November 2019

Perceived Discrimination and Cardiovascular Outcomes in Blacks: A Secondary Data Analysis of the Heart SCORE Study

Marilyn Aluoch
University of South Florida

Perceived Discrimination and Cardiovascular Outcomes in Blacks:

A Secondary Data Analysis of the Heart SCORE Study

by

Marilyn Aluoch

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
Department of Nursing
College of Nursing
University of South Florida

Major Professor: Carmen Rodriguez, Ph.D., ANP-BC, AOCN
Ponrathi Athilingam, Ph.D., RN, A.C.N.P. F.A.A.N.P
Kevin Kip, Ph.D., F.A.H.A.
Ji Ming, Ph.D.

Date of Approval:
November 7, 2019

Keywords: Perceived Discrimination, Cardiovascular Outcomes, Blacks, Cardiovascular Risk Factors

DEDICATION

This dissertation is dedicated to my mother and father. Against the advice of many villagers not to educate a girl you invested in me! I hope I made you proud today and always. This dissertation is also dedicated to my children Zadock, Zawadi and Zachary. I hope I set the right example for you to emulate!

ACKNOWLEDGMENTS

Over the last 7 years, I have pursued my dream of attaining a doctoral degree. This journey has been long and rocky, yet one of the most fulfilling experiences of my life. During this adventure, I was blessed to be joined by many amazing human beings from the university and personal life. First, I would like to thank Dr. LaRon Nelson who was my first adviser and dissertation chair, you took me under your wings and showed me that I could fly! You introduced me to research and a world of possibilities. You believed in me even when I didn't believe in myself. Thank you! Dr. Diane Morrison-Beedy, thank you for all your support and for your wisdom when I couldn't think for myself. I will forever remember you for this advice, "Marilyn family comes first, go take care of your children, come back when you are ready". Thank you!

To Dr. Kevin Kip, I lack words to express my gratitude to you. You literally saved my life when I thought my dream of attaining my PhD had come to a tragic end. When everyone said it was impossible, you said, "it can be done!" Thank you for allowing me to use your data, for your advice and relentless support throughout. Dr. Carmen Rodriguez, what a beacon of hope you are! I came to your office in a hopeless and frustrated state. You didn't know me but believed in me instantaneously, agreed to be my dissertation chair and advised me on a way forward. Your advice and encouragement from that day onwards sustained me. Your wisdom and intellectual guidance were perfect for me. I learnt way more than research from you. I am forever

grateful! Dr. Ponrathi Athilingam, thank you for your intellectual guidance and for reassuring me that I could do it. Dr. Ji, thank you for your stimulating statistical questions and for the video you sent for analysis of my data and for always responding to my emails promptly.

To my children, thank you for teaching me love, strength, determination, resilience and patience. I hope my resilience and determination influence you in same way or form as you pursue your own dreams and strive to beat the odds in your personal lives. To my many wonderful friends who watched my kids when I studied over the years, Miriam, Katherine, Vivian and Winnie, may God bless you. You are a gift to me and to the world!

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Despite the consistent reduction in morbidity and mortality associated with cardiovascular disease (CVD) over the last four decades, CVD remains the leading cause of death globally. In the United States, Blacks are disproportionately affected by CVD compared to Whites. Blacks are also more likely to report incidence of perceived discrimination. Perceived discrimination has been linked to cardiovascular risk factors such as smoking, hypertension (HTN), hyperlipidemia, and obesity. However, the relationship between perceived discrimination and cardiovascular outcomes such as stroke, myocardial infarction, acute ischemic syndrome, coronary revascularization, and cardiac death remains unclear. The primary goal of this study was to examine whether there is a relationship between perceived discrimination and cardiovascular events. The specific aims of this study were: (1) to examine the relationship between perceived discrimination and cardiovascular events in Blacks in the Heart SCORE Study, and (2) to evaluate whether age, sex, education, income, smoking, physical activity, and stress moderate the relationship between perceived discrimination and cardiovascular events in Blacks. Cox regression and Kaplan Meier Methods were utilized to model the relationship between perceived discrimination and cardiovascular events among Blacks in the Heart SCORE study. In relation to Aim 1, the study found not enough evidence to indicate a statistically significant association between the predictor of perceived discrimination and cardiovascular health events in Black participants in the Heart

SCORE study. Model 2 had the best model fit and included four variables of Everyday Discrimination Scale-1, Everyday Discrimination Scale-2, age, and gender. Model 2 (-2 LL = 646.29) was significantly improved over the baseline model with no predictors [-2LL = 670.39; X^2 (5) = 27.21, $p < .0005$]. The second model was also a significant improvement over the first model with 3 predictors (X^2 (2) = 22.53, $p < .0005$). The predictor of age was statistically significant [HR = 1.07; 95% CI for HR [1.03, 1.11], $p < .005$]. The predictor of gender was also statistically significant [HR = 0.46; 95% CI for HR [0.25, 0.76], $p = .003$].

For Aim 2, none of the interaction terms tested reached statistical significance. Therefore, age, gender, physical activity, smoking, income, education and stress did not modify the relationship between perceived discrimination and cardiovascular events among Black participants in the Heart SCORE study.

CHAPTER ONE:

INTRODUCTION

Blacks have been disproportionately affected by chronic diseases and poor health outcomes for decades. In its 1985 report, the Department of Health and Human Services Secretary's Task Force Report on the health of Blacks and other minorities showed that Blacks bore a greater burden of cardiovascular disease (CVD) and were more likely to die from CVD related complications compared to their White counterparts (Nickens, 1986). Fourteen years after that report, a 1999 study presented a widening of the gap between Blacks and Whites for diseases of the heart, Human Immunodeficiency Virus (HIV) infection for males, and cancers for females (Feldman & Fulwood, 1999). Today, research continues to confirm a stark disparity in CVD with Blacks at a greater risk than White individuals (Benjamin et al., 2017; Mozaffarian et al., 2016a). Furthermore, studies show that despite the consistent reduction in morbidity and mortality associated with CVD over the last four decades (Mensah, 2005; Safford et al., 2012), CVD remains the leading cause of death worldwide (Gillespie, Wigington, & Hong, 2013). According to the World Health Organization (WHO; 2018), 17.9 million deaths, or 31% of all deaths every year, are attributed to CVD.

The American Heart Association's (AHA) report on heart disease and stroke statistics has shown that the overall prevalence of CVD (comprising Coronary Heart Disease {CHD}, Heart Failure {HF}, Stroke, and Hypertension {HTN}) in American

adults 20 years of age or older was 48% (121.5 million) in 2016 and that this prevalence increases with aging (Mozaffarian et al., 2016a). When HTN prevalence was omitted from the overall CVD prevalence (CHD, HF, and stroke only), this prevalence is 24.3 million, a decreased by 39% (Benjamin et al., 2019). It is estimated that 45.1% of all Americans will have some form of CVD by 2035. Moreover, 19.2% of deaths among Americans who are 65 years of age and younger were attributed to CVD in 2016 (Benjamin et al., 2019). Additionally, heart disease, HTN, and stroke were among the 15 leading causes of disability in the United States. The direct and indirect costs of CVD were estimated at $351.2 billion dollars and are expected to rise to $1.1 trillion dollars annually by 2035 (Benjamin et al., 2019). Even though mortality associated with CHD decreased from 2005 to 2015, and is expected to continue to decline, the WHO predicts that excess mortality and morbidity among racial minorities will continue to persist (Benjamin et al., 2018; Okhomina, Glover, Taylor, & Sims, 2018).

According to the United States Census Bureau (2018) report on demographics by race, Blacks only constitute approximately 13.4% of the U.S. population. However, CVD risk factors prevalence within black communities stands at 46% for both males and females compared to 38% and 32% among white males and females, respectively (Kanchi et al., 2018; Yano et al., 2017). Notably and in comparison, the prevalence of HTN remains starkly disproportionate among American adults, with Blacks at 43% and whites at 29%. In addition, Blacks continue to bear a greater burden to cardiovascular risk factors such as obesity, HTN, hypercholesterolemia, smoking, and diabetes (Kanchi et al., 2018), as well as a higher risk of dying from cardiovascular-related illness or complication (Mozaffarian et al., 2016b). This disparity between Blacks and Whites in

CVD prevalence and mortality continues to widen despite a steady decline of cardiovascular-related morbidity and mortality rates among all other racial groups in the United States (Pool, Ning, Lloyd-Jones, & Allen, 2017). It is for this reason that cardiovascular health research in this area has recently shifted focus to identification of modifiable risk factors that contribute to this racial disparity. Recent studies have recognized several factors that contribute to this disparity, which can be broadly categorized as structural and institutional barriers, healthcare, safe environments, educational opportunities, and stress-related psychosocial factors (Ferdinand et al., 2017; Pool et al., 2017). In relation to stress-related psychosocial factors, Dunlay and colleagues (2017) identified perceived discrimination as a potential contributing factor to the disparate prevalence of cardiovascular disease among Blacks.

The harmful effects of discrimination on health are well documented in the literature, particularly in the context of stress and coping framework (Ahmed, Mohammed, & Williams, 2007). Even though overt expression of acts of discrimination have substantially reduced over the years, subtle forms of discrimination continue to thrive in the United States, particularly towards racial minorities (Paradies et al., 2015). Racism or racial discrimination refers to complex societal systems characterized by inequal distribution of power, resources, capacities, and opportunities across racial or ethnic groups and usually manifest in the form of discrimination, beliefs, prejudices, attitudes, or stereotypes as well as racialized structures and practices (Paradies et al., 2015; Paradies, 2006). Racial discrimination has been recognized as a social determinant of health that is responsible for at least some part of the health disparity among racial minorities (Marmot, Friel, Bell, Houweling, & Taylor, 2008). Williams and

Mohammed (2009) defined perceived discrimination as the belief that an individual has been unfairly treated by another individual or an institution and that this unfair treatment was solely due to personal attributes of the victim, such as age, race, gender, religion, socioeconomic status, sexual orientation, or weight. Several studies have associated perceived discrimination with cardiovascular health risk factors such as hypertension, obesity, Diabetes mellitus type II, C-reactive Protein, and hypercholesterolemia. For instance, in a cross-sectional observational study of 1,005 Whites and Blacks with almost equal representation, Krieger and colleagues (2013) used multiple racial discrimination measures to assess the prevalence of racial discrimination and to examine the association between racial discrimination and risk for developing CVD. They found a higher prevalence of racial discrimination among Blacks compared to their White counterparts. With regard to the impact of racial discrimination on risk for CVD, Blacks had higher Framingham risk scores and excess risk for elevated systolic blood pressure (SBP) and hypertension (Krieger et al., 2013). Other studies with Blacks have found perceived discrimination to be significantly associated with higher levels of C-reactive Protein (Goosby, Malone, Richardson, Cheadle, & Williams, 2015), smoking (Guthrie, Young, Williams, Boyd, & Kintner, 2002; Landrine & Klonoff, 2000), and HTN (Williams & Neighbors, 2001).

Studies that have assessed the relationship between cardiovascular events and perceived discrimination are scant and show conflicting results (Dunlay et al., 2017; Everson-Rose et al., 2015). In their multi-ethnic cohort study, Everson-Rose et al. (2015) found a modest increase in risk of cardiovascular events in participants who reported discrimination. In contrast, Dunlay and colleagues (2017) found no

associations of perceived discrimination with risk for incident cardiovascular events. In fact, their study found an unexpected inverse association of everyday discrimination and all-cause mortality.

Statement of the Problem

Cardiovascular disease remains the leading cause of death globally (Gillespie et al., 2013). Research has shown a higher burden of cardiovascular health risk factors, such as obesity and hypertension, Diabetes mellitus II, and smoking, among Blacks (Kanchi et al., 2018; Lin et al., 2018). In addition, Blacks are more likely to report incidence of perceived discrimination. While the association between perceived discrimination and cardiovascular health risk factors such as HTN and obesity among Blacks is well established (Kanchi et al., 2018), the link between perceived discrimination and cardiovascular health outcomes such as cerebrovascular accident (stroke), acute ischemic syndrome, coronary revascularization, cardiac death, and cardiovascular-related hospitalization remains unclear. In order to design public health prevention initiatives aimed at decreasing cardiovascular health events within Black communities, it is imperative that we first understand the role of perceived discrimination on cardiovascular health outcomes.

Statement of Purpose

Previous studies have shown a higher prevalence of cardiovascular disease and adverse outcomes within Black communities. Studies have also shown that Blacks are more likely to report discrimination compared to their White counterparts. However, it is not clear whether perceived discrimination is a contributing factor to adverse cardiovascular outcomes among Blacks. The purpose of this secondary data analysis

was to examine whether there is a relationship between perceived discrimination and cardiovascular health outcomes, including stroke, non-fatal myocardial infarction (MI), acute ischemic syndrome, coronary revascularization, and cardiac death, and how demographic, socioeconomic, behavioral and psychosocial variables may moderate this relationship.

Specific Aims

1. To examine the association of perceived discrimination and cardiovascular health outcomes including; non-fatal myocardial infarction, cerebrovascular accident, acute ischemic syndrome, coronary revascularization and cardiac death in Blacks in the Heart SCORE Study.
2. To evaluate whether demographic (age, sex), socioeconomic (education and income) behavioral (smoking and physical) and psychological (stress) variables moderate the relationship between perceived discrimination and cardiovascular health outcomes in Blacks in the Heart SCORE study.

Research Questions

1. Among Blacks in the Heart SCORE study, does perceived discrimination predict cardiovascular health outcomes?
2. Does demographic (age, sex), socioeconomic (education and income) behavioral (smoking and physical) and psychological (stress) variables moderate this postulated relationship?

Significance of the Study

Studies on the association of perceived discrimination and cardiovascular disease outcomes are scant. Moreover, the few studies that were available for review

showed conflicting results. This study sought to add to the limited empirical evidence on the association between perceived discrimination and cardiovascular health events. In addition, this study also hoped to help streamline cardiovascular risk assessment tools to better address CVD and CVD events within black communities.

Definition of Terms

For the purposes of this secondary data analysis, the following terms are defined and will be used throughout the study.

Cardiovascular disease (CVD) – CVD refers to conditions of heart and vascular system, including congenital malformations of the heart and/or vasculature, hypertension, arrhythmias or irregular electrical activity of the heart, stroke, peripheral artery disease, diseases of the veins, valvular heart disease, and ischemic heart disease or atherosclerosis (Benjamin et al., 2018).

Cardiovascular/cardiac events – For the purposes of this study, cardiovascular events refer to non-fatal myocardial infarction, cerebrovascular accident (stroke), coronary revascularization procedure, acute ischemic syndrome, and a diagnosis of death related to cardiac disease.

Myocardial Infarction (MI) – MI refers to the presence of a complete blockage of blood flow to an area of the heart most commonly caused by atherosclerosis (Benjamin et al., 2018).

Coronary revascularization – Coronary revascularization refers to any medical procedure that is conducted to restore blood flow to the heart – for example, percutaneous transluminal coronary angioplasty (PTCA), arterial stenting, and coronary artery bypass surgery (CABG;(Bonaca et al., 2012).

Cerebral vascular accident (CVA) – CVA refers to a condition that results from an insufficient flow of oxygen to the brain due to obstruction of blood flow to the brain, resulting in the death of brain cells. It is also commonly known as a stroke (Benjamin et al., 2018).

Coronary heart disease (CHD) – Coronary heart disease refers to any disease affecting the coronary arteries and includes acute ischemic coronary disease, atherosclerotic cardiovascular disease, acute myocardial infarction, angina pectoris, and all other forms of chronic ischemic coronary heart disease (Benjamin et al., 2019; Go et al., 2014)

Mortality – Mortality refers to the total number of deaths attributable to a given disease in a population during a specific interval of time, usually one year, reported (Go et al., 2014).

Morbidity – Morbidity refers to any physical or psychological state considered to be outside the realm of normal well-being. The term morbidity is often used to describe illness, impairment, or degradation of health, especially when discussing chronic and age-related diseases that can worsen over time (Go et al., 2014).

Perceived discrimination – Perceived discrimination refers to an individual's subjective viewpoint on receiving unfair treatment by others (Coley et al., 2017).

CHAPTER TWO:

REVIEW OF LITERATURE

Introduction

Chapter Two focuses on the conceptual framework that guided this study and includes a review of empirical literature on the variables that were examined in this study. First, the conceptual framework is discussed. Second, traditional cardiovascular risk factors of interest for this secondary analysis are reviewed including age, gender, race, smoking, physical activity, income, education and stress. Finally, literature on nontraditional and the potentially modifiable risk factor of perceived discrimination and its association with health in general and cardiovascular disease are discussed.

Conceptual Framework

The conceptual model that guided this study was adapted from Pascoe and Smart Richman (2009). Pascoe and Richman (2009) developed and tested a discrimination model to explain the relationship between perceived discrimination and health outcomes. Their model included three pathways through which perceived discrimination potentially affects physical and mental health. The first pathway is direct effect of discriminatory acts on health. The second potential pathway posits that the relationship of perceived discrimination to health has the potential to be mediated through stress response to discriminatory events. In other words, it can be mediated through a psychological response to lowered positive emotions and heightened

negative emotions. When an individual experiences discriminatory acts, this stress response would often be activated, leading to consistent negative emotional state. This pathway also includes chronic heightened physiological stress response such as cortisol and cardiovascular reactivity responses. The third mechanism postulates that health outcomes can be influenced via health behaviors. Participants who experience discriminatory acts may engage in unhealthy behaviors or fail to participate in healthy behaviors as a way of coping with the stress related to discrimination.

In the current study, direct relationship between perceived discrimination and cardiovascular events (non-fatal myocardial infarction, acute ischemic syndrome, coronary revascularization, cerebrovascular accident and cardiac death) was evaluated. In addition, potential moderating effect of demographic (age and sex), socioeconomic (income and education), behavioral (smoking and exercise) and psychosocial (stress) variables on the relationship between perceived discrimination and cardiovascular events of interest were also examined.

Relationship Between Age, Gender, Race, and Cardiovascular Disease

Age, gender, and race have long been recognized as nonmodifiable cardiovascular risk factors (Bibbins-Domingo et al., 2009; Ennezat et al., 2008; Goldstein et al., 2014; Kanchi et al., 2018; Lakatta, 2002; Maggioni et al., 1993; O'Neil, Scovelle, Milner, & Kavanagh, 2018; Regitz-Zagrosek, Oertelt-Prigione, Seeland, & Hetzer, 2010; Safford et al., 2012). Aging is typically characterized by a progressive decline in physiological function that originates at the cellular and tissue level (Campisi, 2013). Empirical evidence has suggested that this decline in physiological function is a major risk factor for cardiovascular morbidity and mortality and that this risk increases

with age (Lakatta, 2002). Studies have also suggested that older individuals are more likely to experience a Myocardial Infarction (MI) than their younger counterparts (Shih, Lee, Lee, & Boyle, 2011). Moreover, older individuals who experience an MI are also more likely to develop heart failure compared to younger individuals (Ennezat et al., 2008; Torabi, Cleland, Rigby, & Sherwi, 2014). Mortality due to ischemic heart disease increases progressively with age (Maggioni et al., 1993). In a study that assessed in-hospital mortality of patients treated with thrombolytic therapy after experiencing an MI, Maggioni et al. (1993) found that the mortality rate among participants increased with age. While the mortality rate among participants 40 years or younger was 1.9%, mortality in participants who were 80 years or older increased to 31.9%. Additionally, autopsies performed on patients who died showed an increase in cardiac rapture frequency, from 19% in participants who were 60 years or younger to 86% in participants 70 years or older (Maggioni et al., 1993).

Aging has also been associated with hypertension (HTN), with older individuals more likely to develop HTN, particularly isolated systolic HTN, than younger individuals (Bavishi, Goel, & Messerli, 2016; Vasan et al., 2002). According the Framingham Heart Study, individuals 65 years of age or older had a 90% lifetime risk of developing HTN if they lived for another 20 to 25 years (Vasan et al., 2002).

There is ample evidence to support gender differences in relation to etiopathogenesis and prognosis of CVD (O'Neil et al., 2018; Okunrintemi et al., 2018; Regitz-Zagrosek & Kararigas, 2017; Vaccarino et al., 2016). Recent studies have explored the role of gonadal steroids and sex hormones in gender differences in cardiovascular risk factors and CVD (Falkner, Sherif, Sumner, & Kushner, 1999;

Gyllenborg et al., 2001; Pugeat et al., 1995; Rexrode et al., 2003; Shakir et al., 2007). Using the data from the Women's Health Study, Rexrode and colleagues (2003) explored the relationship between sex hormone levels and risk for cardiovascular events in postmenopausal women. Two hundred women with CVD were matched with controls who were free of CVD at intake. The participants were matched based on age, smoking status, and post-menopausal hormone therapy use. The researchers measured sex hormone binding globulin (SHBG), estradiol, and testosterone. They then calculated free androgen index (FAI), free estradiol index (FEI), and their ratio (FAI/FEI). The results were stratified based on hormone therapy use, and findings suggested that non-hormone-therapy users had significantly higher levels of androgen profiles compared to controls. Additionally, postmenopausal women who did not use hormone therapy had lower SHBG and higher FAI levels. There was no relationship between estradiol levels and CVD risk in either of the groups (Rexrode et al., 2003).

In another study of gender and racial differences in cardiovascular disease among veterans, Goldstein and colleagues (2014) found that women had higher low-density lipoproteins than their male counterparts. With regard to coronary heart disease (CHD), studies have indicated that men are consistently disproportionately affected compared to women and that while CHD prevalence increases from 45 years of age among men, prevalence among women remains stable until 55 years of age (Gosswald, Schienkiewitz, Nowossadeck, & Busch, 2013). Studies have also suggested differences in symptom presentation and how patients who present with cardiovascular-related symptoms are managed by healthcare providers. In general, women are more likely to present with atypical symptoms, to be misdiagnosed, and for their conditions to be

poorly managed, which results in higher post-hospitalization mortality rates (Regitz-Zagrosek & Kararigas, 2017; Vaccarino & Bremner, 2017; Vaccarino et al., 2016). For example, in a study that explored gender differences in relative survival, treatments, and excess mortality after acute MI, researchers found that women were more likely to experience atypical symptoms of acute MI, typically had more comorbidities, and were less likely to receive approved treatment guideline procedures and therapies such as reperfusion and revascularization (Gosswald et al., 2013). Women were also less likely to be discharged from the hospital without guideline-indicated pharmacological therapies, which resulted in higher mortality rates after discharge among women (Goldstein et al., 2014; Gosswald et al., 2013). In relation to congestive heart failure (CHF), women were more likely to present with tiredness and exhaustion, which were more likely to be misinterpreted as signs of depression instead of CHF, thus leading to mismanagement (Regitz-Zagrosek et al., 2010). Evidence has also shown that women who present to primary care physicians with symptoms of paroxysmal supraventricular tachycardia (PSVT) are also likely to experience delayed referral for ablation. In addition, they are more likely to be misdiagnosed with panic attacks, stress, anxiety, or depression, which leads to mismanagement (Carnlof, Iwarzon, Jensen-Urstad, Gadler, & Insulander, 2017).

In relation to race/ethnicity, evidence has suggested that racial/ethnic disparities in cardiovascular risk factors, morbidity, and mortality continue to persist despite a general declining trend across all ethnicities (Benjamin et al., 2018; Hozawa, Folsom, Sharrett, & Chambless, 2007). Blacks are more likely to be diagnosed with CVD and experience higher mortality rates compared to their White counterparts (Benjamin et al.,

2019; Rosamond et al., 2012). The Black population is also disproportionately affected by traditional cardiovascular risk factors such as hypertension, diabetes mellitus, obesity, and atherosclerosis (Carnethon et al., 2017) and their related complications such as stroke, ischemic heart disease, and incident acute coronary heart disease events (Bibbins-Domingo et al., 2009; Carnethon et al., 2017; Safford et al., 2012). In a study that assessed racial/ethnic differences in the prevalence, treatment, and control of cardiovascular disease among US adults, non-Hispanic Blacks were found to have a higher prevalence in cardiovascular disease, treatment eligibility, and unmet treatment goals. In addition, Mexican Americans and participants who identified themselves as "other races/minorities" were less likely to receive cardiovascular disease treatment even when they had indications for treatment (Al Kibria, 2019).

Empirical evidence has suggested that the Black race has a higher prevalence and morbidity of atherosclerotic diseases. For example, the Atherosclerosis Risk in Communities (ARIC) study, which prospectively explored the natural history of atherosclerotic disease in four communities across the United States, showed a general decline in the rate of CHD among all participants. However, the decline among Blacks was less than that of their White counterparts. Among men, the decline in Blacks was half (-3.2%/year) compared to that of Whites (-6.5%/year). Among women, Blacks had a decline of -4.0% per year compared to -5.2% per year among Whites (Rosamond et al., 2012). These findings highlight the importance of more research on possible cardiovascular risk factors that may be unique to Blacks.

Research has shown that members of the Black race experience higher rates of cardiovascular fatalities compared to other races. According to results of the Reasons

for Geographic and Racial Differences in Stroke (REGARDS) study that examined the association of race and risk of incident acute coronary heart disease (CHD) events, Black men had higher incidence of fatal CHD at 4.0 (95% CI [2.9, 5.3]) compared to 1.9 (95% CI [1.4, 2.6]) for White men. Black men also had lower incidence of nonfatal CHD at 4.9 (95% CI [3.8, 6.2]) compared to 6.2 (95% CI [5.2, 7.4]) for White men. Even though the incidence rates per 1,000 person-years among women were lower than that of men, the incidence rates among Black women in particular were higher than that of White women at 5.0 (95% CI [4.2, 6.1]) and 3.4 (95% CI [2.8, 4.2]), respectively (Safford et al., 2012). In a recent analysis of data obtained from the Coronary Artery Risk Development in Young Adults (CARDIA) study, Yano et al. (2017) assessed the association between blood pressure and cardiovascular outcomes based on race. The findings of that study indicated that Blacks were twice as likely to experience premature cardiac events compared to Whites. Among young adults with a mean age of 25 years, systolic blood pressure was the strongest indicator of premature cardiovascular disease risk in Blacks, while among Whites, diastolic blood pressure was a better indicator for premature CVD. Among middle-age adults with a mean age of 40 years old, diastolic blood pressure was a better indicator of CVD in both races (Yano et al., 2017). In another secondary data analysis of the CARDIA study that examined racial disparities and predictors of heart-failure-related deaths and hospitalizations, Bibbins-Domingo et al. (2009) found that among young adults under 50 years old, Blacks had significantly higher heart-failure incidence, with 26 out of the 27 incidents incurred in the study being Black. In a more recent Multi-Ethnic Study of Atherosclerosis (MESA), using 5,004 participants, Akintoye and colleagues (2018) examined the racial/ethnic disparities in

the relationship between incident cardiovascular disease and Left ventricular mass index (LVMI). Among the 360 CVD events that occurred over an average of 10.2 years of follow up, LVMI was most predictive of future CVD events in Chinese (Hazard Ratio [HR] per 10-unit increase in LVMI: 1.7, 95% CI [1.1, 2.8]) and Hispanics (HR per 10-unit increase in LVMI: 1.9, 95% CI [1.5, 2.2]). The predictive utility of LVMI was lowest among non-Hispanic Whites, with HR per 10-unit increase in LVMI being 1.3 (95% CI [1.1, 1.5]; (Akintoye et al., 2018).

A more recent study explored the additive or multiplicative effect of race and gender in the cardiovascular risk factors disparity. Using data from the 2013-2014 New York City Health and Nutrition Examination Survey (NYC HANES), Kanchi et al. (2018) analyzed data from 1,527 male and female participants with multiple racial/ethnic backgrounds. Cardiovascular risk factors examined in the study included obesity, hypertension, hypercholesterolemia, smoking, and diabetes. In relation to gender, the study found women to be less likely to have cardiovascular risk factors compared to men. Specifically, women had lower prevalence of hypertension ($p < .040$), lower triglycerides ($p < .001$), higher HDL ($p < .001$), and were more likely to be nonsmokers and healthier eaters ($p < .05$). Regarding race/ethnicity, this benefit only persisted among non-Latino White women. Non-Latino Black women were more likely to be obese, hypertensive, and diabetic compared to non-Latino White men or women and non-Latino Black men ($p < .05$). Non-Latino Black women were also more likely to have elevated total cholesterol compared to non-Latino Black men (184.4 vs 170.5 mg/dL, $p = .010$), making them the most disproportionately affected gender and race (Kanchi et al., 2018).

Contrary to the above reviews, which depict a consistent negative relationship between Blacks and cardiovascular risk factors and disease in almost every metric, a few studies have shown that even though Blacks bear the greatest burden of HTN and its sequelae, they are also more likely to be aware of their HTN diagnosis and to be on treatment for the same (Cutler et al., 2008; Ostchega, Dillon, Hughes, Carroll, & Yoon, 2007; Yoon et al., 2015).

In summary, there is ample evidence to indicate that age, gender, and race/ethnicity do play a role in the development of CVD and its outcomes. Despite great efforts and progress made towards the fight against CVD, there remains glaring disparities in cardiovascular disease awareness, prevalence, diagnosis, treatment, morbidity, and mortality based on age, gender, and race/ethnicity. The elderly and Blacks appear to be the most disparately affected by CVD, making research that focuses on potential modifiable risk factors that drive these disparities among Blacks and the elderly of utmost public health importance.

Relationship Between Income, Education, and Cardiovascular Health

Socioeconomic status has been recognized as a risk factor in the development of CVD (Albus et al., 2019; Davis, Gebreab, Quarells, & Gibbons, 2014; Harper, Lynch, & Smith, 2011; Karlamangla, Merkin, Crimmins, & Seeman, 2010). Variables that have been typically used to measure an individual's socioeconomic status include level of education, income status, occupation, and housing condition, among others (Danelia, 2006; Davis et al., 2014; Di Chiara et al., 2015; Harper et al., 2011; Metcalf, Scragg, & Davis, 2007; Reddy et al., 2007; Svedberg, Nygren, Staland-Nyman, & Nyholm, 2016; Winkleby, Jatulis, Frank, & Fortmann, 1992). However, research has suggested that

level of education is the strongest predictor of an individual's socioeconomic status (Metcalf et al., 2007; Svedberg et al., 2016; Winkleby et al., 1992). Other studies have highlighted the importance of using more than one measure of socioeconomic status in a study to reduce the risk of cohort effects that could be introduced due to the increasing number of individuals who obtain a high-school education. The inability of education to unmask individual's circumstances, such as their salary or whether or not they have an income, has also been raised as a potential disadvantage of using education as the only measure (Winkleby et al., 1992).

Nearly every epidemiological study that has examined the relationship between education level and cardiovascular risk factors and disease has shown a higher risk association with low education level (Danelia, 2006; Di Chiara et al., 2015; Reddy et al., 2007; Winkleby et al., 1992). For example, Di Chiara and colleagues (2015) evaluated the link between level of education and global cardiovascular risk in a southern Italian urban population. They engaged 433 men and women who were 18 years or older. Education status was categorized as low education, which included participants who had less than 10 years of education, or medium-high education, which included participants who had 10 to 15 years of education. Global cardiovascular risk as well as cardiometabolic comorbidities, such as obesity, visceral obesity, diabetes, dyslipidemia, metabolic syndrome, microalbuminuria, and left ventricular hypertrophy were analyzed. Their findings indicated that participants in the low education group were more likely to be obese, hypertensive, have metabolic syndrome, microalbuminuria, and greater global cardiovascular risk. Moreover, participants' education level was independently associated with global cardiovascular risk. In another study that examined the predictive

ability of level of education on risk factors for coronary heart disease in Indians, Reddy and colleagues (2007) found a significantly higher prevalence of tobacco use and hypertension (56.6% and 33.8%, respectively) in the low-education group in comparison to the high-education group (12.5% and 22.7%, respectively; $p < .001$). However, participants in the high-education group had higher prevalence for dyslipidemia at 27.1% compared to 16.9% in the low-education group ($p < .01$; (Reddy et al., 2007). In contrast, in a study that examined the association between level of education and coronary artery disease in Georgia, investigators found no association between level of education and coronary artery disease. They concluded that lack of association was due to the fact that Georgia was in a transition period at the time of the study and predicted a negative correlation between education and CHD in the future (Danelia, 2006).

There has been ample evidence that supports a negative association between low income and cardiovascular mortality and morbidity (Davis et al., 2014; Harper et al., 2011; Metcalf et al., 2007; Winkleby et al., 1992). Generally, a person's level of income determines their spending power. That is, the kind of housing, diet, and medical care they can afford, which in turn influence their health status (Winkleby et al., 1992). Empirical evidence has shown that cardiovascular disease burden and risk factors are greater among high- and middle-income persons, regions, and countries compared to people, regions, and countries of low income (Yan, Li, Yin, Wang, & Bo, 2017; Yusuf et al., 2014). For example, in an international study with 156,424 participants from three high-income countries, 10 middle-income countries, and four low-income countries, Yusuf and colleagues (2014) used a prospective cohort approach to assess

participants' cardiovascular risk using the INTERHEART risk score, a validated score for quantifying risk-factor burden without the use of laboratory testing (with higher scores indicating greater risk-factor burden) over a 4.1-year period. Their findings indicated that the high-income countries had the highest mean INTERHEART risk score. Middle-income countries and low-income countries had intermediate and lowest mean INTERHEART risk scores, respectively. Even though INTERHEART risk score was lowest in the low-income countries, most major cardiovascular events, including death from cardiovascular causes, myocardial infarction, stroke, or heart failure, occurred in low-income countries, underscoring the impacts of low income on cardiovascular events and outcomes. Furthermore, high-income countries recorded the highest number of participants who used preventive medicine and revascularization procedures compared to middle- and low-income countries (Yusuf et al., 2014).

Relationship Between Smoking and Cardiovascular Disease

Tobacco smoking has been recognized as a modifiable risk factor for a wide range of chronic diseases including multiple types of cancers, respiratory diseases, reproductive diseases, and cardiovascular disease (Lim et al., 2012). According to a 2014 report by the Surgeon General, smoking is the single leading cause of preventable deaths, chronic diseases, and economic losses in the US (Warren, Alberg, Kraft, & Cummings, 2014). A 2008 Center for Disease Control and Prevention's weekly report on deaths attributable to smoking and years of potential life lost indicated that cigarette smoking and secondhand smoke were responsible for 443,000 deaths and 5.1 million years of potential life lost every year between 2000 and 2004 ("State-specific smoking-attributable mortality and years of potential life lost--US, 2000-2004," 2009).

To understand the relationship between tobacco smoke and cardiovascular disease, it is important to first understand the pathophysiology of cigarette smoke and how it influences and affects the cardiovascular system and health. Cigarette smoke contains approximately 4,000 chemicals in its natural state (Csordas & Bernhard, 2013; Messner & Bernhard, 2014). Once inhaled into the body, these chemicals are metabolized into highly complex compounds by the human detoxification systems, such as the cytochrome P450 (Csordas & Bernhard, 2013). These compounds then interact with an individual's genetics and environment to inform the onset and progression of cardiovascular disease (Messner & Bernhard, 2014). More specifically, tobacco smoke reduces the bioavailability of nitric oxide in the body, thereby inducing vascular (endothelial) dysfunction, particularly flow mediated dilatation (FMD). This dysfunction causes an increase in platelets and macrophages adherence to the endothelium, thus creating a procoagulant and inflammatory milieu (Messner & Bernhard, 2014). In a study that examined the effect of smoke cessation on brachial artery FMD, Johnson and colleagues (2010) found a 1% (6.2 +/- 4.4% to 7.2 +/- 4.2%) increase in FMD after participants quit smoking for one year. In another similar study, FMD was strongly associated with pack years smoked and smoke cessation resulted in reversal of endothelial damage (Zeiher, Schachinger, & Minners, 1995).

With regard to cardiovascular disease, smoking has been linked to increased risk for incident atrial fibrillation (Chamberlain et al., 2011; Zhu, Yuan, Shen, Wan, & Hong, 2016), to higher prevalence of coronary atherosclerotic plaque (Cheezum et al., 2017; Hou et al., 2019), to increased risk for non-obstructive and obstructive coronary artery disease (Cheezum et al., 2017), and to being inversely associated with HTN (Kim, Han,

Kang, Kim, & Kang, 2017). In addition, studies have found secondhand tobacco smoking to be a significant predictor of coronary artery calcification in participants who had no history of smoking (Yankelevitz et al., 2013). Secondhand tobacco smoking has also been linked to coronary atherosclerosis in individuals who were never smokers (Yankelevitz et al., 2017).

Relationship Between Physical Activity and Health

Physical activity references movements of the skeletal muscles that cause energy expenditure (Caspersen, Powell, & Christenson, 1985). The literature is replete with evidence that support a positive association between physical activity and cardiovascular health and the negative effects of physical inactivity on health (Caspersen et al., 1985; Chandrashekhar & Anand, 1991; Kyu et al., 2016; Williams, 2001). Consistent physical activity over a long period of time has been linked to multiple positive health effects on the cardiovascular system. These include enhanced cardiopulmonary fitness, physiologic remodeling of the heart including adaptive molecular and cellular reprogramming, and cardioprotective effects (Makar & Siabrenko, 2018). People who engage in physical activity over long periods of time have also been shown to have significantly improved myocardium contractility, central and peripheral blood circulation, increased cardiac output, and myocardial mass, which all contribute to cardiovascular risk reduction (Adams, Reich, Uhlemann, & Niebauer, 2017).

In contrast to the beneficial effects of physical activity, a sedentary lifestyle has been associated with negative cardiovascular outcomes. For example, in a study that examined the impacts of sitting and viewing television in the United States, a decrease in sedentary lifestyle was associated with increase in life expectancy. More precisely,

people who reduced their excessive sitting time to less than 3 hours per day and less than 2 hours per day were predicted to gain 2 and 1.38 years of life expectancy, respectively (Katzmarzyk & Lee, 2012). In a more recent study that examined the association between sedentary lifestyle and blood pressure in 31 patients with multiple sclerosis (MS), sitting time was measured using the International Physical Activity Questionnaire and blood pressure was measured using automated oscillometric monitor. Participants were put to rest in a supine position in a quiet room for 10 minutes prior to their BP and mean arterial pressure being taken. Their findings revealed a significant association between sitting time and systolic BP (r = .365, p = .044, 95% CI [0.013, 0.636]), diastolic BP (r = .382, p = .034, 95% CI [0.032, 0.648]), and mean arterial pressure (r = .425, p = .017, 95% CI [0.084, 0.677]) in patients with MS but not in controls (p > .05), underscoring the ill effects of physical inactivity on cardiovascular health (Hubbard, Motl, & Fernhall, 2018).

The WHO has recommended a minimum of 600 metabolic equivalent (MET) minutes of physical activity per week for an individual to experience the health benefits associated with such (Bull, Maslin, & Armstrong, 2009). The MET is a measure of resting energy expenditure. That is, the amount of oxygen that an individual consumes while sitting at rest; this amount is approximated to be 3.5 ml of oxygen per kilogram body weight per minute (Jette, Sidney, & Blumchen, 1990). This measure (MET) is typically considered to be independent of body weight and thus is relatively constant in all individuals (Franklin et al., 2018). Other studies have also suggested that there is a specific amount (dose) of physical activity that is required to experience its associated health benefits. For example, Kyu and colleagues (2016) conducted a systematic review

including 43 articles on ischemic heart disease and 26 articles on ischemic stroke to quantify the dose-response association between total physical activity and risk for ischemic heart disease and ischemic stroke events, among other diseases. They found a significant association between higher levels of total physical activity and lower risk for ischemic heart disease and ischemic stroke. However, contrary to the WHO's recommendation of 600 MET minutes per week, their study showed that highest health benefits were achieved at total physical activity levels between 3000 and 4000 MET minutes per week (Kyu et al., 2016). In a recent systematic review of one systematic review and 17 meta-analyses, Pescatello and colleagues (2019) examined and updated evidence on the relationship between physical activity and hypertension. Their study findings revealed a strong inverse dose-response relationship between physical activity and normal blood pressure. Among participants with hypertension, physical activity was found to reduce risk for progression of CVD. Furthermore, physical activity was also found to reduce blood pressure among prehypertensive and hypertensive participants (Pescatello et al., 2019). In summary, empirical evidence has shown that engaging in physical activity lowers the risk of CVD in all individuals and that sedentary lifestyle increases risk for CVD.

Relationship Between Stress and Cardiovascular Health

Selye defined stress as a response to pressure or stain exerted upon the body (Szabo, 1998; Szabo, Tache, & Somogyi, 2012). The exact mechanisms through which stress adversely affects cardiovascular health remains unclear. However, empirical evidence suggest multiple possibilities including increased activity of the hypothalamic pituitary axis (Bao, Meynen, & Swaab, 2008; Kageyama & Suda, 2009, 2010;

Kageyama, Tamasawa, & Suda, 2011; Smith & Vale, 2006; Tsigos & Chrousos, 2002), increased sympathetic outflow (Dampney, Michelini, Li, & Pan, 2018), and altered behaviors causing insulin resistance and consequently central obesity (Hewagalamulage, Lee, Clarke, & Henry, 2016; Raikkonen, Keltikangas-Jarvinen, Adlercreutz, & Hautanen, 1996; Tsigos & Chrousos, 2002). The term “stress” often has a negative connotation in most settings around the world. Nonetheless, this perception is deceptive as the activation of stress response to acute stressor and its judicious termination after a stressful event is vital to maintenance of homeostasis (Charmandari, Tsigos, & Chrousos, 2005). In contrast, traumatic and chronic stress can be detrimental and has been associated with negative psychological and physiological outcomes. With regards to psychological outcomes, stress has been linked to neuropsychiatric disorders such as post-traumatic stress disorder (PTSD), depression, anxiety and somatoform disorders (Herzig et al., 2012; Smith & Vale, 2006). In relation to physiological outcomes, stress has been linked to cardiometabolic diseases such as Diabetes Mellitus, (Domingueti et al., 2016; Rochette, Zeller, Cottin, & Vergely, 2014) and cardiovascular disease (Blom et al., 2014; Gianaros & Jennings, 2018; Gianaros et al., 2008; Ginty, Kraynak, Fisher, & Gianaros, 2017; Khanna, Kan, Failinger, Jain, & Finkel, 2006; Kivimaki & Kawachi, 2015; Kivimaki & Steptoe, 2018; Lagraauw, Kuiper, & Bot, 2015; Li, Loerbroks, Bosma, & Angerer, 2016; Li, Ho, & Yew, 2019; O'Keefe, Poston, Haddock, Moe, & Harris, 2004; Pickering, 2007; Wirtz & von Kanel, 2017; Zhang, Liu, Li, & Cai, 2012).

With regard to cardiovascular disease, stress has been linked to the development of atherosclerosis and subclinical disease in otherwise healthy individuals

(Kivimaki & Kawachi, 2015; Kivimaki & Steptoe, 2018; Lagraauw et al., 2015; Li et al., 2016; Richardson et al., 2012). Stress has also been identified as a trigger to acute cardiac or cerebrovascular events (Fransson et al., 2015; Hagstrom et al., 2018). Among individuals diagnosed with CVD, stress has been shown to worsen prognosis by impairing recovery, hastening progression of disease and contributing to cardiovascular mortality (Richardson et al., 2012; Steptoe & Kivimaki, 2013).

Richardson and colleagues (2012) conducted a meta analytic review to examine the association between perceived stress and incident coronary heart disease. Their review included 6 large prospective cohort studies with 118,696 participants conducted for over a ten-year period on average. Their analysis associated 27% increase in risk for incident coronary heart disease to high perceived stress. In a more recent study, Kivimaki and Steptoe (2018) found that chronic stress increased the odds of developing coronary heart disease.

In relation to HTN, a plethora of studies have examined the relationship between stress and elevation in blood pressure (Cuffee, Ogedegbe, Williams, Ogedegbe, & Schoenthaler, 2014; Liu, Li, Li, & Khan, 2017; Spruill, 2010). In a recent meta- analysis of the association between chronic psychosocial stress and hypertension, Liu et al. (2017) found that psychosocial stress was associated with increased risk of hypertension (OR = 2.40, 95% CI [1.65, 3.49]). In addition, their study showed a higher incidence of psychosocial stress among hypertensive participants compared to their normotensive counterparts (OR = 2.69, 95% CI [2.32, 3.11]).

With regard to ischemic events, Hagstrom et al. (2018) conducted a prospective study to examine the relationship between psychosocial stress and ischemic events.

Their study showed that psychosocial stress expressed as financial stress, depressive symptoms and loss of interest were associated with increased risk for cardiovascular death, nonfatal MI, and stroke. In another study that examined the association between ischemic stroke and work related stress, Fransson et al. (2015) conducted a meta-analysis involving 196, 380 participants from 14 European countries who were followed for an average of 9.2 years. There were 2023 first time strokes recorded during the study period with a 1.24 (95% CI [1.05, 1.47]) sex and age adjusted hazard ratio for job strain compared to no job strain for ischemic stroke, 1.01 (95% CI [0.75, 1.36]) for hemorrhagic stroke, and 1.09 (95% CI [0.94 1.26]) for overall stroke.

Psychosocial stress has also been associated with atrial fibrillation (Fransson et al., 2015; Kivimaki et al., 2017; Toren, Schioler, Soderberg, Giang, & Rosengren, 2015). For instance, in a prospective multi-cohort study of 85,494 working individuals with no history of atrial fibrillation followed over 10 years, Kivimaki et al. (2017) investigated the risk of atrial fibrillation among participants who worked long hours (>55hr/week) compared to workers who worked standard hours (35-40 hours/week). There were 1061 new cases of atrial fibrillation over the 10-year period. The study found that participants who worked long hours had a 1.4-fold increased risk of atrial fibrillation compared to those who worked standard hours (hazard ratio = 1.42, 95% CI [1.13, 1.80], p = .003). The risk remained after adjustment for confounders and after excluding participants with coronary artery disease or stroke at baseline or during follow up (N = 2006, hazard ratio = 1.36, 95% CI [1.05, 1.76], p = .0180).

In summary, the literature supports an association between stress and cardiovascular disease. Stress has been linked to development of atherosclerosis,

subclinical disease, acute cardiovascular events, disease progression and mortality. Furthermore, this association appears to persist even after adjusting for potential confounders.

Relationship Between Perceived Discrimination and Health

Williams and Mohammed (2009) defined perceived discrimination as the belief that an individual has been unfairly treated by another individual or an institution and that this unfair treatment was solely due to personal attributes of the victim such as, age, race, gender, religion, socioeconomic status, sexual orientation, or weight. Empirical evidence is replete with studies supporting an association between perceived discrimination and poor health in general (Chilunga et al., 2019; Lee & Ahn, 2011; Pascoe & Smart Richman, 2009; Pieterse, Todd, Neville, & Carter, 2012; Williams & Mohammed, 2009) and cardiovascular disease in particular (Everson-Rose et al., 2015; Lockwood, Marsland, Matthews, & Gianaros, 2018). Perceived discrimination has been associated with both poorer mental health (Lee & Ahn, 2011; Pascoe & Smart Richman, 2009; Pieterse et al., 2012; Schmitt, Branscombe, Postmes, & Garcia, 2014) and physical health and their outcomes (Everson-Rose et al., 2015; Lockwood et al., 2018; Pieterse et al., 2012; Williams & Mohammed, 2009). Furthermore, recent evidence has suggested that increased levels of perceived discrimination are associated with an increase in participation in high-risk health related behaviors and a decrease in participation in healthy behaviors (Pascoe & Smart Richman, 2009).

In relation to mental health, research has suggested that higher levels of perceived discrimination have harmful effects on quality of life and psychological well-being (Mays & Cochran, 2001; Pieterse et al., 2012; Schmitt et al., 2014). More

precisely, increased levels of perceived discrimination have been associated with higher odds of depression, anxiety, substance abuse, psychological distress, and psychiatric morbidity (Britt-Spells, Slebodnik, Sands, & Rollock, 2018; Kohlbrenner, Deuba, Karki, & Marrone, 2016; Mays & Cochran, 2001). For example, in a study that sought to examine the role of perceived discrimination in generating risk for mental illness among lesbian, gay, and bisexual adults in the United States, Mays and Cochran (2001) assessed a 1-year prevalence of depression, anxiety, and substance dependence. The study also assessed for psychological distress and overall mental health status of participants at the time of study. Their findings indicated a positive association between perceived discrimination and both quality of life and all psychiatric morbidity indicators explored in the study. In another study that examined the predictability of perceived discrimination on suicidal ideation among sexual and gender minorities in Nepal, perceived discrimination was found to be an independent risk factor for suicidal ideation (Kohlbrenner et al., 2016).

With regard to physical health, higher levels of perceived discrimination have been associated with cardiovascular risk factors such as elevated blood pressure (Brondolo et al., 2008; Dolezsar, McGrath, Herzig, & Miller, 2014), increased carotid-intima thickness (Brondolo et al., 2008), and heart rate variability (Pascoe & Smart Richman, 2009). Perceived discrimination has also been associated with multiple health conditions, including poor diabetes mellitus control and outcomes in Blacks (Achuko, Walker, Campbell, Dawson, & Egede, 2016; Dawson, Walker, Campbell, & Egede, 2016; Williams, Clay, Ovalle, Atkinson, & Crowe, 2018), higher prevalence and poorly controlled asthma among Blacks (Thakur et al., 2017), and cardiovascular disease and

outcomes (Everson-Rose et al., 2015; Lockwood et al., 2018). Studies that have examined the relationship between cardiovascular health outcomes and perceived discrimination are scarce, and the two that were reviewed revealed conflicting findings. More precisely, Everson-Rose et al. (2015) conducted a multiethnic cohort study to examine the relationship between perceived discrimination and incident cardiovascular events. The participants consisted of 6,058 adults between the ages of 45 and 84 and were free of cardiovascular diagnosis at intake. They found a positive association between discrimination and CVD risk even after adjusting for behavioral factors. In contrast to Everson-Rose and colleagues' (2015) earlier research, in a metanalysis that focused on the association between perceived discrimination and cardiovascular outcomes, Dunlay et al. (2017) found no association between these two variables. However, unlike the earlier study, Dunlay and colleagues conducted a meta-analysis and, by design, were limited by the methodology itself. That is, the meta-analysis method did not allow for variable adjustment or manipulation (Everson-Rose et al., 2015). These conflicting findings underscore the need for further investigation into the relationship between perceived discrimination and cardiovascular outcomes or events to better inform cardiovascular health policy and practice.

Studies that have assessed the relationship between perceived discrimination and health-related behaviors have focused on both healthy and high-risk health-related behaviors. The high-risk health-related behaviors reviewed included smoking, substance use and abuse, alcohol use and abuse, missing doctor appointments, and risky sexual behaviors (Assari, Mistry, Lee, Caldwell, & Zimmerman, 2019; Bennett, Wolin, Robinson, Fowler, & Edwards, 2005; Halim, Yoshikawa, & Amodio, 2013;

Sanchez, Whittaker, Hamilton, & Zayas, 2016; Visser, Ikram, Derks, Snijder, & Kunst, 2017; Wetter et al., 2004). Regarding healthy behaviors, physical activity, medication adherence, sleep, and diet were reviewed. Generally, evidence has suggested that individuals who believed that they were treated unfairly for whatever reason had a higher risk for cigarette smoking (Bennett et al., 2005; Visser et al., 2017; Wetter et al., 2004), alcohol use and abuse (Visser et al., 2017), and substance use and abuse (Assari et al., 2019), and were more likely to miss an appointment or visit the physician more frequently (Halim, Moy, & Yoshikawa, 2017; Halim et al., 2013). Evidence has also suggested that individuals who experience discriminatory acts or behaviors are more likely to engage in risky sexual behaviors (Sanchez, Whittaker, & Hamilton, 2016; Sanchez, Whittaker, Hamilton, et al., 2016).

With regard to healthy behaviors, studies have suggested that individuals who experience discrimination are less likely to engage in healthy behaviors (Jackson & Steptoe, 2017; Turan et al., 2017). For example, in a longitudinal study of 5,480 English men and women that examined the relationship between perceived weight discrimination and physical activity, Jackson and Steptoe (2017) found that almost 60% of the odds of being inactive and 30% of the odds of not participating in moderate or vigorous exercise at least once a week were related to perceived weight discrimination by study participants. In another cross-sectional analysis of the relationship between perceived discrimination and antiretroviral therapy (ART) adherence among women living with HIV, researchers found perceived discrimination to be indirectly related to suboptimal ART adherence via internalization of HIV-related stigma, which led to depressive symptoms and hence nonadherence (Turan et al., 2017).

In summary, the evidence from the literature reviewed shows that age, gender, race, physical activity, smoking, income, education, and stress play a role in the development of CVD, morbidity, and mortality. The literature reviewed also identified perceived discrimination as a potential modifiable cardiovascular risk factor. Perceived discrimination is thought to affect CVD via direct effect, psychological and physiological stress response pathways, as well as by influencing the health behaviors of an individual. However, there remains a gap in knowledge regarding the association between perceived discrimination and cardiovascular events, warranting this research study.

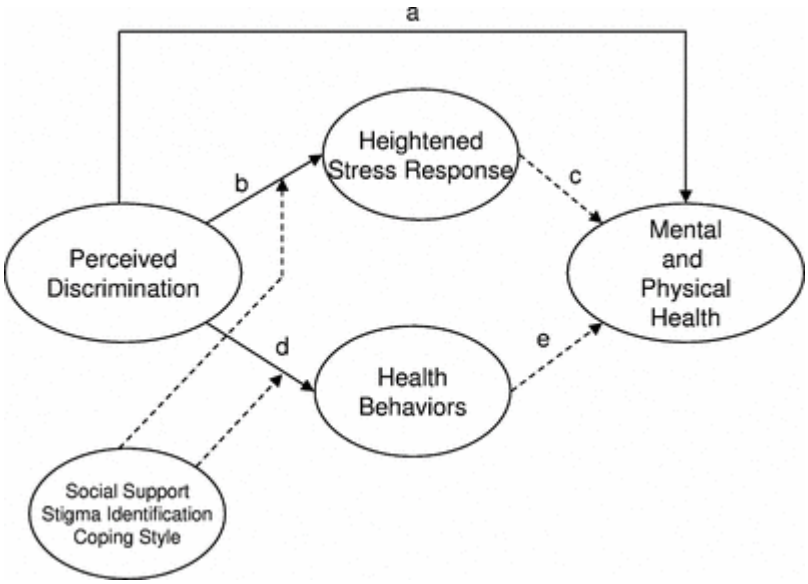

Figure 1. Pathways by which perceived discrimination influences health outcomes. Solid lines indicate pathways analyzed by Pascoe and Richman (2009); dashed lines represent pathways hypothesized by past research.

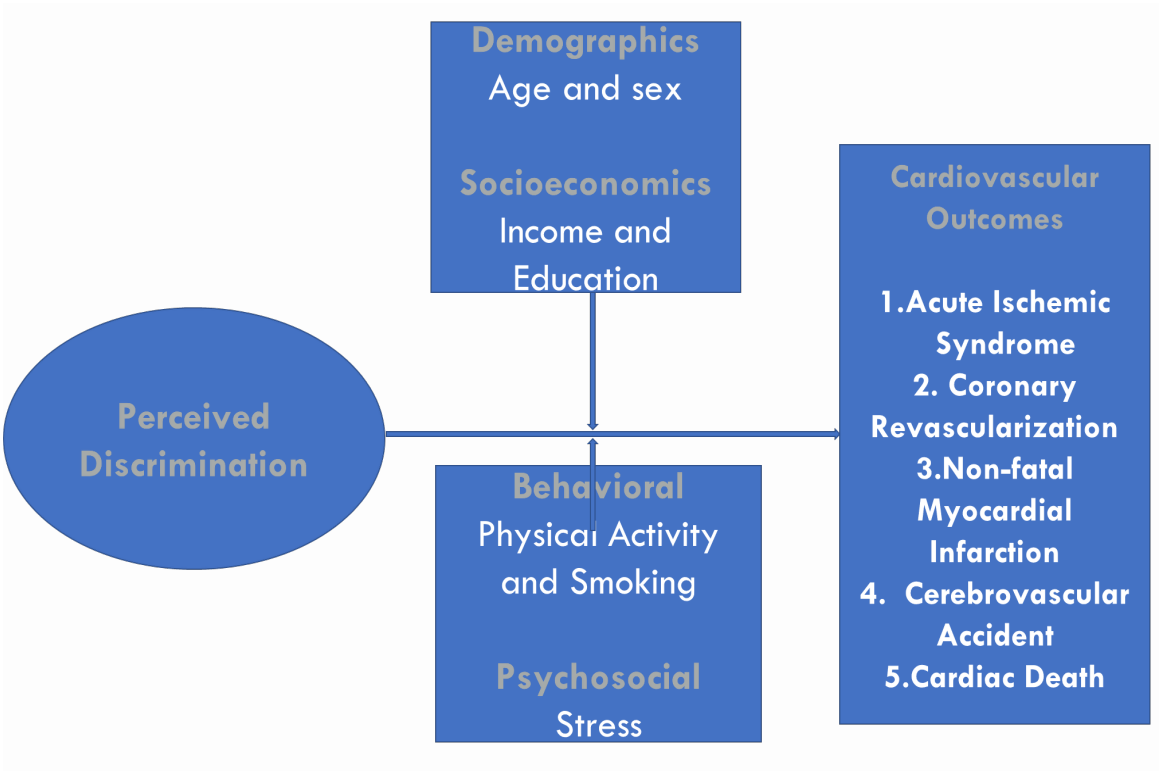

Figure 2. Diagram showing direct effect of perceived discrimination on cardiovascular outcomes and potential moderators of that relationship.

CHAPTER THREE:

METHODS

Introduction

In Chapter 3, the research methodology for the community-based prospective cohort study known as the Heart Strategies Concentrating on Risk Evaluation (Heart SCORE) study as well as the methodology for the secondary data analysis are discussed. This chapter is organized into topics including study design, setting and sample, protection of human subjects and secondary data access and management procedures, data collection methods, and statistical analysis. The aims of the Heart SCORE study were to address cardiovascular disease by improving risk stratification, identify racial disparities, and evaluate a multi-disciplinary community-based intervention program to decrease CVD risk in high-risk populations (Aiyer et al., 2007; Bambs et al., 2011; Bambs et al., 2013). The purpose of the current secondary data analysis was to examine whether there is a relationship between perceived discrimination and cardiovascular events among Blacks, and to evaluate whether other modifiable risk factors moderate that relationship.

Study Design

The Heart SCORE study is an ongoing community-based prospective cohort study that was initiated in 2003 (Bambs et al., 2013). The current study was a secondary data analysis that was performed to utilize the extensive data available from the Heart SCORE study to evaluate the relationship between perceived discrimination

and cardiovascular events in Blacks. The Heart SCORE dataset used in this study also included information for following of change over time among the study participants within the cohort (Erqou, Echouffo-Tcheugui, Kip, Aiyer, & Reis, 2017).

Study Setting and Sample

Data obtained from The Heart SCORE study data base was used in this secondary data analysis. The Heart SCORE is a prospective cohort study that began in 2003 in Allegheny County, Pennsylvania. Enrollment of participants concluded on October 11, 2006 (Aiyer et al., 2007). The primary and only study site for the Heart SCORE study is the University of Pittsburgh Medical Center (UPMC) Cardiovascular Institute in Pittsburgh, PA (Bambs et al., 2011; Shpilsky et al., 2018). All participants were residents of the greater Pittsburgh area of Pennsylvania at time of entry into the study. The Heart SCORE study data base has almost equal representation of Whites (56%) and Blacks (44%). About 2.6% of the participants are categorized as race other than White or Black (Aiyer et al., 2007).

The data set used in this secondary data analysis included participant records with approximately 13.5 years of information from the beginning of participant enrollment and includes N = 854 records of Black individuals.

Recruitment and Enrollment

The developers of the Heart SCORE study utilized multiple approaches to recruit study participants and included: mailing of recruitment letters to potential participants based on ZIP codes, advertisements, and direct promotion through community organizations such as community centers, community-based blood pressure and lipid screening programs and at places of worship. The investigators set *a priori* recruitment

goal of enrolling 2000 participants with approximately 50% Blacks. To make this possible, investigators made purposeful efforts to recruit traditionally underserved and high-risk communities through partnering with several organizations within the community including: The Urban League of Pittsburgh, Cardiovascular Institute at the University of Pittsburgh, Metro Urban Institute Office of Applied Religion (MUI-OAR) of the Pittsburgh Theological Seminary, and other community based and academic partners. Baseline enrollment into the study commenced on June 16, 2003 and was concluded on October 11, 2006 (Aiyer et al., 2007).

To be eligible for the Heart SCORE study, potential participants had to meet the following criteria;

1. Age between 45 to 75 years.
2. Residence in the greater Pittsburgh metropolitan area.
3. Ability to undergo baseline and annual follow up visits.
4. Absence of known comorbidities expected to limit life expectancy to less than 5 years.

For this secondary data analysis, the exclusion criteria included:

1. Participants who identified themselves as Whites or other than White or Black race.
2. Participants who reported a history of a major cardiac event prior to baseline data collection.
3. Participants who had missing data on independent and dependent variables in the study.

Protection of Human Subjects and Secondary Data Management Procedures

Prior to the initiation of the Heart SCORE study, approval was obtained from the University of Pittsburgh Institutional Review Board (IRB) to ensure protection and privacy of the study participants. Participants were informed about all possible risks associated with the study including required time commitments to participate in the study. Potential participants' questions were addressed, written informed consents were signed by each participant, and a copy of the signed consent was given to each participant.

The investigator for the current secondary data analysis sought and obtained the permission of the Heart SCORE investigators at the University of Pittsburgh to conduct the current secondary data analysis. Dr. Kevin Kip, who is a dissertation committee member of the secondary study, is involved in the Heart SCORE study and provided access to required data for this analysis. The Heart SCORE data are stored in a passcode protected site. Dr. Kevin Kip obtained de-identified data sets that were copied and provided to the Primary Investigator for this secondary data analysis. A separate University of South Florida (USF)IRB approval has been in place for years for secondary analyses of the Heart SCORE data set. In addition, the Primary Investigator for this secondary analysis sought and obtained approval from the USF IRB to conduct this secondary analysis. The original de-identified data set and subsequent analysis files of this secondary` study were stored in password-protected computers of the Primary Investigator. The data set and subsequent analysis files were made available to all committee members on an as-needed basis.

Power Analysis and Sample Size

A series of Cox regressions were modeled to address the aims of this research. An a priori power analysis was performed to determine the required sample size for this study. Power is (1-β), where β is the chance of Type II error (when one rejects the null hypothesis when it is in fact false). At a power of .80, one has an 80% chance of seeing significance that is truly in the data. PASS software (Hintze, 2008) was used in this determination. The largest regression model included one strata variable and 25 predictor variables. The power was computed for the largest regression model with criteria of a two-tailed test, an alpha level of .05, power of .80, and a log hazard ratio of ln (1.25) = 0.223. The hazard ratio of 1.25 was computed by taking the overall hazard rate of the data set (8.4%) then assuming the EDS Level 1 group of higher discrimination will have a hazard of 11%. The EDS Level 1 group of lower discrimination was assumed to have a hazard of 8%. The hazard ratio of .11/.08 = 1.375 and was used for estimation of the sample size. The results indicated that a sample of 410 participants was required to achieve power at 80%. The secondary data that was used in this study contained 854 records. Therefore, even with some attrition due to incomplete records, the sample size remained adequate to perform the analyses.

Data Collection

As previously stated, data collected from the Heart SCORE study was used for this secondary data analysis. The researchers of the Heart SCORE study had collected data on over six hundred variables at the time of this analyses. For the purposes of this secondary data analysis, only data collection methods for the variables of interest to the current study are discussed in the following sections. Table 1 presents the variables of

interest for the current study as well as each variable's level of measurement and coding schema for analysis.

Predictor Variables of Secondary Data Analysis

The role of psychosocial factors in the development of cardiovascular disease is well documented in the literature (Dunlay et al., 2017; Erqou et al., 2017; Everson-Rose et al., 2015; Goosby et al., 2015). The Heart SCORE study investigators made efforts to include several psychosocial factors in their study. In this secondary data analysis, perceived discrimination was the predictor variable. Perceived discrimination was assessed using the everyday discrimination scale (EDS). The EDS is a 10-item scale that was adapted from the original 9-item scale that was used in the Detroit Area study (Williams & Mohammed, 2009; Williams, Yan, Jackson, & Anderson, 1997). For the Heart SCORE study, this scale was adapted to include a second level of assessment. More specifically, the first level of the EDS (EDS-1) included 10 items that were scored to assess the frequency of occurrence of unfair treatment without reference to age, gender and other demographic variables. Precisely, participants were asked how often they felt that the following statements applied to them: 1) You are treated with less courtesy than other people; 2) You are treated with less respect than other people; 3) You receive poorer service than other people at restaurants or stores; 4) People act as if they think you are not smart; 5) People act as if they are afraid of you; 6) People act as if they think you are dishonest; 7) People act as if they are better than you; 8) You or your family members are called names or insulted; 9) You are threatened or harassed; 10) People ignore you or act as if you are not there. Responses from the EDS-1 were rated on a Likert-type scale ranging from often (4) to sometimes (3), to rarely (2), to

never (1). The total were summed up and ranged from 10 to 40 with a higher score indicating a higher frequency of perceived discrimination (Sullivan et al., 2019).The second level of the EDS (EDS-2) included questions used to assess if participants thought that the unfair treatments they experienced were due to race, ethnicity, gender, age, income level, language, religion, body weight, or other physical appearance. The responses of “yes” or “no” for the specific questions within the questionnaire were summed up (Erqou et al., 2017). A higher frequency of “Yes” answers indicated more varied types of discrimination and higher frequency of “No” answers indicated less variation in types of discrimination. The EDS has been shown to have acceptable psychometric properties among Blacks with no significant differences in scores for males and females ($p > .18$). The alpha reliability coefficient was 0.87 with item-correlations that ranged from 0.50 to 0.70 ($M = 0.61$).The split-half reliability was 0.83 ($p < 0.0001$; (Clark, Coleman, & Novak, 2004).

The data set that was used for this secondary data analysis included scores of 0 to 3 for the 10 Likert-type scaled items instead of the range of 1 to 4 noted in the study by Sullivan et al. (2019). Additionally, the data set included records for which one or more of the 10 items were missing values. Therefore, in order to keep with the structure of the models in which a referent score of 0 on the EDS-1 scale would be meaningful, and also to help in the preservation of records for analysis, responses from the EDS were rated on a Likert-type scale ranging from often (3) to sometimes (2), to rarely (1), to never (0). The 10 items were then averaged instead of totaled to account for missing-ness in responses. Thus, the EDS-1 score ranges from 0 – 30 with a higher score indicating a higher frequency of perceived discrimination.

The EDS-1 variable was included in the Cox regression models as a “strata” variable for grouping. The EDS-1 variable was first scored and then each participant’s scores were averaged. The participants’ scores were then divided into two groups. Participants with average score of 0 to 2 were given EDS-1 score of 0 and participants with scores of > 2 were given EDS-1 score of 1. Scores of 0 were associated with lesser discrimination. Scores of 1 were associated with greater discrimination. The Kaplan-Meier curves and Cox regression models reflect a comparison of survival between the two EDS-1 groups.

Outcome Variable of the Secondary Analysis

The outcome variable in this secondary data analysis was time to cardiac event and referred to the number of days from study entry until the time of the event. The researchers of the Heart SCORE study collected data on a variety of cardiac events. Occurrence of cardiac events was assessed during follow up visits using a standard questionnaire. Specifically, the patients were asked; since the last follow up assessment or since study entry, have you experienced any of the following: any inpatient hospitalization, out-patient hospitalization, documented MI, suspected MI, chest pain, diagnostic cardiac catheterization, percutaneous coronary intervention (PCI), coronary bypass surgery (CABG), cerebrovascular accident (stroke), carotid stent or surgery, non-coronary vascular surgery, new onset/diagnosed malignancy. Participants were also asked to include the first date (date, month, year) of occurrence of the events. Participants who reported in-patient hospitalization were also asked to specify if it was due to unstable angina, acute ischemic syndrome, other CVD condition or non-cardiac condition.

For the purposes of this secondary analysis, the status variable of cardiovascular event was a composite outcome variable defined as first occurrence of any of the following: (a) non-fatal myocardial infarction, (b) acute ischemic syndrome, (c) coronary revascularization, (d) cerebrovascular accident, or (e) cardiac death. The number of days from study entry until the time of the cardiac event for each participant was used as the time to cardiac event variable. Participants without a cardiac event, or who were dropped from study for other reasons, were included as censored participants based on their late date of contact in the study.

Status Variable – Cardiac Event

For the purposes of this secondary analysis, the status variable of cardiovascular event was defined as (a) non-fatal myocardial infarction, (b) acute ischemic syndrome, (c) coronary revascularization, (d) cerebrovascular accident, or (e) cardiac death. Table 1 includes the names of the five cardiovascular event variables included in the time to event cardiovascular event outcome, the data set variables that were used to derive the time to event in days. The time to cardiac event variable is dichotomous and coded as 0 = no cardiac event and 1 = cardiac event.

Covariates

Covariates for this secondary analysis were chosen based on prior knowledge of potential confounders of the relationship between perceived discrimination and cardiovascular events. These potential confounders were grouped into four categories of (a) demographic factors, (b) behavioral factors, and (c) socioeconomic factors, and (d) psychosocial factors (Benjamin et al., 2019). For the behavioral variables, smoking and physical activity were included. For socioeconomic variables, education and income

were included. For demographic variables age and sex were included and for psychosocial variables, stress was included. Stress was score derived from the 4-item perceived stress scale (Cohen, Kamarck, & Mermelstein, 1983). These four groups (demographic, socioeconomic factors, behavioral variables, and psychosocial variable) were used to sequentially adjust the models in this study.

Demographic variables of age and sex. The data included in the Heart SCORE Study were obtained via self-report at baseline visit. For the purposes of this secondary analysis, age is reported in years and sex is classified with two groups coded as (a) male = 0 or (b) female = 1. Age and sex were included as covariates in each model. Age is a continuous variable and sex is a dichotomous variable.

Behavioral variables of smoking and physical activity. Smoking status was measured in the Heart SCORE study via self-report of smoking history. Participants were asked about status of smoking and was reported as former, current or never a smoker. Physical activity data were collected through the Lipids Research Clinics (LRC) questionnaire. The LRC questionnaire is a standard questionnaire that has been previously validated for use in population research with a high test-retest reliability of r = 0.85 (Ainsworth, Jacobs, & Leon, 1993). The questionnaire includes seven questions that assess the type and frequency of physical activities that participants engage in at work as well as during leisure time. It provides approximations of amount of physical activity required to achieve ideal cardiovascular health as defined by the American Heart Association (AHA). The AHA defines physical activity for ideal cardiovascular health in adults over 20 years of age as physical activity of moderate intensity for 150 or more minutes per week or 75 minutes or more of vigorously intensive activity or a

combination. For children 12-19 years of age, 60 or more minutes of moderate or vigorous intensity activity every day is considered ideal (Lloyd-Jones et al., 2010). Participants can be classified as active or inactive (Ainsworth et al., 1993; Erqou et al., 2017).

The smoking and physical activity variables were treated as ordinal measurements in this secondary analysis. Smoking was coded as 1 = never smoked, 2 = former smoker, and 3 = current smoker. Physical activity was coded as 1 = sedentary, 2 = mild, 3 = moderate, and 4 = strenuous.

Socioeconomic variables of income and education. Participants in the Heart SCORE study completed a detailed demographic and medical history questionnaire that captured socioeconomic status of the participants. Level of income was assessed through a question that asked for annual income of the participants. Annual income was reported as less than $10, 000, $10,000 to less than $20,000, $20,000 to less than $40,000, $ 40,000 to less than $80,000, and $80,000 or more. Low income was defined as annual income of $20k or less. Education level was assessed through a question that asked participants to state their highest level of school completed and reported as some college or higher or less than college (Bambs et al., 2013). Low educational level was defined as participants who did not complete high school diploma. (Erqou et al., 2017). The income and education variables were treated as ordinal measurements in the analysis. Income represented dollars per year and was coded as 1 = less than 10,000, 2 = 10,000 to less than 20,000, 3 = 20,000 to less than 40,000, 4 = 40,000 to less than 80,000, and 5 = 80,000 or more. Education was coded according to the

highest level completed by a participant as 1 = less than high school, 2 = high school diploma, 3 = some college, 4 = Bachelor's degree, and 5 = advanced degree.

Psychosocial variable of stress. Participants in the Heart SCORE study completed the Perceived Stress Scale 4 (Cohen et al., 1983). The score range of the stress variable is 0 to 16, with higher scores indicative of greater stress.

Statistical Analysis

The *Statistical Packages for the Social Sciences* (SPSS) version 25.0 software was utilized in this secondary data analysis for all descriptive and inferential analyses. Descriptive statistics are reported for all variables. Continuous variables are presented as measures of central tendency and variability. Ordinal and nominal variables are presented as frequencies and percentages. Other descriptive analyses and inferential tests are guided by the aims of the study.

Aim 1. To examine the relationship between perceived discrimination and cardiovascular health events including; non-fatal myocardial infarction, cerebrovascular accident, acute ischemic syndrome, coronary revascularization and cardiac death in Blacks in the Heart SCORE Study.

To examine the relationship between perceived discrimination and cardiovascular events, Cox regression methods were used. Logistic regression and Cox regression are two methods mostly used for risk factors identification and the development of risk factors estimation equations for cardiovascular disease (Knuiman, Vu, & Segal, 1997). In this analysis, the Cox regression was chosen for various reasons. First, unlike a logistic regression model, a Cox proportional regression model allows for simultaneous consideration of multiple risk factors (covariates) to determine

survival time and also provides individual effects known as hazard ratios (McArtor et al., 2017). Second, unlike other research designs, epidemiological studies such as the Heart SCORE study are typically prone to censoring due to the extended amount of time (years) that it takes to observe events in such studies. Censoring occurs when there is incomplete information about survival time of some participants in a study (Leung, Elashoff, & Afifi, 1997). Typically, this happens when participants survive the entire study or drop out of the study before the end of the study observation time without experiencing the event of interest (Jackson et al., 2014). Cox regression includes censoring as an element of the survival analysis making it the most suitable statistical method for this analysis (Gong & Schaubel, 2018).

Despite the advantages of the Cox proportional hazard model discussed above, this statistical analysis method has some assumptions which must be considered and include:

1. Independence between individual subjects.
2. Multiplicative relationship between hazard and predictors.
3. The hazard ratio is constant (i.e. proportional) over the time tested.
4. The hazard ratio represents a 1 unit of change in risk of the outcome of interest holding all other predictors constant (Moolgavkar, Chang, Watson, & Lau, 2018).

To address Aim 1, cumulative incidence of cardiovascular events of interest including; non-fatal MI, acute ischemic syndrome, coronary revascularization, cerebrovascular accident, and cardiac death, were calculated and plotted using Kaplan-Meier (KM) methods. To assess for differences by quartile, log-rank test was used to

investigate statistically significant differences in the curves generated from the KM analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may contribute to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and cardiovascular events of interest.

The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in model four plus psychosocial variable of stress

The remainder of the models addressed the second aim of the study as detailed below.

Aim 2. To evaluate whether demographic (age, sex), socioeconomic (education and income) behavioral (smoking and physical) and psychosocial (stress) variables moderate the relationship between perceived discrimination and cardiovascular health events in Blacks.

To assess for potential effect modification, seven interaction terms were added to the fifth model separately on a second step. The interaction terms added to the fifth model separately included: (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income,

and (g) EDS-1 X Stress. Exploratory follow up tests were also completed for each of the five events (cardiovascular accident, myocardial infarction, acute ischemic syndrome, coronary revascularization and cardiac death). To test for the proportional hazard's assumption, an interaction term was added between log of survival time and perceived discrimination measure in each model.

The Cox regression models were nested and therefore model fit for the Cox regressions included evaluation of log likelihood statistics and comparison of the models with likelihood ratio hypothesis tests (Singer & Willett, 2003).

Table 1. List of Study Variables with Associated Data Set Variable Names, Levels of Measurement, and Coding Schema

Variable Name	Data Set Variable Name(s)	Level of Measurement	Variable Coding/Notes
Dependent Variable			
Time to event	Time to event is a derived variable computed for the 5 categories of outcomes.	Continuous	Number of days from baseline (study entry) until time of event.
Non-fatal myocardial infarction	*event_MI* = 1 AND *event_death* = 0 AND *time_event_MI*		
Acute ischemic syndrome	*event_AIS* = 1 AND *time_event_AIS*		
Coronary revascularization	*event_REVASC* AND *time-event_REVASC*		
Cerebrovascular accident	*Event_CVA* AND *time-event_CVA*		
Cardiac death	*event_death_doc_cardiac* = 1 AND *time_event_cardiac_death*		
Status Variable			
Cardiovascular event	This variable was derived to indicate which participants had one of the 5 cardiovascular events.	Dichotomous	0 = Did not have an event 1 = Had an event
Predictor Variables			
EDS Level 1 (EDS-1) (EDS-1 is the strata Variable)	Average of variables *DIS_Q1* through *DIS_Q10* for each participant.	Continuous	This variable was used as the “strata” variable for grouping. Score ranges from 0 to 3, with higher scores indicative of greater perceived discrimination. Participants were divided into two groups with participants with a score of less than or equal to 2=0 (lower discrimination) and score >2=1 (higher discrimination)

Table 1 (Continued)

Variable Name	Data Set Variable Name(s)	Level of Measurement	Variable Coding/Notes
Predictor Variables (cont.)			
EDS Level 2 (EDS-2)	Sum of the number of "yes" responses to variables *DIS_QA* through *DIS_QI*	Dichotomous	The number of "Yes" answers were counted and summed up.
Covariates			
Age (in years)	*SCR_AGE*	Continuous	Age in years. The variable was mean centered in the regression model.
Sex	*SCR_SEX*	Dichotomous	Male = 0 Female = 1
Physical Activity	*PA_Q6*	Ordinal	1 = Sedentary 2 = Mild 3 = Moderate 4 = Strenuous
Stress	*COHEN_Revised*	Continuous	Score of Perceived Stress Scale 4-items (Cohen) Scores range from 0 to 16 with higher scores indicative of higher stress
Smoking	*LIFE_SMOKING, c*oded in dataset as: 1 = Current smoker 2 = Former smoker 3 = Never smoker	Ordinal	The smoking variable was reverse coded from the LIFE_SMOKING variable to derive an ordinal variable that was ranked from never smoker to current smoker, in order to more easily interpret the regression findings: 1 = Never smoked 2 = Former smoker 3 = Current smoker

Table 1 (Continued)

Variable Name	Data Set Variable Name(s)	Level of Measurement	Variable Coding/Notes
Covariates (cont.)			
Income (in dollars)	*DEMO_INCOME*	Ordinal	1 = less than 10,000 2 = 10,000 to less than 20,000 3 = 20,000 to less than 40,000 4 = 40,000 to less than 80,000 5 = 80,000 or more
Education	*DEMO_EDUCCAT5*	Ordinal	1 = Less than high school 2 = High school diploma 3 = Some college 4 = Bachelor's degree 5 = Advanced degree
Interaction Terms			
EDS-1 X Age			The product of the EDS-1 X Age variables.
EDS-1 X Sex			The product of the EDS-1 X Sex variables.
EDS-1 X Education			The product of the EDS-1 X Education variables.
EDS-1 X Income			The product of the EDS-1 X Income variables.
EDS-1 X Smoking			The product of the EDS-1 X Smoking variables.
EDS-1 X Physical Activity			The product of the EDS-1 X Physical Activity variables.
EDS-1 X Stress			The product of EDS-1 X Stress variables

Note. Variables in italics are the variable names from the Heart SCORE data set. EDS = Everyday Discrimination Scale; SCR = Screening Variable.

CHAPTER FOUR:

RESULTS

Introduction

In Chapter 4, the results of the research are presented in a descriptive format as well as with tables. The results of Chapter 4 are divided into four sections: (a) descriptive findings, (b) investigation of assumptions as relates to the inferential analyses, (c) inferential tests to address the two specific aims of the study, and (d) additional exploratory follow-up tests performed separately for each of the five cardiovascular events. The chapter concludes with a summary of the results. SPSS V25 was used for all descriptive and inferential analyses. A 0.05 level of significance was used for all inferential analyses.

This study involved a secondary data analysis that was performed to utilize the extensive data available from the Heart SCORE study to evaluate the relationship between perceived discrimination and cardiovascular events in Blacks. The dataset also included information for following of change over time among the study participants within the cohort (Erqou et al., 2017). The aims of the Heart SCORE study were to address cardiovascular disease by improving risk stratification, identify racial disparities, and evaluate a multi-disciplinary community-based intervention program to decrease CVD risk in high-risk populations (Bambs et al., 2011). The purpose of this secondary data analysis was to determine whether there is a relationship between perceived discrimination and cardiovascular events among Blacks, and to evaluate whether other

modifiable risk factors moderate that relationship. Hypothesis tests were performed to investigate two aims:

Aim 1. To examine the relationship between perceived discrimination and cardiovascular health events in Black participants in the Heart SCORE study.

Aim 2. To evaluate whether demographic (age, sex), socioeconomic (education and income), behavioral (smoking and physical activity), and psychosocial (stress) factors moderate the relationship between perceived discrimination and cardiovascular events in Black participants.

Population and Descriptive Statistics

The data set used in this secondary analysis included participant records with approximately 13.5 years of information from the beginning of participant enrollment and included N = 653 records of Black individuals. A total of 854 records of Black individuals were in the full Heart SCORE dataset. However, only records with complete information on all model variables were included for the study according to the exclusion criteria noted in Chapter 3.

Table 2 includes the measures of central tendency and variability, and Cronbach's alpha coefficients for the four continuous variables which were included in the study analyses. The participants ranged in age from 45 to 74 years (M = 57.93 years, SD = 7.28 years). The 10-item Everyday Discrimination Scale – Level 1 (EDS-1) was used as the strata variable for partitioning the participants to two groups of low and high perceived discrimination. The possible range of the EDS-1 is 0 to 3, with higher scores indicative of greater perceived discrimination. The range of the EDS-1 for the study participants was 0.70 to 3.0 (M = 2.08, SD = 0.45). Thus, the EDS-1 was divided

into two groups of lower perceived discrimination (scores of <=2, n=296) and higher discrimination (scores of >2, n=357), n= lower discrimination group had scores of 2 or less and the higher discrimination group has scores of >2. Lower discrimination group had a mean age of 57.35 with a 7.11 standard deviation while the higher discrimination group had a mean age of 58.40 with a standard deviation of 7.40. The predictor variable of Everyday Discrimination Scale – Level 2 (EDS-2) contained eight items and was scored from 0 to 8 by counting the number of "yes" responses. The final continuous variable was stress, and the variable was scored from the 4-item Cohen Stress Scale. Possible scores ranged from 0 to 16, with higher scores indicative of greater stress. The scores for the participants in this study ranged from 0 to 16 and were lower on average (M = 4.51, SD = 3.13). "Lower than average" means that the mean or median of the Cohen Stress Scale, given the possible range of scores, would be 8.5. Thus, a mean of 4.51 is indicative of the participants as a whole scoring lower in stress on average.

Table 3 includes the frequency counts and percentages of the demographic, behavioral, and socioeconomic variables used in the Cox regression models. Most participants were female (69%). Most participants reported a level of mild or moderate physical activity (82%). A small percentage of the participants (14%) were current smokers. Over one-half of the participants (65%) claimed an income of $20,000 to $80,000 per year. Most participants (81%) had completed at least some college or greater for their education.

Table 4 includes the frequency counts and percentages of the 5 events that constituted the dependent variable of the study, as well as the measures of central tendency and variability for the time to each event for all participants in the study (both

censored and non-censored). A total of 84 events were noted for a total of 54 participants. When a participant had more than one event, the type and time of the first event was counted as the event of interest. Time to event was recorded as the time frame from entry of a participant into the study until the time (in days) the first cardiac event occurred. Participants who did not experience an event were defined as right censored.

A total of 84 events were noted for a total of 54 participants. When a participant had more than one event, the type and time of the first event was counted as the event of interest.

Assumptions for Inferential Tests

Two types of inferential analyses, (a) Kaplan-Meier survival analysis, and (b) Cox proportional hazard model (Cox regression) were performed to address the aims of this study. The assumptions for each test and their relation to the data of this study are presented according to the inferential test type.

Aim 1 was investigated via a Kaplan-Meier Survival Analysis (KMSA) which included a log-rank test. KMSA assumes that events (survival or event) are dependent only on time. The data set was checked, and the event status of each participant was mutually exclusive (each participant was classified as either “event” or “censored” but not both. The survival time was clearly defined and precisely measured in days. Left censoring was not present because each participant’s starting time in the study was time of enrollment. The assumption of independence of censoring and the event was also met, because censoring was not related to the event. The assumption of no secular trends was met because new drugs or treatments were not given to patients recruited

into the study later than the earlier recruited patients. And finally, a similar amount and pattern of censorship was noted for the two EDS-1 groups by calculating the percentage of censored cases per group. Thus, it was assumed for this study that event probabilities depended only on time, all subjects were assumed to behave similarly, and the survival functions were assumed to describe all subjects. The implication of these assumptions is that censored and uncensored cases behaved similarly. All assumptions for the KMSA were met.

Cox proportion hazard models (Cox regressions) were used to test Aims 1 and 2. Assumptions for Cox regression include (a) independence between individual subjects, (b) multiplicative relationship between the hazard and predictors, (c) constant hazard ratio over time between the two EDS-1 groups, and (d) the hazard ratio represents a 1 unit change in risk of the outcome of interest holding all other predictors constant. The status of any given participant was not influenced by the status of other participants on any of the model variables. Thus, the assumption of independence between subjects was met. The Cox regression model by design has a multiplicative relationship between the hazard and predictors and this assumption was met. The constant hazard ratio over time between the two EDS-1 groups of lower perceived discrimination and higher perceived discrimination (proportional hazards assumption), was tested in each model with an interaction term between log of survival time and perceived discrimination measure levels. The interaction term was not statistically significant in any of the tested models and therefore the proportional hazards assumption was met. All results are reported without the Ln Survival Time X EDS-1 interaction term.

Multicollinearity between predictors and covariates could be a problem in regression models. Multicollinearity diagnostics for the variables used in the Cox regression models were performed using SPSS via correlational analysis. Multicollinearity may be assumed if a correlation coefficient between two variables is .90 or greater (Pallant, 2013). No violations were noted and the assumption of absence of multicollinearity was met. Spearman's correlation coefficients for the bivariate relationships between the variables of the study are presented in Table 5.

Inferential Tests to Address the Two Specific Aims of the Study

Prior to testing the two aims of study, bi-variate correlations were investigated for the variables which were utilized in the analyses. Spearman's Rank Order Correlation analyses were conducted to check the bi-variate relationships between the tested variables. Spearman's correlation analyses were conducted, as opposed to Pearson's correlation, because many of the variable measures were ordinal in nature and did not follow the normal distribution or necessarily have a linear relationship with each other. Spearman's correlations can be used when variables are at least ordinal (Pallant, 2013). Correlations should not be interpreted as indicating cause-and-effect relationships, as correlation analyses are not designed to detect cause and effect, only to indicate associations. Direct (positive) correlations indicate the values of two variables move in a like manner, values either increase or decrease similarly. An indirect (negative) correlation indicates the values of two variables move in opposing directions, i.e. when the values of one variable increase, the values of the other variable decrease.

Table 5 presents the findings of the Spearman's rank order correlational analyses. Cohen suggests that the measured effects of correlation coefficients with absolute values between .10 to .29 are weak, between .30 to .49 are moderate, and between .50 to 1.0 are strong. Due to the larger sample size of N = 653 participants, many statistically significant correlations were found between the variables of study, even when the correlational effect was weak. Therefore, only the significant correlations pertaining to the outcome of cardiovascular event and the significantly moderate to strong correlational effects, (an absolute magnitude of correlation between .30 and 1.0) between the remaining variables are reported in the text to preserve parsimony.

Cardiovascular event had a weak but statistically significant positive correlation with age (r = .143, p = .000). The positive correlation between cardiovascular event and age indicated that as the age of the participants increases, so did the risk of a cardiovascular event. The variable of EDS-1 was moderately and negatively correlated with the variable of EDS-2 (r = -.323, p < .000). The negative association between the two variables indicates that the scores on two measures move oppositely to each other. Both variables were scored such that higher scores were indicative of greater perceived discrimination and therefore a positive correlation was expected. However, the raw scores of the EDS-2 variable could range from 0 to 8 and the variable assessed the types or quality of unfair treatment rather than the frequency or quantity of unfair treatment experienced by participants as measured by EDS-1. The variables of income and education were moderately and positively correlated (r = .418, p < .000). The positive correlation indicated that as education increased, so did income.

The relationship between variables of cardiovascular event (including all five categories of events) and gender were tested via chi-square test of independence as both variables were binary. Overall, 54 participants experienced some form of cardiovascular event. Out of the 54 cardiovascular events that were recorded, both men and women had 27 (50%) cardiovascular events each. However, men only contributed 31% of the sample in this study, this was significant ($\times^2=10.016$, df=1, $p<0.002$).

KMSA and Cox regression analyses for the outcome of cardiovascular event (all five events). One KMSA and a series of 12 nested Cox regression models were tested to address Aims 1 and 2 of this study. To address Aim 1, cumulative incidence of cardiovascular events of interest including; non-fatal MI, acute ischemic syndrome, coronary revascularization, cerebrovascular accident, and cardiac death, were calculated and plotted using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and cardiovascular events of interest. The four additional Cox regression models were used to examine other potential main effect predictors of the relationship of perceived discrimination and cardiovascular event. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables

including smoking and physical activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in Model 4 plus the variable of stress.

To assess for potential effect modification (Aim 2), interaction for each of the seven potential effect modifiers were assessed in separate models to avoid overfitting. This included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The Cox regression models were nested models and therefore model fit for the Cox regressions included evaluation of log likelihood statistics and comparison of the models with likelihood ratio hypothesis tests (Singer & Willett, 2003). The model with the best fit is reported in the text. Models that were not significant or not improved over the reported model are not reported in the text, but the syntax to the model output can be found in Appendix I. The model tests and findings are presented according to each of the two study aims.

Aim 1. To examine the relationship between perceived discrimination and cardiovascular health events in Black participants in the Heart SCORE study.

A total of 54 records with cardiovascular events were recorded in the dataset. Five hundred and ninety-nine records were right censored, indicating that a cardiovascular event did not occur during the timeframe of study (approximately 13.5 years) for those participants. Table 6 presents the incidence rates and means for survival time as well as standard errors and associated 95% confidence levels of the mean survival time for the entire sample and the two EDS-1 perceived discrimination

groups. Quartiles of survival time were not available because the number of events did not reach 25% or greater. The syntax for the survival tables for the entire sample and for the two EDS-1 groups are attached in Appendix I.

The incidence rate of all cardiovascular events, which represents a median of 13.5 years of follow-up, for the entire sample was 8.3%. The incidence rate for the EDS-1 lower perceived discrimination group was 9.1% and the incidence rate for the EDS-1 higher perceived discrimination group was 7.7%. The mean survival time for all cardiovascular events (N = 54) was M = 4657.16 days (SEM = 39.52 days). The mean survival time was greater for the EDS-1 lower perceived discrimination group (M = 4660.59, SEM = 57.16) compared to the EDS-1 higher perceived discrimination group (M = 4555.97 days, SEM = 51.33 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 0.018, p = .893, indicating that the two EDS-1 groups did not differ statistically in mean times to event despite a numerically higher rate in the low perceived discrimination group (i.e. 9.09% vs. 7.7%). The syntax for survival function curves for the EDS-1 groups are presented in Appendix I. The survival plots present the survival function on a linear scale. The x-axis is time to failure; the y-axis is the cumulative survival. The curves slope down, with fewer surviving in the risk pool as time progressed. The cumulative survival percent at any given time on the x-axis can be interpreted as the probability of survival to that time (the probability of not experiencing an event).

The syntax for cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix I. The x-axis is the survival time; the y-axis is

the cumulative hazard. The curve represents a hypothetical individual with mean values at any given time as represented on the x-axis. The curve shows how cumulative hazard increases over time for such hypothetical individuals. The hazard function describes the probability of failure during a very small time increment (if no failures have occurred prior to that time). This is also called the instantaneous failure rate. Hazard is the slope of the survival curve – a measure of how rapidly participants are having the failure.

A Cox regression model was also tested for Aim 1. The first Cox regression model was unadjusted and was used to examine the independent relationship between the perceived discrimination strata variable of EDS-1, the perceived discrimination predictor variable of EDS-2, and the outcome of cardiovascular event. The model findings are presented in Table 7 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The model with two variables of EDS-1 and EDS-2 (-2 Log Likelihood = 667.32) was not significantly improved over the baseline model with no predictors (-2 Log Likelihood = 670.39; $X^2(2) = 3.07$, $p = .216$. The non-significance indicates the model with the two predictors did not provide more information than the baseline model.

Conclusion as relates to Aim 1. There was not enough evidence to indicate a statistically significant association between the predictors of perceived discrimination and cardiovascular health events in Black participants in the Heart SCORE study. In relation to examination for potential main effect predictors, the second Cox regression model was adjusted for demographic variables including gender and age. Gender was coded as 0 = male and 1 = female. Age was mean centered prior to the analysis. The

second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination. The model findings are presented in Table 8 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The second model with four variables of EDS-1, EDS-2, age, and gender (-2 LL = 646.03) was significantly improved over the baseline model with no predictors (-2LL = 670.39; $X^2(4) = 24{,}36$, $p < .0005$. The second model was also a significant improvement over the first model with 2 predictors (See Table 7; $X^2(2) = 21.76$, $p < .0005$)

The predictor of age was statistically significant [HR = 1.07; 95% CI for HR [1.03, 1.11]), $p = .00$]. The HR of 1.07 indicated that each year increase in age above the mean of 57.93 years was associated with a 7% increase in the risk of a cardiovascular event, holding the other variables constant. The predictor of gender was also statistically significant [HR = 0.46; 95% CI for HR [0.27, 0.79], $p = .005$. The HR of 0.46 indicated that females were associated with a 54% decrease in the risk of a cardiovascular event when compared to males, holding the other variables constant.

The third Cox regression model was adjusted for demographics of the second Cox regression and behavioral variables including smoking and physical activity. Smoking was an ordinal variable coded as 1 = never smoked, 2 = former smoker, and 3 = current smoker. Physical activity was an ordinal variable coded as 1 = sedentary, 2 = mild, 3 = moderate, and 4 = strenuous. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, and had mild physical activity. The gender and age variables remained statistically significant in the third model, but no other variables

reached statistical significance. The -2 log likelihood of the third model (-2LL = 645.44) was compared with the -2 log likelihood of the second model [-2LL = 646.03; $X^2(5) = 24.36$, $p < .0005$]. Model 3 was not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. Education was an ordinal variable coded as 1 = less than high school, 2 = high school diploma, 3 = some college, 4 = bachelor's degree, and 5 = advanced degree. Income was coded as 1 = less than $10,000 per year, 2 = $10,000 to less than $20,000 per year, 3 = $20,000 to less than $40,000 per year, 4 = $40,000 per year to less than $80,000 per year and 5 = $80,000 or more per year. The fourth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, and made less than $10,000 per year. The gender and age variables remained statistically significant in the fourth model, but no other variables reached statistical significance. The -2 log likelihood of the fourth model (-2LL = 644.54) was compared with the -2 log likelihood of the third model [- 2LL = 643.03; $X^2(8) = 3.63$, $p = .889$] Model 4 was not a statistically significant improvement over Model 3. The -2 log likelihood of the fourth model (-2LL = 635.22) was also compared with the -2 log likelihood of the second model [643.95; $X^2(13) = 8.73$, $p = .793$]. Model 4 was also not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fifth model was adjusted for the variables in model four plus the variable of stress, a continuous variable scored from 0 to 16, with higher scores indicative of greater stress. The fifth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, made less than $10,000 per year, and had a stress score of 0. The -2 log likelihood of the Model 5, step 1 (-2LL = 637.86) was compared with the -2 log likelihood of the fourth model [- 2LL = 637.87; $X^2(1) = 0.22$, $p = .639$]. Model 5 step 1 was not a statistically significant improvement over Model 4. The -2 log likelihood of the Model 5 step 1 was also compared with the -2 log likelihood of the third model [-2LL = 641.15; $X^2(9) = 2.55$, $p = .980$]. Model 5 step 1 was not a significant improvement over Model 3. Finally, the -2 log likelihood of Model 5, step 1 was compared with Model 2 [- 2LL = 646.29; $X^2(14) = 97.95$, $p = .892$]. Model 5, step 1 was not a significant improvement over Model 2. Thus, the best fitting model remained Model 2 (see Table 8).

Aim 2. To evaluate whether demographic, socioeconomic, behavioral and psychosocial factors modify the relationship between perceived discrimination and cardiovascular events in Black participants in the Heart SCORE study.

To assess for potential effect modification, seven interaction terms were individually added to the fifth model on a second step. These included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress. Fifth model step 2 (i) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X age. Model 5 step 2 (ii) was adjusted for all variables in model 5 step 1 plus interaction of

EDS-1 X gender. Model 5 step 2 (iii) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X physical activity. Model 5 step 2 (iv) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X smoking. Model 5 step 2 (v) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X Income. Model 5 step 2 (vi) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X education. Finally, model 5 step 2 (vii) was adjusted for all variables in model 5 step 1 plus interaction of EDS-1 X stress. When interaction terms were added to model 5 step 1, the variables of age and gender were not statistically significant in all models except for model with the interaction of EDS-1 and age (model 5 step 2-1) ($\times^2$ (1) =3.937, p= 0.047). None of the seven interaction models were statistically significant (see table 10) and no further investigations of the model or variable were performed

Conclusion as relates to Aim 2. None of the interaction term models tested were statistically significant. Therefore, age, gender, physical activity, smoking, income, education and stress did not modify the relationship between perceived discrimination and cardiovascular events among Blacks in the Heart SCORE study. Next, exploratory follow -up tests for individual cardiovascular events will be discussed. The event of acute ischemic syndrome (AIS) will not be reported as there were only 6 events recorded and were not enough for this analysis.

Exploratory Follow-up Tests for Each Individual Cardiovascular Event

KMSA and Cox regression analyses for the outcome of cardiovascular event non-fatal myocardial infarction (non-fatal MI). One KMSA and a series of five nested Cox regression models were tested to explore the event of MI only. Cumulative incidences of the cardiovascular events of non-fatal MI were calculated and plotted

using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and the cardiovascular events of non-fatal MI. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in model four plus the variable of stress. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The Cox regression models were nested models and therefore model fit for the Cox regressions included evaluation of log likelihood statistics and comparison of the models with likelihood ratio hypothesis tests (Singer & Willett, 2003). The model with the best fit is reported in the text. Models that were not significant or not improved over the reported model are not reported in the text, but the syntax for the model output can be found in Appendix II.

A total of 19 records with the cardiovascular event of non-fatal MI were recorded in the dataset. Six hundred and thirty-four records were right censored, indicating that a cardiovascular event of non-fatal MI did not occur during the timeframe of study for those participants. Table 11 presents the means for survival time as well as standard errors and associated 95% confidence levels for the entire sample and the two EDS-1 perceived discrimination groups. Quartiles of survival time were not available because the number of events did not reach 25% or greater. The syntax for the survival tables for the entire sample and for the two EDS-1 groups are attached in Appendix II.

The incidence rate of all non-fatal MI events for the entire sample was 2.9%. The incidence rate for the EDS-1 lower perceived discrimination group was 3.7% and the incidence rate for the EDS-1 higher perceived discrimination group was 1.8%. The mean survival time for cardiovascular events of non-fatal MI (N = 19) was M = 4768.06 days (SEM = 40.47 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (M = 4784.73, SEM = 40.17) compared to the EDS-1 lower perceived discrimination group (M = 4722.82 days, SEM = 53.91 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 2.26, p = .133, indicating that at the two EDS-1 groups did not differ in their mean times to event. The Syntax for the survival function curves and Cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix II.

A Cox regression model was also tested for the cardiovascular event of non-fatal MI. The Cox regression model was unadjusted and was used to examine the independent relationship between the perceived discrimination strata variable of EDS-1,

the perceived discrimination predictor variable of EDS-2, and the outcome of cardiovascular event of non-fatal MI. The model findings are presented in Table 12 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The model with two variables of EDS-1 and EDS-2 (-2 Log Likelihood = 202.14) was not significantly improved over the baseline model with no predictors (-2 Log Likelihood = 205.01; $X^2(2) = 2.87$, $p = .238$). The non-significance indicates the model with the two predictors did not provide more information than the baseline model.

The second Cox regression model was adjusted for demographic variables including gender and age. Gender was coded as 0 = male and 1 = female. Age was mean centered prior to the analysis. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination. The model findings are presented in Table 13 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The second model with four variables of EDS-1, EDS-2, age, and gender (-2 LL = 192.76) was significantly improved over the baseline model with no predictors (-2LL = 205.01; $X^2(4) = 12.25$, $p = .016$). The second model was also a significant improvement over the first model with 2 predictors (See Table 12; $X^2(2) = 9.38$, $p = .009$).

The predictor of age was statistically significant (HR = 1.09; 95% CI for HR [1.02, 1.17], $p = .011$). The HR of 1.09 indicated that each year increase in age above the mean of 57.93 years was associated with a 9% increase in the risk of a cardiovascular event of non-fatal MI, holding the other variables constant.

The third Cox regression model was adjusted for demographics of the second Cox regression and behavioral variables including smoking and physical activity. Smoking was an ordinal variable coded as 1 = never smoked, 2 = former smoker, and 3 = current smoker. Physical activity was an ordinal variable coded as 1 = sedentary, 2 = mild, 3 = moderate, and 4 = strenuous. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, and had mild physical activity. The age variable remained statistically significant in the third model, but no other variables reached statistical significance The -2 log likelihood of the third model (-2LL = 187.47) was compared with the -2 log likelihood of the second model [-2LL = 192.76; $X^2(5) = 5.29$, $p = .382$]. Model 3 was not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. Education was an ordinal variable coded as 1 = less than high school, 2 = high school diploma, 3 = some college, 4 = bachelor's degree, and 5 = advanced degree. Income was coded as 1 = less than $10,000 per year, 2 = $10,000 to less than $20,000 per year, 3 = $20,000 to less than $40,000 per year, 4 = $40,000 per year to less than $80,000 per year and 5 = $80,000 or more per year. The fourth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, and made less than $10,000 per year. The age variable remained statistically significant in the fourth model, but no other variables reached statistical significance The -2 log

likelihood of the fourth model (-2LL = 175.50) was compared with the -2 log likelihood of the third model [- 2LL = 187.47; $X^2(8) = 11.97$, $p = .153$]. Model 4 was not a statistically significant improvement over Model 3. The -2 log likelihood of the fourth model (-2LL = 175.50) was also compared with the -2 log likelihood of the second model [192.76; $X^2(13) = 17.26$, $p = .188$]. Model 4 was also not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fifth model was adjusted for the variables in model four plus the variable of stress, a continuous variable scored from 0 to 16, with higher scores indicative of greater stress. The fifth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, made less than $10,000 per year, and had a stress score of 0. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The variables of age (HR = 1.13, $p = .001$) and gender (HR = 0.35, $p = .048$) were statistically significant in step 1 of the fifth model, but no other variables reached statistical significance. Age was a significant variable in step 2 of Model 5, but step 2 was not statistically significant when compared to step 1 $X^2(7) = 4.82$, $p = .682$.

The -2 log likelihood of the Model 5, step 1 (-2LL = 172.66) was compared with the -2 log likelihood of the fourth model [- 2LL = 175.50; $X^2(1) = 2.84$, $p = .092$]. Model 5

step 1 was not a statistically significant improvement over Model 4. The -2 log likelihood of the Model 5 step 1 was also compared with the -2 log likelihood of the third model [- 2LL = 187.47; $X^2(9) = 14.81$, $p = .096$]. Model 5 step 1 was not a significant improvement over Model 3. Finally, the -2 log likelihood of Model 5, step 1 was compared with Model 2 [- 2LL = 192.76; $X^2(14) = 20.10$, $p = .127$]. Model 5, step 1 was not a significant improvement over Model 2. Thus, the best fitting model remained Model 2 (see Table 12). A listing of the -2 log likelihood values and number of variables for each of the six models relating to the outcome of cardiovascular event of non-fatal MI is presented in Table 14.

KMSA and Cox regression analyses for the outcome of cardiovascular event coronary revascularization (REVASC). One KMSA and a series of five nested Cox regression models were tested to explore the event of REVASC only. Cumulative incidences of the cardiovascular events of REVASC were calculated and plotted using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and the cardiovascular events of REVASC. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical

activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in Model 4 plus the variable of stress. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress

The Cox regression models were nested models and therefore model fit for the Cox regressions included include evaluation of log likelihood statistics and comparison of the models with likelihood ratio hypothesis tests (Singer & Willett, 2003). The model with the best fit is reported in the text. Models that were not significant or not improved over the reported model are not reported in the text, but the syntax for the model output can be found in Appendix IV.

A total of 26 records with the cardiovascular event of REVASC were recorded in the dataset. Six hundred and twenty seven records were right censored, indicating that a cardiovascular event of REVASC did not occur during the timeframe of study for those participants. Table 17 presents the means for survival time as well as standard errors and associated 95% confidence levels for the entire sample and the two EDS-1 perceived discrimination groups. Quartiles of survival time were not available because the number of events did not reach 25% or greater. The syntax for the survival tables for the entire sample and for the two EDS-1 groups are attached in Appendix IV.

The incidence rate of all REVASC events for the entire sample was 3.9%. The incidence rate for the EDS-1 lower perceived discrimination group was 4.8% and the

incidence rate for the EDS-1 higher perceived discrimination group was 2.9%. The mean survival time for cardiovascular events of REVASC (*N* = 26) was *M* = 4784.76 days (*SEM* = 31.05 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (*M* = 4763.87 days, *SEM* = 39.20 days) compared to the EDS-2 lower perceived discrimination group (*M* = 4755.29 days, *SEM* = 41.16 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 1.57, *p* = .210, indicating that at the two EDS-1 groups did not differ in their mean times to event. Syntax for the survival function curves and cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix IV.

A Cox regression model was also tested for the cardiovascular event of REVASC. The Cox regression model was unadjusted and was used to examine the independent relationship between the perceived discrimination strata variable of EDS-1, the perceived discrimination predictor variable of EDS-2, and the outcome of cardiovascular event of REVASC. The model findings are presented in Table 18 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The model with two variables of EDS-1 and EDS-2 (-2 Log Likelihood = 322.14) was not significantly improved over the baseline model with no predictors (-2 Log Likelihood = 325.46; $X^2(2) = 3.17$, $p = .205$). The non-significance indicates the model with the two predictors did not provide more information than the baseline model.

The second Cox regression model was adjusted for demographic variables including gender and age. Gender was coded as 0 = male and 1 = female. Age was

mean centered prior to the analysis. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination. The model findings are presented in Table 19 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The second model with four variables of EDS-1, EDS-2, age, and gender (-2 LL = 312.98) was significantly improved over the baseline model with no predictors (-2LL = 325.46; $X^2(4) = 12.48$, $p = .014$. The second model was also a significant improvement over the first model with two predictors (See Table 17; $X^2(2) = 9.16$, $p = .010$).

The predictor of gender was statistically significant [HR = 0.38; 95% CI for HR (0.18, 0.84), $p = .017$]. The HR of 0.38 indicated that females were associated with a 62% decrease in the risk of a cardiovascular event of REVASC when compared to males, holding the other variables constant.

The third Cox regression model was adjusted for demographics of the second Cox regression and behavioral variables including smoking and physical activity. Smoking was an ordinal variable coded as 1 = never smoked, 2 = former smoker, and 3 = current smoker. Physical activity was an ordinal variable coded as 1 = sedentary, 2 = mild, 3 = moderate, and 4 = strenuous. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, and had mild physical activity. The gender variable remained statistically significant in the third model, and the variables of age and physical activity = moderate also reached statistical significance However, Model 3 (-2LL = 307.55) was not statistically significantly improved over Model 2 (-2LL = 312.98;

$X^2(5) = 5.43$, $p = .366$]. Since Model 3 was not a statistically significant improvement over Model 2, no further investigation into significance of the model or variables was performed.

The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. Education was an ordinal variable coded as 1 = less than high school, 2 = high school diploma, 3 = some college, 4 = bachelor's degree, and 5 = advanced degree. Income was coded as 1 = less than $10,000 per year, 2 = $10,000 to less than $20,000 per year, 3 = $20,000 to less than $40,000 per year, 4 = $40,000 per year to less than $80,000 per year and 5 = $80,000 or more per year. The fourth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, and made less than $10,000 per year. The age, gender, and physical activity = moderate variables remained statistically significant in the fourth model, but no other variables reached statistical significance The -2 log likelihood of the fourth model (-2LL = 302.87) was compared with the -2 log likelihood of the third model [- 2LL = 307.55; $X^2(8) = 4.68$, $p = .791$]. Model 4 was not a statistically significant improvement over Model 3. The -2 log likelihood of the fourth model (-2LL = 302.87) was also compared with the -2 log likelihood of the second model [312.98; $X^2(13) = 10.11$, $p = .694$]. Model 4 was also not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fifth model was adjusted for the variables in Model 4 plus the variable of stress, a continuous variable scored from 0 to 16, with higher scores indicative of

greater stress. The fifth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, made less than $10,000 per year, and had a stress score of 0. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The age, gender, and physical activity = moderate variables remained statistically significant in step 1 of the fifth model, but no other variables reached statistical significance. Physical activity = moderate and the interaction between EDS-1 X Smoking were significant predictors in step 2 Model 5, but step 2 was not statistically significant when compared to step 1 [-2LL = 290.61; $X^2(7) = 12.26$, $p = .092$].

The -2 log likelihood of the Model 5, step 1 (-2LL = 302.87) was compared with the -2 log likelihood of the fourth model [- 2LL = 302.87; $X^2(1) = 0$, $p = 1.0$]. Model 5 step 1 was not a statistically significant improvement over Model 4. The -2 log likelihood of the Model 5 step 1 was also compared with the -2 log likelihood of the third model [-2LL = 307.55; $X^2(9) = 4.68$, $p = .861$]. Model 5 step 1 was not a significant improvement over Model 3. Finally, the -2 log likelihood of Model 5, step 1 was compared with Model 2 [-2LL = 312.98; $X^2(14) = 10.11$, $p = .754$]. Model 5, step 1 was not a significant improvement over Model 2. Thus, the best fitting model remained Model 2 (see Table 18). A listing of the -2 log likelihood values and number of variables for each of the six

models relating to the outcome of cardiovascular event of REVASC is presented in Table 19.

KMSA and Cox regression analyses for the outcome of cardiovascular event cerebrovascular accident (CVA). One KMSA and a series of five nested Cox regression models were tested to explore the event of CVA only. Cumulative incidence of the cardiovascular events of CVA was calculated and plotted using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and the cardiovascular events of CVA. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical activity. The fourth model was adjusted for variables in Model 3 plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in model four plus the variable of stress. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress. The syntax for the SPSS output of the Cox regression models with the event of CVA are attached in Appendix V.

A total of 16 records with the cardiovascular event of CVA were recorded in the dataset. Six hundred and twenty seven records were right censored, indicating that a cardiovascular event of CVA did not occur during the timeframe of study for those participants. Table 20 presents the means for survival time as well as standard errors and associated 95% confidence levels for the entire sample and the two EDS-1 perceived discrimination groups. Quartiles of survival time were not available because the number of events did not reach 25% or greater. The syntax for survival tables for the entire sample and for the two EDS-1 groups are attached in Appendix V.

The incidence rate of all CVA events for the entire sample was 2.5%. The incidence rate for the EDS-1 lower perceived discrimination group was 2.7% and the incidence rate for the EDS-1 higher perceived discrimination group was 2.2%. The mean survival time for cardiovascular events of CVA (N = 16) was M = 4968.01 days (SEM = 76.92 days). The mean survival time was greater for the EDS-1 lower perceived discrimination group (M = 4914.32 days, SEM = 95.65 days) compared to the EDS-1 higher perceived discrimination group (M = 4825.30 days, SEM = 21.91 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 0.00, p = .989, indicating that at the two EDS-1 groups did not differ in their mean times to event. The syntax for survival function curves and Cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix V.

A series of five Cox regression models were developed for the outcome of cardiovascular event of CVA. However, Models 1 and 2 were not statistically significant, and convergence failed on Models 3, 4, and 5 when modeled as specified. None of the

coefficients for the predictors or covariates in any of the five models were statistically significant, and the second step in Model 5 did not run in the model. The specified models were determined to be a poor fit with the dataset and no further investigation for the outcome of the cardiovascular event of CVA was performed.

KMSA and Cox regression analyses for the outcome of cardiovascular event of cardiac death. One KMSA and a series of five nested Cox regression models were tested to explore the event of cardiac death only. Cumulative incidences of the cardiovascular events of non-fatal MI were calculated and plotted using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and the cardiovascular events of cardiac death. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in Model 4 plus the variable of stress. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X

Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The Cox regression models were nested models and therefore model fit for the Cox regressions included evaluation of log likelihood statistics and comparison of the models with likelihood ratio hypothesis tests (Singer & Willett, 2003). The model with the best fit is reported in the text. Models that were not significant or not improved over the reported model are not reported in the text, but the syntax for model output can be found in Appendix VI.

A total of 17 records with the cardiovascular event of cardiac death were recorded in the dataset. Six hundred and thirty-six records were right censored, indicating that a cardiovascular event of cardiac death did not occur during the timeframe of study for those participants. Table 22 presents the means for survival time as well as standard errors and associated 95% confidence levels for the entire sample and the two EDS-1 perceived discrimination groups. Quartiles of survival time were not available because the number of events did not reach 25% or greater. The syntax for survival tables for the entire sample and for the two EDS-1 groups are attached in Appendix VI.

The incidence rate of all cardiac death events for the entire sample was 2.6%. The incidence rate for the EDS-1 lower perceived discrimination group was 3.2% and the incidence rate for the EDS-1 higher perceived discrimination group was 1.8%. The mean survival time for cardiovascular events of cardiac death (N = 17) was M = 5010.91 days (SEM = 39.93 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (M = 5061.80 days, SEM = 25.47 days) compared to the

EDS-1 lower perceived discrimination group (*M* = 4898.85 days, *SEM* = 54.83 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 1.29, *p* = .256, indicating that at the two EDS-1 groups did not differ in their mean times to event. The syntax for the survival function curves and Cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix VI.

A Cox regression model was also tested for the cardiovascular event of cardiac death. The Cox regression model was unadjusted and was used to examine the independent relationship between the perceived discrimination strata variable of EDS-1, the perceived discrimination predictor variable of EDS-2, and the outcome of cardiovascular event of cardiac death. The model findings are presented in Table 23 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The model with two variables of EDS-1 and EDS-2 (-2 Log Likelihood = 197.66) was not significantly improved over the baseline model with no predictors (-2 Log Likelihood = 199.01; X^2(2) = 1.35, *p* = .509). The non-significance indicates the model with the two predictors did not provide more information than the baseline model.

The second Cox regression model was adjusted for demographic variables including gender and age. Gender was coded as 0 = male and 1 = female. Age was mean centered prior to the analysis. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination. The model findings are presented in Table 24 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95%

confidence intervals for the hazard ratios. The second model with five variables of EDS-1, EDS-2, interaction between log of survival time and EDS-1, age, and gender (-2 LL = 179.04) was significantly improved over the baseline model with no predictors (-2LL = 199.01; $X^2(4) = 19.97$, $p = .001$). The second model was also a significant improvement over the first model with two predictors (See Table 23; $X^2(2) = 18.62$, $p < .0005$).

The predictor of age was statistically significant [HR = 1.15; 95% CI for HR (1.07, 1.24), $p < .0005$]. The HR of 1.15 indicated that each year increase in age above the mean of 57.93 years was associated with a 15% increase in the risk of a cardiovascular event of cardiac death, holding the other variables constant.

The third Cox regression model was adjusted for demographics of the second Cox regression and behavioral variables including smoking and physical activity. Smoking was an ordinal variable coded as 1 = never smoked, 2 = former smoker, and 3 = current smoker. Physical activity was an ordinal variable coded as 1 = sedentary, 2 = mild, 3 = moderate, and 4 = strenuous. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, and had mild physical activity. The age variable remained statistically significant in the third model. The physical activity variable of moderate activity was also statistically significant but no other variables reached statistical significance The -2 log likelihood of the third model (-2LL = 168.09) was compared with the -2 log likelihood of the second model [-2LL = 179.04; $X^2(5) = 10.95$, $p = .052$]. Model 3 was not a statistically significant improvement over Model 2, and no further investigation into significance of the model or variables was performed.

The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. Education was an ordinal variable coded as 1 = less than high school, 2 = high school diploma, 3 = some college, 4 = bachelor's degree, and 5 = advanced degree. Income was coded as 1 = less than $10,000 per year, 2 = $10,000 to less than $20,000 per year, 3 = $20,000 to less than $40,000 per year, 4 = $40,000 per year to less than $80,000 per year, and 5 = $80,000 or more per year. The fourth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, and made less than $10,000 per year. The age variable remained statistically significant in the fourth. Other significant variables included gender, and the physical activity variables of mild and moderate. The -2 log likelihood of the fourth model (-2LL = 158.25) was compared with the -2 log likelihood of the third model [- 2LL = 168.09; $X^2(8) = 9.84$, $p = .276$]. Model 4 was not a statistically significant improvement over Model 3. The -2 log likelihood of the fourth model (-2LL = 158.25) was also compared with the -2 log likelihood of the second model [179.04; $X^2(13) = 20.79$, $p = .077$]. Model 4 was also not a statistically significant improvement over Model 2, and no further investigation into significance of Model 4 was performed.

The fifth model was adjusted for the variables in Model 4 plus the variable of stress, a continuous variable scored from 0 to 16, with higher scores indicative of greater stress. The fifth regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination, never smoked, had mild physical activity, had an education of less than high school, made

less than $10,000 per year, and had a stress score of 0. To assess for potential effect modification, seven interaction terms were added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

The variables of age, gender, physical activity = mild, and physical activity = moderate remained significant in step 1 of Model 5. Step two of Model 5 was statistically significant from step 1 of model 5. However, step 1 of Model 5 was not statistically different from Models 2, 3, or 4. Step 2 of model 5 (-2LL = 141.77) was statistically significant improvement from Model 2 [- 2LL = 179.04; $X^2(21) = 37.27$, $p = .016$] and Models 3, 4, and 5 step 1 (see Table 24). Therefore, the best model for the event of cardiac death was the saturated model (with steps 1 and 2) of Model 5. A listing of the -2 log likelihood values and number of variables for each of the six models relating to the outcome of cardiovascular event of cardiac death presented in Table 24. The model findings for Model 5 steps 1 and 2 are presented in Table 25 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. A statistically significant interaction was found between EDS-1 X Age [HR = 1.40; 95% CI for HR (1.05, 1.86), $p = .023$]. The size and direction of the hazard ratio of 1.40 indicated that participants who experienced higher stress as indicated the EDS-1 variable experienced a 40% greater risk of cardiac death for each year of age above the average age, when compared to participants who scored as lower stress on the EDS-1 variable. The main effects of EDS-1 and age were also statistically significant. However, the interaction term takes precedence over the main

effect in the model. Of note, the main effect of EDS-1 had a very high hazard ratio and large associated 95% confidence interval of the hazard ratio. It is conventional to include main terms in the model with the interaction terms (Tabachnick and Fidell, 2013). Therefore, the model was kept intact as specified.

Other statistically significant interactions included EDS-1 X Physical Activity [HR = 0.14; 95% CI for HR (0.02, 0.98), p = .048] and the interaction of EDS-1 X Smoking [HR = 0.08; 95% CI for HR (0.01, 0.98), p = .048]. The HR of 0.14 for the EDS-1 X Physical Activity interaction indicated that participants with higher perceived discrimination reduced their risk of cardiac death by 86% for each level of physical activity when compared to participants who had lower perceived discrimination. The HR of 0.08 for the EDS-1 X Smoking interaction indicated the participants reduced their risk of cardiac death by 92% for each level of smoking from never smoked to current smoker. The finding for the EDS-1 X Smoking interaction did not make sense when compared to the main effects and the extant literature.

The main effects in the model indicated that current smokers had a 521% greater risk of having a cardiac death when compared to participants who never smoked [HR = 6.21; 95% CI for HR (1.14, 33.57), *p* = .033]. A review of crosstabs on the EDS-1 groups and smoking status for the participants who experienced a cardiac death indicated that of the five participants who were classified having higher perceived discrimination on the EDS-1 variable, none were classified as current smokers. The classification schema and the small number of records with a recorded cardiac death (*n* = 17 records) may have skewed the results on the EDS-1 X Smoking interaction term.

Summary

Chapter 4 began with a description of the demographics of the participants in the study. Descriptive statistics for the variables tested during inferential analysis were presented and information pertaining to required assumptions for the inferential analysis was presented and all assumptions were met. Correlational analysis was then performed on the variables used for hypothesis testing to check for bi-variate associations and multicollinearity. Inferential testing with one KMSA and a series of 5 nested Cox regression models were tested to address Aims 1 and 2 of this study. To address Aim 1, cumulative incidence of cardiovascular events of interest including; non-fatal MI, acute ischemic syndrome, coronary revascularization, cerebrovascular accident, and cardiac death, were calculated and plotted using KMSA. To assess for differences by quartile, a log-rank test was used to investigate statistically significant differences in the curves generated from the KMSA analysis for the two EDS-1 groups of (a) lower discrimination, and (b) higher discrimination. Cox regression models were then developed to investigate other factors that may have contributed to the time to event outcome. Covariates were added to the regression models in a sequential fashion. The first model was unadjusted and was used to examine the independent relationship between perceived discrimination and cardiovascular events of interest.

There was not enough evidence to indicate a statistically significant association between the predictors of perceived discrimination and cardiovascular health events in Black participants in the Heart SCORE study. Aim 1 was not supported. Model 2 (see Table 9) had the best model fit and included four variables of EDS-1, EDS-2, age, and gender. (-2 LL = 643.95) was significantly improved over the baseline model with no

predictors (-2LL = 670.39; $X^2(4) = 26.44$, $p < .0005$. The second model was also a significant improvement over the first model with 2 predictors (See Table 7; $X^2(2) = 21.76$, $p < .0005$). Main effect predictors were investigated via four additional cox regression models. The second Cox regression model was adjusted for demographic variables including sex and age. The third Cox regression model was adjusted for demographics and behavioral variables including smoking and physical activity. The fourth model was adjusted for variables in model three plus the socioeconomic variables of education and income. The fifth model was adjusted for the variables in model four plus the variable of stress. The predictor of age was statistically significant [HR = 1.07; 95% CI for HR (1.03, 1.11), $p < .005$]. The HR of 1.07 indicated that each year increase in age above the mean of 57.93 years was associated with a 7% increase in the risk of a cardiovascular event, holding the other variables constant. The predictor of gender was also statistically significant [HR = 0.46; 95% CI for HR (0.25, 0.76), $p = .003$]. The HR of 0.44 indicated that females were associated with a 54% decrease in the risk of a cardiovascular event when compared to males, holding the other variables constant

To assess for potential effect modification (Aim 2), seven interaction terms were separately added on a second step in the fifth model and included interactions of (a) EDS-1 X Gender, (b) EDS-1 X Age, (c) EDS-1 X Smoking, (d) EDS-1 X Physical Activity, (e) EDS-1 X Education, (f) EDS-1 X Income, and (g) EDS-1 X Stress.

None of the interaction term or variables in the models reached statistical significance except for variable of age in the model with all variables in model 5 step 1 and interaction of EDS-1 X age. Therefore, aim 2 was not supported as relates to the seven interaction terms tested in this study. The relationship between perceived

discrimination and cardiovascular events in Black participants was not supported by any of the tested models. A listing of the -2 log likelihood values and number of variables for each of the 5 models relating to the outcome of cardiovascular event (all five events) is presented in Table 9 and the results of the omnibus tests of significance for the 7 interactions tested in this study are presented in Table 10.

Additional sets of KMSA analyses and Cox regression models were tested to explore relationships between the predictors, covariates, and each of the five cardiac event outcomes separately. The incidence rate of all non-fatal MI events for the entire sample was 2.9%. The incidence rate for the EDS-1 lower perceived discrimination group was 3.7% and the incidence rate for the EDS-1 higher perceived discrimination group was 1.8%. The mean survival time for cardiovascular events of non-fatal MI (*N* = 19) was *M* = 4768.06 days (*SEM* = 40.47 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (*M* = 4784.73, *SEM* = 40.17) compared to the EDS-1 lower perceived discrimination group (*M* = 4722.82 days, *SEM* = 53.91 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 2.26, *p* = .133, indicating that the two EDS-1 groups did not differ in their mean times to event. The predictor of age was statistically significant [HR = 1.09; 95% CI for HR (1.02, 1.17), *p* = .011]. The HR of 1.09 indicated that each year increase in age above the mean of 57.93 years was associated with a 9% increase in the risk of a cardiovascular event of non-fatal MI, holding the other variables constant.

The incidence rate of all AIS events for the entire sample was 0.9%. The incidence rate for the EDS-1 lower perceived discrimination group was 0.8% and the

incidence rate for the EDS-1 higher perceived discrimination group was 1.1%. The mean survival time for cardiovascular events of AIS (N = 6) was M = 4904.76 days (SEM = 14.76 days). The mean survival time was greater for the EDS-1 lower discrimination group (M = 4908.19 days, SEM = 18.88 days) compared to the EDS-1 higher perceived discrimination group (M = 4834.70 days, SEM = 23.35 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 0.14, p = .712, indicating that at the two EDS-1 groups did not differ in their mean times to AIS.

A Cox regression model was also tested for the cardiovascular event of AIS. The Cox regression model was unadjusted and was used to examine the independent relationship between the perceived discrimination strata variable of EDS-1, the perceived discrimination predictor variable of EDS-1, and the outcome of cardiovascular event of non-fatal MI. The model findings are presented in Table 16 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. The model with two variables of EDS-1 and EDS-2 (-2 Log Likelihood = 63.02) was significantly improved over the baseline model with no predictors (-2 Log Likelihood = 76.97; $X^2(2) = 13.95$, p = .001. The significant finding indicates the model with the two predictors provided more information than the baseline model. However, the hazard ratio for the variable of EDS-1 was very large and had a very wide 95% confidence interval [HR = 89.29, 95% CI for HR (5,18, 1536.44). Although the model was statistically significant from the baseline model, the model structure was not good. Only six participants experienced the AIS event which may not have provided enough information to specify a good model fit. The

significance of the model was thus determined as misleading. The second through 5th models also contained large standard errors and large hazard ratios and 95% confidence intervals for the hazard ratios on many of the covariates (see Appendix III). Therefore, the models were determined as not tenable as specified and no further reporting was done for the cardiac event of AIS.

The incidence rate of all REVASC events for the entire sample was 3.9%. The incidence rate for the EDS-1 lower perceived discrimination group was 4.8% and the incidence rate for the EDS-1 higher perceived discrimination group was 2.9%. The mean survival time for cardiovascular events of REVASC (*N* = 26) was *M* = 4784.76 days (*SEM* = 31.05 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (*M* = 4763.87 days, *SEM* = 39.20 days) compared to the EDS-1 lower perceived discrimination group (*M* = 4755.29 days, *SEM* = 41.16 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 1.57, *p* = .210, indicating that at the two EDS-1 groups did not differ in their mean times to event. The syntax for the survival function curves and Cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix IV.

The second Cox regression model was adjusted for demographic variables including gender and age. Gender was coded as 0 = male and 1 = female. Age was mean centered prior to the analysis. The second regression baseline was therefore modeled with referents of males who were 57.93 years of age and had lower perceived discrimination. The model findings are presented in Table 18 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95%

confidence intervals for the hazard ratios. The second model with four variables of EDS-1, EDS-2, age, and gender (-2 LL = 312.98) was significantly improved over the baseline model with no predictors (-2LL = 325.46; $X^2(4) = 12.48$, $p = .014$. The second model was also a significant improvement over the first model with two predictors (See Table 17; $X^2(2) = 9.16$, $p = .010$).

The predictor of gender was statistically significant [HR = 0.38; 95% CI for HR (0.18, 0.84), $p = .017$]. The HR of 0.38 indicated that females were associated with a 62% decrease in the risk of a cardiovascular event of REVASC when compared to males, holding the other variables constant.

The incidence rate of all CVA events for the entire sample was 2.5%. The incidence rate for the EDS-1 lower perceived discrimination group was 2.7% and the incidence rate for the EDS-1 higher perceived discrimination group was 2.2%. The mean survival time for cardiovascular events of CVA ($N = 16$) was $M = 4968.01$ days ($SEM = 76.92$ days). The mean survival time was greater for the EDS-1 lower perceived discrimination group ($M = 4914.32$ days, $SEM = 95.65$ days) compared to the EDS-1 higher perceived discrimination group ($M = 4825.30$ days, $SEM = 21.91$ days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, $\chi^2 (1) = 0.00$, $p = .989$, indicating that at the two EDS-1 groups did not differ in their mean times to event. The syntax for the survival function curves and Cumulative hazard function plots for the entire sample and for the EDS-1 groups are presented in Appendix V.

A series of five Cox regression models were developed for the outcome of cardiovascular event of CVA. However, Models 1 and 2 were not statistically significant,

and convergence failed on Models 3, 4, and 5 when modeled as specified. None of the coefficients for the predictors or covariates in any of the five models were statistically significant, and the second step in model 5 did not run in the model. The specified models were determined to be a poor fit with the dataset and no further investigation for the outcome of the cardiovascular event of CVA was performed.

The incidence rate of all cardiac death events for the entire sample was 2.6%. The incidence rate for the EDS-1 lower perceived discrimination group was 3.2% and the incidence rate for the EDS-1 higher perceived discrimination group was 1.8%. The mean survival time for cardiovascular events of cardiac death (*N* = 17) was *M* = 5010.91 days (*SEM* = 39.93 days). The mean survival time was greater for the EDS-1 higher perceived discrimination group (*M* = 5061.80 days, *SEM* = 25.47 days) compared to the EDS-1 lower perceived discrimination group (*M* = 4898.85 days, *SEM* = 54.83 days). A log rank test was performed to investigate differences in the mean time to failure between the two EDS-1 groups. Results were not statistically significant, χ^2 (1) = 1.29, *p* = .256, indicating that at the two EDS-1 groups did not differ in their mean times to event.

The best Cox regression model for the event of cardiac death was the saturated model (with steps 1 and 2) of Model 5. A listing of the -2 log likelihood values and number of variables for each of the 6 models relating to the outcome of cardiovascular event of cardiac death presented in Table 25. The model findings for Model 5 steps 1 and 2 are presented in Table 26 and include B coefficients with associated standard errors, Wald statistics, p-values, hazard ratios, and the 95% confidence intervals for the hazard ratios. A statistically significant interaction was found between EDS-1 X Age [HR

= 1.40; 95% CI for HR (1.05, 1.86), *p* = .023]. The size and direction of the hazard ratio of 1.40 indicated that participants who experienced higher perceived discrimination as indicated on the EDS-1 variable experienced a 40% greater risk of cardiac death for each year of age above the average age, when compared to participants who scored as lower stress on the EDS-1 variable. The main effects of EDS-1 and age were also statistically significant. However, the interaction term takes precedence over the main effect in the model. Of note, the main effect of EDS-1 had a very high hazard ratio and large associated 95% confidence interval of the hazard ratio. It is conventional to include main terms in the model with the interaction terms (Tabachnick and Fidell, 2013). Therefore, the model was kept intact as specified.

Other statistically significant interactions included EDS-1 X Physical Activity [HR = 0.14; 95% CI for HR (0.02, 0.98), p = .048] and the interaction of EDS-1 X Smoking [HR = 0.08; 95% CI for HR (0.01, 0.98), p = .048]. The HR of 0.14 for the EDS-1 X Physical Activity interaction indicated that participants with higher perceived discrimination reduced their risk of cardiac death by 86% for each level of physical activity when compared to participants who had lower perceived discrimination. The HR of 0.08 for the EDS-1 X Smoking interaction indicated the participants reduced their risk of cardiac death by 92% for each level of smoking from never smoked to current smoker. The finding for the EDS-1 X Smoking interaction did not make sense when compared to the main effects and the extant literature.

The main effects in the model indicated that Current smokers had an approximate 6-fold greater risk of having a cardiac death when compared to participants who never smoked [HR = 6.21; 95% CI for HR (1.14, 33.57), *p* = .033]. A review of

crosstabs on the EDS-1 groups and smoking status for the participants who experienced a cardiac death indicated that of the five participants who were classified as having higher perceived discrimination on the EDS-1 variable, none were classified as current smokers. The classification schema and the small number of records with a recorded cardiac death (*n* = 17 records) may have skewed the results on the EDS-1 X Smoking interaction term.

Table 2. Measures of Central Tendency for the Continuous Variables of Age, Everyday Discrimination Scale Scores, and Stress Scores (*N* = 653)

Variable/Cohort	*M*	*SD*	*Mdn*	Range	α
Age in years	57.93	7.28	57.00	45 – 74	---
Everyday discrimination scale – level 1 (EDS-1)	2.08	0.45	2.10	0.70 – 3.00	.845
Everyday discrimination scale – level 2 (EDS-2)	2.39	1.88	2.00	0.00 – 8.00	.686
Cohen's Stress Scale	4.51	3.13	4.00	0.00 – 16.00	.780

Note. M = Mean*; SD* = Standard Deviation*; Mdn* = Median; α = Cronbach's alpha coefficient.

Table 3. Frequency Counts and Percentages of Categorical Demographic, Behavioral, and Socioeconomic Variables of Study (*N* = 653)

Variable/Cohort	Frequency	%
Gender		
Male	202	30.9
Female	451	69.1
Physical Activity		
Sedentary	86	13.2
Mild	225	34.5
Moderate	307	47.0
Strenuous	35	5.4
Smoking (current smoking status)		
Never smoked	274	42.0
Former smoker	288	44.1
Current smoker	91	13.9
Income		
Less than $10,000 per year	64	9.8
$10,000 to less than $20,000 per year	115	17.6
$20,000 to less than $40,000 per year	213	32.6
$40.000 to less than $80,000 per year	209	32.0
$80,000 or more a year	52	8.0
Education		
Less than high school	21	3.2
High school diploma	104	15.9
Some college	288	44.1
Bachelor's degree	124	19.0
Advanced degree	116	17.8

Table 4. Frequency Counts of Events and Measures of Central Tendency for the Time to Event for the 6 Event Type Classifications for All Study Participants (*N* = 653)

		Time to Event (In Days)			
Variable/Cohort	**Freq.**	***M***	***SD***	***Mdn***	**Range**
All Events	54	3631.14	1286.49	4396.00	9 – 4941
Acute ischemic syndrome	6	3769.22	1166.52	4403.00	191 – 4941
Cardiac death	17	3704.86	1206.51	4399.00	14 – 5115
Cerebrovascular accident	16	3775.58	1165.17	4405.00	33 – 5178
Coronary revascularization	26	3697.46	1236.59	4400.00	9 – 4941
Non-fatal myocardial infarction	19	3764.22	1188.41	4405.00	9 - 4941

Note. Freq. = Frequency Count*; M* = Mean*; SD* = Standard Deviation*; Mdn* = Median.

Table 5. Correlations for Bi-Variate Relationships of Variables Utilized for Inferential Analysis

	Variable	1	2	3	4	5	6	7	8	
1.	Cardiovascular event = yes									
2.	EDS-2	-.059	1.000							
3.	EDS-1 _group	.006	-.323**	1.000						
4	Age (mean centered)	.143**	-.166**	.072*	1.000					
5.	Smoking	.009	.023	-.044	-.027	1.000				
6.	Physical Activity	-.008	-.035	.004	-.107**	.063	1.000			
7.	Income	-.019	-.007	-.060	-.140**	-.116**	.066**	1.000		
8.	Education	-.055	.144**	-.004	-.132**	-.106**	-.048**	.418**	1.000	
9.	Stress	-.029	.223**	-.199**	-.179**3	-.038**	.125**	-.192**	.126**	1.000

Note. EDS = Everyday Discrimination Scale.
* $p < .05$
** $p < .01$

Table 6. Incident Rates and Measures of Central Tendency of Time Until Cardiovascular Event (in days) for the Entire Sample and the Two EDS-1 Groups of Study (N = 653)

Group	*N* Total	*N* Events	Incidence Rate (%)	*M*	SE *M*	95% CI for *M*		Censored	
						Lower	Upper	*N*	%
All Records	653	54	8.3	4657.16	39.52	4579.70	4734.62	599	91.7
EDS_1_group Lower Discrimination	296	25	8.4	4660.59	57.164	4548.55	4772.63	271	91.6
EDS_1_group Higher Discrimination	357	29	8.1	4555.97	51.327	4455.37	4656.57	328	91.9

Note. *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 7. Cox Regression Analysis of Cardiovascular Event as a Function of EDS-2 Predictor and EDS-1 Grouping Variables (Model 1) to Address Study Aim 1 (*N* = 653)

Variable	B	*SE* B	Wald χ^2	*p*	Hazard Ratio	95% CI for Hazard Ratio	
						Lower	Upper
EDS-2	-.140	.083	2.854	.091	.870	.74	1.02
EDS_1_group	-.210	.289	.524	.469	.810	.46	1.43

Note. CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value.

Table 8. Cox Regression Analysis of Cardiovascular Event as a Function of EDS-2 Predictor, EDS-1 Grouping Variable, and Demographic Covariates (Model 2) to Assess main effect predictors Aim 1 (*N* = 653)

Variable	B	*SE* B	Wald χ^2	*p*	Hazard Ratio	95% CI for Hazard Ratio	
						Lower	Upper
EDS-2	-.058	.084	.480	.488	.943	.800	1.112
EDS_1_group	-.113	.295	.148	.701	.893	.501	1.592
Age (mean centered)	.071	0.019	13.785	.000	1.073	1.034	1.114
Gender = Female	-.775	.277	7.807	.005	.461	.268	.794

Note. CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value. Age was mean centered for analysis at *M* = 57.93. Gender reference category = Male.

Table 9. Model Comparisons via Likelihood Ratio Tests for the Outcome of Cardiovascular Event (All Five Events; *N* = 635)

Model	-2LL	Number of Variables	Test Results		Decision
			X^2	*p*	
Baseline	670.39		---	---	---
Model 1	667..71	2	$X^2(2) = 3.06$	.217	Not improved over baseline
Model 2**	646.29	4	$X^2(4) = 24.36$ $X^2(2) = 21.30$	<.0005 <.0005	Improved over baseline Improved over Model 1
Model 3	641.15	9	$X^2(5) = 5.40$	.369	Not improved over Model 2
Model 4	637.87	17	$X^2(13) = 7.98$ $X^2(8) = 2.57$	.845 .958	Not Improved over Model 2 Not improved over Model 3
Model 5					
Step 1	637.86	18	$X^2(14) = 7.95$ $X^2(9) = 2.55$ $X^2(1) = 0.22$	.892 .980 .639	Not improved over Model 2 Not improved over Model 3 Not improved over Model 4

Note. -2LL = Negative 2 Log Likelihood Statistic; *p* = p-value.
** Best model fit of model series.

Table 10. *Results of Omnibus Tests of Model Coefficients for Step 2 (Interaction Term) Models.*

Model	Interaction Term	X^2	*df*	p-value
1	EDS1 X Age	0.859	1	.354
2	EDS1 X Gender	0.243	1	.622
3	EDS1 X Physical Activity	1.858	3	.602
4	EDS1 X Smoking	1.651	2	.438
5	EDS1 X Income	1.777	4	.777
6	EDS1 X Education	3.089	4	.543
7	EDS1 X Stress	0.426	1	.514

Note. EDS1 = Everyday Discrimination Scale - Level 1.

Table 11. Measures of Central Tendency of Time Until Cardiovascular Event: Non-fatal MI (in days) for the Entire Sample and the Two EDS-1 Groups of Study (*N* = 653)

			Incidence			95% CI for Mean		Censored	
Group	***N* Total**	***N* Events**	**Rate (%)**	***M***	**SE *M***	**Lower**	**Upper**	***N***	**%**
All Records	653	19	2.9	4768.06	40.47	4688.74	4847.32	634	97.1
EDS-1 Lower Discrimination	296	14	3.7	4722.82	53.91	4617.15	4828.48	282	95.2
EDS-1 Higher Discrimination	357	5	1.8	4784.73	40.17	4706.00	4863.46	352	98.6

Note. MI = Myocardial Infarction; *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 12. Cox Regression Analysis of Cardiovascular Event: Non-Fatal MI as a Function of EDS-2 Predictor and EDS-1 Grouping Variables: Model 1 (*N* = 653)

						95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Hazard Ratio**	**Lower**	**Upper**
EDS-2	-0.89	0.55	2.60	.107	0.41	0.14	1.21
EDS_1 _grouping	-0.41	0.58	0.50	.480	0.66	0.21	2.07

Note. MI = Myocardial Infarction; CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value.

Table 13. Cox Regression Analysis of Cardiovascular Event: Non-Fatal MI as a Function of EDS-2 Predictor, EDS-1 Grouping Variable, and Demographic Covariates: Model 2 (*N* = 653)

						95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Hazard Ratio**	**Lower**	**Upper**
EDS-2	-0.58	0.56	1.06	.302	0.56	0.19	1.68
EDS_1_grouping	-0.25	0.55	0.20	.653	0.78	0.26	2.31
Age (mean centered)	0.09	0.03	6.52	.011	1.09	1.02	1.17
Gender = Female	-0.85	0.50	2.91	.088	0.43	0.16	1.13

Note. MI = Myocardial Infarction; CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value. Age was mean centered for analysis at *M* = 57.93. Gender reference category = Male.

Table 14. Model Comparisons via Likelihood Ratio Tests for the Outcome of Cardiovascular Event: Non-Fatal MI (*N* = 635)

Model	-2LL	Number of Variables	Test Results		Decision
			X^2	*p*	
Baseline	205.01	0	---	---	---
Model 1	202.14	2	$X^2(2) = 2.87$	.238	Not improved over baseline
Model 2**	192.76	4	$X^2(4) = 12.25$ $X^2(2) = 9.38$	.016 .009	Improved over baseline Improved over Model 1
Model 3	187.47	9	$X^2(5) = 5.29$	.381	Not improved over Model 2
Model 4	175.50	17	$X^2(13) = 17.26$ $X^2(8) = 11.97$	.187 .153	Not improved over Model 2 Not improved over Model 3
Model 5					
Step 1	172.66	18	$X^2(14) = 20.10$ $X^2(9) = 14.81$ $X^2(1) = 2.84$	.127 .096 .092	Not improved over Model 2 Not improved over Model 3 Not improved over Model 4
Step 2	167.85	25	$X^2(7) = 4.82$	.682	Not improved over Model 5, step 1

Note. MI = Myocardial Infarction; -2LL = Negative 2 Log Likelihood Statistic; *p* = p-value.
** Best model fit of model series.

Table 15. Measures of Central Tendency of Time Until Cardiovascular Event: AIS (in days) for the Entire Sample and the Two EDS-1 Groups of Study (*N* = 653)

Group	*N* Total	*N* Events	Incidence Rates (%)	*M*	SE *M*	95% CI for Mean		Censored	
						Lower	Upper	*N*	%
All Records	653	6	0.9	4904.86	14.76	4875.93	4933.79	647	99.1
EDS-1 Lower Discrimination	296	3	0.8	4908.19	18.88	4871.18	4945.19	293	99.0
EDS-1 Higher Discrimination	357	3	1.1	4834.70	23.35	4788.93	4880.46	354	99.2

Note. AIS = Acute Ischemic Syndrome; *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 16. Cox Regression Analysis of Cardiovascular Event: AIS as a Function of EDS-2 Predictor and EDS-1 Grouping Variables: Model 1 (*N* = 653)

						95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Hazard Ratio**	**Lower**	**Upper**
EDS_2	1.39	0.86	2.61	.106	4.02	0.74	21.69
EDS_1_grouping	4.49	1.45	9.57	.002	89.29	5.18	1536.44

Note. AIS = Acute Ischemic Syndrome; CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value.

Table 17. Measures of Central Tendency of Time Until Cardiovascular Event: REVASC in Days for the Entire Sample and the Two EDS-1 Groups of Study (*N* = 653)

Group	*N* Total	*N* Events	Incidence Rate (%)	*M*	SE *M*	95% CI for Mean		Censored	
						Lower	Upper	*N*	%
All Records	653	26	3.9	4768.06	40.47	4688.74	4847.32	627	96.0
EDS-1 Lower Discrimination	296	18	4.8	4722.82	53.91	4617.15	4828.48	278	94.9
EDS-1 Higher Discrimination	357	8	2.9	4784.73	40.17	4706.00	4863.46	349	97.8

Note. REVASC = Coronary Revascularization; *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 18. Cox Regression Analysis of Cardiovascular Event: REVASC as a Function of EDS-2 Predictor and EDS-1 Grouping Variables: Model 1 (*N* = 653)

						95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Hazard Ratio**	**Lower**	**Upper**
EDS-2	-0.33	0.45	0.54	.465	0.72	0.30	1.74
EDS_1_grouping	0.63	0.49	1.65	.199	1.87	0.72	4.87

Note. REVASC = Coronary Revascularization; CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value.

Table 19. Cox Regression Analysis of Cardiovascular Event: REVASC as a Function of EDS-2 Predictor, EDS-1 Grouping Variable, and Demographic Covariates: Model 2 (*N* = *653*)

					Hazard	95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Ratio**	**Lower**	**Upper**
EDS-2	-0.06	0.46	0.02	.894	0.94	0.38	2.32
EDS_1_grouping	0.66	0.47	1.98	.160	1.93	0.77	4.81
Age (mean centered)	0.05	0.03	3.44	.064	1.05	0.99	1.11
Gender = Female	-0.96	0.40	5.68	.017	0.38	0.18	0.84

Note: REVASC = Coronary Revascularization; CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value. Age was mean centered for analysis at *M* = 57.93. Gender reference category = Male.

Table 20. Model Comparisons via Likelihood Ratio Tests for the Outcome of Cardiovascular Event: REVASC (*N* = 635)

Model	-2LL	Number of Variables	Test Results		Decision
			X^2	*p*	
Baseline	325.46	0	---		---
Model 1	322.14	2	$X^2(2) = 3.32$	.190	Not improved over baseline
Model 2**	312.98	4	$X^2(4) = 12.48$ $X^2(2) = 9.16$	.014 .010	Improved over baseline Improved over Model 1
Model 3	307.55	9	$X^2(5) = 5.43$	.366	Not improved over Model 2
Model 4	302.87	17	$X^2(13) = 10.11$ $X^2(8) = 4.68$	.694 .791	Not improved over Model 2 Not improved over Model 3
Model 5					
Step 1	302.87	18	$X^2(14) = 10.11$ $X^2(9) = 4.68$ $X^2(1) = 0$	.754 .861 1.00	Not improved over Model 2 Not improved over Model 3 Not improved over Model 4
Step 2	290.61	25	$X^2(7) = 12.26$	.092	Not improved over Model 5, step 1

Note. REVASC = Coronary Revascularization; -2LL = Negative 2 Log Likelihood Statistic; *p* = p-value.
** Best model fit of model series.

Table 21. Measures of Central Tendency of Time Until Cardiovascular Event: CVA in Days for the Entire Sample and the Two EDS-1 Groups of Study (*N* = 653)

Group	*N* Total	*N* Events	Incidence Rate (%)	*M*	SE *M*	95% CI for Mean		Censored	
						Lower	Upper	*N*	%
All Records	653	16	2.5	4968.01	76.92	4817.24	5118.78	637	97.5
EDS-1 Lower Discrimination	296	10	2.7	4914.32	95.65	4726.86	5101.79	286	96.6
EDS-1 Higher Discrimination	357	6	2.2	4825.30	21.91	4782.37	5118.78	351	98.3

Note. CVA = Cerebrovascular Accident; *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 22. Measures of Central Tendency of Time Until Cardiovascular Event: Cardiac Death (in days) for the Entire Sample and the Two EDS-1 Groups of Study (*N* = 653)

Group	*N* Total	*N* Events	Incidence Rate (%)	*M*	SE *M*	95% CI for Mean		Censored	
						Lower	Upper	*N*	%
All Records	653	17	2.6	5010.91	39.93	4932.64	5089.17	636	97.4
EDS-1 Lower Discrimination	296	12	3.2	4898.85	54.83	4791.38	5006.32	284	95.9
EDS-1 Higher Discrimination	357	5	1.8	5061.80	25.47	5011.87	5111.72	352	98.6

Note. *N* = Number of Participants; *M* = Mean; *SE* = Standard Error; *Mdn* = Median; *CI* = Confidence Interval.

Table 23. Cox Regression Analysis of Cardiovascular Event: Cardiac Death as a Function of EDS-2 Predictor and EDS-1 Grouping Variables: Model 1 (*N* = 653)

Variable	B	*SE* B	Wald χ^2	*p*	Hazard Ratio	95% CI for Hazard Ratio	
						Lower	Upper
EDS-2	-0.58	0.56	1.07	.302	0.56	0.19	1.69
EDS_1_grouping	0.05	0.59	0.01	.936	1.05	0.33	3.33

Note: CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value.

Table 24. Cox Regression Analysis of Cardiovascular Event: Cardiac Death as a Function of EDS-2 Predictor, EDS-1 Grouping Variable, and Demographic Covariates: Model 2 (*N* = 653)

Variable	B	*SE* B	Wald χ^2	*p*	Hazard Ratio	95% CI for Hazard Ratio	
						Lower	Upper
EDS-2	-0.15	0.57	0.07	.799	0.86	0.28	2.66
EDS_1_grouping	0.03	0.56	<0.01	.963	1.03	0.35	3.06
Age (mean centered)	0.14	0.04	13.97	<.0005	1.15	1.07	1.24
Gender = Female	-0.83	0.50	2.77	.096	0.44	0.16	1.16

Note. CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value. Age was mean centered for analysis at *M* = 57.93. Gender reference category = Male.

Table 25. Model Comparisons via Likelihood Ratio Tests for the Outcome of Cardiovascular Event: Cardiac Death (*N* = 635)

Model	-2LL	Number of Variables	Test Results		
			X^2	*p*	Decision
Baseline	199.01	0	---	---	---
Model 1	197.66	2	$X^2(2) = 1.35$	.509	Not improved over baseline
Model 2	179.04	4	$X^2(4) = 19.97$	.001	Improved over baseline
			$X^2(2) = 18.62$	<.0005	Improved over Model 1
Model 3	168.09	9	$X^2(5) = 10.95$	.052	Not improved over Model 2
Model 4	158.25	17	$X^2(13) = 20.79$	.077	Not improved over Model 2
			$X^2(8) = 9.84$	.276	Not improved over Model 3
Model 5					
Step 1	157.22	18	$X^2(14) = 21.82$	.082	Not improved over Model 2
			$X^2(9) = 10.87$	.285	Not improved over Model 3
			$X^2(1) = 1.03$	.310	Not improved over Model 4
Step 2**	141.77	25	$X^2(21) = 37.27$	.016	Improved over Model 2
			$X^2(16) = 26.32$	.0496	Improved over Model 3
			$X^2(8) = 16.48$	.036	Improved over Model 4
			$X^2(7) = 15.45$	.031	Improved over Model 5, step 1

Note. -2LL = Negative 2 Log Likelihood Statistic; *p* = p-value.
** Best model fit of model series.

Table 26. Cox Regression Analysis of Cardiovascular Event as a Function of EDS-2 Predictor, EDS-1 Grouping Variable, and Demographic Covariates to Address Study Aim 2 (*N* = 653)

						95% CI for Hazard Ratio	
Variable	**B**	***SE* B**	**Wald χ^2**	***p***	**Hazard Ratio**	**Lower**	**Upper**
Step 1							
EDS-2	12.26	5.62	4.76	.029	2.10×10^5	3.46	1.05×10^{12}
EDS_1_grouping	0.39	0.75	0.26	.608	1.47	0.34	6.41
Age (mean centered)	0.14	0.05	7.10	.008	1.15	1.04	1.28
Gender = female	-1.10	0.69	2.54	.111	0.33	0.09	1.29
Smoking = former smoker	-0.29	0.81	0.13	.722	0.75	0.15	3.65
Smoking = current smoker	1.83	0.86	4.50	.034	6.21	1.15	33.57
Physical activity = mild	-1.68	0.81	4.26	.039	0.19	0.04	0.91
Physical activity = moderate	-1.51	0.83	3.32	.068	0.22	0.04	1.12
Physical activity = strenuous	-0.94	1.34	0.49	.482	0.39	0.03	5.40
Income $10K to < $20K	0.02	1.07	<0.01	.988	1.02	0.12	8.33
Income $20K to < $40K	1.10	1.17	0.88	.348	3.00	0.30	29.90
Income $40K to < $80K	0.41	1.35	0.09	.764	1.50	0.11	21.22
Income $80K or more	-12.18	498.86	<0.01	.981	<0.01	<0.01	Unspecified
Education = high school	-2.87	1.50	3.66	.056	0.06	<0.01	1.07
Education = some college	-0.77	1.04	0.55	.458	0.46	0.06	3.53
Education = Bachelor's deg.	-2.17	1.52	2.04	.153	0.11	0.01	2.25
Education = Advanced deg.	-0.82	1.40	0.35	.557	0.44	0.03	6.84
Stress	0.05	0.12	0.17	.678	1.05	0.83	1.34

Table 26 (Continued)

Variable	B	*SE* B	Wald χ^2	*p*	Hazard Ratio	95% CI for Hazard Ratio	
						Lower	Upper
Step 2							
EDS-1 X Gender	-1.40	1.56	0.81	.368	0.25	0.01	5.20
EDS-1 X Age	0.33	0.15	5.17	.023	1.40	1.05	1.86
EDS-1 X Education	-1.21	0.70	3.00	.083	0.30	0.08	1.17
EDS-1 X Income	-1.27	0.88	2.08	.149	0.28	0.05	1.58
EDS-1 X Physical Activity	-1.94	0.98	3.93	.048	0.14	0.02	0.98
EDS-1 X Stress	0.21	0.25	0.74	.391	1.23	0.76	1.99
EDS-1 X Smoking	-2.50	1.26	3.92	.048	0.08	0.01	0.98

Note. CI = Confidence Interval; *SE* B = Standard Error of B; *p* = p-value. Age was mean centered for analysis at *M* = 57.93. Gender reference category = Male. Smoking reference category = Never smoked. Physical activity reference category = Sedentary. Income reference category = Less than $10K per year. Education reference category = Less than high school.

CHAPTER FIVE:

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

In Chapter five, the results of this secondary analysis are synthesized in the context of study theory and review of the literature. This chapter is divided into subheadings including; discussion and conclusion, implications of findings and recommendations, limitations, and strengths of the study. The purpose of this secondary data analysis was to examine whether there was a relationship between perceived discrimination and cardiovascular health outcomes, including stroke, non-fatal myocardial infarction (MI), acute ischemic syndrome, coronary revascularization, and cardiac death, and how demographic, socioeconomic, behavioral and psychosocial variables may moderate that relationship.

Specific Aims

1. To examine the impact of perceived discrimination on cardiovascular health outcomes including; non-fatal myocardial infarction, cerebrovascular accident, acute ischemic syndrome, coronary revascularization and cardiac death in Blacks in the Heart SCORE Study.
2. To evaluate whether demographic (age, sex), socioeconomic (education and income) behavioral (smoking and physical) and psychological (Stress) variables moderate the relationship between perceived discrimination and cardiovascular health outcomes in Blacks in the Heart SCORE study.

Cox regression and Kaplan Meier Methods were utilized to model the relationship between perceived discrimination and cardiovascular events among Blacks in the Heart SCORE study.

Discussion and Recommendations

In reference to Aim 1, and consistent with Dunlay et al. (2017) this study found no sufficient evidence to indicate a statistically significant association between the predictor of perceived discrimination and cardiovascular health events in Black participants in the Heart SCORE study. In contrast to this finding, Everson-Rose et al. (2015) found a modest independent association of perceived discrimination and cardiovascular outcomes which persisted after adjustment for potential confounders. Nonetheless, this study adds to the limited empirical evidence related to the association of perceived discrimination and cardiovascular outcomes. Of note, this study as well as the study conducted by Dunlay et al. (2017) focused solely on Blacks and had similar findings while the study by Everson-Rose et al. (2015) used a multiethnic sample with about 26% Blacks in the study sample. Perhaps the two studies that used purely Black samples are a better reflection of the association of perceived discrimination and cardiac outcomes among Blacks. Model 2 (see Table 8) had the best model fit and included four variables of EDS-1, EDS-2, age, and gender. Model 2 (-2 LL = 643.18) was significantly improved over the baseline model with no predictors [-2LL = 670.39; X 2 (5) = 27.21, $p < .0005$]. The second model was also a significant improvement over the first model with 3 predictors (See Table 7; X 2 (2) = 22.53, $p < .0005$). The predictor of age was statistically significant [HR = 1.07; 95% CI for HR (1.03, 1.11), p < .005]. The HR of 1.07 indicated that each year increase in age above the mean of 57.93 years was

associated with a 7% increase in the risk of a cardiovascular event, holding the other variables constant. This is consistent with previous studies of cardiovascular risk factors that have associated aging with increased risk for CVD morbidity and mortality (Lakatta, 2002), ischemic heart disease (Ennezat et al., 2008), hypertension (Krieger & Sidney, 1996; Lim et al., 2012; Lockwood et al., 2018; Sims et al., 2012; Williams & Neighbors, 2001), and myocardial infarction (Shih et al., 2011). The predictor of gender was also statistically significant [HR = 0.44; 95% CI for HR (0.25, 0.76), $p < .003$). This is also consistent with previous studies that have shown men to be at an increased risk for myocardial infarction and CHD compared to women (Gosswald et al., 2013). Everson-Rose et al. (2015), also found gender to interact with perceived discrimination with men at an increased risk of cardiac outcomes with each increase in standard deviation score.

There are several possible reasons that could contribute to the lack of association between perceived discrimination and cardiovascular events among Blacks as found in this study. First, while Blacks are disproportionately affected by cardiovascular risk factors such as HTN, diabetes, smoking, and obesity (Kanchi et al., 2018; Lin et al., 2018), they are also more likely to be aware of their HTN diagnosis and to be on treatment for the same compared to their white counterparts (Cutler et al., 2008; Ostchega et al., 2007; Yoon et al., 2015). Second, there could be other confounders of the relationship between perceived discrimination and cardiovascular events that were not included in this study. For example, in their metanalytic review of association of perceived discrimination and health and investigation of possible pathways through which perceived discrimination may influence health outcomes,

Pascoe and Smart Richman (2009) found social support and coping styles to have a buffering effect on health outcomes.

Third, the use of EDS which captures the frequency of everyday experience of unfair treatment over the last week or month as the only measure for perceived discrimination in this study may not have captured the intended effect of perceived discrimination on cardiovascular events given that cardiovascular disease develop sub-clinically over a longer period of time than when a person is exposed to the discrimination. Perhaps, a more appropriate measure to capture the complex chain between perceived discrimination and cardiovascular event would be one that measures the chronicity or average change over time of perceived discrimination and its association of cardiovascular events (Dunlay et al., 2017). Fourth, this study had a relatively low number of events (54 out of possible 653) which may have affected the results of this study. Lastly, previous studies have shown that Blacks who experienced higher levels of discrimination were also likely to engage in healthy activities such as diet and physical activity (Dubbert et al., 2010). In relation to study theory, Pascoe and Smart Richman (2009) postulated three potential pathways through which perceived discrimination could influence health outcomes. The first pathway is direct effect of discriminatory acts on health. The second potential pathway posits that the relationship of perceived discrimination to health has the potential to be mediated through stress response to discriminatory events. Third, health outcomes can be influenced via health behaviors. Participants who experience discriminatory acts may engage in unhealthy behaviors or fail to participate in healthy behaviors as a way of coping with the stress related to discrimination. Previous studies have also suggested that younger Blacks

who experience higher perceived discrimination were also more likely to engage in physical activity as a coping mechanism for discrimination related stress (Borrell, Kiefe, Diez-Roux, Williams, & Gordon-Larsen, 2013) . In this study, 82% of participants reported that they engaged in mild to moderate physical activity. However, the level of perceived discrimination in this study was generally low which may have affected the potential effects of physical activity on the association of perceived discrimination and cardiovascular events.

Implications of Findings and Recommendations

The purpose of this secondary data analysis was to examine whether there was a relationship between perceived discrimination and cardiovascular health outcomes including: stroke, non-fatal myocardial infarction (MI), acute ischemic syndrome, coronary revascularization, and cardiac death, and how demographic, socioeconomic, behavioral and psychosocial variables may moderate this relationship. Consistent with Dunlay et al. (2017), this study did not find perceived discrimination to be a predictor of development of any of the cardiovascular events that were examined. In relation to potential moderating effects of socioeconomic factors on the relationship between perceived discrimination and cardiovascular events, it may be more beneficial to examine institutional forms of discrimination that are more likely to affect socioeconomic factors such as income and education. More precisely, discrimination in job promotion, treatment by employers and academic institutions, unfair or unrealistic job and academic expectations are more likely to affect an individual's socioeconomic status and may consequently have a greater impact on cardiovascular outcomes compared to interpersonal forms of discrimination such as unfair treatment at a restaurant. Future

studies that focus on relationship between perceived discrimination and cardiovascular outcomes should include multiple measures of perceived discrimination to better capture the chronicity of perceived discrimination at it relates to cardiovascular outcomes.

Previous studies have shown an association of cardiovascular risk factors such as HTN, obesity, smoking and diabetes mellitus II and perceived discrimination (Kanchi et al., 2018), however the reasons that make it possible for perceived discrimination to influence cardiovascular risk factors but not result into cardiovascular event remains unclear. Future research should seek to unravel the mechanistic pathways that make this possible. Mediated pathways through stress-response and health behaviors should be explored in future studies.

Limitations

This study was a secondary data analysis and by design had a few limitations worth acknowledging. First, this study utilized data from the Heart SCORE study and was therefore restricted by the available data. The Heart SCORE study used the Everyday Discrimination Scale (EDS) as the only measure of perceived discrimination which made it difficult to compare multiple measures. The use of multiple measures of perceived discrimination particularly one that captures the chronicity or average change over time of perceived discrimination is recommended in future studies as this may better reflect the true relationship between perceived discrimination and cardiovascular events. The EDS captures the frequency of day to day discrimination over the past week or month which may not capture the long-term effects of perceived discrimination on cardiovascular events which develop over years (Dunlay et al., 2017). This may be

one of the reasons why perceived discrimination is associated with cardiovascular risk factors such as HTN and smoking (Kanchi et al., 2018) but not cardiovascular events. Second, the Heart SCORE study collected data for over 600 variables with many potential confounders, moderators and mediators of the relationship between perceived discrimination and cardiovascular events. While this study included several of potential moderators, it is still possible that some confounders that affects this relationship were not included in this study. In addition, the small number of events (54 out of 653) observed among blacks in the Heart SCORE study may have affected the findings of the study. Lastly, perceived discrimination was generally low among the Heart SCORE study participants with a mean of 4.51 out of possible 16 and a standard deviation of 3.13. Perhaps the findings would be different in a population with higher levels of perceived discrimination.

Strengths

This study used data from the Heart SCORE study which is a longitudinal study with data collected for over a decade with a large sample of Blacks. While power and sample size analysis for this study showed that 410 participants were required to achieve power of 80%, this study included 653 participants making the findings reliable.

REFERENCES

Achuko, O., Walker, R. J., Campbell, J. A., Dawson, A. Z., & Egede, L. E. (2016). Pathways Between Discrimination and Quality of Life in Patients with Type 2 Diabetes. *Diabetes Technol Ther, 18*(3), 151-158. doi:10.1089/dia.2015.0305

Adams, V., Reich, B., Uhlemann, M., & Niebauer, J. (2017). Molecular effects of exercise training in patients with cardiovascular disease: focus on skeletal muscle, endothelium, and myocardium. *Am J Physiol Heart Circ Physiol, 313*(1), H72-h88. doi:10.1152/ajpheart.00470.2016

Ahmed, A. T., Mohammed, S. A., & Williams, D. R. (2007). Racial discrimination & health: pathways & evidence. *Indian J Med Res, 126*(4), 318-327.

Ainsworth, B. E., Jacobs, D. R., Jr., & Leon, A. S. (1993). Validity and reliability of self-reported physical activity status: the Lipid Research Clinics questionnaire. *Med Sci Sports Exerc, 25*(1), 92-98.

Aiyer, A. N., Kip, K. E., Mulukutla, S. R., Marroquin, O. C., Hipps, L., Jr., & Reis, S. E. (2007). Predictors of significant short-term increases in blood pressure in a community-based population. *Am J Med, 120*(11), 960-967. doi:10.1016/j.amjmed.2007.06.021

Akintoye, E., Mahmoud, K., Shokr, M., Sandio, A., Mallikethi-Reddy, S., Sheikh, M., . . . Afonso, L. (2018). Racial/ethnic differences in the prognostic utility of left ventricular mass index for incident cardiovascular disease. *Clin Cardiol, 41*(4), 502-509. doi:10.1002/clc.22914

Al Kibria, G. M. (2019). Racial/ethnic disparities in prevalence, treatment, and control of hypertension among US adults following application of the 2017 American College of Cardiology/American Heart Association guideline. *Prev Med Rep, 14*, 100850. doi:10.1016/j.pmedr.2019.100850

Albus, C., Waller, C., Fritzsche, K., Gunold, H., Haass, M., Hamann, B., . . . Herrmann-Lingen, C. (2019). Significance of psychosocial factors in cardiology: update 2018 : Position paper of the German Cardiac Society. *Clin Res Cardiol*. doi:10.1007/s00392-019-01488-w

Assari, S., Mistry, R., Lee, D. B., Caldwell, C. H., & Zimmerman, M. A. (2019). Perceived Racial Discrimination and Marijuana Use a Decade Later; Gender Differences Among Black Youth. *Front Pediatr, 7*, 78. doi:10.3389/fped.2019.00078

Bambs, C., Kip, K. E., Dinga, A., Mulukutla, S. R., Aiyer, A. N., & Reis, S. E. (2011). Low prevalence of "ideal cardiovascular health" in a community-based population: the heart strategies concentrating on risk evaluation (Heart SCORE) study. *Circulation, 123*(8), 850-857. doi:10.1161/circulationaha.110.980151

Bambs, C. E., Kip, K. E., Mulukutla, S. R., Aiyer, A. N., Johnson, C., McDowell, L. A., . . . Reis, S. E. (2013). Sociodemographic, clinical, and psychological factors associated with attrition in a prospective study of cardiovascular prevention: the Heart Strategies Concentrating on Risk Evaluation study. *Ann Epidemiol, 23*(6), 328-333. doi:10.1016/j.annepidem.2013.02.007

Bao, A. M., Meynen, G., & Swaab, D. F. (2008). The stress system in depression and neurodegeneration: focus on the human hypothalamus. *Brain Res Rev, 57*(2), 531-553. doi:10.1016/j.brainresrev.2007.04.005

Bavishi, C., Goel, S., & Messerli, F. H. (2016). Isolated Systolic Hypertension: An Update After SPRINT. *Am J Med, 129*(12), 1251-1258. doi:10.1016/j.amjmed.2016.08.032

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., . . . Muntner, P. (2017). Heart Disease and Stroke Statistics-2017 Update: A Report From the American Heart Association. *Circulation, 135*(10), e146-e603. doi:10.1161/cir.0000000000000485

Benjamin, E. J., Muntner, P., Alonso, A., Bittencourt, M. S., Callaway, C. W., Carson, A. P., . . . Virani, S. S. (2019). Heart Disease and Stroke Statistics-2019 Update: A Report From the American Heart Association. *Circulation, 139*(10), e56-e528. doi:10.1161/cir.0000000000000659

Benjamin, E. J., Virani, S. S., Callaway, C. W., Chamberlain, A. M., Chang, A. R., Cheng, S., . . . Muntner, P. (2018). Heart Disease and Stroke Statistics-2018 Update: A Report From the American Heart Association. *Circulation, 137*(12), e67-e492. doi:10.1161/cir.0000000000000558

Bennett, G. G., Wolin, K. Y., Robinson, E. L., Fowler, S., & Edwards, C. L. (2005). Perceived racial/ethnic harassment and tobacco use among African American young adults. *Am J Public Health, 95*(2), 238-240. doi:10.2105/ajph.2004.037812

Bibbins-Domingo, K., Pletcher, M. J., Lin, F., Vittinghoff, E., Gardin, J. M., Arynchyn, A., . . . Hulley, S. B. (2009). Racial differences in incident heart failure among young adults. *N Engl J Med, 360*(12), 1179-1190. doi:10.1056/NEJMoa0807265

Blom, K., Baker, B., How, M., Dai, M., Irvine, J., Abbey, S., . . . Tobe, S. W. (2014). Hypertension analysis of stress reduction using mindfulness meditation and yoga: results from the HARMONY randomized controlled trial. *Am J Hypertens, 27*(1), 122-129. doi:10.1093/ajh/hpt134

Bonaca, M. P., Wiviott, S. D., Braunwald, E., Murphy, S. A., Ruff, C. T., Antman, E. M., & Morrow, D. A. (2012). American College of Cardiology/American Heart Association/European Society of Cardiology/World Heart Federation universal definition of myocardial infarction classification system and the risk of cardiovascular death: observations from the TRITON-TIMI 38 trial (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition With Prasugrel-Thrombolysis in Myocardial Infarction 38). *Circulation, 125*(4), 577-583. doi:10.1161/circulationaha.111.041160

Borrell, L. N., Kiefe, C. I., Diez-Roux, A. V., Williams, D. R., & Gordon-Larsen, P. (2013). Racial discrimination, racial/ethnic segregation, and health behaviors in the CARDIA study. *Ethn Health, 18*(3), 227-243. doi:10.1080/13557858.2012.713092

Britt-Spells, A. M., Slebodnik, M., Sands, L. P., & Rollock, D. (2018). Effects of Perceived Discrimination on Depressive Symptoms Among Black Men Residing in the United States: A Meta-Analysis. *Am J Mens Health, 12*(1), 52-63. doi:10.1177/1557988315624509

Brondolo, E., Brady, N., Thompson, S., Tobin, J. N., Cassells, A., Sweeney, M., . . . Contrada, R. J. (2008). PERCEIVED RACISM AND NEGATIVE AFFECT: ANALYSES OF TRAIT AND STATE MEASURES OF AFFECT IN A COMMUNITY SAMPLE. *J Soc Clin Psychol, 27*(2), 150-173. doi:10.1521/jscp.2008.27.2.150

Bull, F. C., Maslin, T. S., & Armstrong, T. (2009). Global physical activity questionnaire (GPAQ): nine country reliability and validity study. *J Phys Act Health, 6*(6), 790-804.

Campisi, J. (2013). Aging, cellular senescence, and cancer. *Annu Rev Physiol, 75*, 685-705. doi:10.1146/annurev-physiol-030212-183653

Carnethon, M. R., Pu, J., Howard, G., Albert, M. A., Anderson, C. A. M., Bertoni, A. G., . . . Yancy, C. W. (2017). Cardiovascular Health in African Americans: A Scientific Statement From the American Heart Association. *Circulation, 136*(21), e393-e423. doi:10.1161/cir.0000000000000534

Carnlof, C., Iwarzon, M., Jensen-Urstad, M., Gadler, F., & Insulander, P. (2017). Women with PSVT are often misdiagnosed, referred later than men, and have more symptoms after ablation. *Scand Cardiovasc J, 51*(6), 299-307. doi:10.1080/14017431.2017.1385837

Caspersen, C. J., Powell, K. E., & Christenson, G. M. (1985). Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep, 100*(2), 126-131.

Chamberlain, A. M., Agarwal, S. K., Folsom, A. R., Duval, S., Soliman, E. Z., Ambrose, M., . . . Alonso, A. (2011). Smoking and incidence of atrial fibrillation: results from the Atherosclerosis Risk in Communities (ARIC) study. *Heart Rhythm, 8*(8), 1160-1166. doi:10.1016/j.hrthm.2011.03.038

Chandrashekhar, Y., & Anand, I. S. (1991). Exercise as a coronary protective factor. *American Heart Journal, 122*(6), 1723-1739. doi:https://doi.org/10.1016/0002-8703(91)90290-X

Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annu Rev Physiol, 67*, 259-284. doi:10.1146/annurev.physiol.67.040403.120816

Cheezum, M. K., Kim, A., Bittencourt, M. S., Kassop, D., Nissen, A., Thomas, D. M., . . . Villines, T. C. (2017). Association of tobacco use and cessation with coronary atherosclerosis. *Atherosclerosis, 257*, 201-207. doi:10.1016/j.atherosclerosis.2016.11.016

Chilunga, F. P., Boateng, D., Henneman, P., Beune, E., Requena-Mendez, A., Meeks, K., . . . Agyemang, C. (2019). Perceived discrimination and stressful life events are associated with cardiovascular risk score in migrant and non-migrant populations: The RODAM study. *Int J Cardiol, 286*, 169-174. doi:10.1016/j.ijcard.2018.12.056

Clark, R., Coleman, A. P., & Novak, J. D. (2004). Brief report: Initial psychometric properties of the everyday discrimination scale in black adolescents. *J Adolesc, 27*(3), 363-368. doi:10.1016/j.adolescence.2003.09.004

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *J Health Soc Behav, 24*(4), 385-396.

Coley, S. L., Mendes de Leon, C. F., Ward, E. C., Barnes, L. L., Skarupski, K. A., & Jacobs, E. A. (2017). Perceived discrimination and health-related quality-of-life: gender differences among older African Americans. *Qual Life Res, 26*(12), 3449-3458. doi:10.1007/s11136-017-1663-9

Csordas, A., & Bernhard, D. (2013). The biology behind the atherothrombotic effects of cigarette smoke. *Nat Rev Cardiol, 10*(4), 219-230. doi:10.1038/nrcardio.2013.8

Cuffee, Y., Ogedegbe, C., Williams, N. J., Ogedegbe, G., & Schoenthaler, A. (2014). Psychosocial risk factors for hypertension: an update of the literature. *Curr Hypertens Rep, 16*(10), 483. doi:10.1007/s11906-014-0483-3

Cutler, J. A., Sorlie, P. D., Wolz, M., Thom, T., Fields, L. E., & Roccella, E. J. (2008). Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988-1994 and 1999-2004. *Hypertension, 52*(5), 818-827. doi:10.1161/hypertensionaha.108.113357

Dampney, R. A., Michelini, L. C., Li, D. P., & Pan, H. L. (2018). Regulation of sympathetic vasomotor activity by the hypothalamic paraventricular nucleus in normotensive and hypertensive states. *Am J Physiol Heart Circ Physiol, 315*(5), H1200-h1214. doi:10.1152/ajpheart.00216.2018

Danelia, M. (2006). Education and coronary heart disease. *Georgian Med News*(133), 51-52.

Davis, S. K., Gebreab, S., Quarells, R., & Gibbons, G. H. (2014). Social determinants of cardiovascular health among black and white women residing in Stroke Belt and Buckle regions of the South. *Ethn Dis, 24*(2), 133-143.

Dawson, A. Z., Walker, R. J., Campbell, J. A., & Egede, L. E. (2016). Validation of theoretical pathway between discrimination, diabetes self-care and glycemic control. *J Diabetes Complications, 30*(5), 858-863. doi:10.1016/j.jdiacomp.2016.03.014

Di Chiara, T., Scaglione, A., Corrao, S., Argano, C., Pinto, A., & Scaglione, R. (2015). Association between low education and higher global cardiovascular risk. *J Clin Hypertens (Greenwich), 17*(5), 332-337. doi:10.1111/jch.12506

Dolezsar, C. M., McGrath, J. J., Herzig, A. J. M., & Miller, S. B. (2014). Perceived racial discrimination and hypertension: a comprehensive systematic review. *Health Psychol, 33*(1), 20-34. doi:10.1037/a0033718

Domingueti, C. P., Dusse, L. M., Carvalho, M., de Sousa, L. P., Gomes, K. B., & Fernandes, A. P. (2016). Diabetes mellitus: The linkage between oxidative stress, inflammation, hypercoagulability and vascular complications. *J Diabetes Complications, 30*(4), 738-745. doi:10.1016/j.jdiacomp.2015.12.018

Dubbert, P. M., Robinson, J. C., Sung, J. H., Ainsworth, B. E., Wyatt, S. B., Carithers, T., . . . Taylor, H., Jr. (2010). Physical activity and obesity in African Americans: the Jackson Heart Study. *Ethn Dis, 20*(4), 383-389.

Dunlay, S. M., Lippmann, S. J., Greiner, M. A., O'Brien, E. C., Chamberlain, A. M., Mentz, R. J., & Sims, M. (2017). Perceived Discrimination and Cardiovascular Outcomes in Older African Americans: Insights From the Jackson Heart Study. *Mayo Clin Proc, 92*(5), 699-709. doi:10.1016/j.mayocp.2017.01.024

Ennezat, P. V., Lamblin, N., Mouquet, F., Tricot, O., Quandalle, P., Aumegeat, V., . . . Bauters, C. (2008). The effect of ageing on cardiac remodelling and hospitalization for heart failure after an inaugural anterior myocardial infarction. *Eur Heart J, 29*(16), 1992-1999. doi:10.1093/eurheartj/ehn267

Erqou, S., Echouffo-Tcheugui, J. B., Kip, K. E., Aiyer, A., & Reis, S. E. (2017). Association of cumulative social risk with mortality and adverse cardiovascular disease outcomes. *BMC Cardiovascular Disorders, 17*(1), 110. doi:10.1186/s12872-017-0539-9

Everson-Rose, S. A., Lutsey, P. L., Roetker, N. S., Lewis, T. T., Kershaw, K. N., Alonso, A., & Diez Roux, A. V. (2015). Perceived Discrimination and Incident Cardiovascular Events: The Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol, 182*(3), 225-234. doi:10.1093/aje/kwv035

Falkner, B., Sherif, K., Sumner, A., & Kushner, H. (1999). Hyperinsulinism and sex hormones in young adult African Americans. *Metabolism, 48*(1), 107-112.

Feldman, R. H., & Fulwood, R. (1999). The three leading causes of death in African Americans: barriers to reducing excess disparity and to improving health behaviors. *J Health Care Poor Underserved, 10*(1), 45-71.

Ferdinand, K. C., Yadav, K., Nasser, S. A., Clayton-Jeter, H. D., Lewin, J., Cryer, D. R., & Senatore, F. F. (2017). Disparities in hypertension and cardiovascular disease in blacks: The critical role of medication adherence. *J Clin Hypertens (Greenwich), 19*(10), 1015-1024. doi:10.1111/jch.13089

Franklin, B. A., Brinks, J., Berra, K., Lavie, C. J., Gordon, N. F., & Sperling, L. S. (2018). Using Metabolic Equivalents in Clinical Practice. *Am J Cardiol, 121*(3), 382-387. doi:10.1016/j.amjcard.2017.10.033

Fransson, E. I., Nyberg, S. T., Heikkila, K., Alfredsson, L., Bjorner, J. B., Borritz, M., . . . Kivimaki, M. (2015). Job strain and the risk of stroke: an individual-participant data meta-analysis. *Stroke, 46*(2), 557-559. doi:10.1161/strokeaha.114.008019

Gianaros, P. J., & Jennings, J. R. (2018). Host in the machine: A neurobiological perspective on psychological stress and cardiovascular disease. *Am Psychol, 73*(8), 1031-1044. doi:10.1037/amp0000232

Gianaros, P. J., Sheu, L. K., Matthews, K. A., Jennings, J. R., Manuck, S. B., & Hariri, A. R. (2008). Individual differences in stressor-evoked blood pressure reactivity vary with activation, volume, and functional connectivity of the amygdala. *J Neurosci, 28*(4), 990-999. doi:10.1523/jneurosci.3606-07.2008

Gillespie, C. D., Wigington, C., & Hong, Y. (2013). Coronary heart disease and stroke deaths - United States, 2009. *MMWR Suppl, 62*(3), 157-160.

Ginty, A. T., Kraynak, T. E., Fisher, J. P., & Gianaros, P. J. (2017). Cardiovascular and autonomic reactivity to psychological stress: Neurophysiological substrates and links to cardiovascular disease. *Auton Neurosci, 207*, 2-9. doi:10.1016/j.autneu.2017.03.003

Go, A. S., Mozaffarian, D., Roger, V. L., Benjamin, E. J., Berry, J. D., Blaha, M. J., . . . Turner, M. B. (2014). Executive summary: heart disease and stroke statistics--2014 update: a report from the American Heart Association. *Circulation, 129*(3), 399-410. doi:10.1161/01.cir.0000442015.53336.12

Goldstein, K. M., Melnyk, S. D., Zullig, L. L., Stechuchak, K. M., Oddone, E., Bastian, L. A., . . . Bosworth, H. B. (2014). Heart matters: Gender and racial differences cardiovascular disease risk factor control among veterans. *Womens Health Issues, 24*(5), 477-483. doi:10.1016/j.whi.2014.05.005

Gong, Q., & Schaubel, D. E. (2018). Tobit regression for modeling mean survival time using data subject to multiple sources of censoring. *Pharm Stat, 17*(2), 117-125. doi:10.1002/pst.1844

Goosby, B. J., Malone, S., Richardson, E. A., Cheadle, J. E., & Williams, D. T. (2015). Perceived discrimination and markers of cardiovascular risk among low-income African American youth. *Am J Hum Biol, 27*(4), 546-552. doi:10.1002/ajhb.22683

Gosswald, A., Schienkiewitz, A., Nowossadeck, E., & Busch, M. A. (2013). [Prevalence of myocardial infarction and coronary heart disease in adults aged 40-79 years in Germany: results of the German Health Interview and Examination Survey for Adults (DEGS1)]. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz, 56*(5-6), 650-655. doi:10.1007/s00103-013-1666-9

Guthrie, B. J., Young, A. M., Williams, D. R., Boyd, C. J., & Kintner, E. K. (2002). African American girls' smoking habits and day-to-day experiences with racial discrimination. *Nurs Res, 51*(3), 183-190.

Gyllenborg, J., Rasmussen, S. L., Borch-Johnsen, K., Heitmann, B. L., Skakkebaek, N. E., & Juul, A. (2001). Cardiovascular risk factors in men: The role of gonadal steroids and sex hormone-binding globulin. *Metabolism, 50*(8), 882-888.

Hagstrom, E., Norlund, F., Stebbins, A., Armstrong, P. W., Chiswell, K., Granger, C. B., . . . Held, C. (2018). Psychosocial stress and major cardiovascular events in patients with stable coronary heart disease. *J Intern Med, 283*(1), 83-92. doi:10.1111/joim.12692

Halim, M. L., Moy, K. H., & Yoshikawa, H. (2017). Perceived ethnic and language-based discrimination and Latina immigrant women's health. *J Health Psychol, 22*(1), 68-78. doi:10.1177/1359105315595121

Halim, M. L., Yoshikawa, H., & Amodio, D. M. (2013). Cross-generational effects of discrimination among immigrant mothers: perceived discrimination predicts child's healthcare visits for illness. *Health Psychol, 32*(2), 203-211. doi:10.1037/a0027279

Harper, S., Lynch, J., & Smith, G. D. (2011). Social determinants and the decline of cardiovascular diseases: understanding the links. *Annu Rev Public Health, 32*, 39-69. doi:10.1146/annurev-publhealth-031210-101234

Herzig, L., Muhlemann, N., Burnand, B., Favrat, B., Haftgoli, N., Verdon, F., . . . Vaucher, P. (2012). Development of mental disorders one year after exposure to psychosocial stressors; a cohort study in primary care patients with a physical complaint. *BMC Psychiatry, 12*, 120. doi:10.1186/1471-244x-12-120

Hewagalamulage, S. D., Lee, T. K., Clarke, I. J., & Henry, B. A. (2016). Stress, cortisol, and obesity: a role for cortisol responsiveness in identifying individuals prone to obesity. *Domest Anim Endocrinol, 56 Suppl*, S112-120. doi:10.1016/j.domaniend.2016.03.004

Hou, Z. H., Lu, B., Li, Z. N., An, Y. Q., Gao, Y., & Yin, W. H. (2019). Coronary Atherosclerotic Plaque Volume Quantified by Computed Tomographic Angiography in Smokers Compared to Nonsmokers. *Acad Radiol*. doi:10.1016/j.acra.2019.03.017

Hozawa, A., Folsom, A. R., Sharrett, A. R., & Chambless, L. E. (2007). Absolute and attributable risks of cardiovascular disease incidence in relation to optimal and borderline risk factors: comparison of African American with white subjects--Atherosclerosis Risk in Communities Study. *Arch Intern Med, 167*(6), 573-579. doi:10.1001/archinte.167.6.573

Hubbard, E. A., Motl, R. W., & Fernhall, B. (2018). Sedentary Behavior and Blood Pressure in Patients with Multiple Sclerosis. *Int J MS Care, 20*(1), 1-8. doi:10.7224/1537-2073.2016-021

Jackson, D., White, I. R., Seaman, S., Evans, H., Baisley, K., & Carpenter, J. (2014). Relaxing the independent censoring assumption in the Cox proportional hazards model using multiple imputation. *Stat Med, 33*(27), 4681-4694. doi:10.1002/sim.6274

Jackson, S. E., & Steptoe, A. (2017). Association between perceived weight discrimination and physical activity: a population-based study among English middle-aged and older adults. *BMJ Open, 7*(3), e014592. doi:10.1136/bmjopen-2016-014592

Jette, M., Sidney, K., & Blumchen, G. (1990). Metabolic equivalents (METS) in exercise testing, exercise prescription, and evaluation of functional capacity. *Clin Cardiol, 13*(8), 555-565. doi:10.1002/clc.4960130809

Johnson, H. M., Gossett, L. K., Piper, M. E., Aeschlimann, S. E., Korcarz, C. E., Baker, T. B., . . . Stein, J. H. (2010). Effects of smoking and smoking cessation on endothelial function: 1-year outcomes from a randomized clinical trial. *J Am Coll Cardiol, 55*(18), 1988-1995. doi:10.1016/j.jacc.2010.03.002

Kageyama, K., & Suda, T. (2009). Regulatory mechanisms underlying corticotropin-releasing factor gene expression in the hypothalamus. *Endocr J, 56*(3), 335-344. doi:10.1507/endocrj.k09e-075

Kageyama, K., & Suda, T. (2010). Transcriptional regulation of hypothalamic corticotropin-releasing factor gene. *Vitam Horm, 82*, 301-317. doi:10.1016/s0083-6729(10)82016-3

Kageyama, K., Tamasawa, N., & Suda, T. (2011). Signal transduction in the hypothalamic corticotropin-releasing factor system and its clinical implications. *Stress, 14*(4), 357-367. doi:10.3109/10253890.2010.536279

Kanchi, R., Perlman, S. E., Chernov, C., Wu, W., Tabaei, B. P., Trinh-Shevrin, C., . . . Thorpe, L. E. (2018). Gender and Race Disparities in Cardiovascular Disease Risk Factors among New York City Adults: New York City Health and Nutrition Examination Survey (NYC HANES) 2013-2014. *J Urban Health, 95*(6), 801-812. doi:10.1007/s11524-018-0287-x

Karlamangla, A. S., Merkin, S. S., Crimmins, E. M., & Seeman, T. E. (2010). Socioeconomic and ethnic disparities in cardiovascular risk in the United States, 2001-2006. *Ann Epidemiol, 20*(8), 617-628. doi:10.1016/j.annepidem.2010.05.003

Katzmarzyk, P. T., & Lee, I. M. (2012). Sedentary behaviour and life expectancy in the USA: a cause-deleted life table analysis. *BMJ Open, 2*(4). doi:10.1136/bmjopen-2012-000828

Khanna, D., Kan, H., Failinger, C., Jain, A. C., & Finkel, M. S. (2006). Emotional stress and reversible myocardial dysfunction. *Cardiovasc Toxicol, 6*(3-4), 183-198.

Kim, B. J., Han, J. M., Kang, J. G., Kim, B. S., & Kang, J. H. (2017). Association between cotinine-verified smoking status and hypertension in 167,868 Korean adults. *Blood Press, 26*(5), 303-310. doi:10.1080/08037051.2017.1344539

Kivimaki, M., & Kawachi, I. (2015). Work Stress as a Risk Factor for Cardiovascular Disease. *Curr Cardiol Rep, 17*(9), 630. doi:10.1007/s11886-015-0630-8

Kivimaki, M., Nyberg, S. T., Batty, G. D., Kawachi, I., Jokela, M., Alfredsson, L., . . . Tabak, A. G. (2017). Long working hours as a risk factor for atrial fibrillation: a multi-cohort study. *Eur Heart J, 38*(34), 2621-2628. doi:10.1093/eurheartj/ehx324

Kivimaki, M., & Steptoe, A. (2018). Effects of stress on the development and progression of cardiovascular disease. *Nat Rev Cardiol, 15*(4), 215-229. doi:10.1038/nrcardio.2017.189

Knuiman, M. W., Vu, H. T., & Segal, M. R. (1997). An empirical comparison of multivariable methods for estimating risk of death from coronary heart disease. *J Cardiovasc Risk, 4*(2), 127-134.

Kohlbrenner, V., Deuba, K., Karki, D. K., & Marrone, G. (2016). Perceived Discrimination Is an Independent Risk Factor for Suicidal Ideation among Sexual and Gender Minorities in Nepal. *PLoS One, 11*(7), e0159359. doi:10.1371/journal.pone.0159359

Krieger, N., & Sidney, S. (1996). Racial discrimination and blood pressure: the CARDIA Study of young black and white adults. *Am J Public Health, 86*(10), 1370-1378. doi:10.2105/ajph.86.10.1370

Krieger, N., Waterman, P. D., Kosheleva, A., Chen, J. T., Smith, K. W., Carney, D. R., . . . Freeman, E. R. (2013). Racial discrimination & cardiovascular disease risk: my body my story study of 1005 US-born black and white community health center participants (US). *PLoS One, 8*(10), e77174. doi:10.1371/journal.pone.0077174

Kyu, H. H., Bachman, V. F., Alexander, L. T., Mumford, J. E., Afshin, A., Estep, K., . . . Forouzanfar, M. H. (2016). Physical activity and risk of breast cancer, colon cancer, diabetes, ischemic heart disease, and ischemic stroke events: systematic review and dose-response meta-analysis for the Global Burden of Disease Study 2013. *Bmj, 354*, i3857. doi:10.1136/bmj.i3857

Lagraauw, H. M., Kuiper, J., & Bot, I. (2015). Acute and chronic psychological stress as risk factors for cardiovascular disease: Insights gained from epidemiological, clinical and experimental studies. *Brain Behav Immun, 50*, 18-30. doi:10.1016/j.bbi.2015.08.007

Lakatta, E. G. (2002). Age-associated Cardiovascular Changes in Health: Impact on Cardiovascular Disease in Older Persons. *Heart Failure Reviews, 7*(1), 29-49. doi:10.1023/a:1013797722156

Landrine, H., & Klonoff, E. A. (2000). Racial discrimination and cigarette smoking among Blacks: findings from two studies. *Ethn Dis, 10*(2), 195-202.

Lee, D. L., & Ahn, S. (2011). Discrimination Against Latina/os: A Meta-Analysis of Individual-Level Resources and Outcomes Ψ. *The Counseling Psychologist, 40*(1), 28-65. doi:10.1177/0011000011403326

Leung, K. M., Elashoff, R. M., & Afifi, A. A. (1997). Censoring issues in survival analysis. *Annu Rev Public Health, 18*, 83-104. doi:10.1146/annurev.publhealth.18.1.83

Li, J., Loerbroks, A., Bosma, H., & Angerer, P. (2016). Work stress and cardiovascular disease: a life course perspective. *J Occup Health, 58*(2), 216-219. doi:10.1539/joh.15-0326-OP

Li, K. F. C., Ho, H. H., & Yew, M. S. (2019). A case report of dipyridamole stress-induced ST depression progressing to ST-elevation myocardial infarction despite intravenous aminophylline: steal, spasm, or something else? *Eur Heart J Case Rep, 3*(2). doi:10.1093/ehjcr/ytz054

Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., . . . Memish, Z. A. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet, 380*(9859), 2224-2260. doi:10.1016/s0140-6736(12)61766-8

Lin, J. S., Evans, C. V., Johnson, E., Redmond, N., Burda, B. U., Coppola, E. L., & Smith, N. (2018). U.S. Preventive Services Task Force Evidence Syntheses, formerly Systematic Evidence Reviews. In *Nontraditional Risk Factors in Cardiovascular Disease Risk Assessment: A Systematic Evidence Report for the U.S. Preventive Services Task Force*. Rockville, MD: Agency for Healthcare Research and Quality (US).

Liu, M. Y., Li, N., Li, W. A., & Khan, H. (2017). Association between psychosocial stress and hypertension: a systematic review and meta-analysis. *Neurol Res, 39*(6), 573-580. doi:10.1080/01616412.2017.1317904

Lloyd-Jones, D. M., Hong, Y., Labarthe, D., Mozaffarian, D., Appel, L. J., Van Horn, L., . . . Rosamond, W. D. (2010). Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation, 121*(4), 586-613. doi:10.1161/circulationaha.109.192703

Lockwood, K. G., Marsland, A. L., Matthews, K. A., & Gianaros, P. J. (2018). Perceived discrimination and cardiovascular health disparities: a multisystem review and health neuroscience perspective. *Ann N Y Acad Sci, 1428*(1), 170-207. doi:10.1111/nyas.13939

Maggioni, A. P., Maseri, A., Fresco, C., Franzosi, M. G., Mauri, F., Santoro, E., & Tognoni, G. (1993). Age-related increase in mortality among patients with first myocardial infarctions treated with thrombolysis. The Investigators of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2). *N Engl J Med, 329*(20), 1442-1448. doi:10.1056/nejm199311113292002

Makar, O., & Siabrenko, G. (2018). INFLUENCE OF PHYSICAL ACTIVITY ON CARDIOVASCULAR SYSTEM AND PREVENTION OF CARDIOVASCULAR DISEASES (REVIEW). *Georgian Med News*(285), 69-74.

Marmot, M., Friel, S., Bell, R., Houweling, T. A., & Taylor, S. (2008). Closing the gap in a generation: health equity through action on the social determinants of health. *Lancet, 372*(9650), 1661-1669. doi:10.1016/s0140-6736(08)61690-6

Mays, V. M., & Cochran, S. D. (2001). Mental health correlates of perceived discrimination among lesbian, gay, and bisexual adults in the United States. *Am J Public Health, 91*(11), 1869-1876.

McArtor, D. B., Lin, B. D., Hottenga, J. J., Boomsma, D. I., Willemsen, G., & Lubke, G. H. (2017). Using a multivariate model to assess the interactive effects of demographics and lifestyle on the hematological profile. *Biomark Med, 11*(6), 427-438. doi:10.2217/bmm-2016-0285

Mensah, G. A. (2005). Eliminating disparities in cardiovascular health: six strategic imperatives and a framework for action. *Circulation, 111*(10), 1332-1336. doi:10.1161/01.Cir.0000158134.24860.91

Messner, B., & Bernhard, D. (2014). Smoking and cardiovascular disease: mechanisms of endothelial dysfunction and early atherogenesis. *Arterioscler Thromb Vasc Biol, 34*(3), 509-515. doi:10.1161/atvbaha.113.300156

Metcalf, P., Scragg, R., & Davis, P. (2007). Relationship of different measures of socioeconomic status with cardiovascular disease risk factors and lifestyle in a New Zealand workforce survey. *N Z Med J, 120*(1248), U2392.

Moolgavkar, S. H., Chang, E. T., Watson, H. N., & Lau, E. C. (2018). An Assessment of the Cox Proportional Hazards Regression Model for Epidemiologic Studies. *Risk Anal, 38*(4), 777-794. doi:10.1111/risa.12865

Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., . . . Turner, M. B. (2016a). Executive Summary: Heart Disease and Stroke Statistics--2016 Update: A Report From the American Heart Association. *Circulation, 133*(4), 447-454. doi:10.1161/cir.0000000000000366

Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., . . . Turner, M. B. (2016b). Heart Disease and Stroke Statistics-2016 Update: A Report From the American Heart Association. *Circulation, 133*(4), e38-360. doi:10.1161/cir.0000000000000350

Nickens, H. (1986). Report of the Secretary's Task Force on Black and Minority Health: a summary and a presentation of health data with regard to blacks. *J Natl Med Assoc, 78*(6), 577-580.

O'Keefe, J. H., Jr., Poston, W. S., Haddock, C. K., Moe, R. M., & Harris, W. (2004). Psychosocial stress and cardiovascular disease: how to heal a broken heart. *Compr Ther, 30*(1), 37-43.

O'Neil, A., Scovelle, A. J., Milner, A. J., & Kavanagh, A. (2018). Gender/Sex as a Social Determinant of Cardiovascular Risk. *Circulation, 137*(8), 854-864. doi:10.1161/circulationaha.117.028595

Okhomina, V. I., Glover, L., Taylor, H., & Sims, M. (2018). Dimensions of and Responses to Perceived Discrimination and Subclinical Disease Among African-Americans in the Jackson Heart Study. *J Racial Ethn Health Disparities, 5*(5), 1084-1092. doi:10.1007/s40615-017-0457-7

Okunrintemi, V., Valero-Elizondo, J., Patrick, B., Salami, J., Tibuakuu, M., Ahmad, S., . . . Michos, E. D. (2018). Gender Differences in Patient-Reported Outcomes Among Adults With Atherosclerotic Cardiovascular Disease. *J Am Heart Assoc, 7*(24), e010498. doi:10.1161/jaha.118.010498

Ostchega, Y., Dillon, C. F., Hughes, J. P., Carroll, M., & Yoon, S. (2007). Trends in hypertension prevalence, awareness, treatment, and control in older U.S. adults: data from the National Health and Nutrition Examination Survey 1988 to 2004. *J Am Geriatr Soc, 55*(7), 1056-1065. doi:10.1111/j.1532-5415.2007.01215.x

Pallant, J. (2013). SPSS survival manual: a step by step guide to data analysis using IBM SPSS*,* 597.

Paradies, Y., Ben, J., Denson, N., Elias, A., Priest, N., Pieterse, A., . . . Gee, G. (2015). Racism as a Determinant of Health: A Systematic Review and Meta-Analysis. *PLoS One, 10*(9), e0138511. doi:10.1371/journal.pone.0138511

Paradies, Y. C. (2006). Defining, conceptualizing and characterizing racism in health research. *Critical Public Health, 16*(2), 143-157. doi:10.1080/09581590600828881

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: a meta-analytic review. *Psychol Bull, 135*(4), 531-554. doi:10.1037/a0016059

Pescatello, L. S., Buchner, D. M., Jakicic, J. M., Powell, K. E., Kraus, W. E., Bloodgood, B., . . . Piercy, K. L. (2019). Physical Activity to Prevent and Treat Hypertension: A Systematic Review. *Med Sci Sports Exerc, 51*(6), 1314-1323. doi:10.1249/mss.0000000000001943

Pickering, T. G. (2007). Stress, inflammation, and hypertension. *J Clin Hypertens (Greenwich), 9*(7), 567-571.

Pieterse, A. L., Todd, N. R., Neville, H. A., & Carter, R. T. (2012). Perceived racism and mental health among Black American adults: a meta-analytic review. *J Couns Psychol, 59*(1), 1-9. doi:10.1037/a0026208

Pool, L. R., Ning, H., Lloyd-Jones, D. M., & Allen, N. B. (2017). Trends in Racial/Ethnic Disparities in Cardiovascular Health Among US Adults From 1999-2012. *J Am Heart Assoc, 6*(9). doi:10.1161/jaha.117.006027

Pugeat, M., Moulin, P., Cousin, P., Fimbel, S., Nicolas, M. H., Crave, J. C., & Lejeune, H. (1995). Interrelations between sex hormone-binding globulin (SHBG), plasma lipoproteins and cardiovascular risk. *J Steroid Biochem Mol Biol, 53*(1-6), 567-572.

Raikkonen, K., Keltikangas-Jarvinen, L., Adlercreutz, H., & Hautanen, A. (1996). Psychosocial stress and the insulin resistance syndrome. *Metabolism, 45*(12), 1533-1538. doi:10.1016/s0026-0495(96)90184-5

Reddy, K. S., Prabhakaran, D., Jeemon, P., Thankappan, K. R., Joshi, P., Chaturvedi, V., . . . Ahmed, F. (2007). Educational status and cardiovascular risk profile in Indians. *Proc Natl Acad Sci U S A, 104*(41), 16263-16268. doi:10.1073/pnas.0700933104

Regitz-Zagrosek, V., & Kararigas, G. (2017). Mechanistic Pathways of Sex Differences in Cardiovascular Disease. *Physiol Rev, 97*(1), 1-37. doi:10.1152/physrev.00021.2015

Regitz-Zagrosek, V., Oertelt-Prigione, S., Seeland, U., & Hetzer, R. (2010). Sex and gender differences in myocardial hypertrophy and heart failure. *Circ J, 74*(7), 1265-1273.

Rexrode, K. M., Manson, J. E., Lee, I. M., Ridker, P. M., Sluss, P. M., Cook, N. R., & Buring, J. E. (2003). Sex hormone levels and risk of cardiovascular events in postmenopausal women. *Circulation, 108*(14), 1688-1693. doi:10.1161/01.Cir.0000091114.36254.F3

Richardson, S., Shaffer, J. A., Falzon, L., Krupka, D., Davidson, K. W., & Edmondson, D. (2012). Meta-analysis of perceived stress and its association with incident coronary heart disease. *Am J Cardiol, 110*(12), 1711-1716. doi:10.1016/j.amjcard.2012.08.004

Rochette, L., Zeller, M., Cottin, Y., & Vergely, C. (2014). Diabetes, oxidative stress and therapeutic strategies. *Biochim Biophys Acta, 1840*(9), 2709-2729. doi:10.1016/j.bbagen.2014.05.017

Rosamond, W. D., Chambless, L. E., Heiss, G., Mosley, T. H., Coresh, J., Whitsel, E., . . . Folsom, A. R. (2012). Twenty-two-year trends in incidence of myocardial infarction, coronary heart disease mortality, and case fatality in 4 US communities, 1987-2008. *Circulation, 125*(15), 1848-1857. doi:10.1161/circulationaha.111.047480

Safford, M. M., Brown, T. M., Muntner, P. M., Durant, R. W., Glasser, S., Halanych, J. H., . . . Howard, G. (2012). Association of race and sex with risk of incident acute coronary heart disease events. *Jama, 308*(17), 1768-1774. doi:10.1001/jama.2012.14306

Sanchez, D., Whittaker, T. A., & Hamilton, E. (2016). Perceived Discrimination, Peer Influence and Sexual Behaviors in Mexican American Preadolescents. *J Youth Adolesc, 45*(5), 928-944. doi:10.1007/s10964-016-0420-7

Sanchez, D., Whittaker, T. A., Hamilton, E., & Zayas, L. H. (2016). Perceived discrimination and sexual precursor behaviors in Mexican American preadolescent girls: The role of psychological distress, sexual attitudes, and marianismo beliefs. *Cultur Divers Ethnic Minor Psychol, 22*(3), 395-407. doi:10.1037/cdp0000066

Schmitt, M. T., Branscombe, N. R., Postmes, T., & Garcia, A. (2014). The consequences of perceived discrimination for psychological well-being: a meta-analytic review. *Psychol Bull, 140*(4), 921-948. doi:10.1037/a0035754

Shakir, Y. A., Samsioe, G., Nyberg, P., Lidfeldt, J., Nerbrand, C., & Agardh, C. D. (2007). Do sex hormones influence features of the metabolic syndrome in middle-aged women? A population-based study of Swedish women: the Women's Health in the Lund Area (WHILA) Study. *Fertil Steril, 88*(1), 163-171. doi:10.1016/j.fertnstert.2006.11.111

Shih, H., Lee, B., Lee, R. J., & Boyle, A. J. (2011). The aging heart and post-infarction left ventricular remodeling. *J Am Coll Cardiol, 57*(1), 9-17. doi:10.1016/j.jacc.2010.08.623

Shpilsky, D., Bambs, C., Kip, K., Patel, S., Aiyer, A., Olafiranye, O., . . . Erqou, S. (2018). Association between ideal cardiovascular health and markers of subclinical cardiovascular disease. *Clin Cardiol, 41*(12), 1593-1599. doi:10.1002/clc.23096

Sims, M., Diez-Roux, A. V., Dudley, A., Gebreab, S., Wyatt, S. B., Bruce, M. A., . . . Taylor, H. A. (2012). Perceived discrimination and hypertension among African Americans in the Jackson Heart Study. *Am J Public Health, 102 Suppl 2*, S258-265. doi:10.2105/ajph.2011.300523

Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis modeling change and event occurrence*. New York ; Oxford: Oxford University Press.

Smith, S. M., & Vale, W. W. (2006). The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. *Dialogues Clin Neurosci, 8*(4), 383-395.

Spruill, T. M. (2010). Chronic psychosocial stress and hypertension. *Curr Hypertens Rep, 12*(1), 10-16. doi:10.1007/s11906-009-0084-8

State-specific smoking-attributable mortality and years of potential life lost--United States, 2000-2004. (2009). *MMWR Morb Mortal Wkly Rep, 58*(2), 29-33.

Steptoe, A., & Kivimaki, M. (2013). Stress and cardiovascular disease: an update on current knowledge. *Annu Rev Public Health, 34*, 337-354. doi:10.1146/annurev-publhealth-031912-114452

Sullivan, S., Hammadah, M., Al Mheid, I., Shah, A., Sun, Y. V., Kutner, M., . . . Lewis, T. T. (2019). An investigation of racial/ethnic and sex differences in the association between experiences of everyday discrimination and leukocyte telomere length among patients with coronary artery disease. *Psychoneuroendocrinology, 106*, 122-128. doi:10.1016/j.psyneuen.2019.03.021

Svedberg, P., Nygren, J. M., Staland-Nyman, C., & Nyholm, M. (2016). The validity of socioeconomic status measures among adolescents based on self-reported information about parents occupations, FAS and perceived SES; implication for health related quality of life studies. *BMC Med Res Methodol, 16*, 48. doi:10.1186/s12874-016-0148-9

Szabo, S. (1998). Hans Selye and the development of the stress concept. Special reference to gastroduodenal ulcerogenesis. *Ann N Y Acad Sci, 851*, 19-27. doi:10.1111/j.1749-6632.1998.tb08972.x

Szabo, S., Tache, Y., & Somogyi, A. (2012). The legacy of Hans Selye and the origins of stress research: a retrospective 75 years after his landmark brief "letter" to the editor# of nature. *Stress, 15*(5), 472-478. doi:10.3109/10253890.2012.710919

Thakur, N., Barcelo, N. E., Borrell, L. N., Singh, S., Eng, C., Davis, A., . . . Burchard, E. G. (2017). Perceived Discrimination Associated With Asthma and Related Outcomes in Minority Youth: The GALA II and SAGE II Studies. *Chest, 151*(4), 804-812. doi:10.1016/j.chest.2016.11.027

Torabi, A., Cleland, J. G., Rigby, A. S., & Sherwi, N. (2014). Development and course of heart failure after a myocardial infarction in younger and older people. *J Geriatr Cardiol, 11*(1), 1-12. doi:10.3969/j.issn.1671-5411.2014.01.002

Toren, K., Schioler, L., Soderberg, M., Giang, K. W., & Rosengren, A. (2015). The association between job strain and atrial fibrillation in Swedish men. *Occup Environ Med, 72*(3), 177-180. doi:10.1136/oemed-2014-102256

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *J Psychosom Res, 53*(4), 865-871. doi:10.1016/s0022-3999(02)00429-4

Turan, B., Rogers, A. J., Rice, W. S., Atkins, G. C., Cohen, M. H., Wilson, T. E., . . . Weiser, S. D. (2017). Association between Perceived Discrimination in Healthcare Settings and HIV Medication Adherence: Mediating Psychosocial Mechanisms. *AIDS Behav, 21*(12), 3431-3439. doi:10.1007/s10461-017-1957-5

Vaccarino, V., & Bremner, J. D. (2017). Behavioral, emotional and neurobiological determinants of coronary heart disease risk in women. *Neurosci Biobehav Rev, 74*(Pt B), 297-309. doi:10.1016/j.neubiorev.2016.04.023

Vaccarino, V., Wilmot, K., Al Mheid, I., Ramadan, R., Pimple, P., Shah, A. J., . . . Quyyumi, A. A. (2016). Sex Differences in Mental Stress-Induced Myocardial Ischemia in Patients With Coronary Heart Disease. *J Am Heart Assoc, 5*(9). doi:10.1161/jaha.116.003630

Vasan, R. S., Beiser, A., Seshadri, S., Larson, M. G., Kannel, W. B., D'Agostino, R. B., & Levy, D. (2002). Residual Lifetime Risk for Developing Hypertension in Middle-aged Women and MenThe Framingham Heart Study. *Jama, 287*(8), 1003-1010. doi:10.1001/jama.287.8.1003

Visser, M. J., Ikram, U. Z., Derks, E. M., Snijder, M. B., & Kunst, A. E. (2017). Perceived ethnic discrimination in relation to smoking and alcohol consumption in ethnic minority groups in The Netherlands: the HELIUS study. *Int J Public Health, 62*(8), 879-887. doi:10.1007/s00038-017-0977-2

Warren, G. W., Alberg, A. J., Kraft, A. S., & Cummings, K. M. (2014). The 2014 Surgeon General's report: "The health consequences of smoking--50 years of progress": a paradigm shift in cancer care. *Cancer, 120*(13), 1914-1916. doi:10.1002/cncr.28695

Wetter, D. W., Kenford, S. L., Welsch, S. K., Smith, S. S., Fouladi, R. T., Fiore, M. C., & Baker, T. B. (2004). Prevalence and predictors of transitions in smoking behavior among college students. *Health Psychol, 23*(2), 168-177. doi:10.1037/0278-6133.23.2.168

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: evidence and needed research. *J Behav Med, 32*(1), 20-47. doi:10.1007/s10865-008-9185-0

Williams, D. R., & Neighbors, H. (2001). Racism, discrimination and hypertension: evidence and needed research. *Ethn Dis, 11*(4), 800-816.

Williams, D. R., Yan, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial Differences in Physical and Mental Health: Socio-economic Status, Stress and Discrimination. *J Health Psychol, 2*(3), 335-351. doi:10.1177/135910539700200305

Williams, I. C., Clay, O. J., Ovalle, F., Atkinson, D., & Crowe, M. (2018). The Role of Perceived Discrimination and Other Psychosocial Factors in Explaining Diabetes Distress Among Older African American and White Adults. *J Appl Gerontol*, 733464817750273. doi:10.1177/0733464817750273

Williams, P. T. (2001). Physical fitness and activity as separate heart disease risk factors: a meta-analysis. *Med Sci Sports Exerc, 33*(5), 754-761.

Winkleby, M. A., Jatulis, D. E., Frank, E., & Fortmann, S. P. (1992). Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health, 82*(6), 816-820. doi:10.2105/ajph.82.6.816

Wirtz, P. H., & von Kanel, R. (2017). Psychological Stress, Inflammation, and Coronary Heart Disease. *Curr Cardiol Rep, 19*(11), 111. doi:10.1007/s11886-017-0919-x

Yan, R., Li, W., Yin, L., Wang, Y., & Bo, J. (2017). Cardiovascular Diseases and Risk-Factor Burden in Urban and Rural Communities in High-, Middle-, and Low-Income Regions of China: A Large Community-Based Epidemiological Study. *J Am Heart Assoc, 6*(2). doi:10.1161/jaha.116.004445

Yankelevitz, D. F., Cham, M. D., Hecht, H., Yip, R., Shemesh, J., Narula, J., & Henschke, C. I. (2017). The Association of Secondhand Tobacco Smoke and CT Angiography-Verified Coronary Atherosclerosis. *JACC Cardiovasc Imaging, 10*(6), 652-659. doi:10.1016/j.jcmg.2016.07.003

Yankelevitz, D. F., Henschke, C. I., Yip, R., Boffetta, P., Shemesh, J., Cham, M. D., . . . Hecht, H. S. (2013). Second-hand tobacco smoke in never smokers is a significant risk factor for coronary artery calcification. *JACC Cardiovasc Imaging, 6*(6), 651-657. doi:10.1016/j.jcmg.2013.02.004

Yano, Y., Reis, J. P., Tedla, Y. G., Goff, D. C., Jr., Jacobs, D. R., Jr., Sidney, S., . . . Lloyd-Jones, D. M. (2017). Racial Differences in Associations of Blood Pressure Components in Young Adulthood With Incident Cardiovascular Disease by Middle Age: Coronary Artery Risk Development in Young Adults (CARDIA) Study. *JAMA Cardiol, 2*(4), 381-389. doi:10.1001/jamacardio.2016.5678

Yoon, S. S., Gu, Q., Nwankwo, T., Wright, J. D., Hong, Y., & Burt, V. (2015). Trends in blood pressure among adults with hypertension: United States, 2003 to 2012. *Hypertension, 65*(1), 54-61. doi:10.1161/hypertensionaha.114.04012

Yusuf, S., Rangarajan, S., Teo, K., Islam, S., Li, W., Liu, L., . . . Dagenais, G. (2014). Cardiovascular risk and events in 17 low-, middle-, and high-income countries. *N Engl J Med, 371*(9), 818-827. doi:10.1056/NEJMoa1311890

Zeiher, A. M., Schachinger, V., & Minners, J. (1995). Long-term cigarette smoking impairs endothelium-dependent coronary arterial vasodilator function. *Circulation, 92*(5), 1094-1100.

Zhang, W. Y., Liu, S., Li, H. D., & Cai, H. L. (2012). Chronic unpredictable mild stress affects myocardial metabolic profiling of SD rats. *J Pharm Biomed Anal, 70*, 534-538. doi:10.1016/j.jpba.2012.04.032

Zhu, W., Yuan, P., Shen, Y., Wan, R., & Hong, K. (2016). Association of smoking with the risk of incident atrial fibrillation: A meta-analysis of prospective studies. *Int J Cardiol, 218*, 259-266. doi:10.1016/j.ijcard.2016.05.013

APPENDICES

Appendix I: SPSS Syntax for All Events

```
/* Kaplan-Meier analyses
  KM TIME_TO_EVENT BY EDS_1_group
  /STATUS=cardiovascular_event(1)
  /PRINT TABLE MEAN
  /PERCENTILES
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-2 groups visually
COXREG TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group=Indicator(1)
  /METHOD=ENTER EDS_1_group  EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
 TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
```

```
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-2, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /PATTERN BY EDS-1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER EDS-1_group  EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

```
/*  Cox regression model 4 with added demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
COXREG TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group_2 EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
    /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/* Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
    /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

```
/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and age.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_mean_centered_age
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).


/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and gender.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and physical activity.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_physical activity
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and smoking.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_smoking
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
```

```
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and income.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_income
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and education.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_education
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

 /*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, interaction terms for EDS-1 and stress.
```

```
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   TIME_TO_EVENT
  /STATUS=cardiovascular_event(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_stress
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix II: SPSS Syntax for Myocardial Infarction

```
/* Kaplan-Meier analyses
  KM time_event_MI BY EDS_1_group
  /STATUS=event_MI(1)
  /PRINT TABLE MEAN
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-1 groups visually
COXREG time_event_MI
  /STATUS=event_MI(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_MI
  /STATUS=event_MI(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG time_event_MI
  /STATUS=event_MI(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
```

```
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-1, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_MI
  /STATUS=event_MI(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG time_event_MI
  /STATUS=event_MI(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_MI
  /STATUS=event_MI(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with added  demographic variables of sex and age,
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG time_event_MI
  /STATUS=event_MI(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_MI
  /STATUS=event_MI(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
COXREG time_event_MI
  /STATUS=event_MI(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS-1_grouping)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS-1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS-1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress
EDS_1_group_X_smoke_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_MI
  /STATUS=event_MI(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress
EDS_1_group_X_smoke_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix III: SPSS Syntax for Acute Ischemic Syndrome

```
/* Kaplan-Meier analyses
  KM time_event_AIS BY EDS_1_group
  /STATUS=event_AIS(1)
  /PRINT TABLE MEAN
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-1 groups visually
COXREG time_event_AIS
  /STATUS=event_AIS(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_AIS
  /STATUS=event_AIS(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG time_event_AIS
  /STATUS=event_AIS(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
```

```
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-1, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_AIS
  /STATUS=event_AIS(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG time_event_AIS
  /STATUS=event_AIS(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER median_EDS_1_group EDS_2  mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*ED_1_group.
COXREG   time_event_AIS
  /STATUS=event_AIS(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2  mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with added  demographic variables of sex and age,
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG time_event_AIS
  /STATUS=event_AIS(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_AIS
  /STATUS=event_AIS(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
COXREG time_event_AIS
  /STATUS=event_AIS(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group _X_sex_coded EDS_1_group
_X_mean_centered_age EDS_1_group _X_education EDS_1_group _X_income
EDS_1_group _X_PA EDS_1_group _X_stress EDS_1_group _X_smoke_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_AIS
  /STATUS=event_AIS(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group _X_sex_coded EDS_1_group
_X_mean_centered_age EDS_1_group _X_education EDS_1_group _X_income
EDS_1_group _X_PA EDS_1_group _X_stress EDS_1_group _X_smoke_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix IV: SPSS Syntax for Coronary Revascularization

```
/* Kaplan-Meier analyses
  KM time_event_REVASC BY EDS_1_group
  /STATUS=event_REVASC(1)
  /PRINT TABLE MEAN
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-1 groups visually
COXREG time_event_REVASC
  /STATUS=event_REVASC(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_REVASC
  /STATUS=event_REVASC(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG time_event_REVASC
  /STATUS=event_REVASC(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
```

```
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-1, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_REVASC
  /STATUS=event_REVASC(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG time_event_REVASC
  /STATUS=event_REVASC(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_REVASC
  /STATUS=event_REVASC(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with added  demographic variables of sex and age,
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG time_event_REVASC
  /STATUS=event_REVASC(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_REVASC
  /STATUS=event_REVASC(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
COXREG time_event_REVASC
  /STATUS=event_REVASC(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group _X_sex_coded EDS_1_group
_X_mean_centered_age EDS_1_group _X_education EDS_1_group _X_income
EDS_1_group _X_PA EDS_1_group _X_stress EDS_1_group _X_smoke_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_REVASC
  /STATUS=event_REVASC(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_1 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group _X_sex_coded EDS_1_group
_X_mean_centered_age EDS_1_group _X_education EDS_1_group _X_income
EDS_1_group _X_PA EDS_1_group _X_stress EDS_1_group _X_smoke_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix V: SPSS Syntax for Cerebral Vascular Accident

```
/* Kaplan-Meier analyses
  KM time_event_CVA BY EDS_1_group
  /STATUS=event_CVA(1)
  /PRINT TABLE MEAN
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-1 groups visually
COXREG time_event_CVA
  /STATUS=event_CVA(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group .
COXREG   time_event_CVA
  /STATUS=event_CVA(1)
  /CONTRAST (EDS_1_group )=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG time_event_CVA
  /STATUS=event_CVA(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group )=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
```

```
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-1, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)* EDS_1_group.
COXREG   time_event_CVA
  /STATUS=event_CVA(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG time_event_CVA
  /STATUS=event_CVA(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_CVA
  /STATUS=event_CVA(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with added  demographic variables of sex and age,
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG time_event_CVA
  /STATUS=event_CVA(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_CVA
  /STATUS=event_CVA(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
COXREG time_event_CVA
  /STATUS=event_CVA(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress
EDS_1_group_X_smoke_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_CVA
  /STATUS=event_CVA(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress
EDS_1_group_X_smoke_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix VI: SPSS Syntax for Cardiac Death

```
/* Kaplan-Meier analyses
  KM time_event_cardiac_death BY EDS_1_group
  /STATUS=event_death_doc_cardiac(1)
  /PRINT TABLE MEAN
  /PLOT SURVIVAL HAZARD
  /TEST LOGRANK BRESLOW TARONE
  /COMPARE OVERALL POOLED.

/*Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at LML function graphs to check proportional hazards assumption between
the two EDS-1 groups visually
COXREG time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/**Cox regression analysis unadjusted baseline model for hypothesis 1
/*Looking at time dependent interaction term of log survival time and perceived
discrimination (EDS-1) to test
/* proportional hazards assumptions. The effect N/S and therefore time dependent
covariate of EDS-1 does not
/* contribute significantly to the model, and proportional hazards assumption is met.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with added  demographic variables of sex and age
COXREG time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
```

```
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 2 with Time dependent covariate of EDS-1, and the 2 added
demographic variables of sex and age
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with added  demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
COXREG time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 3 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/*  Cox regression model 4 with added  demographic variables of sex and age,
```

```
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
COXREG time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/* Cox regression model 4 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

*  Cox regression model 5 with added demographic variables of sex and age,
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
COXREG time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /PATTERN BY EDS_1_group
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
```

```
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress
EDS_1_group_X_smoke_coded
  /PLOT SURVIVAL HAZARDS LML
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

/* Cox regression model 5 with Time dependent covariate of EDS-1, the 2 added
demographic variables of sex and age
/* and added behavioral variables of smoking and physical activity
/* and added socioeconomic variables of income and education
/* added variable of stress
/* and added on step 2, 7 interaction terms for EDS-1 and covariates.
TIME PROGRAM.
COMPUTE T_COV_ = LN(T_)*EDS_1_group.
COXREG   time_event_cardiac_death
  /STATUS=event_death_doc_cardiac(1)
  /CONTRAST (EDS_1_group)=Indicator(1)
  /CONTRAST (sex_coded)=Indicator(1)
  /CONTRAST (PA_Q6)=Indicator(1)
  /CONTRAST (smoke_coded)=Indicator(1)
  /CONTRAST (DEMO_INCOME)=Indicator(1)
  /CONTRAST (DEMO_EDUCCAT5)=Indicator(1)
  /METHOD=ENTER T_COV_ EDS_1_group EDS_2 mean_centered_age sex_coded
smoke_coded PA_Q6 DEMO_INCOME DEMO_EDUCCAT5 COHEN_REVISED
  /METHOD=ENTER EDS_1_group_X_sex_coded
EDS_1_group_X_mean_centered_age EDS_1_group_X_education
EDS_1_group_X_income EDS_1_group_X_PA EDS_1_group_X_stress EDS_1_group
EDS_1_group_X_smoke_coded
  /PRINT=CI(95)
  /CRITERIA=PIN(.05) POUT(.10) ITERATE(20).
```

Appendix VII: IRB Approval

RESEARCH INTEGRITY & COMPLIANCE
Institutional Review Boards, FWA No. 00001669
12901 Bruce B. Downs Blvd, MDC35, Tampa, FL 33612-4799
(813) 974-5638 FAX (813) 974-7091

8/21/2019

Marilyn Aluoch
College of Nursing
Tampa, FL 33612

RE: **Not Human Subjects Research Determination**
IRB#: Pro00041525
Title: Perceived Discrimination and Cardiovascular Outcomes in Blacks : A Secondary Data Analysis of the Heart SCORE study

Dear Ms. Aluoch:

The Institutional Review Board (IRB) has reviewed your application. The activities presented in the application involve methods of program evaluation, quality improvement, needs analysis, and/or research that does not involve human subjects. As such, USF IRB approval and oversight are not required.

While not requiring USF IRB approval and oversight, your study activities should be conducted in a manner that is consistent with the ethical principles of your profession. If the scope of your project changes in the future, please contact the IRB for further guidance.

If you will be obtaining consent to conduct a program evaluation, quality improvement project, or needs assessment, please remove any references to "research" and do not include the assigned Protocol Number or USF IRB contact information.

If your study activities involve collection or use of health information, please note that there may be requirements under the HIPAA Privacy Rule that apply. For further information, please contact a HIPAA Program administrator at (813) 974-5638.

Sincerely,

V Jorgensen MD

E. Verena Jorgensen, M.D., Chairperson
USF Institutional Review Board