67	.000	1.96	1.37	2.80
Nutrition: poor vs. intermediate health	-.33	.024	.72	.54	.96
HDL	-.43	.031	.65	.44	.96
LDL	-.27	.001	.76	.65	.89
Hypertension: no vs. yes (HTN)[1]	.15	.338	1.17	.85	1.60
SES-Racism Effect for education[1]		.159			
Increasing SES - Low Racism (SESe-R 1)	-.27	.433	.77	.39	1.49
Diminishing SES - Low Racism (SESe-R 2)	.50	.189	1.64	.78	3.44
Stable low SES - Low Racism (SESe-R 3)	.19	.498	1.21	.69	2.13
Stable high SES - High Racism (SESe-R 4)	-.56	.289	.57	.20	1.61
Increasing SES - High Racism (SESe-R 5)	-.02	.950	.98	.49	1.97
Diminishing SES - High Racism (SESe-R 6)	.13	.812	1.14	.39	3.29
Stable low SES - High Racism (SESe-R 7)	.37	.234	.45	.79	.69

Note. Only results for fourth and final step are reported. Overall ft for the fourth step, $\chi^2(13) = 49.39$, $p < .001$. [1] Reference categories represents Stable high SES – Low Racism, which is consider optimal.

Table 15

Logistic Regression Results for SES-Racism Effect (Income), Hypertension, and Cardiovascular Disease (N = 2289)

Variables	*B*	Sig.	*OR*	95% CI for *OR* Lower	Upper
Age group in years[1]		.000			
34 to 54 vs. 55 to 64 (Age 1)	.38	.031	1.47	1.04	2.08
34 to 54 vs. 65 to 84 (Age 2)	.70	.000	2.02	1.42	2.87
Nutrition: poor vs. intermediate health	-.36	.013	.70	.52	.93
HDL	-.41	.038	.66	.45	.98
LDL	-.27	.001	.76	.65	.89
Hypertension: no vs. yes (HTN)[1]	.18	.270	1.19	.87	1.63
SES-Racism Effect for income[1]		.709			
Increasing SES - Low Racism (SESe-R 1)	.30	.319	1.35	.78	2.46
Diminishing SES - Low Racism (SESe-R 2)	.07	.720	1.07	.74	1.56
Stable low SES - Low Racism (SESe-R 3)	-.14	.632	.87	.50	1.53
Stable high SES - High Racism (SESe-R 4)	.21	.329	1.23	.81	1.85
Increasing SES - High Racism (SESe-R 5)	.16	.747	1.17	.45	3.08
Diminishing SES - High Racism (SESe-R 6)	.01	.978	1.01	.60	1.68
Stable low SES - High Racism (SESe-R 7)	.71	.181	.49	.17	1.39

Note. Only results for fourth and final step are reported. Overall ft for the fourth step, $\chi^2(13) = 49.2$, $p < .001$. [1] Reference categories represents Stable high SES – Low Racism, which is consider optimal.

Summary of Findings

The first research question sought to determine whether SES mobility would predict level of exposure to discrimination. The findings indicated that SES mobility, as measured by education, predicted level of exposure to racial discrimination. Specifically, respondents categorized as having stable low SES mobility based on education were less likely to experience a high level of racial discrimination than respondents who were categorized as having increasing and stable high SES mobility.

The second research question sought to determine whether SES mobility would predict burden levels due to exposure to discrimination. The findings indicated that SES mobility, as measured by education, predicted burden levels due to exposure to discrimination. Specifically, respondents categorized as having stable low SES mobility in terms of education were more likely to experience a high burden due to racial discrimination than respondents who were categorized as having diminishing SES mobility.

The third research question sought to determine whether SES mobility, level of exposure to racial discrimination, and level of burden would moderate the relationship between hypertension and cardiovascular disease. None of these variables had a moderating effect on the relationship between hypertension and cardiovascular disease.

Finally, the fourth research question sought to determine whether the SES-racism effect (i.e., the interaction between SES mobility and level of discrimination exposure) would moderate the relationship between hypertension and cardiovascular disease. The

findings reveal that the SES-racism effect did not moderate the relationship between hypertension and cardiovascular disease.

Chapter 5: Discussion

Introduction

The purpose of this study was to evaluate whether there was a relationship between perceived lifetime racial discrimination and SES mobility, if the relationship was moderated by age or gender, and if the interaction of these variables moderated the relationship between hypertension and CVD. This study was conducted as an opportunity to enhance public health research methodologies regarding the measurement of racial discrimination's influence of CVD related health outcomes in Black populations in the United States. This study was also conducted to fill gaps in understanding how the role of racial dynamics, coupled with changes in socioeconomic mobility, influence poor health outcomes among Blacks in Jackson, Mississippi, and to provide evidence for needed changes in policies, practices, infrastructure, and/or social norms. Data from the Jackson Heart Study (JHS) were analyzed to measure outcomes to the research questions. This chapter addresses the findings of this research study, limitations of the study, recommendations for continued research, and implications for social change.

Summary and Interpretation of the Findings

There were four research questions used to explore whether or not exposure to racial discrimination had any bearing on the relationship between hypertension and CVD. The findings indicated that SES mobility, as measured by education, predicted both the exposure to perceived lifetime racial discrimination and the burden that participants experienced. Participants with stable low SES mobility based on education were less likely to experience a high level of racial discrimination exposure than respondents who

were categorized as having increasing and stable high SES mobility. Conversely, the same group was more likely to experience a greater burden from the exposure that they did experience than respondents who were categorized as having diminishing SES mobility. However, the remaining models illustrated that neither SES mobility nor racial discrimination had any effect in moderating the relationship between hypertension and cardiovascular disease when examined individually or collectively.

In Chapter 2, the literature review described the role of both SES mobility and racial discrimination in contributing to adverse CVD-related outcomes. Studies that examined racial discrimination historically reported widely varied results, indicators of measurement, and population groups (Chae et al., 2010; Din-Dzietham et al., 2004; Dolezsar et al., 2014; Krieger et al., 2013; Krieger & Sidney, 1996; Roberts et al., 2008; Sims et al., 2012). While little evidence is available on strategies to comprehensively assess lifetime racial discrimination, this study does extend the results found in the CARDIA Study in which professional Blacks were found to be more strongly effected by racial discrimination experiences than working class Blacks (Krieger & Sidney, 1996).

However, unlike the CARDIA Study, expected elevated blood pressure results were not observed in this dissertation study. Bastos et al. (2010) and Williams et al. (2012) agreed, however, that the effect of racism on disease pathways vary based on how racism is manifested (e.g., job or provider-related, aggressive behaviors or actions, social rejection), and others (Chae et al., 2010; Williams & Mohammed, 2013) advise that understanding of whether or not any dose-response from exposure exists is unknown. Furthermore, previous research illustrated that differences in racial discrimination

experiences were often influenced by gender and/or age (Chae et al., 2010; Dolezsar et al., 2014; Krieger & Sidney, 1996), yet that was not confirmed as a moderating effect by this study. Studies that demonstrated age or gender differences in racial discrimination were often smaller studies and had no or unequal comparable population. JHS has not only a large sample size, but also an appropriately representative cohort, which provides statistical power that may minimize age and gender differences that might otherwise be observed in smaller studies.

Similar to previous racial discrimination research, the literature depicted the SES mobility and SES constructs as being inconsistently measured across studies. SES mobility studies have based this construct on varied combinations of education, income, occupation, and/or wealth measurements (Adler & Stewart, 2010; Conroy et al., 2010; Pollitt et al., 2005). As expected, the findings of this study aligned with previous JHS research in which Sims et al. (2012) found that participants experiencing high levels of lifetime discrimination had high SES. However, the results of this dissertation study did not confirm increased risk of hypertension translating to the expected CVD outcome. Sims et al. (2012) included all measures of lifetime discrimination (e.g., weight, gender, racial) and only adult SES measurements, subtle differences that may have resulted in altered findings. This study also confirms the findings of Sellers et al. (2009) and Sellers, Neighbors, and Bonham (2011) demonstrating that middle-class Blacks of higher educational status more frequently encountered racially motivated experiences.

Conversely, research that actually examined SES mobility documented an inverse relationship between lifecourse SES and CVD (Adler & Stewart, 2010; Berry et al., 2012;

Hogberg et al., 2011; Johnson-Lawrence et al., 2013; Roberts et al., 2010; Wamala et al., 2001), with individuals of high and increasing SES mobility having the lowest risk (Hogberg et al., 2011; James et al., 2006; Johnson-Lawrence et al., 2013). The SES mobility indicators used in this study, based on singular measurements of education and income, may attribute to why these findings were not confirmed. Measuring these indicators of SES in isolation as a proxy for SES mobility deprives the researcher from considering the long-term effects of additional socioeconomic circumstances that were not included. Singular measurements of SES mobility are therefore not only incomplete, but also fail to capture how health status is impacted by variations in the dimension of social stratification (Blank et al., 2004).

Moreover, aforementioned SES mobility investigations do not address any aspect of racial discrimination. Since racism serves to create unequal opportunities and worth, it makes sense that more affluent Blacks, who have the potential to possess similar privileges, resources, and power, pose the greatest threat. While previous racial discrimination studies may have taken into account participants of different SES levels, no studies were found that addressed the role that SES mobility plays in the extent of exposure or burden due to racial discrimination which individuals experience. To this end, the research presented in this study extends previous research by considering the addition of this element. The research questions for this study attempted to establish an interaction between commonly assessed contributors to health disparities and bridge research gaps that potentially attributed to the variations observed across studies. This study extends our understanding of how SES indicators are not only strongly patterned by

race (Williams et al., 2010), but potentially also the social conditions of the community in which an individual lives.

This investigation was rooted in two conceptual frameworks. First, the framework created by Harper et al. (2011) suggested that CVD outcomes are the result of SES position guiding the prevalence of risk factors an individual is exposed to. However, the framework cautions that the effect of this relationship is conditional based on the strength of the linkage between macrosocial factors and proximal causes of disease over an individual's lifecourse, historical context, and geographic location. Although racism is considered a macrosocial factor, there are numerous other factors (e.g., political ideologies, cultural belief systems, economic philosophies) that may work in concert to effect this relationship differently than racism alone. This framework also examines the interaction of these dynamics across place and time. This study considers the impact of time, related to lifetime measurements, but not historical or geographic contexts. It is plausible that not only racial discrimination, but also SES mobility, are impacted differently as these contexts are observed individually and collectively.

The second framework, created by Williams and Mohammed (2013) contends that health disparities occur as a response to a multi-layered and cumulative impact of social factors negatively intervening on the proximal pathways that link risk factors to health outcomes. As Williams and Mohammed's (2013) framework emphasizes various social factors as intervening mechanisms on intricate and multidimensional processes, it is noted that there are numerous elements of this framework that were not included in the analysis of this study (e.g., societal institutions, societal resources, physiological

responses). Given that this study only examines racism as it intervenes on some indicators of SES mobility, additional studies are warranted that can further extend this research to include more broadly measured constructs.

Both frameworks observe the interaction between SES mobility and racial discrimination as part of a larger, more complex model. This study lends support to the relationship between racial discrimination and social status; however, exclusion of the additional aspects of both models that were not measured in this study may have limited the ability to illustrate linkage to distal risk factors (i.e., hypertension) and adverse health outcomes (i.e., CVD).

Strengths of the study

Historically, Blacks are underrepresented in research studies, even though they are more likely to be disproportionately affected by chronic diseases; this combination increases the difficulty in exploring the possible influences that contribute to these health disparities (Diaz, Mainous, McCall, & Geesey, 2008; Fuqua et al., 2005; Schmotzer, 2012). The participation of Blacks in research studies across the United States is reported to range from 3% to 20% (Fuqua et al., 2005). Researchers have identified numerous reasons that contribute to these low participation rates, such as mistrust of researchers and/or healthcare systems, lack of minority researchers, cultural barriers, and failure of researchers to actively recruit Blacks (Diaz et al., 2008; Durant et al., 2014). The JHS was established as a follow-up to the ARIC study, a familiar and trusted research study among Blacks in metro Jackson, MS. There are few studies to date that have specifically focused and collected longitudinal data on CVD and a multitude of interconnected factors

that influence the manifestation of disease outcomes to the extent of the JHS (Taylor et al., 2005).

The JHS is the largest study of CVD among Blacks (Taylor et al., 2005). While this population cohort is restricted to a single site, the historical awareness of both blatant and subtle forms of racial discrimination in Mississippi creates an unparalleled snapshot in which to investigate issues that may affect Blacks across the nation. The large sample size of participants in the JHS provided the necessary power to detect small, but significant relationships between the variables of interest; thereby, giving strength to study findings and allowing for an in-depth examination of a complex phenomena.

In addition, JHS is a longitudinal study with data spanning more than 10 years, collected on a wide array of indicators. The vast array of indicators that have been collected and linked to CVD in JHS have supported advances to the field of social epidemiology and the role of social determinants of health in shaping health outcomes (Fuqua et al., 2005; Payne et al., 2005). It also provides objective data and outcomes, as well as identifies concrete and plausible areas around which to intervene.

Finally, this study investigated the complex, multidimensional experiences of Blacks from a unique perspective. Exploration of the literature to date found that there is little or no research that has been conducted to examine the relationship between SES mobility and perceived lifetime discrimination (Adler & Stewart, 2010), or how the combination of these two variables (i.e., SES-Racism effect) may influence the relationship between hypertension and CVD. Hence, this study conceptualizes a new mechanism for investigating the multifaceted pathway by which racism impacts health

outcomes. While this study did not demonstrate SES-Racism effect to be a moderator, the positive relationship between SES mobility and lifetime racial discrimination exposure warrants further investigation.

Limitations of the Study

As previous reported in Chapter 1, there are overall limitations in the strength of this study and its findings; therefore, these findings should be considered with caution. The Jackson Heart Study is a unique cohort in which to measure both perceived discrimination, as well as SES mobility, given that the data was collected in the backdrop of a geographic region known to have historical racial issues, SES disadvantage, and a high prevalence of CVD (Payne et al., 2005; Sims et al., 2009; Taylor, 2003) This study was based on secondary data analysis, which has limited specificity due to the use of fixed survey questions. The data ascertained measured aspects of discrimination attributed to race, and do not entirely encompass the definition of racism (Sims et al., 2012; Sims et al., 2009).

Racial discrimination is a very subjective construct with a wide range of interpretation that is personally mediated based on an individual's vantage point; whereas, racism may be thought of as more overt acts which could also be included under the umbrella of racial discrimination (Bonilla-Silva, 2001; Jones, 2000; Kumanyika & Jones, 2015). The survey questions used to measure discrimination do not account for differences in perceived discrimination. Differences in how an individual perceives racial discrimination may vary based on several factors such as coping mechanisms, situation or circumstances of the event, historical exposure, tolerance, or generational beliefs. The

development of a standardized definition and understanding of what constitutes racial discrimination may reveal that the prevalence of both racial discrimination exposure and burden has been substantial under reported. Factors such as these may prevent a cause-effect relationship from being demonstrated using this correlational study to assess how the relationship between hypertension and CVD over the lifecourse is moderated by racial discrimination and SES.

Finally, the study measures only Blacks located in the metro Jackson, MS area, and any findings are not generalizable to geographic areas or other racial/ethnic populations that may experience racial discrimination. Blacks in Mississippi have exposures and experiences that are different from Blacks in other geographic areas. Furthermore, this study did not have a White population cohort to compare exposures and outcomes. The inability to compare differences in racial discrimination exposure between Whites and Blacks eliminates the opportunity to understand how the membership to a racial/ethnic group, including coping mechanisms or health-related behaviors commonly observed or practiced by a particular population group, may influence health outcomes (Brondolo et al., 2005). These limitations provide justification for further research to be conducted in a wider population, additional geographic locations, and using more specific methodologies.

SES mobility could not be measured as originally planned. Although derived categories for adult occupational data were provided by JHS, equivalent aggregate data for parental occupational was not (M. Sims, personal communication, December 16, 2014). As such, there was an inability to demonstrate SES mobility based on occupation.

Furthermore, the SES mobility construct was initially a cumulative measurement based on previously developed research, which was later determined to be incomplete. Examining singular measures of SES limits the extent to which we can understand an individual's true SES mobility relative to other factors. This study assumes that individuals of stable low or diminishing SES mobility for education do not fair as well as an individual of higher educational status. However, it is quite plausible that these assumptions to not always hold true.

A person with only a high school diploma may have an income that categorizes them as being affluent. More than 80% of the participants in this study were found to have lower educational status (i.e., high school diploma or less), but roughly 66% had at least upper-middle income. Also, previous studies have found that even well-educated Blacks may have relatively poor health outcomes because they frequently reside in neighborhoods with less than desirable characteristics (Bucholz, Ma, Normand, & Krumholz, 2015; Jackson, Rowley, & Owens, 2012; Jones-Jack et al., 2010; LaVeist et al., 2011; Logan, 2011); hence, residential neighborhood characteristics may also need to be considered as a SES mobility component.

Furthermore, the childhood SES measurements (i.e., education and income) potentially introduce biases into the study. These measurements required adult participants to recall information about their parents' socioeconomic status, which they may not have been fully aware of or have the ability to recall accurately. It is difficult to determine whether this recall bias would be overestimated or underestimated. Prediction

about the direction of this bias would require knowledge of the fluctuations in major socioeconomic influences that may have occurred over the wide timespan of the cohort.

Finally, this study only examined a cross-section of data collected at baseline. However, there are two additional exam periods that warrant further investigation (JHS, 2015). The snapshot of data analyzed may not provide a true synopsis of the effect racial discrimination on hypertension and CVD outcomes. Further investigation, such as a time series analysis, may provide additional insight on whether participants experience delayed adverse health outcomes associated with varying frequencies of racial discrimination exposure or burden.

Recommendations for Future Research

There continue to be inconsistencies in research examining the influence of racial discrimination on the health outcomes of Blacks. Chapter 2 documented previously conducted studies, described differences the population groups explored, and deficiencies in measurement strategies. While the JHS Discrimination Instrument Survey provided a more comprehensive approach for ensuring that the multiple factors that contribute to the layered mechanisms that drive the institution of racism were accounted for, there were additional aspects of this study that warrant further analysis. Focusing on lifetime racial discrimination exposure and burden did not consider how an individual's physiology prepares an armored defense and heighten sense of expectation of subsequent incidents. Sims et al. (2012) indicated that recent studies have suggested that "clinic measures of hypertension might be insufficient to detect associations with discrimination" (p. S263). Some researchers (Dolezsar et al., 2014) posited that ambulatory blood pressure

measurements may be necessary to more accurately link the effects of racially discriminatory experiences, particularly when focusing on everyday experiences. However, in an environment where racial discrimination has long been tolerated as commonplace, researchers must identify improved techniques to control for the acceptance and normalization of minimizing policies, practices, and social norms.

Although the JHS researchers utilized previous research to develop a discrimination construct that was more inclusive of aspects of discrimination found to either not be consistently measured across studies or found to be gaps in investigations (Sims et al., 2009), racial discrimination is still a subjective measurement. Additional questions about how racial discrimination is measured have yet to be addressed for this population cohort. For example, the magnitude to which one experiences an event identified as being *racially charged* may easily vary from one person to the next based on their level of tolerance, previous experiences/exposures, and coping mechanisms. Racial discrimination is a social stressor, and further study aligning JHS participants' reporting of racial discrimination exposure and burden with cortisol levels may enhance the validity of this measurement.

Substantial evidence has been generated to indicate that elevated, prolonged stress invokes physiological and hormonal responses that increase an individual's risk for adverse health outcomes (Thoits, 2010). However, social inequality is a source of various psychosocial stressors that are subtly, but often relentlessly, embedded in the daily aspects of disadvantaged populations. Researchers agree that individuals who experience social and economic inequality are at risk of greater emotional distress (Lamech &

Haynes, 2015; Mildestvedt & Meland, 2007; Thoits, 2010). Social and economic inequality are often associated with increased feelings or perceptions of vulnerability, helplessness, deprivation, and dependence (Baum et al., 1999), as well as limited coping ability and motivation toward health-promoting behaviors (Mildestvedt & Meland, 2007). Further refinement of the JHS Discrimination Instrument Survey to address these gaps is warranted.

A strategy to expand this study would be to include subsequent examination periods. This study was conducted using only baseline data from the JHS cohort collected in 2004; however, the Discrimination Instrument was also used to collect data from this cohort during Exam 3 (2009-2012). A prospective analysis of the JHS cohort over time would provide a greater opportunity to understand if and how additional contextual factors (e.g., political changes, societal traumas) or continued personal experiences modify how individuals report their lifetime racial discrimination exposure and any burden it causes in their life. Similarly, SES mobility was only measured at baseline. The data collected during this exam period do not take into consideration recent changes in the economic climate of this country. Subsequent analysis may reveal a shift in the prevalence of individual who report upper-middle or affluent socioeconomic classification during adulthood. Finally, roughly 60% of JHS study participants had hypertension at baseline, and 10% had been diagnosed with CVD. However, hypertension and CVD are both health conditions that increase in prevalence as individuals age. Since the majority of study participants were <65 years of age, it is likely that the prevalence of CVD will increase as this longitudinal study progresses. The

influence of such factors on each of these constructs has the potential to demonstrate that SES-Racism Effect moderates the relationship between hypertension and CVD.

Future research needs to better understand how the impact of differing racial discrimination burden effect health outcomes. More specifically, additional research should examine whether or not there is a threshold of racial discrimination that an individual much reach before it considered to be detrimental to their health. Williams and Mohammed (2013) maintain that the age at which the initial experience occurs, the accumulation of those lifetime experiences, and the trajectory of illness is not clearly understood. Also, are Blacks predisposed to the impact of racial discrimination before birth? There have been generations of Blacks that have been exposed to structural, sociopolitical, and institutionalized racism. Gee and Ford (2011) suggest that more comprehensive research examining racism and health include the concept of intergenerational drag, which is the passing of "social assets and liabilities on to their descendants" (Darity Jr., Dietrich, & Guilkey, 2001, p. 435). In the same way that White populations a historically benefited from passing down wealth from one generation to the next (Domhoff, 2011; Kochhar et al., 2011), Blacks are also hypothesized to also pass down their experiences of historical trauma. The JHS cohort is uniquely positioned to have participants that not only span the continuum of adulthood, but also have family connections in some cases. Future research with JHS data may serve as a viable source to explore the exposure and burden of racial discrimination with family cohorts and their linkage to CVD related health outcomes.

The research maintains that a linkage between racial discrimination and CVD-related health outcomes exists. However, this is undoubtedly a complex pathway, and identifying how to demonstrate this connection has been challenging. This research originally sought to examine SES mobility as a cumulative construct. However because details about the construct were not available, single indices of the construct were examined. From this perspective, the measurement of SES mobility observed in this study was incomplete because education and income alone are not explicitly predictive of changes in an individual's lifetime SES; meaning, an individual of low educational status may have a high income and vice versa. Therefore, future studies need to examine the role of the cumulative impact of not only education and income, but also occupation and wealth. Furthering this study with the use of a more complete SES mobility construct may yield different results.

Implications for Social Change

Because Blacks experience rates of hypertension and CVD that are higher than other racial ethnic populations, regardless of education and income, one must consider the influence of nontraditional social determinants of health. Since education-based SES mobility was found to predict level of exposure to racial discrimination and the burden that participants experienced, this study supports the fact that the institutional dynamics that create racial factors, as well as the impact of such experiences on individuals, are complex. Furthermore, translating this research into public health practice also requires the implementation of multifaceted investments to ameliorate these gaps in addressing

health disparities (Krieger, 2012b; Kumanyika, 2012; Kumanyika & Jones, 2015; Williams & Mohammed, 2013).

Research has continued to peel back the layers of the onion in tackling chronic disease health improvements, addressing risk factors and social determinants of health, and developing modified methodologies. While this would certainly include patient-provider relationships, health promoting messages, and the availability of resources that are respectful, trustworthy, and culturally sensitive, history has shown that this barely scratches the surface. There are yet additional layers to be uncovered. Using traditional public health approaches to address social issues, like racism, seems to be the equivalent of tossing a pebble into raging rapids with the expectation of it having some impact. Focusing on the social determinants of health (e.g., education, food choices, improving physical activity, neighborhood poverty) continues to amplify that all is not fair or just, and fails to force discussions that sincerely unpack the underlying historical issues with transparency and demonstrate a true sense of equality for all lives (Kumanyika & Jones, 2015).

This country's sense of fairness influences how individuals of all backgrounds, SES groups, etc. can equitably take advantage of health care and resources, and are encouraged and supported to do so. The American Public Health Association's President, Camara Jones, argues that "Disparities arise from differences in quality of care, access to care & life opportunities, exposures & stresses" (2015). Unlike social determinants of health, social determinants of equity include systems of power which are mechanisms for decision making processes that can distribute resources in populations (e,g, racism,

capitalism) (Kumanyika & Jones, 2015). In order to make impactful and sustainable social changes in minimizing adverse health outcomes among Blacks attributed to upstream racially discriminatory factors, society must embrace our common humanity to understand and accept that regardless of the tenacity of individual's effort, they will continue to experience health disparities in the face of contextual and structural lack (Krieger, 2012b; Kumanyika & Jones, 2015; Williams & Mohammed, 2013). The understanding that our health outcomes begin to be shaped early in life, long before the manifestation of any health condition, supports the need for Blacks to have the opportunity to level the playing field in a manner that is both equal and equitable. Consequently, the implementation of policies, practices, and changes to social norms are also necessary.

Williams and Mohammed (2013) posited that in order to rectify the institutionalized racism that lies within longstanding practices, policies, and social norms, a three-pronged approach is needed. Such as approach consists of:

1. cultivating improved living, educational, and employment conditions, as well as income potential, that enhance access to resources and services that will improve health;
2. minimizing the societal messages and images that undergird the perpetuation of discrimination and prejudice at the societal and individual level; and
3. implementing policies to support sustainable behavior change and empowerment over health outcomes at individuals and communities levels (Williams & Mohammed, 2013).

Implementing such strategies as a multi-layered effort may substantially minimize the extent to which Blacks perceive their experiences across domains (e.g., work/school, receiving services, public settings, receiving medical care) as racially discriminatory, as well as reduce the burden that such racism has had on their lives.

This research effort was important because it provides further evidence to show that while linkages between societal influences and social determinants of health exist, how individuals are ultimately impacted by the culmination of these experienced requires continued advancement. This study examined a new approach for measuring the relationship of racial discrimination on health outcomes, as well as understanding the contextual and relational factors that provide structure for a complex area of study. The JHS itself provides support for the fact that health is a function of a multifactorial interaction that includes biological changes, psychosocial functioning, environmental attributes, and institutional responsibility. The next step in advancing this field of study lies in creating a platform to engage both public health and nontraditional professionals in collaborative efforts to redefine and improve future parameters used to articulate strategies for routinely monitoring differential exposures, identifying the mechanisms (e.g., policies, structures, values, practices) that allow racism to exist, and actively engaging a national effort to eliminate the factors that perpetuate these conditions (Kumanyika & Jones, 2015).

Conclusions

In summary, the literature provides evidence to support perceived racial discrimination as a factor predisposing Blacks to elevated rates of hypertension. This

research illustrates that Blacks in the metro Jackson, MS area are not only likely to experience perceive racial discrimination over their lifecourse, but also attribute racial discrimination to having a burdensome impact on their life. This research also suggests that there is an association between levels of SES mobility based on education and levels of perceived racial discrimination exposure. While Blacks of stable high and increasing SES were found to be more likely to be impacted by exposure and burden attributed to racism, assumptions should not be made that Blacks of lower SES are not impacted.

Continued research is important to improve measurement strategies that more comprehensively capture these social constructs. In addition, both public health professionals and health care providers must be more astutely aware of the pervasiveness of racially discriminatory policies, practices, institutional barriers, and social norms that continue to exist in our society. Understanding that willingness to openly label and discuss the systems that allow racism to exist, not necessarily singling out an individual, may have tremendous influence in healing historical wounds, and ultimately diminishing health disparities.

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Appendix A: JHS Eligibility Form

Eligibility Form

FORM CODE: ELG
VERSION A10/11/2000

ID NUMBER: _____ CONTACT YEAR: 0 1

LAST NAME: _____ INITIALS: _____

INSTRUCTIONS: This form is to be completed for each eligible person living in the household of the sample person. ID Number, Contact Year, and Name must be entered above. Whenever numerical responses are required, enter the number so that the last digit appears in the rightmost box. Enter leading zeroes where necessary to fill all boxes. If a number is entered incorrectly, mark through the incorrect entry with an "X". Code the correct entry clearly above the incorrect entry. For "multiple choice" and "yes/no" type questions, circle the letter corresponding to the most appropriate response. If a letter is circled incorrectly, mark through it with an "X" and circle the correct response.

"We appreciate your cooperation in this important study. This will involve an interview today. At the end of today's interview, we will schedule an appointment for a physical examination and other tests to be conducted at a later date at the Jackson Heart Study clinic located in the Jackson Medical Mall. Do you have any questions before we begin?"

"Let me record the date and time and we will begin the interview."

1. Date of Interview: ..... __ __ / __ __ / __ __ __ __
   m m d d y y y y

2a. Time Interview Began: ..... __ __ : __ __
   h h m m

2b. AM A
PM P

3. Interviewer ID #: ..... ___

4. Record Participant's gender: ..... Male M
Female F

Please give me your complete date of birth. [VERIFY ELIGIBILITY. IF INELIGIBLE, SAY: "Thank you very much for your help but only people who are 35 through 84 years old are eligible for our study. The assistance of many people is important to ensure the success of the study. We would like to place you on our mailing list to receive notification of free community events and educational offerings if you would like. You can help assure the success of the study in a variety of ways such as spreading the word about the study, or volunteering a few hours of time. Additional details about how you might help, as well as a contact number, will be included in the mailing." [TERMINATE INTERVIEW. ENTER CODE "S" IN HOME INDUCTION RECORD OF CONTACT.]

5a. Birthdate: ..... __ __ / __ __ / __ __ __ __
   m m d d y y y y

5b. Is respondent eligible? ..... Yes Y → Go to Item 6

No N

5c. May I send you this information on educational offerings and volunteering in the Jackson Heart Study? ..... Yes Y

No N

IF "YES" CONCLUDE WITH: "Thank you for your time today. We look forward to having you join the Jackson Heart Study family as a supporter. You may call the Jackson Heart Study number on the brochure to discuss how you would like to help."

IF "NO" CONCLUDE WITH: "Thank you for your time today. We will send you information on free Jackson Heart Study community events. Should you decide to volunteer at a later time, feel free to call the Jackson Heart Study number on the brochure for additional information."

"Since the Jackson Heart Study is a long-term study which will include a brief telephone interview with you each year and may include a second clinic examination three years from now, I would like to ask you about your future plans."

6. Do you have definite plans to move outside of Hinds, Madison, or Rankin county in the next year? ..... Yes Y → Read script and terminate interview

No N

IF "YES" SAY: "Since the Jackson Heart Study is a long term study, and because you will be unable to participate in the follow-up due to your moving plans, we will not be able to include you in the study. Thank you for your help. If your plans change and you remain in the Jackson area, will you call the Jackson Heart Study staff to discuss your participation in the study?" [GIVE RESPONDENT JACKSON HEART STUDY BROCHURE WITH TELEPHONE NUMBER CIRCLED, WITH JACKSON HEART STUDY INTERVIEWER BUSINESS CARD ATTACHED, AND TERMINATE INTERVIEW.]

[ENTER CODE "N" IN HOUSEHOLD INDUCTION RECORD OF CONTACT (IRC)]

7. Are you currently or have you ever been a participant of the ARIC study? ..... Yes Y

No N

Don't know D

8. Are you currently or have you ever been a participant of the GENOA study? ..... Yes Y

No N

Don't know D

9a. Are you currently or have you ever been a participant in any other heart or blood pressure study? ..... Yes Y

Go to Item 10a — No N

Which studies?

9b. ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

9c. ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

9d. ☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

"Since heart disease often runs in families, the Jackson Heart Study includes studying families, as well as individuals. We would like to include other members of your family in this study if they are eligible. We are looking for families with 11 or more blood relatives living in Hinds, Madison, or Rankin counties. I have a few questions about relatives you might have living nearby."

10a. Do you have any grandparents living in Hinds, Madison, or Rankin County now? ..... Yes Y

Go to Item 11a — No N

10b. How many? ..... ☐☐
(# Live in tri-county)

11a. Is your father alive? ..... Yes Y

Go to Item 12a — No N

11b. Does he live in Hinds, Madison, or Rankin counties? ..... Yes Y

No N

12.a Is your mother alive? ..... Yes Y

Go to Item 13 — No N

12b. Does she live in Hinds, Madison, or Rankin counties? ..... Yes Y

No N

	Aunts (a)	Uncles (b)	# Live in tri-county (c)	# ≥ 21 years (d)
13. How many aunts and uncles do you have living who are related to you by blood?				

	Brothers (a)	Sisters (b)	# Live in tri-county (c)	# ≥ 21 years (d)
14. How many living, biological brothers and sisters do you have, that is with the same mother and father?				

	Half Brothers (a)	Half Sisters (b)	# Live in tri-county (c)	# ≥ 21 years (d)
15. How many living, half brothers and sisters do you have, that is with the same mother OR the same father?				

	Sons (a)	Daughters (b)	# Live in tri-county (c)	# ≥ 21 years (d)
16. How many living, biological sons and daughters do you have?				

17a. [ASK ONLY IF ITEM 16d IS "01" OR MORE. IF ITEM 16d IS "00," GO TO ITEM 18.] Does the (mother/father) of your children aged 21 years or older live in Hinds, Madison, or Rankin County? ..... Yes Y

Go to Item 18 — No N

17b. Does (she/he) live at this address? ..... Yes Y
No N

17c. Has (she/he) ever been a participant in the ARIC study or Jackson Heart Study? ..... Yes Y
No N

17d. Does most of (her/his) family live in Hinds, Madison, or Rankin county? ..... Yes Y
No N

	Nieces (a)	Nephews (b)	# Live in tri-county (c)	# ≥ 21 years (d)
18. How many nieces and nephews related by blood are alive?				

	Number (a)	# Live in tri-county (b)	# ≥ 21 years (c)
19. How many grandchildren do you have living, who are related to you by blood?			

20a. If your family is selected for the study, do we have your permission to contact your family members? ..... Yes Y
Go to Item 21 — No N

20b. Are there any family members that we should not contact? ..... Yes Y
Go to Item 21 — No N

20c. How many family members shouldn't be contacted? .....

"Now, in an effort to make sure that we do everything WITHIN OUR MEANS to make your participation in the Jackson Heart Study possible, I would like to ask you a couple of questions about your decision to take part (or not) in the study. I would also like to know about any expectations that you have as a participant so that we can attempt to better meet your needs."

21. Before your household received the letter from the Jackson Heart Study, had you heard about the study? ..... Yes Y

Go to Item 25 — No N

22. How did you hear about the Jackson Heart Study? Please tell me all the ways that apply. [READ ALL CATEGORIES TO PARTICIPANT]

		Yes	No
a.	A friend or relative told you about it	Y	N
b.	A doctor or nurse told you about it	Y	N
c.	Saw a billboard	Y	N
d.	Saw a poster or flyer	Y	N
e.	Received a brochure	Y	N
f.	Heard a presentation	Y	N
g.	Heard about it at church	Y	N
h.	Heard about it at work	Y	N
i.	From an information booth at Metro Mall	Y	N
j.	From a health fair or other community event	Y	N
k.	Someone came to your door or called to tell you about it	Y	N
l.	Heard about it on radio or TV	Y	N
m.	Read about it in the newspaper	Y	N
n.	Other (Specify)	Y	N — Go to Item
o.			

If zero or one "YES" response to Item 22, Go to Item 25

23. Of all the ways that you heard about the study, which would you say was the MOST important in your decision to (take part/not to take part)? [RECORD LETTER FROM ITEM 22 FOR MOST IMPORTANT] ..... ☐

24. Which was the LEAST important? [RECORD LETTER FROM ITEM 22 FOR LEAST IMPORTANT] ..... ☐

25. There are many reasons that people decide to (take part/not to take part) in studies such as the Jackson Heart Study. Using the response card, please tell me for each of these if it was very important, moderately important, or not important in your decision about taking part. [READ EACH RESPONSE TO PARTICIPANT] [RC #1]

		Very Important	Moderately Important	Not Important
a.	Getting free medical tests and examinations.....	V	M	N
b.	Getting information about your health.....	V	M	N
c.	Curiosity about medical research.....	V	M	N
d.	Thinking it can't hurt to give it a try.....	V	M	N
e.	Concerns about being a "guinea pig".....	V	M	N
f.	Finding out things you don't want to know.....	V	M	N
g.	Having a regular doctor.....	V	M	N
h.	Taking part will improve your health.....	V	M	N
i.	Taking part will help people in general.....	V	M	N
j.	Not having enough time.....	V	M	N
k.	Seeing nothing in it for you.....	V	M	N
l.	Taking part will help Black people.....	V	M	N
m.	Taking part will help your family in particular.....	V	M	N
n.	Taking part will help the scientists more than your people.....	V	M	N
o.	Are there any other reasons involved with your decision to (take part/not take part) in studies such as the Jackson Heart Study?		Yes	Y
	Go to Item 26		No	N

p. Please specify the reason:

q. Is this reason very important, moderately important, or not important in your decision about taking part? [RC #1]

Very important	V
Moderately important	M
Not important	N

26. What expectations do you have as a participant in the Jackson Heart Study?

[INTERVIEWER ONLY: WERE EXPECTATIONS EXPRESSED?]

Yes Y

No N

27. What concerns do you have about the study or coming to the clinic examination?

[INTERVIEWER ONLY: WERE CONCERNS EXPRESSED?]

Yes Y

No N

28. Some people have indicated that it would help to talk with others who have already taken part in research like the Jackson Heart Study. Would you like to have a volunteer who has taken part in another research study similar to the Jackson Heart Study call you to talk about what it is really like to be in a study?..... Yes Y

Go to Item 30 — No N

29. Is there a particular day or time that would be best for you?

	Yes	No
29a. Weekday morning?	Y	N
29b. Weekday afternoon?	Y	N
29c. Weekday evening?	Y	N
29d. Week-end morning?	Y	N
29e. Week-end afternoon?	Y	N
29f. Week-end evening?	Y	N

29g. Specific day..... Sunday S

Monday M

Tuesday T

Wednesday W

Thursday H

Friday F

Saturday A

Not indicated N

30. Do you have any recommendations for us on how to better recruit people like you to take part in the Jackson Heart Study?

_____

_____

_____

[INTERVIEWER ONLY:
WERE RECOMMENDATIONS EXPRESSED?]

Yes Y

No N

31. [COMPUTED FIELD. FAMILY STUDY ELIGIBILITY SCORE.] ..... ☐☐☐☐

Appendix B: JHS Parental SES Form

First Year Questionnaire

FORM CODE: AF1
VERSION A 11-8-2001

ID NUMBER: | | | | | | | | | CONTACT YEAR: | |

LAST NAME: | | | | | | | | | | | | | INITIALS: | |

INSTRUCTIONS: This form should be completed during the first year annual follow-up call. ID Number, Contact Year, and Name must be entered above. Whenever numerical responses are required, enter the number so that the last digit appears in the rightmost box. Enter leading zeroes where necessary to fill all boxes. If a number is entered incorrectly, mark through the incorrect entry with an "X". Code the correct entry clearly above the incorrect entry. For "multiple choice" and "yes/no" type questions, circle the letter corresponding to the most appropriate response. If a letter is circled incorrectly, mark through it with an "X" and circle the correct response.

"Now I am going to read a list of negative or stressful events that may happen in one's life. Across America, these events are among the unfortunate things that may happen to people no matter what their circumstances in life. Studies show that these negative or stressful events may have an important effect on one's health. After each one, please tell me if it has happened to you in the last 12 months."

		Yes	No
1.	First, have you had a serious illness or injury that started or got worse in the last year?	Y	N
2.	Have you been the victim of a serious physical attack, mugging, sexual assault or other assault?	Y	N
3.	Have you been robbed or was your home burglarized?	Y	N
4.	Have you lost a loved one due to violence?	Y	N
5.	Has your house been shot at, or has there been gunfire in your neighborhood?	Y	N
6.	Has anyone close to you died?	Y	N
7.	Has a family member or close friend had a major illness or injury?	Y	N

In the last 12 months . . .

		Yes	No
8.	Have you moved to a worse residence or neighborhood?	Y	N
9.	Have you or anyone in your household lost a job?	Y	N
10.	Have you retired from a job when you did not want to?	Y	N
11.	Have you had a divorce or separation from your (husband/wife)?	Y	N

[FOR JHS/ARIC ONLY, SAY:] "Now I have a series of questions that are similar to ones you have just answered about your childhood experiences. Those earlier questions were for your ARIC annual follow up, while these are for JHS. Where questions are nearly identical, I will do my best to first make sure your earlier response applies without asking you the full question. Thank you for your patience."

[OR:]

[FOR JHS ONLY, SAY:] "Some studies suggest that the experiences we have in early life may be related to the occurrence of illness throughout our lives. The following questions are designed to assess some of your early life experiences. We realize these things happened long ago. Please try to remember and answer as best you can."

12. Were you raised up to age 16 by anyone other than your natural parents? ..... Yes Y

No N → Go to Item 14a

13a. Was that because one of your parents died, because they divorced or separated, or from some other reason? .....

Parents died	A
Parents divorced or separated	B
Other reason	C
Don't Know	D
Refused	R

→ Go to Item 14a

13b. Specify:

[IF YES TO ITEM 12 SAY:] "The following questions refer to the persons whom you consider to be the most important in raising you up to age 16." [THIS CAN BE PARENTS OR ANY OTHER 2 PERSONS/CARETAKERS WHOM RESPONDENT THINKS WERE MOST IMPORTANT].

14a. Did your father (or other important male caretaker) ever work for pay while you were growing up? ..... Yes — Y

No — N — Go to Item 15a

There was no father/ male caretaker in household — T — Go to Item 16a

Does not know — D — Go to Item 15a

14b. When you were growing up, what was your father's (or other important male caretaker's) main job (the most important one)? [PROBE FOR WHAT FATHER DID, NOT WHERE HE WORKED].

14c. What were his most important activities or duties? For example selling cars, hearing legal cases, keeping books or office work, teaching school, etc.

14d. What kind of business or industry was that? [IF UNSURE, ASK:] "What did they make or do where he worked?" [PROBE FOR NAME OF BUSINESS OR INDUSTRY].

15a. What is the highest degree or years of school your father (or important male caretaker) completed, including trade or vocational school or college?

[RECORD NUMBER OF YEARS FOR GRADES 1-12:] ..... ☐☐

Some vocational or trade school, but no certificates	14
Vocational or trade certificate	15
Some college, but no degree	16
Associate degree, (junior college) (AA or AS)	17
Bachelor's degree (BA, BS, AB)	18
Graduate or professional schools (MA, MS, Master's Doctorate, MD, JD, DDS, DVM, etc.)	19

15b. [IF LESS THAN 12, ASK:] Did he complete a GED? ..... Yes Y

No N

16a. Did your mother (or other important female caretaker) ever work for pay while you were growing up? ..... Yes Y

No N — Go to Item 17a

There was no mother/ female caretaker in household T — Go to Item 18

Does not know D — Go to Item 17a

16b. What was her main occupation or job while you were growing up? [PROBE FOR WHAT MOTHER DID, NOT WHERE SHE WORKED].

16c. What were her most important activities or duties? For example selling cars, hearing legal cases, keeping books or office work, teaching school, etc.

16d. What kind of business or industry was that? [IF UNSURE, ASK:] "What did they make or do where she worked?" [PROBE FOR NAME OF BUSINESS OR INDUSTRY].

17a. What is the highest degree or years of school your mother (or important female caretaker) completed, including trade or vocational school or college?

[RECORD NUMBER OF YEARS FOR GRADES 1-12:] ..... | | |

Some vocational or trade school, but no certificates	14
Vocational or trade certificate	15
Some college, but no degree	16
Associate degree, (junior college) (AA or AS)	17
Bachelor's degree (BA, BS, AB)	18
Graduate or professional schools (MA, MS, Master's Doctorate, MD, JD, DDS, DVM, etc.)	19

17b. [IF LESS THAN 12, ASK:] Did she complete a GED? ..... Yes Y

No N

These next questions are about the place you lived when you were growing up until age 10. I realize this was a long time ago."

18. When you were growing up, did your parents (persons who raised you) own or were buying their home, pay rent, or had some other living arrangement, such as living with relatives, etc,? .....

Own or buying	B
Pay rent	R
Some other living arrangement	O
Unsure	U

19. Thinking about the place you lived until you were age 10, did it:

	Yes	No
a. have indoor plumbing?	Y	N
b. have electricity?	Y	N
c. and how many rooms did it have?		

20. When you were growing up, that is up until you were 10 years old or so, did your family own or have:

	Yes	No
a. a refrigerator?	Y	N
b. a car?	Y	N
c. a telephone?	Y	N
d. a television?	Y	N
e. air conditioning?	Y	N

21. That is all the questions I have. Do you have any other comments or questions?

ADMINISTRATIVE INFORMATION

22. Date of data collection: ____/____/____
m m d d y y y y

23. Method of data collection: Computer C
Paper form P

24. Code number of person completing this form: ____

Appendix C: JHS Personal Data/SES Form

Personal Data – Socioeconomic Status

FORM CODE: PDS
VERSION A 09/20/2000

ID NUMBER: | | | | | | | | CONTACT YEAR: 0 1

LAST NAME: | | | | | | | | | | | | INITIALS: | |

"Now I would like to ask you a few questions about yourself. In studies like this we often compare the ideas of men and women, young and old persons, and people of different economic backgrounds. The following questions are designed to assess some of your current and early life experiences. We realize that many of these refer to events that happened a long time ago. Please try to remember and answer as best you can. We will start our questions by gathering information about your current occupation, education and so forth. These questions are very important to this study. Can you agree to give us this information?"

Where were you born?

1a. City or Town: …. | | | | | | | | | | | | | | | | | | | |

1b. County: .. | | | | | | | | | | | | | | | | | | | |

1c. State (or Country if not US)…….. | |

2a. Think of this ladder with ten steps as representing where people stand in their communities. People define community in different ways. Please define it in whatever way is meaningful to you. At step 10 are people who have the highest standing in their community. At step 1 are people who have the lowest standing in their community. Tell me a number that represents where you think you stand at this time in your life, relative to other people in your community.

[SHOW RC #1]

Specify step on ladder: …….. | |

2b. People think of their communities in different ways. When you answered the last question, what did you think of as your community?

| | | | | | | | | | | | | | | | | | | |

| | | | | | | | | | | | | | | | | | | |

3. We would like to know about the kind of work you do. Looking at this card, [SHOW RC #2] please tell me the letter which best describes your current job?

Working now, full-time	A → Go to Item 5
Working now, part-time	B → Go to Item 5
Employed, but temporarily laid off	C
Sick or on leave for health reasons	D
Unemployed, looking for work	E
Unemployed, not looking for work	F
Homemaker, not working outside the home	G
Retired from my usual job and not working	H
Retired from my usual job but working for pay	I

4. When was your last regular job for pay? [RECORD YEAR] ..... [ ][ ][ ][ ] → Go to Item 6a
y y y y

[ENTER '9999' IF NEVER HAD A REGULAR JOB FOR PAY]

5. Are you currently working in one or more than one job? ... One O
More than one M

6a. What (is/was) your occupation on your main job? (For example: registered nurse, personnel manager, auto mechanic, accountant, machine operator, etc.)

[IF MORE THAN ONE, ASK:] Which do you consider your main occupation or job?

[PROBE FOR WHAT RESPONDENT DOES, NOT WHERE RESPONDENT WORKS, OBTAIN JOB TITLE]

6b. (Are/were) you self-employed for this occupation? ..... Yes Y

No N

7. Tell me a little more about your main job. What are your most important activities or duties? For example patient care, directing hiring policies, repairing automobiles, reviewing financial records, operating machinery, etc.)

8. What kind of business or industry (is/was) that? (For example, hospital newspaper publishing, auto repair shop, bank, etc.)

[IF UNSURE, ASK:] What do they make or do where you (work/worked)?

[PROBE FOR NAME OF BUSINESS OR INDUSTRY]

9a. How long (have you had/did you have) your main job? (Number of years).....

9b. (Is/was) your job one that (provides/provided) regular steady work throughout the year, (is/was) it seasonal, (are/were) there frequent layoffs, or what? .....

Regular, steady work	R
Seasonal	S
Frequent layoffs	F
Don't know	D
Other	O

→ Go to Item 10a

9c. Specify:

10a. Thinking over your entire work life, have you ever been unemployed (that is looking for a job but unable to find one) for 6 months or longer? ..... Yes Y

Go to Item 11 — No N

10b. How many times have you been unemployed for more than 6 months? [SPECIFY NUMBER OF TIMES] ..... ☐☐

11. How satisfied are you with your job? Are you satisfied, dissatisfied, or neither? ..... Satisfied S

Dissatisfied D

Neither N

12. Now I would like you to think about the kind of jobs you had when you were younger. Please tell me what your first full-time occupation or job was.

[PROBE FOR WHAT RESPONDENT DID, NOT WHERE RESPONDENT WORKED. OBTAIN JOB TITLE.]

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

13. Pretend that the steps on this ladder stand for 10 possible steps in your life. The tenth step stands for the best possible way of life for you and the first step stands for the worst possible way of life for you. Keeping in mind that step 10 represents your best way of life and step 1 represents your worst way of life, will you tell me the step number that best describes where you are now?

[SHOW RC #3]

Specify step on ladder: ..... ☐☐

14. Would you please tell me the step number that best describes where you were ten years ago?

Specify step on ladder: ..... ☐☐

15. Will you please tell me the step number that best describes where you would like to be next year?

Specify step on ladder: ..... ☐☐

16. Will you please tell me the step number that best describes where you expect to be next year?

Specify step on ladder:..... ☐☐

17. How disappointed would you be if you found out that you could never reach (STEP # IN Q#15)? Would you be very disappointed, fairly disappointed, slightly disappointed, or not at all disappointed? .....

Very disappointed	V
Fairly disappointed	F
Slightly disappointed	S
Not at all disappointed	N

18a. What is the highest degree or years of school you have completed, including trade or vocational school or college?

[IF CURRENTLY ENROLLED, MARK HIGHEST GRADE COMPLETED OR HIGHEST DEGREE RECEIVED.]

[RECORD NUMBER OF YEARS FOR GRADES 1-12:] ..... ☐☐

Some vocational or trade school, but no certificates	14
Vocational or trade certificate	15
Some college, but no degree	16
Associate degree, (junior college) (AA or AS)	17
Bachelor's degree (BA, BS, AB)	18
Graduate or professional schools (MA, MS, Master's Doctorate, MD, JD, DDS, DVM, etc.)	19

18b. [IF LESS THAN 12, ASK:] Did you complete a GED? .....

Yes	Y
No	N

19a. Have you completed any other training or education outside of formal school programs? ..... Yes Y

Go to Item 20 — No N

19b. Specify:

20. [DO NOT ASK; REFER TO SOCIAL SUPPORT FORM]

[HAS RESPONDENT EVER HAD SPOUSE OR CURRENTLY HAS SPOUSE/PARTNER?] ..... Yes Y

Go to Item 24 — No N

21. [SELECT APPROPRIATE WORDING]

Is your (husband/wife/partner) presently working for pay?

OR

Did your (husband/wife/partner) ever work for pay? ..... Yes, currently Y

Yes, in the past P

Go to Item 23a — No N

22a. What (is/was) (his/her) occupation or main job? (For example: registered nurse, personnel manager, auto mechanic, accountant, machine operator, etc.)

[PROBE FOR WHAT SPOUSE/PARTNER DOES, NOT WHERE SPOUSE/PARTNER WORKS, OBTAIN JOB TITLE]

22b. What are/were (his/her) most important activities or duties? (For example patient care, directing hiring policies, repairing automobiles, reviewing financial records, operating machinery, etc.)

22c. What kind of business or industry (is/was) that? (For example, hospital newspaper publishing, auto repair shop, bank, etc.)

[IF UNSURE, ASK:] What do they make or do where your (spouse/partner) (works/worked)?

[PROBE FOR NAME OF BUSINESS OR INDUSTRY]

23a. What is the highest degree or years of school your (husband/wife/partner) ever completed, including trade or vocational school or college?

[IF CURRENTLY ENROLLED,
MARK HIGHEST GRADE COMPLETED
OR HIGHEST DEGREE RECEIVED]

RECORD NUMBER OF YEARS FOR GRADES 1–12	
Some vocational or trade school, but no certificates	14
Vocational or trade certificate	15
Some college, but no degree	16
Associate degree, (junior college) (AA or AS)	17
Bachelor's degree (BA, BS, AB)	18
Graduate or professional school (MA, MS, Master's Doctorate, MD, JD, DDS, DVM, etc)	19

23b. [IF LESS THAN 12, ASK:] Did (he/she) complete a GED? .....Yes Y

No N

"The following questions have to do with family finances. We know from other research that financial strain is common and very important to consider in understanding people's health. These questions will help give a picture of the various financial situations experienced by persons in the Jackson Heart Study. I want to remind you that key information you provide is strictly confidential and will never be identified with you as an individual."

24.	Are you or your family renting, buying (paying a mortgage), or do you own (paid off) the house or apartment where you live now?	Pays rent	P
		Buying (paying a mortgage)	B
		Owns	O
		Neither owns nor pays rent	N
		Don't know	D
25.	Do you own or are buying/leasing one or more cars?	Yes, one	O
		Yes, more than one	M
		No	N
26.	Suppose you needed money quickly and you cashed in all of your (and your spouse's/ partner's) checking and savings accounts, cars, jewelry, or other possessions and any stocks, bonds, or real estate (other than your principal home). If you added up what you get, about how much would it amount to? Just give me your best estimate from the list. [HAND RC #4]	$0 – 499	A
		$500 – 999	B
		$1,000 – 4,999	C
		$5,000 – 9,999	D
		$10,000 – 19,999	E
		$20,000 – 49,999	F
		$50,000 – 99,999	G
		$100,000 – 199,999	H
		$200,000 or more	I
		Don't know	J
		Refused	K

27. In the past year, did you or anyone living in your household receive any income from the following sources?

	YES	NO/ DON'T KNOW	REFUSED
27a. Investments?	Y	N	R
27b. Social Security?	Y	N	R
27c. Worker's Compensation?	Y	N	R
27d. Unemployment Compensation?	Y	N	R
27e. ADC or AFDC? (Aid to Dependent Children)	Y	N	R
27f. Food Stamps?	Y	N	R
27g. Other Welfare Programs?	Y	N	R
27h. Supplemental Security Income (SSI)?	Y	N	R
27i. Gambling?	Y	N	R

28a. Now, thinking of all these sources as well as money from jobs, income from a business, or farm, rent from property, social security or retirement benefits, help from friends or family, or any other income not reported, what was your total combined family income before taxes in (YEAR)? Using this card [RC #5] tell me the letter that most closely matches your total combined family income.

Less than $5,000	A
$5,000 – 7,999	B
$8,000 – 11,999	C
$12,000 – 15,999	D
$16,000 – 19,999	E
$20,000 – 24,999	F → Go to Item 29
$25,000 – 34,999	G
$35,000 – 49,999	H
$50,000 –74,999	I
$75,000 – 99,999	J
$100,000 or more	K
Don't Know	L
Refused	M

28b. You may not be able to give me an exact range for your family income, but can you tell me if your family received $35,000 or more? ..... Yes Y

Go to Item 28f — No N

Go to Item 29 — Don't know D

Refused R

28c. Was it $50,000 or above? ..... Yes Y

No N

Go to Item 29 — Don't know D

Refused R

28d. Was it $75,000 or above? ..... Yes Y

No N

Go to Item 29 — Don't know D

Refused R

28e. Was it $100,000 or above? ..... Yes Y

No N

Go to Item 29 — Don't know D

Refused R

[IF THE FAMILY DID NOT RECEIVE $35,000 OR MORE IN (YEAR)]

28f. Was it $10,000 or above? ..... Yes Y

No N

Go to Item 29 — Don't know D

Refused R

28g. Was it $25,000 or above? ..... Yes Y

No N

Don't know D

Refused R

29. How much of that income do you contribute? Using this card tell me the letter that most closely matches your total income before taxes in (year).
[HAND RC #5] .....

Less than $5,000	A
$5,000 – 7,999	B
$8,000 – 11,999	C
$12,000 – 15,999	D
$16,000 – 19,999	E
$20,000 – 24,999	F
$25,000 – 34,999	G
$35,000 – 49,999	H
$50,000 – 74,999	I
$75,000 to 99,999	J
$100,000 or more	K
Don't know	L
Refused	M

30. On average, how many people, including yourself does your total family income support?

Number of persons: ..... ☐☐

31a. Including yourself, how many people lived in your house during the past 12 months?

Number of persons: ..... ☐☐

31b. Of these, how many are under the age of 18?

Number of persons: ..... ☐☐

32. [SHOW RC #6] Now, think of a ladder with 10 steps representing where people stand in the United States. At step 10 are the people who are the best off—those who have the most money, the most education and the most respected jobs. At step 1 are the people who are the worst off--who have the least money, least education, and the worst jobs or no job. The higher up you are on this ladder, the closer you are to the people at the very top, and the lower you are, the closer you are to the people at the very bottom. Where would you place yourself on this ladder? Tell me a number that represents where you think you stand at this point in time relative to other people in the United States.

Specify number of step: ..... | | |

ADMINISTRATIVE INFORMATION

33. Date of data collection:..... | | | / | | | / | | | | |
m m d d y y y y

34. Code number of person completing this form: ..... | | | |

Appendix D: JHS Sitting Blood Pressure Form

Sitting Blood Pressure Form

FORM CODE: SBP
VERSION A 04/03/2000

ID NUMBER: | | | | | | | | CONTACT YEAR: 0 1

LAST NAME: | | | | | | | | | | | | INITIALS: | |

INSTRUCTIONS: This form should be completed during the participant's visit. ID Number, Contact Year, and Name must be entered above. Whenever numerical responses are required, enter the number so that the last digit appears in the rightmost box. Enter leading zeroes where necessary to fill all boxes. If a number is entered incorrectly, mark through the incorrect entry with an "X". Code the correct entry clearly above the incorrect entry. For "multiple choice" and "yes/no" type questions, circle the letter corresponding to the most appropriate response. If a letter is circled incorrectly, mark through it with an "X" and circle the correct response.

A. TEMPERATURE

1. Room Temperature (degrees centigrade):..... | |

B. TOBACCO AND CAFFEINE USE

2. Have you smoked or used chewing tobacco, nicotine gum or snuff today or do you wear a nicotine patch?.....Yes Y

   Go to Item 4 — No N

3. How long ago did you last smoke or last use chewing tobacco or snuff?

   3a. | | hours 3b. | | minutes

4. Have you had any caffeinated beverages, such as coffee, tea, or colas, or any chocolate today?.....Yes Y

   Go to Item 6 — No N

5. How long ago did you last have any caffeinated beverage, or chocolate?

   5a. | | hours 5b. | | minutes

C. PRELIMINARY MEASUREMENTS

6. Right Arm Circumference (cm):..... ☐☐

7. Cuff Size:
{arm circumference in brackets}..... Small adult {<24 cm} S
Regular Arm {24-32 cm} R
Large Arm {33-41 cm} L
Thigh {>41cm} T

8. Heart Rate (30 seconds):..... ☐☐

9a. Time of Day: ☐☐:☐☐ (h h m m) 9b. AM A
PM P

10. Pulse Obliteration Pressure:..... ☐☐☐

11. Maximum Zero:..... ☐☐

+ 3 0

12. Peak Inflation Level
{Computation--Item #10
+ Item #11 + 30}:..... ☐☐☐

D. FIRST BLOOD PRESSURE MEASUREMENT

13. Systolic:..... ☐☐☐

14. Diastolic:..... ☐☐☐

15. Zero Reading:..... ☐☐☐

E. SECOND BLOOD PRESSURE MEASUREMENT

16. Systolic:..... | | | |

17. Diastolic:..... | | | |

18. Zero Reading:..... | | | |

F. COMPUTED NET AVERAGE OF FIRST AND SECOND BLOOD PRESSURE MEASUREMENTS (See Worksheet)

19. Systolic:..... | | | |

20. Diastolic:..... | | | |

G. ADMINISTRATIVE INFORMATION

21. Date of data collection:..... | | | / | | | / | | | | |
m m d d y y y y

22. Method of Data Collection: .....Computer C
Paper Form P

23. Code number of person completing this form:..... | | | |

ID: _____ CONTACT YEAR: _____ FORM CODE: SBP VERSION: A 03/15/2000

WORKSHEET FOR COMPUTING AVERAGE OF 1ST AND 2ND READINGS (ITEMS 19 AND 20)

	SYSTOLIC		DIASTOLIC	
First Measurement	☐☐☐	(#13)	☐☐☐	(#14)
1st Zero Reading	– ☐☐☐	(#15)	– ☐☐☐	(#15)
First Corrected	☐☐☐		☐☐☐	
Second Measurement	☐☐☐	(#16)	☐☐☐	(#17)
2nd Zero Reading	☐☐☐	(#18)	☐☐☐	(#18)
Second Corrected	☐☐☐		☐☐☐	
Average Corrected	☐☐☐	(#19)	☐☐☐	(#20)

Appendix E: JHS Medication Survey Form

Medication Survey Form

FORM CODE: MSR
VERSION A 09/25/2000

ID NUMBER: | | | | | | | |

CONTACT YEAR: 0 1

LAST NAME: | | | | | | | | | | | | |

INITIALS: | | |

INSTRUCTIONS: This form is completed during the participant's clinic visit in several stages by appropriately trained persons at the workstations identified for this purpose. If the paper form is used for data collection, data are keyed into the data entry system as soon as possible following its completion. ID Number, Contact Year, and Name are entered above. Whenever numerical responses are required, enter the number so that the last digit appears in the rightmost box. Enter leading zeros where necessary to fill all boxes. If a number is entered incorrectly on a paper form, mark through the correct entry with an "X". Code the correct entry clearly above the incorrect entry. For "multiple choice" and "yes/no" type questions, circle the letter corresponding to the most appropriate response. If a letter is circled incorrectly, mark through it with an "X" and circle the correct response.

At the reception station, verify that the medication bag is clearly identified with the participant's name and ID number. Do not open the medication bag or transcribe medications until the participant has signed the informed consent. The transcription section of Section B is completed while the participant proceeds with the visit. Medications are coded by trained field center personnel after the transcription and interview portions have been completed. Code numbers of the interviewer, transcriber and coder are recorded in the appropriate locations.

A. RECEPTION

1. Did you bring all the medications you used in the past two weeks, or their containers? This includes all prescription medications, all over-the-counter medications, all vitamins, minerals, herbs and dietary supplements? .....

Yes, all	Y — Go to Item 4: Begin transcription while participant proceeds with clinic visit.
Some of them	S — Go to Item 3a and transcribe those medications which were brought at this time.
No	N — Go to Item 3a and transcribe those medications which were brought at this time.

2. Is this because you forgot, because you have not taken any medications at all in the last two weeks, or because you could not bring your medications? .....

Took no medications	T — Go to Item 31a
Forgot or was unable to bring medications	F

"That's alright. Since the information on medications is so important, we would still like to ask you about it during the interview."

3a. Could we follow up on this after the visit so that we can get the information from the (other) medication labels? [EXPLAIN FOLLOW-UP OPTIONS] .... Yes Y

No or not applicable N

ATTEMPT TO CONVERT REFUSALS; INDICATE ON ITINERARY FORM

3b. Describe method of follow-up to be used:

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

B. MEDICATION TRANSCRIPTION

Transcribe the <u>NAME</u> followed by the <u>CONCENTRATION and INSTRUCTIONS FOR ADMINISTRATION</u> of each medication in the spaces below. List all ingredients for nutritional supplements OR make a copy of label and affix to form (continue on the second line if needed).

	A MEDICATION NAME	B CONCENTRATION	C INSTRUCTIONS FOR ADMINISTRATION	D "DID YOU TAKE THIS MEDICATION IN PAST 24 HOURS?" YES – Y, NO – N DON'T KNOW – D	E CODE NUMBER
4.	______	______	______		
	______	______	______	Y N D	______
5.	______	______	______		
	______	______	______	Y N D	______

	A MEDICATION NAME	B CONCENTRATION	C INSTRUCTIONS FOR ADMINISTRATION	D "DID YOU TAKE THIS MEDICATION IN PAST 24 HOURS?" YES – Y, NO – N DON'T KNOW – D	E CODE NUMBER
6.				Y N D	
7.				Y N D	
8.				Y N D	
9.				Y N D	
10.				Y N D	
11.				Y N D	
12.				Y N D	

	A MEDICATION NAME	B CONCENTRATION	C INSTRUCTIONS FOR ADMINISTRATION	D "DID YOU TAKE THIS MEDICATION IN PAST 24 HOURS?" YES – Y, NO – N DON'T KNOW – D	E CODE NUMBER
13.				Y N D	
14.				Y N D	
15.				Y N D	
16.				Y N D	
17.				Y N D	
18.				Y N D	
19.				Y N D	

	A MEDICATION NAME	B CONCENTRATION	C INSTRUCTIONS FOR ADMINISTRATION	D "DID YOU TAKE THIS MEDICATION IN PAST 24 HOURS?" YES – Y, NO – N DON'T KNOW – D	E CODE NUMBER
20.				Y N D	
21.				Y N D	
22.				Y N D	
23.				Y N D	
24.				Y N D	
25.				Y N D	
26.				Y N D	

27. Total number of medications in bag: ..... ☐☐

28. Number of medications unable to transcribe: ..... ☐☐

Code numbers of person transcribing and coding medications:

29a. Transcriber code number: ..... ☐☐☐

29b. Medication coder code number: ..... ☐☐☐

29c. Date of medication coding: ..... ☐☐/☐☐/☐☐☐☐
m m d d y y y y

C. INTERVIEW

"Now I would like to ask about a few specific medications."

Were any of the medications you took during the past two weeks for:
[IF YES, VERIFY THAT MEDICATION NAME IS ON MEDICATION RECORD.]

	Yes	No	Don't Know
30a. High blood pressure?	Y	N	D
30b. High blood cholesterol?	Y	N	D
30c. Angina or chest pain?	Y	N	D

27. Total number of medications in bag: ..... ☐☐

28. Number of medications unable to transcribe: ..... ☐☐

Code numbers of person transcribing and coding medications:

29a. Transcriber code number: ..... ☐☐☐

29b. Medication coder code number: ..... ☐☐☐

29c. Date of medication coding: ..... ☐☐/☐☐/☐☐☐☐ (m m / d d / y y y y)

C. INTERVIEW

"Now I would like to ask about a few specific medications."

Were any of the medications you took during the past two weeks for:
[IF YES, VERIFY THAT MEDICATION NAME IS ON MEDICATION RECORD.]

	Yes	No	Don't Know
30a. High blood pressure?	Y	N	D
30b. High blood cholesterol?	Y	N	D
30c. Angina or chest pain?	Y	N	D

	Yes	No	Don't Know
30d. Control of heart rhythm?	Y	N	D
30e. Heart failure?	Y	N	D
30f. Blood thinning?	Y	N	D
30g. Diabetes or high blood sugar?	Y	N	D
30h. Stroke?	Y	N	D
30i. Leg pain when walking?	Y	N	D

D. MEDICATION-TAKING BEHAVIORS

"There are many things that keep people from taking medicines exactly as prescribed. I am going to read a list of typical reasons people have for not taking prescribed medicines. For each reason I list, please tell me if you have not taken a prescribed medicine for this reason."

	Reason Indicated	Not a Reason	Don't Know
31a. You were in a hurry, too busy, or forgot	Y	N	D
31b. It was inconvenient, for example, the medication needed to be refrigerated, or had to be taken with food	Y	N	D
31c. You thought the medication wouldn't do you any good	Y	N	D

		Reason Indicated	Not a Reason	Don't Know	
31d.	The medication made you feel bad	Y	N	D	
31e.	If you took the medication, you wouldn't be able to carry out your normal activities—for example, driving	Y	N	D	
31f.	You thought you might become addicted or hooked on the medication	Y	N	D	
31g.	You don't like to take medicine	Y	N	D	
31h.	You were trying to do without it	Y	N	D	
31i.	You did not have money to purchase the medication (or its refills)	Y	N	D	
31j.	Did not have the medication available	Y	N	D	
31k.	Are there any other reasons why you haven't taken a prescribed medication?	Y	N	D	Go to Item 32

31l. If yes, specify reason:

E. ASPIRIN AND NSAID USE

32. During the past two weeks, did you take any aspirin, Alka-Seltzer, cold medicine or headache powder? ..... Yes Y

Go to Item 35a — No N

Don't know D

"Next I would like to ask you about your regular use of aspirin alone or an aspirin-containing medication, for example, aspirin+caffeine+codeine. By regular, I mean at least once a week for several months."

33. Are you NOW taking aspirin, or a medicine containing aspirin, on a regular basis? This does not include Tylenol nor Advil..... Yes Y

Go to Item 35a — No N

Don't know D

34a. What is the strength of aspirin in the pill? [CHECK THE PREPARATION, IF AVAILABLE; OTHERWISE SHOW RC #1] ..... Less than 300 mg (Baby) A

300 - 499 mg (Regular) B

500 mg or greater (Extra strength) C

Don't know D

34b. How many days a week, on average, are you taking this medication? ..... ☐ Days

34c. How many pills are you taking per week, on average? ..... ☐☐ Pills

34d. For what purpose are you taking this medication? ..... Participant mentioned to avoid heart attack or stroke H

Participant did NOT mention to avoid heart or attack or stroke O

34e. When did you start taking aspirin, or a medicine containing aspirin, on a regular basis? ..... m m / y y y y

35a. Except for aspirin or Tylenol, are you NOW taking other non-steroidal anti-inflammatory drugs or arthritis medicines on a regular basis? Examples include Ibuprofen, Advil, Nuprin, Motrin, Naprosyn, Feldene and Clinoril.....

- Yes — Y
- No — N — Go to Item 36a
- Don't know — D

35b. What is the brand name of the medicine? [CHECK THE PREPARATION, IF AVAILABLE] .....

- Ibuprofen or Advil — I — Go to Item 35d
- Other — O

35c. If "Other", specify:

35d. How many pills per week are you taking, on average? ..... Pills

35e. When did you start taking [INSERT NAME] on a regular basis?..... m m / y y y y

F. FOLK MEDICINE

"Other than medicines prescribed by your doctor or health professional, what other home remedies, teas, roots or herbs have you used in the last 2 weeks for medical reasons only: Have you used..."

36a. Vinegar? ..... Yes Y

Go to Item 37a — No N

36b. How many days during the past 2 weeks? ..... ☐☐ Days

36c. For what purpose?

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

37a. Epsom Salts? ..... Yes Y

Go to Item 38a — No N

37b. How many days during the past 2 weeks? ..... ☐☐ Days

37c. For what purpose?

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

38a. Lemon juice or lemon? .... Yes Y

[Go to Item 39a] — No N

38b. How many days during the past 2 weeks? .... [ ][ ] Days

38c. For what purpose?

[ ]

39a. Garlic? .... Yes Y

[Go to Item 40a] — No N

39b. How many days during the past 2 weeks? .... [ ][ ] Days

39c. For what purpose?

[ ]

40a. Teas? ..... Yes Y

Go to Item 41a — No N

40b. How many days during the past 2 weeks? ..... | | |

Days

40c. For what purpose?

40d. Specify type:

41a. Roots? ..... Yes Y

Go to Item 42a — No N

41b. How many days during the past 2 weeks? ..... | | |

Days

41c. For what purpose?

41d. Specify type:

42a. Have you taken any other home remedies, teas, roots or herbs in the last 2 weeks? ..... Yes Y

Go to Item 43a — No N

42b. How many days during the past 2 weeks? ..... ☐☐ Days

42c. For what purpose?

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

42d. Specify type:

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

43a. Have you ever used any other home remedies, teas, roots, herbs or other medicines? ..... Yes Y

Go to Item 44 — No N

43b. Was this for your heart or for other symptoms? ..... Heart H — Go to Item 43d

Other O

43c. For what other symptoms?

☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐☐

43d. About how often would you say you have used any of these remedies? Would you say daily, weekly, several times a month, monthly, several times a year, yearly, rarely, almost never, or never? [SHOW RC #2] .....

Daily	D
Weekly	W
Several times a month	S
Monthly	M
Several times a year	T
Yearly	Y
Rarely	R
Almost never	A
Never	N

G. ADMINISTRATIVE INFORMATION

44. Date of data collection: ..... | | | / | | | / | | | | |
m m d d y y y y

45. Method of data collection: ..... Computer C

Paper form P

46. Code number of person completing this form: ..... | | | |

Appendix F: JHS Discrimination Form

Discrimination Form

FORM CODE: DIS
VERSION A 10/24/2000

ID NUMBER: | | | | | | | | CONTACT YEAR: | 0 | 1 |

LAST NAME: | | | | | | | | | | | | | INITIALS: | | |

INSTRUCTIONS: This form should be completed during the participant's clinic visit. ID Number, Contact Year, and Name must be entered above. Whenever numerical responses are required, enter the number so that the last digit appears in the rightmost box. Enter leading zeroes where necessary to fill all boxes. If a number is entered incorrectly, mark through the incorrect entry with an "X". Code the correct entry clearly above the incorrect entry. For "multiple choice" and "yes/no" type questions, circle the letter corresponding to the most appropriate response. If a letter is circled incorrectly, mark through it with an "X" and circle the correct response.

"These next questions have to do with things that may have happened to you and the way you have been treated over your lifetime. We know from other research that experiences of unfair treatment are common and very important to consider in understanding people's health. These questions will give a picture of the various kinds of experiences of people in the Jackson Heart Study. There are no right or wrong answers; only your experiences. I want to remind you that any information you provide is strictly confidential and will never be identified with you as an individual. Let's start with experiences you may have had on a day-to-day basis."

1. Using the responses on this card, tell me how often each of the following things happen to you in your day-to-day life. Just tell me the letter beside the response that most closely matches your experience. **[HAND RC #1]** .....

Several times a day	A
Almost every day	B
At least once a week	C
A few times a month	D
A few times a year	E
Less than a few times a year	F
Never	G

How often on a day-to-day basis do you have the following experiences? [CIRCLE CODE]

1a.	You are treated with less courtesy than other people.....	A	B	C	D	E	F	G
1b.	You are treated with less respect than other people.....	A	B	C	D	E	F	G

A – Several times a day
B – Almost every day
C – At least once a week
D – A few times a month
E – A few times a year
F – Less than a few times a year
G – Never

1c.	You receive poorer service than others at restaurants.....	A	B	C	D	E	F	G
1d.	People act as if they think you are not smart.....	A	B	C	D	E	F	G
1e.	People act as if they are afraid of you.....	A	B	C	D	E	F	G
1f.	People act as if they think you are dishonest.....	A	B	C	D	E	F	G
1g.	People act as if they think you are not as good as they are.....	A	B	C	D	E	F	G
1h.	You are called names or insulted.....	A	B	C	D	E	F	G
1i.	You are threatened or harassed.....	A	B	C	D	E	F	G

If all responses in Item 1 are "NEVER," Code G, then go to Item 4a

2a. Thinking over these day-to-day experiences, what is the main reason for this treatment?

Your age	A
Your gender	B
Your race	C
Your height or weight	D
Some other reason for discrimination	E

Go to Item 3a

2b. Specify other reason:

3a. And when you receive lesser or unfair treatment in your day-to-day life, do you usually:

Speak up	A
Accept it	B
Ignore it	C
Try to change it	D
Keep it to yourself	E
Work harder to prove them wrong	F
Pray	G
Avoid it	H
Get violent	I
Forget it	J
Blame yourself	K
Other	L

Go to Item 4a

3b. Specify other:

"Now let's talk about things that may have happened over your lifetime because of such issues as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics."

4a. Have you ever felt unfairly treated at school or during training? (For example, you were discouraged by a teacher or advisor from seeking higher education, were denied a scholarship, etc.) ..... Yes Y

Go to Item — No N

Over your entire life, how many times has this happened?

4b. Specify number of times: ..... ☐☐☐ times

When was the last time?

4c. Specify years ago: ..... ☐☐☐ years

4d. Specify months ago: ..... ☐☐☐ months

5a. Have you ever felt unfairly treated in getting a job? (For example, you were not hired or you were told you could not apply.)..... Yes Y

Go to Item 6a — No N

Over your entire life, how many times has this happened?

5b. Specify number of times: ..... ☐☐☐ times

When was the last time?

5c. Specify years ago: ..... ☐☐☐ years

5d. Specify months ago: ..... ☐☐☐ months

6a. Have you ever felt unfairly treated at work?

(For example, you were not promoted, you were overworked or hassled, you were fired or you were unable to get health insurance.) ..... Yes Y

Go to Item 7a — No N

Never worked W

Over your entire life, how many times has this happened?

6b. Specify number of times: ..... ☐☐☐ times

When was the last time?

6c. Specify years ago: ..... ☐☐☐ years

6d. Specify months ago: ..... ☐☐☐ months

7a. Have you ever felt unfairly treated in getting housing or finding a place to live? (For example, you were prevented from renting or buying a home in the neighborhood you wanted or you were prevented from remaining in a neighborhood because neighbors made life so uncomfortable.) ..... Yes Y

Go to Item — No N

Over your entire life, how many times has this happened?

7b. Specify number of times: ..... ☐☐☐ times

When was the last time?

7c. Specify years ago: ..... ☐☐☐ years

7d. Specify months ago: ..... ☐☐☐ months

8a. Have you ever felt unfairly treated in getting resources or money? (For example, you were denied a bank loan,

a credit card or some other form of credit.) .....Yes Y

Go to Item — No N

Over your entire life, how many times has this happened?

8b. Specify number of times: ..... ☐☐☐ times

When was the last time?

8c. Specify years ago: ..... ☐☐☐ years

8d. Specify months ago: ..... ☐☐☐ months

9a. Have you ever felt unfairly treated in getting medical care? (For example, you were denied or provided inferior medical care, you were made to wait long periods of time before getting care or you could not get care from a medical specialist such as a heart doctor.) ..... Yes Y

Go to Item 10a — No N

Over your entire life, how many times has this happened?

9b. Specify number of times: ..... ☐☐☐ times

When was the last time?

9c. Specify years ago: ..... ☐☐☐ years

9d. Specify months ago: ..... ☐☐☐ months

10a. Have you ever felt unfairly treated on the street or in a public place? (For example, you were hassled by the

police, were the target of public ridicule, etc.) .... Yes Y

Go to Item — No N

Over your entire life, how many times has this happened?

10b. Specify number of times: .... [ ][ ][ ] times

When was the last time?

10c. Specify years ago: .... [ ][ ][ ] years

10d. Specify months ago: .... [ ][ ][ ] months

11a. Have you ever felt unfairly treated in getting services? (For example, you were denied or provided inferior service by a plumber, in a restaurant, the grocery store, or by some other service provider.) .... Yes Y

Go to Item 12a — No N

Over your entire life, how many times has this happened?

11b. Specify number of times: .... [ ][ ][ ] times

When was the last time?

11c. Specify years ago: .... [ ][ ][ ] years

11d. Specify months ago: .... [ ][ ][ ] months

12a. In addition to these areas we have talked about, have you been treated unfairly in any other ways? .... Yes Y

Go to Item 13a — No N

12b. Specify:

Over your entire life, how many times has this happened?

[ ][ ][ ]

12c. Specify number of times: .....
times

When was the last time?

12d. Specify years ago: .....
years

12e. Specify months ago: .....
months

If all responses in Items 4a-12a are "NO" or "NEVER WORKED" (Item 6a), go to Item 15

13a. Thinking about the most recent of these experiences over your lifetime, what was the main reason for the discrimination you experienced?

Your age	A
Your gender	B
Your race	C
Your height or weight	D
Some other reason for discrimination	E

Go to Item 14

13b. Specify other reason:

14. What did you do? ..... Did you do that a lot, some, or a little?

___ ____ ____

			IF YES		
14a. Speak up?	Yes	Y	→	14a1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

			IF YES		
14b. Accept it?	Yes	Y	→	14b1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

			IF YES		
14c. Ignore it?	Yes	Y	→	14c1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

			IF YES		
14d. Try to change it?	Yes	Y	→	14d1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

			IF YES		
14e. Keep to yourself?		Yes	→	Y 14e1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

			IF YES		
14f. Work harder to prove them wrong?	Yes	Y	→	14f1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C

Did you do that a lot, some, or a little?

			IF YES		
14g. Pray?	Yes	Y	→	14g1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C
14h. Avoid it?	Yes	Y	→	14h1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C
14i. Get violent?	Yes	Y	→	14i1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C
14j. Forget it?	Yes	Y	→	14j1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C
14k. Blame yourself?	Yes	Y	→	14k1. [CIRCLE VALUE GIVEN TO RESPONSE]	
	No	N		A lot	A
				Some	B
				A Little	C
14l. Other?	Yes	Y	→	14l1. [CIRCLE VALUE GIVEN TO RESPONSE]	
Go to Item 15	No	N		A lot	A
				Some	B
				A Little	C

14m. Specify other:

15. Thinking back over these types of experiences, compared with when you were younger, are the experiences more frequent, less frequent, or about the same? ..... More frequent A

Less frequent B

About the same C

16. When you have had experiences like these over your lifetime, would you say they have been very stressful, moderately stressful, or not stressful? ..... Very stressful A

Moderately stressful B

Not stressful C

17. Overall, how much has discrimination interfered with you having a full and productive life? Would you say a lot, some, a little, or not at all? ..... A lot A

Some B

A little C

Not at all D

18. Overall, how much harder has your life been because of discrimination? Would you say a lot, some, a little, or not at all? ..... A lot A

Some B

A little C

Not at all D

19. Because of the shade of your skin color, do you think white people treat you a lot better, somewhat better, no different, somewhat worse, or a lot worse than other Blacks? ..... A lot better A

Somewhat better B

No different C

Somewhat worse D

A lot worse E

20. Because of the shade of your skin color, do you think Black people treat you a lot better, somewhat better, no different, somewhat worse, or a lot worse than other Blacks? ..... A lot better A

Somewhat better B

No different C

Somewhat worse D

A lot worse E

ADMINISTRATIVE INFORMATION

21. Date of data collection: ..... | | | / | | | / | | | | |
m m d d y y y y

22. Method of data collection: ..... Computer C

Paper form P

23. Code number of person completing this form: ..... | | | |

Appendix G: JHS Mauscript Proposal Form

Jackson Heart Study Manuscript Proposal Form

Please read JHS Publications & Presentations protocol before completing this proposal.

ADMINISTRATIVE USE

JHS MS #

Date of Submission: _____(mm/yy) **Date of Approval:**_____ (mm/yy)

PART I. OUTLINE OF PAPER

1. Title Information

a. Proposal Title: The Moderating Effects of Socioeconomic Status (SES) Mobility and Lifetime Exposure to Discrimination on Cardiovascular Disease (CVD) Occurrence in the Jackson Heart Study

b. Abbreviated Title: Moderating Effects of SES Mobility and Racism on CVD Occurrence

c. Suggested key words: Childhood SES, Adult SES, SES Mobility, Perceived Lifetime Racism, Hypertension, CVD, African Americans, JHS**.**

2. Lead Author Name: Nkenge H. Jones-Jack, MPH
Institutional Affiliation**:** Walden University, School of Health Sciences, Public Health Doctoral Program
Address: 155 Fifth Avenue South, Suite 100, Minneapolis, MN 55401
Telephone: 678-524-1147 Fax: N/A
Email: nkenge.jack@waldenu.edu

3. Co-authors: (Proposed co-authors email address and/or telephone numbers and proposed responsibilities and/or indicate specific writing assignments. Items not assigned to a co-author are assumed to be the responsibility of the lead author. Non –JHS Lead authors are encouraged to visit the JHS Website

http://www.jsums.edu/~jhs/ for information on relevant JHS investigators. JHS may nominate additional author if special expertise for interpreting JHS data is needed).

Name	Contact Information	Responsibilities
Angela W. Prehn, PhD	angela.prehn@waldenu.edu	Supervise study, assist with the concept and design of the study, critically review and edit draft manuscript
Mario Sims, PhD*	msims2@umc.edu	Interpret results, reviewing, and editing drafts of the manuscript
Jamuir Robinson, PhD	JaMuir.Robinson@email.waldenu.edu	Interpret results, reviewing, and editing drafts of the manuscript
DeMarc A. Hickson, PhD	demarc.a.hickson@jsums.edu	Interpret results, reviewing, and editing drafts of the manuscript

Name(s) of JHS investigators from the writing group list above:
Mario Sims, DeMarc A. Hickson

Name(s) of under-represented minorities from the writing group list above:
Nkenge Jones-Jack, JaMuir Robinson, Mario Sims, DeMarc A. Hickson

4. Background/Rationale:

Socioeconomic status (SES) has been well-documented as a strong predictor of adverse cardiovascular health outcomes. SES is frequently based on several parameters beyond income and education, and interacts with complex demographic, environmental, and social atrributes which further attribute to adverse health outcomes (I. Kawachi et al., 2005; Wamala et al., 2001). Several studies have provided evidence linking economic disadvantage during early life as being strongly linked to adverse adult health outcomes (Hogberg et al., 2011; James et al., 2006; Johnson-Lawrence, Kaplan, & Galea, 2013; Hardaway & McLoyd, 2008; Pollitt et al., 2005). A recent study comparing the SES trajectories of adults from Alameda County for nearly 30 years found that as SES improved over the lifecourse, cardiovascular disease (CVD) mortality risk decreased, even after adjusting for age, race/ethnicity, marital status, and gender (Johnson-Lawrence et al., 2013). Another study conducted among a group of Black men in Pitt County, North Carolina found more than 7 times greater risk of hypertension among a stable low SES group compared to a stable high SES group (James et al., 2006). Many studies typically compare only unwavering low and high SES trajectories (Pollitt et al., 2005); little empirical evidence was found to assess how improved or diminished SES mobility is associated with CVD risk factors or CVD morbidity and mortality. In studies that did

assess trends of upward and downward mobility, many researchers agreed that individuals of high and increasing SES had lower health risk (Johnson-Lawrence et al., 2013; Hogberg et al., 2011; James et al., 2006). For example, in the Pitt County study, individuals with downward mobility had almost 6 times greater hypertension risk, whereas those with upward mobility were less than 4 times greater risk of hypertension compared to the stable high SES group (James et al., 2006). However, Hardaway et al. (2008) argued that SES mobility studies have not adequately considered the significance of race in SES mobility or as a proxy of SES. For example, Roberts et al. (2010) found that not only were Blacks more likely to be exposed to low SES during early life, but they were also more likely to experience a higher prevalence of CVD risk factors regardless of early life SES compared to their White counterparts. Meaning that even Blacks of high summary SES experienced greater incidence of heart failure than Whites of low summary SES. This provides evidence that exploring differences SES mobility alone will not explain the Black-White differences observed in CVD health outcomes.

Jones (2000) illustrated that perceived racial discrimination occurs at multiple levels and contributes to inequities in the allocation of services, goods and resources, and health outcomes. Some researchers argue that although lower SES is not definitively responsible for social stressors (e.g., racism), it has been strongly suggested that the stressors associated with lower SES often directly or indirectly influence health and well-being (Thoits, 2010). At all levels of SES, African Americans are impacted by chronic social and economic stressors, such as racial discrimination, more frequently than whites (Hatch & Dohrenwend, 2007; David R. Williams & Mohammed, 2008). Research has shown that African Americans of higher SES more frequently report experiences of perceived racial discrimination (Sims et al., 2012), though gender differences exist (Dailey, Kasl, Holford, Lewis, & Jones, 2010). Researchers have also explored various approaches of how the stress related to experiences of perceived racial discrimination may transcend multiple aspects of an individual's life (e.g., residentially segregated communities; stereotypical or derogatory media portrayals; level of control or flexibility at work; availability, quality, and affordability of resources and services; understanding of cultural differences). Of greater concern are the multiple pathways through which racial discrimination affects health (Brondolo, Gallo, et al., 2009). Two aspects of racial discrimination that will be address in this study are perceived lifetime exposure and burden. Perceived lifetime racial discrimination is the cumulative exposure to either negative or differential treatment or judgement an individual of a certain racial/ethnic population perceives that they have experienced over their lifetime compared to other racial/ethnic groups. Burden refers to the extent to which racial discrimination exposure has made an individual's life stressful, more difficult, and less productive over the course of their lifetime.

While several studies have examined the impact of these key constructs on hypertension and CVD outcomes independently, Hardaway et al. (2008) acknowledges the failure to understand that the consequences of racial discrimination on social mobility creates

unique challenges for African Americans. Furthermore, there is very little research available to explain the multiple aspects of racial discrimination using a multidimensional instrument. The study conducted by Sims et al. (2012) is one of the first studies to examine the impact of multiple measures of lifetime discrimination exposure and burden on hypertension among Blacks; however this study does not measure lifetime racism discrimination exposure or the burden at different levels of SES mobility. Exploration of these measures of SES mobility can further guide understanding of how CVD health disparities manifest in this population. Because African American may experience different levels of SES mobility and different levels of lifetime racial discrimination simultaneously, it is important to investigate how these factors moderate the extent to which hypertension contributes to the CVD health disparities observed among Blacks in the Jackson Heart Study (JHS) both independently and collectively. The objective of this study is to investigate how African Americans in the JHS cohort experience perceived racial discrimination at different levels of SES mobility, and how the interaction between SES mobility and perceived racial discrimination (SES-Racism Effect) is associated with prevalent and incident CVD.

5. Research Hypotheses/Research Questions:

RQ1: What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of lifetime discrimination attributed to race, as measured by the cumulative occurrence of perceived lifetime discrimination exposure attributed to race?

Hypothesis 1: Increasing levels of SES mobility will be associated with decreasing levels of perceived lifetime discrimination exposure attributed to race after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

Null Hypothesis 1: There will be no association between levels of SES mobility and levels of perceived lifetime discrimination exposure attributed to race after adjusting for identified covariates.

If an association between levels of SES mobility and levels of perceived lifetime exposure attributed to race is identified, the following subhypotheses will also be tested (Figure 1):

> Hypothesis 1b: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race will be inversely moderated by age.

Hypothesis 1c: The association between levels of SES mobility and levels of perceived lifetime exposure attributed to race will be more strongly moderated by males than females.

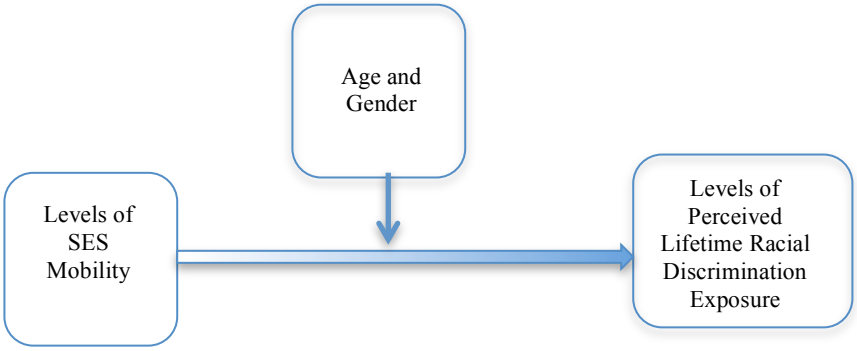

Figure 1. Causal pathway between Levels of SES Mobility and Levels of Perceived Lifetime Racial Discrimination moderated by Age and Gender.

RQ2: What is the relationship between levels of SES mobility, as measured by the change in SES from childhood to adulthood, and levels of burden of lifetime discrimination attributed to race, as measured by the extent of life stressfulness, difficulty, and productivity as a result of perceived lifetime discrimination attributed to race?

Hypothesis 2: Increasing levels of SES mobility will be associated with decreasing levels of burden of lifetime discrimination attributed to race after adjusting for identified covariates.
Null Hypothesis 2: There will be no association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race after adjusting for the following covariates, identified based on previous studies and determined to have a statistical association ($p<0.20$) in the current sample: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

If an association between levels of SES mobility and levels burden attributed to perceived lifetime racial discrimination is identified, the following subhypotheses will also be tested (Figure 2):

Hypothesis 2b: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race will be inversely moderated by age.

Hypothesis 2c: The association between levels of SES mobility and levels of burden of lifetime discrimination attributed to race will be more strongly moderated by males than females.

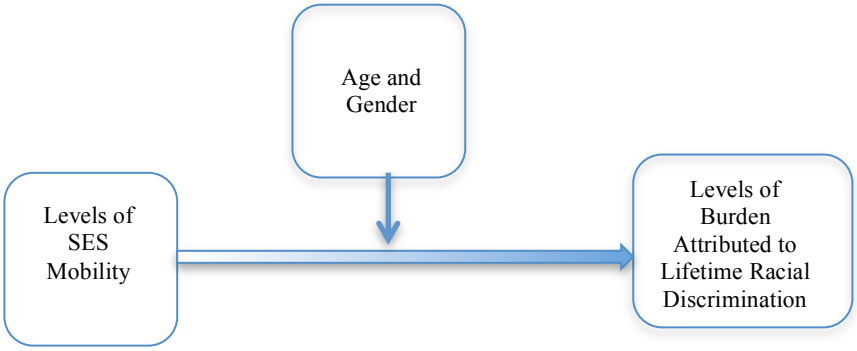

Figure 2. Causal pathway between Levels of SES Mobility and Levels of Burden Attributed to Lifetime Racial Discrimination moderated by Age and Gender.

RQ3: Do the levels of the SES mobility, cumulative perceived lifetime racial discrimination exposure, or burden moderate the relationship between hypertension and cardiovascular disease?

Hypothesis 3: The relationship between hypertension and CVD is inversely moderated by increasing levels of SES mobility.
Null Hypothesis 3: The relationship between hypertension and CVD is not moderated by increasing levels of SES mobility.

Hypothesis 3b: The relationship between hypertension and CVD is positively moderated by increasing levels of perceived lifetime discrimination attributed to race.
Null Hypothesis 3b: The relationship between hypertension and CVD is not moderated by increasing levels of perceived lifetime discrimination attributed to race.

Hypothesis 3b: The relationship between hypertension and CVD is positively moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.
Null Hypothesis 3b: The relationship between hypertension and CVD is not moderated by increasing levels of burden attributed to perceived lifetime racial discrimination.

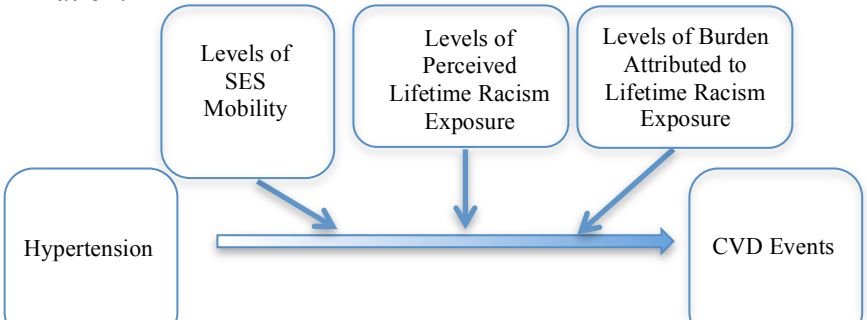

Figure 3. Causal pathway between hypertension and CVD outcomes moderated by levels of the SES mobility, perceived lifetime racial discrimination exposure, or burden.

RQ4: If a relationship between levels of SES mobility and levels of perceived lifetime discrimination exposure attributed to race is found, does the interaction of these variables (i.e., SES-Racism Effect) moderate the relationship between hypertension and cardiovascular disease?

> Hypothesis 4: The relationship between hypertension and CVD is positively moderated by the SES-Racism Effect.
> Null Hypothesis 4: The relationship between hypertension and CVD is not moderated by the SES-Racism Effect.

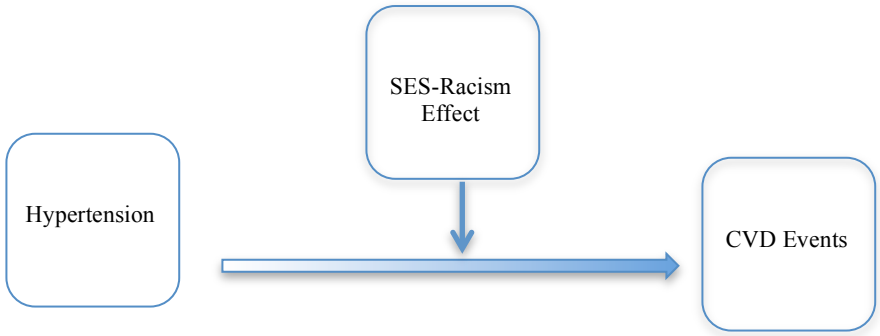

Figure 4. Causal pathway between hypertension and CVD outcomes moderated by the SES-Racism Effect.

6. Data: (Visits and variables to be used, sample inclusions/exclusions)

Data from the baseline examination (Visit 1) and CVD events (2000-2010).

Inclusion criteria include participants who attended the baseline examination conducted between September 2000 and March 2004 of the JHS.

Exclusion criteria include participants with missing discrimination, hypertension, CVD outcomes, and demographic data will be excluded from this study analysis. In addition, participants that were identified by Sims et al. (2012) to attribute their lifetime discrimination exposure to nonracial factors were also excluded from analysis to align with the research questions, which are specifically centers around factors related to racial based discrimination.

Dependent variables
In this study, we will examine the following outcomes:

RQ1: Cumulative perceived lifetime racial discrimination exposure is the frequency of discrimination exposures, as a measure of the number of times discrimination is perceived, will be summed across nine domains (i.e., school, getting a job, at work, getting housing, getting money or resources, getting medical care, in a public place, getting services, or in some other environment).

RQ2: Cumulative burden attributed to perceived lifetime racial discrimination exposure will be determined by combining scores for perceived burden to racial discrimination for the three domains: stress experienced, interfered with having full life, and made life difficult.

RQ3 and RQ4: CVD events observed during the period of risk (2000-2010), including fatal and nonfatal myocardial infarction, coronary insufficiency (prolonged angina with documented electrocardiographic changes), heart failure, and stroke;

Independent variables
The independent variables used in analysis are based on a calculated measure of varying time periods. These variables will be measured at baseline only, and therefore specified as fixed and assumed to maintain a consistent value throughout the duration of the study period.

2. ***Childhood Socioeconomic Status (Collected during Year 1 Annual Follow Up)***
    1. Childhood material resources (number of rooms, availability of plumbing, TV, Car, air conditioning, phone, electricity, and refrigerator)
    2. Father's and Mother's education
    3. Father's and Mother's occupation

3. ***Adult Socioeconomic Status (Collected during Baseline Exam)***
    1. Adult education (<high school, high school/GED, some college, and college graduate)

2. Adult income (low, lower-middle, upper-middle, and affluent)
3. Adult occupation (Production/construction, sales, services, and professionals)

4. SES or SES Mobility

SES mobility will be determined by combining scores for childhood SES and adulthood SES, and then dichotomizing as high or low to create four non-overlapping SES mobility scores as follows:

1. Stable High (HH) (Childhood high, Adult high)
2. Diminishing (HL) (Childhood High, Adult low)
3. Increasing (LH) (Childhood low, Adult high)
4. Stable Low (LL) (Childhood low, Adult low)

The Stable High group is expected to have the lowest risk, followed by Increasing, and Diminishing. The Stable Low group is expected to have the greatest risk. In studies that assessed trends of upward and downward mobility, researchers agreed that individuals of high and increasing SES had lower health risk. If review of the data indicates that inadequate sample sizes are available for testing each of these subgroups, categories will be collapsed into High (including Stable High and Increasing) and Low (Stable Low and Diminishing).

5. Cumulative Racial Discrimination

Cumulative discrimination values will be determined for exposure to lifetime discrimination attributed to race and burden of lifetime discrimination independently.

Cumulative Lifetime Racial Discrimination Exposure - The frequency of discrimination exposures, as a measure of the number of times discrimination is perceived, will be summed across nine domains (i.e., school, getting a job, at work, getting housing, getting money or resources, getting medical care, in a public place, getting services, or in some other environment) to create the cumulative racial discrimination score. A scatterplot will be used to examine the distribution of the scores before determining categorical levels.

Cumulative Burden Attributed to Lifetime Racial Discrimination - A burden score will be determined by combining scores for perceived burden to racial discrimination for the three domains: stress experienced, interfered with having full life, and made life difficult. The cumulative value for burden due to racial discrimination, ranging between 4 and 16 will be dichotomized into lower and upper strata (based on the median). These strata for discrimination burden represent the potential overall impact of racism burden (low vs. high) on JHS participants.

6. ***Hypertension Status***
   Hypertension is determined as the presence or absence of elevated blood pressure based on whether or not the average systolic blood pressure was 140mmHg or greater, and diastolic blood pressure was 90mmHg or greater, or using of anti-hypertensive meds (Sims et al, 2012).

Covariates

The following variables will be used as covariates in this study: BMI ($kg/m^{2)}$, smoking status, physical activity score, diabetes status, alcohol consumption, diet, total cholesterol, LDL, HDL, and John Henryism, financial adversity/stress, and job strain.

Each covariates will independently be tested for collinearity. Variables determined to have high multicollinearity may be either eliminated or combined to create a composite index variable, depending on empirical justification. Covariates will be added last to each model to determine the presence of confounding.

7. **Brief Statistical Analysis Plan and Methods:** (Including power calculations, if necessary.)
   Similar to a previously conducted study, participants will be excluded from analysis if all discrimination data (n=283), education (n=20), or hypertension (n=59) are missing, providing a final sample size of 4939 participants (Sims et al., 2012). To align with the research questions, participants that were identified by Sims et al. (2012) to attribute their lifetime discrimination exposure to nonracial factors were also excluded from analysis (n=1626). Preliminary "posteriori" power analysis was conducted using G*Power 3.1.7 to determine the feasibility of the JHS sample in addressing the proposed research questions. Given that the sample size for the study is known (n=3313), a posteriori power analysis was used to determine whether or not the sample provides adequate power for the study. A multiple regression design was selected to solve for power based on a sample size of 3300, and using a two-sized t-test with an alpha significance level of 0.05 (JHS Coordinating Center, 2008). The analysis controlled for SES and racism as independent variables, and accounted for the adjustment of the 12 identified covariates. The analysis revealed that this study has more than adequate statistical power (80%) to detect a small effect (0.10) of SES mobility and racial discrimination on the relationship between hypertension and CVD (Research Question 3). Further study analysis (including central tendencies) will be conducted using SPSS.

Analysis for Research Questions 1 and 2

RQ1 aims to explore the relationship between levels of SES mobility and levels of perceived lifetime racial discrimination exposure, and factors (i.e., age and gender) that may moderate the relationship. Similarly, the aim of RQ2 is to explore the relationship between levels of SES mobility and levels of perceived lifetime racial discrimination burden. As such, the following analysis plan will be applied to both questions. Multinominal logistic regression will be used to measure the linear relationship between the levels of perceived lifetime racism and levels of SES mobility, and how the relationship is influenced by age and gender. First, a chi-square test will be applied to the categorical variables, based on the appropriate degrees of freedom (df), to determine whether or not the distributions of SES mobility levels and racial discrimination patterns are statistically independent, with *p*-values (0.05) included to illustrate significance. This strategy will be applied to each moderator and covariate to evaluate the contribution in the overall relationship. Only covariates with a bivariate association with the dependent variable at $p<0.20$ will be included in the multivariate model, suggested to be a standard practice. Moderators and covariates will be fit to the logistic model in a stepwise fashion. Model 1 for both research questions includes the independent and dependent variables (i.e., SES mobility and perceived lifetime racial discrimination exposure/burden, respectively). The evaluated covariates will be introduced as blocks in successive models; the stepwise addition will begin with demographics followed by adjustment for lifestyle behaviors, risk factors, and then other social stressors. Included in the output will be a parameter estimates table, which generates the B coefficient and p-value, and a classification table, which determines the accuracy of the model. If the p-value is less than the significance level of $p<0.05$, the $H1a_{Null}$ hypothesis will be rejected; it will be concluded that a relationship between levels of SES mobility and levels of perceived lifetime racism exists.

Finally, if the regression analysis produces a large standard error or B coefficient, additional analysis will be conducted to investigate problems that may not be detected by SPSS version 21.0 (e.g., multicollinearity). A scatterplot will be used to detect whether or not the relationship between the independent and dependent variables monotonically increases or decreases (i.e., in a manner may or may not be linear), and to identify possible outliers.

Analysis for Research Questions 3 and 4

RQ3 aims to explore if the relationship between hypertension and CVD end points is moderated by levels of SES mobility, perceived lifetime racial discrimination, or burden. Each of these moderators will be modeled separately. In addition, RQ4 investigates whether the relationship between hypertension and CVD cumulative incidence is moderated by the SES-Racism Effect. Since the hypotheses for both

research questions have the same independent and dependent variables, the overall plan of analysis (Cox regression) will be the same. Cox regression of CVD cumulative incidence observed during the period of risk will be used to explore the influence of multiple variables on survival time. Estimating the potential impact of social constructs over the lifecourse (i.e., levels of SES mobility, levels of perceived lifetime racial discrimination, burden, and SES-Racism Effect) will provide increased understanding of for whom or under what conditions relationship between hypertension and CVD outcomes may change.

For RQ3, Cox regression models will be used to analyze the association between all independent variables (levels of SES mobility, levels of perceived lifetime discrimination attributed to race, and burden due to racial discrimination) to determine which of these factor has the most robust relation to risk for CVD events, adjusting for covariate factors. Additional Cox regression models will examine the extent to which SES-Racism Effect determines the occurrence of CVD events. Bivariate analysis will be conducted to describe the direction and extent of each association, statistical significance, and intercorrelations among independent and dependent variables. Only covariates with a bivariate association with the dependent variable at $p<0.20$ will be included in the model. Moderators and covariates will be fit to the cox regression model in a stepwise fashion. Three primary models will be analyzed including the independent and dependent variables (i.e., hypertension and CVD, respectively), with each model examining the independent interaction of each moderator (i.e., levels of SES mobility, levels of perceived lifetime racial discrimination exposure, and burden attributed to lifetime racial discrimination). In addition, the evaluated covariates will be introduced as blocks in successive models; the stepwise addition will begin with demographics followed by adjustment for lifestyle behaviors, risk factors, and then other social stressors. The hazard ratios for risk of CVD will be presented in a table will be used to illustrate differences across models. Consideration for time-dependent effects will be made. If the p-value is less than the significance level of $p<0.05$, the $H1a_{Null}$ hypothesis will be rejected; it will be concluded that a relationship between respective independent and dependent variables exists.

8. References: (Maximum 15)

PART II. AUTHOR CONTRIBUTIONS

9 Have all co-authors reviewed and approved this document? **X**Yes (required)

10. . Does the lead author (or designee) agree to present findings at a JHS Colloquium or Seminar? **X** Yes (required)

Note: A lay summary is required when submitting the completed manuscript draft for JHS and NHLBI approvals.

PART III. ADDITIONAL INFORMATION

11. Type of Study:
X Full Cohort Family Study _____ Sub-Study
_____ Ancillary Study _____ Case Control _____ARIC/JHS Combined Cohort
_____ Other (list):

12. Type of Data:
X Longitudinal X Cross-Sectional _____ Other (list):

13. Location of Statistical Analysis:
X Central (by Jackson Heart Study Staff)
_____ Local (list site) _____

14. Genetic Information:
a. Do you propose use of data from a participant's DNA? _____ Yes (see b)
X No
b. If yes, for a primary aim or secondary aim of JHS? (check one or both)
_____ Primary Aim (heart, vascular disease) _____ Secondary Aim (other conditions)

15. Conflict of Interest
a. Are these analyses to involve a for-profit corporation? _____Yes __X__No
b. Do you or any member of your Writing Group intend to patent any process, aspect of outcome of these analyses? _____Yes ___X___No

16. Data Sharing Agreement
Do you agree to have the Lead Author and any co-authors who will have direct access to JHS data sign the JHS Data Sharing Agreement once it has been approved?
___X__Yes (Required)

17. JHS Manuscript Overlap

The Lead Author has reviewed all existing JHS manuscripts / manuscript proposals and found:

a. No similar manuscripts / proposals ___X__Yes
b. The following manuscripts / proposals with similarities: (List MS # title and Lead Author below)

18. Note: Completion of manuscript preparation is expected in less than three years. The manuscript proposal will expire if no manuscript draft is submitted for JHS review at the end of the three years from date of approval. If additional time is needed after three years, please contact JHS for extension.

Appendix H: Reprint Permission from Annual Review of Public Health

Title: Social Determinants and the Decline of Cardiovascular Diseases: Understanding the Links

Author: Sam Harper, John Lynch, George Davey Smith

Publication: Annual Review of Public Health

Publisher: Annual Reviews

Date: Apr 21, 2011

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