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What are the mechanisms of action of stereotype threat and how does it contribute to the development of cardiovascular disease in African Americans

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What are the mechanisms of action of stereotype threat and how does it contribute to the development of cardiovascular disease in African Americans

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Abstract

The rate of hypertension and cardiovascular disease for African Americans is disproportionately higher compared to Whites Americans. Previous research has focused much of its attention on biological and socioeconomic differences as a pathway to explain the disparity in rates of cardiovascular disease. Reviewed is the connection between socioeconomic status and cardiovascular disease, but also extends the reach of etiology to include racially induced cardiovascular reactivity responses in African American. Stereotype threat occurs when a member of a stereotyped group encounters a situation where a stereotype exists, the threat of confirming the stereotype produces an exaggerated cardiovascular response. This model takes into account mediating factors such as perceptions and conceptualizations of race and stereotype as important markers which influence the degree of cardiovascular response demonstrated, thus African Americans who perceived greater racial stress, also demonstrated greater cardiovascular response. Stereotype threat may provide an explanation and a possible pathway to the etiology of hypertension disparity in African Americans.

WHAT ARE THE MECHANISMS OF ACTION OF STEREOTYPE THREAT
AND HOW DOES IT CONTRIBUTE TO THE DEVELOPMENT
OF CARDIOVASCULAR DISEASE IN
AFRICAN AMERICANS

Submitted

in Partial Fulfillment

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Entitled: WHAT ARE THE MECHANISMS OF STEREOTYPE THREAT AND HOW DOES IT CONTRIBUTE TO THE DEVELOPMENT OF CARDIOVASCULAR DISEASE IN AFRICAN AMERICANS

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ABSTRACT

The rate of hypertension and cardiovascular disease for African Americans is disproportionately higher compared to Whites Americans. Previous research has focused much of its attention on biological and socioeconomic differences as a pathway to explain the disparity in rates of cardiovascular disease. Reviewed is the connection between socioeconomic status and cardiovascular disease, but also extends the reach of etiology to include racially induced cardiovascular reactivity responses in African American. Stereotype threat occurs when a member of a stereotyped group encounters a situation where a stereotype exists, the threat of confirming the stereotype produces an exaggerated cardiovascular response. This model takes into account mediating factors such as perceptions and conceptualizations of race and stereotype as important markers which influence the degree of cardiovascular response demonstrated, thus African Americans who perceived greater racial stress, also demonstrated greater cardiovascular response. Stereotype threat may provide an explanation and a possible pathway to the etiology of hypertension disparity in African Americans.

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Chapter 1

Introduction

African Americans have the highest prevalence of hypertension in the world at 44% and consequently die from cardiovascular disease at a much higher rate (387 per 100,000 for African Americans versus 281 per 100,000 for Whites) (Go et al., 2013). There has been much speculation to explain the racial disparity in hypertension and cardiovascular disease rates in the U.S. among African Americans. Many of these explanations have centered on genetic and biological differences, as well as socioeconomic and environmental factors. Until recently, there has been no agreement regarding the etiology which accounts for the drastic differential in cardiovascular morbidity and mortality rates. Additionally, a majority of past and current research focused on determining the causation of racial differences in hypertension, and related disease etiology has centered on SES position and health outcomes.

There is robust literature support for the association between socioeconomic status and health, specifically cardiovascular disease. Those who are socially and economically challenged have limited educational attainment, less access to resources, and marginal autonomy. Additionally, they face considerably higher risk of premature morbidity and mortality than those of higher socio-economic status. Generally, this correlation between SES and health is consistent regardless of gender, ethnicity, and age. In the U.S., a disproportionate percentage of African Americans are disadvantaged and endure poor SES position in society, and thus as a group experience poorer health effects as a result (Mathews, 2010).

The poor health outcomes associated with African Americans who live in low SES are multi factorial. Environmental, social, psychological, and behavioral factors all play a role in the

disparities in health African Americans face. It is commonly understood that low SES manifests itself in risky health behaviors such as smoking, alcohol consumption, and poor nutrition. Also, increased acute and chronic stress, depression, hostility, and anger are common characteristics among those from low SES positions. Additionally, a lack of physical activity, control and/or social support and inadequate or no preventative health care contribute to the poor health outcomes demonstrated by those from low SES position. As the literature illustrates, social economic status contributes to the length and intensity of exposure to many of these factors which lead to poor health (Mathews et al., 2010).

Mathews et al. (2010) explain that living in a lower SES position leads to increased exposure to risk factors, and a decrease of physical and social resources, which act like a buffer called reserve capacity, to protect persons from constant overexposure to a broad range of risk factors and their attendant maladaptive consequences. Without the benefits of a reserve capacity, or the ability to restore it, persons from low SES are much more susceptible to more frequent future stress. People living in a chronic stressful environment will cope by use of tobacco, alcohol, illicit sex, and other maladaptive coping mechanisms, which further contribute to the risk of morbidity and mortality. Besides the common attributes connected to living in low SES position, African Americans suffer from racially induced stress which is more salient in nature as it stems from racism and discrimination. The chronic stress associated with being black is inescapable; it is a condition in which increased SES position may not be effective in reconciling. Interestingly, emerging research suggests increased SES may not be as health protective for African Americans as once believed (Blascovich et al., 2001).

Although SES continues to be an important factor connected to health outcomes for African Americans, emerging research suggests repeated exposure to racism and discrimination

form a unique type of stressor, stereotype threat, which may be the foundation for blacks to perceive their environment as more threatening and negative (Blascovich et al., 2001). The research has suggested African Americans confront a culturally constructed psychological threat, a threat which emerges in situations where there is negative stereotype associated with it. Additionally, the threat comes from the fear of being negatively judged by others, or conforming to a known stereotype, such as mathematical performance, as demonstrated with African Americans.

Historically, in the U.S. there have been many negative stereotypes attributed to African Americans, such as being lazy, unintelligent, thieves, drug users, welfare recipients, aggressiveness, and live in ghettos. In response to being judged by a particular stereotype, many African Americans under the stress and pressure underperform, and ultimately withdraw from activities where they may be judged stereotypically. Many African Americans perceive higher levels of racism in their life and demonstrate elevated cardiovascular reactivity due to stereotype threat. Research has revealed that indistinct interpersonal encounters can be interpreted as racist and elicit elevated cardiovascular responses. According to Steele (1997) African Americans report racial interaction frequently in their life, and combined with cardiovascular reaction to racial stimuli found in the research, this may help clarify the racial disparity in cardiovascular morbidity between African Americans and Whites.

As a result of racially charged stimuli, repeated excursions of cardiovascular reactivity take place which become predictable, chronic, and do not habituate over time. Specifically, the emerging research suggests African Americans who perceive their environment more racially sensitive, demonstrate a much higher level of cardiovascular reactivity than those who do not. Compared to Whites, African Americans demonstrate as a group much more cardiovascular

reactivity to any test battery which integrates racial stereotypes; in the case of mathematical tests where African American participants were told a cultural bias exists, those participants also perform exceedingly worse than their White counterparts. In contrast, when the negative stereotype was removed, African American participants perform equally as well as Whites regardless of SES position, and demonstrate no difference in cardiovascular reactivity (Claire & Croizet, 1998). Cardiovascular reactivity resulting from stereotype threat may be the key to understanding the differential prevalence of hypertension in African Americans.

Statement of the Problem

The purpose of this review of literature is to explore the contributions of stereotype threat to the etiology of racial disparity in cardiovascular disease morbidity and mortality; specifically, the prevalence of hypertension in African Americans. First, an overview of the connection between SES position and poor health and cardiovascular disease will be covered. Secondly, racial stress and cardiovascular reactivity will be evaluated. Lastly, an introduction to the concept of stereotype threat, and mechanisms of action related to chronic stress and cardiovascular reactivity will be reviewed. Numerous mechanisms and pathways arising from living in low SES will be identified, such as greater perceived exposure to detrimental situations, as well as harmful and depressive life events. For example, living in chronic stress can have a direct impact on psychological and biological systems, which lead to downstream peripheral over activation of the CNS which compromises the immune system and increases fat and blood pressure (Mathews et al., 2010). Research suggests as SES is increased health outcomes are

improved, including cardiovascular disease. But other data propose some minority populations may not benefit equally, specifically African Americans, and may even experience decreasing health outcomes as they ascend the SES ladder.

CHAPTER II

REVIEW OF RELATED LITERATURE

Cardiovascular Disease Rates

The following section will compare the rates of cardiovascular disease found in African Americans and White Americans. The purpose of the comparison is to establish the disparity of rates of cardiovascular disease which exists between the two groups.

Pearcy and Keppel (2002) researched better methodological standards for measuring health disparity. The authors attempted to develop the Index of Disparity as one such measurement. The purpose of the study was to find more appropriate methods for measuring levels of health disparity, which often are difficult to operationalize, due to the need to define disparity widely, as well as a more focused comparison between groups.

The study model was based on standard epidemiological methods of determining disparity such as hazard ratios, relative risk, Gini coefficients and indexes of slope and dissimilarity. The ID (index of disparity) incorporated is the mean of the absolute differences of rates among defined groups compared to a population or the population rate, which is then divisible by the overall population rate. This rate is then converted into a percentage or the index of disparity. Utilizing this index, 6 health measures were compared against 5 racial groups (Pearcy and Keppel, 2002).

The results indicate the cardiovascular disease death rate for African Americans is 246 per 100,000 compared to that of Whites at 155 per 100,000. In addition, there are large disparities for African Americans within all 6 health measures when compared to Whites. Specifically alarming are the differences in incidence rates of Tuberculosis. African Americans have an incidence rate 10 times higher than Whites. Consequently, this study illustrated that the health disparity from 1989 to 1998 has not changed. The authors state better measures will better identify areas where more or improved public health interventions can be developed (Pearcy and Keppel, 2002).

Thomas et al. (2005) researched the disparity of cardiovascular disease (CVD) mortality between Black and White men within known cardiovascular risk factors. The purpose of the

study was to investigate the disparity between Black and White men and CVD mortality rates by risk factor type. It is believed there has been no research which separates overall CVD mortality rates by risk factor type. Consequently, the study attempted to discover any dissimilarity in treatment, behaviors or genetics etc. which might lead to a more targeted intervention. The rationale for the study was that in 2000, the life expectancy for White men and women was nearly 6 years longer than for Black men and women. Additionally, the ICD-10 reports cardiovascular disease related deaths among Black men was 51 per 10,000 compared to 40 per 10,000 for White men.

Thomas et al. (2005) assessed 300,647 White and 20,223 Black men selected during the MRFIT intervention trial. Names and contact information was obtained for follow up. All of the participants completed full risk assessment and identified race, medical history, health behaviors, and biometric screenings at the inception of the study. Mortality data was accumulated from the Social Security Administration and the National Death Index, and ICD data was used to determine cause of death.

Overall, the results found Black men had a lower incidence of hyperlipidemia than White men; alternatively, Black men had a higher incidence of hypertension than White men. Furthermore, Black men were classified more often as being at high risk 51% compared to 27% of White men in the study. Additionally, the incidence of elevated risk factors and low incomes among Black men were more than 4 times more prevalent compared to White men in the study. Interestingly, age, income, and risk factors accounted for only 35% of the incidence of CVD mortality in the group of Black men. Also, risk factor mediators were shown to be more predictive than race/ethnicity or income differences, but low risk White men were at much lower relative risk for CVD mortality than low risk Black men. CVD mortality risk based on income

levels was very inconsistent for both groups in this study. It is concluded that very low income in combination with high level of risk is the most deadly combination for Black men, and that risk levels of Black men increases over time which may be due to increased accumulation of psychosocial effects (Thomas et al., 2005).

Social Economic Status and Health Outcomes

The following section will review the association which exists between social economic status (SES) and health outcomes, specifically cardiovascular disease. In addition, the research may establish SES as an independent risk factor for cardiovascular disease, as well as explain the difficulty in evaluating how SES affects the multiple pathways which lead to physical morbidity.

Marmot and Smith (1991) researched the extent of causal factors which lead to the sharp inverse connection between social class and mortality discovered in the original Whitehall (1967) study. Additionally, found was a vast differences in health behaviors existed along a continuum of employment grade, and those in lower grades demonstrated many more negative health behaviors than those from higher grades.

Marmot and Smith (1991) targeted men and women between the ages of 35-55 working in London civil servant positions, which are systematically graded and defined in an orderly hierarchy. All members between these ages were sent invitations to take part. The overall response rate was 73%, with a concluding sample size N= 10,314 consisting of 6,900 men, and 3,414 women.

Participants were sent self-administered questionnaires which covered topics such as: demographics, health status, work characteristics, social networks, health behaviors and Framingham hostility scales. At the subsequent screening evaluation, participants were asked comprehensive disease state questions covering major biological systems. In addition, their work characteristics were assessed, along with major life event occurrences within the past 12 months. Moreover, biological markers of disease, such as blood pressure, lipids, and glycemic levels were assessed through blood panels. Finally, Electrocardiograms were incorporated to establish cardiovascular health and/or detect prior ischemic events (Marmot & Smith, 1991).

Marmot and Smith (1991) integrated linear regression to compare outcomes with employment levels. In addition, Cochran-Mantel-Haenszel tests were used to assess association of binary outcomes with employment grade levels, which were stratified by age. These outcomes were tested using SAS software.

The prevalence rate of smoking, bronchitis, angina, and ischemia was found to be inversely correlated with employment grade. Interestingly, known biomarkers for cardiovascular disease such as lipid levels and blood pressure are similar between all employment grades, but BMI measurements were statistically inverse compared to employment grade. The authors point to smoking as the variable with the largest difference in prevalence between employment grades. They found the prevalence in smoking in the highest grades were 8.3%, whereas the lowest grades had rates of 33%. Moderate exercise was also found to be inversely related to job grade as well, with those in the lower grades acquiring significantly less than those of higher job grades. In addition, a large variation of perceived control exists between subjects in low to high job grades. Participants in lower job grades reported lower levels of perceived control compared to subjects from higher job grades, who consequently reported much more self efficacy in directing

their life outcomes. Additionally, the study found participants from the highest job grade levels reported much higher levels of type A behavior compared to those from lower grade jobs, but those eliciting type A behavior had significantly lower rates of heart disease. Intriguingly, those participants from higher job grades had lower scores on the Cook-Medley hostility scale.

Marmot and Smith (1991) maintained the self reported data on self health rating and angina are powerful mortality predictors, because they correlate to their true pathology, and are consistent along the job classification gradient. They feel the findings can be mapped to the society as a whole, as there is a wide divergence in social class that exists in England as in the U.S. They also allude to the fact that health behaviors correlated strongly within the employment level gradient, where smoking and a lack of activity are persistent among those from lower employment grades.

Marmot and Smith (1991) stated in order to reduce the mortality rates of the lowest grade workers in the study, to those of the highest grades, their current death rates would have to be cut in half. They suggest psycho-social environments of those in lower grades must be attended to, and that the attenuation of income inequity must become a priority in order to offset the consequences of such.

Kaplan and Keil (1993) conducted a literature review of the association between socioeconomic factors and cardiovascular disease. The review authenticated and summarized the effects SES has on cardiovascular disease. In addition, they desired to improve the conceptualization of SES, and to illustrate methods which better operationalized mechanisms of SES. Finally, the authors aimed to illustrate that the effects of SES may function independently of, as well as exacerbate, other known behavioral risk factors.

Kaplan and Keil (1993) incorporated MEDLINE to search for related articles which were associated with SES and cardiovascular disease as well as their risk factors. Publications used for the review were restricted to Nordic countries, Germany, U.K., Canada, and the U.S. The researchers noted there is diversity in the overall quality of the studies chosen to be reviewed, but the overall results were reliable and remarkable. They reported the majority of the articles were peer reviewed, and originated from well known and highly ethical journals, such as the *American Journal of Epidemiology*, *American Journal of Public Health*, *Journal of the American Medical Association*, *Lancet*, and *Circulation*.

Kaplan and Keil (1993) found education as the most frequently utilized measure of SES in epidemiologic studies. The authors reported the non-response rates were low, not complex, and are esteemed by highly educated researchers. Additionally, education as a measure of SES is popular, as its level is fixed early in the life of most adults, and it is improbable to be altered by poor adult health. The authors acknowledged there were shortcomings to education as a measure of SES. An intergenerational limitation may exist; for example, many older adults may not have finished high school, so results from these cohorts may not prove as reliable a predictor as those from more recent generations. Another limitation is the association between education and income, for Women and African-Americans the correlation between education and income is considerably weaker than it is for White men. Finally, many research studies which use education as a measure for SES, failed to take into account that childhood illness may precede educational attainment, and thus confound results of association.

Kaplan and Keil (1993) reported income as a frequently employed indicator of SES. Income was important as it relates to one's ability to purchase necessities, such as food, shelter, medical care etc. The authors suggested measuring income can become difficult, because it can

be measured in many different ways, such as family, individual, non-cash benefits, and wealth. Additionally, non-response rates tended to be much higher, as questions of confidentiality and humility existed.

Occupation is an additional criterion used to measure SES; this measure usually requires categorization such as professional, technical, administrators, sales, and clerical etc. These categories are used 22% of the time as measures of SES. Problems exist in utilization of this measure, for example, a CEO of a large corporation may get grouped along with an owner of a small business, or a skilled laborer such as a master plumber may actually produce more income than a university professor. The authors argued traits such as decision making, time constraint, and personal discretion, may prove to be a more reliable way to measure occupation (Kaplan & Keil, 1993).

Kaplan and Keil (1993) state employment status is another measure of SES, they insist the effects of employment status are more complex than it might seem. They state the problem lies with how employment status is interpreted; for example, in some studies those who are unemployed but able to work, are grouped with those who are unemployed but not physically able to work, due to health reasons. They argue it is important to also collect data on those who may be underemployed, as well as the consideration of job loss.

Kaplan and Keil (1993) suggested living conditions are important measure of SES. They argued even though living conditions are associated with income and education; they also more closely correlated to important health attributes. Whether study participants own a home, car, television or other appliances, can help stratify SES more clearly, which aids in determining which attributes may be exercising more of a force on behavior.

Kaplan and Keil (1993) found there is a significant and robust pool of corroboration for the association between SES and all-cause mortality. For example, U.S. National Longitudinal Mortality Study which interviewed 1.3 million people, which supported the premise that overall mortality dropped as educational level increased. This was true across all age, sex, and ethnicity levels. White men and women with college degrees or more had 66% and 44% lower mortality rates than those with 0-4 years of education. These findings were even more pronounced among Blacks, where results show 73% and 78% lower mortality rates.

Kaplan and Keil (1993) found in a meta-analysis of mortality studies, a relationship between SES and coronary heart disease. In the 1st Whitehall Study 17,530 civil servants were followed for 24 years; after adjusting for age, the prevalence of angina was 53% higher in the lowest quartile of employment level compared to the highest. In addition, electrocardiogram irregularities, due to ischemic episodes were again 72% more frequent among those workers in the lowest quartiles compared to the upper group. Additionally, the researchers found over a 7 year period of the study, those among the lowest work categories had twice the death rates than those who were classified as professional workers in a rural Georgia town.

Kaplan and Keil (1993) suggested one way to reduce the cardiovascular effects of low SES, was to implement targeted, extensive interventions which focus on changing societal living conditions, those which leads to high risk behavior. In addition, they believed research should focus on the intermediate steps which take place between SES and poor health outcomes. Finally, the authors recommended improving economic policies which reduces the percentage of the population living in poverty. Consequently, it is believed reduced poverty will reduce cardiovascular disease rates.

Johnson, Aderson, Bastida, Kramer, Williams, and Wong (1995) researched the health effects of macrosocial and environmental aspects of ethnic minorities. The purpose of this article was to illustrate each individual ethnic group has a distinctive history of sociocultural features such as environmental, political, and economic. These features become major variables in determining health outcomes for each particular group.

Johnson et al. (1995) refuted the genetic model proposed by Cooper and David in 1986, which theorized disparities in Black and White health were attributed to differences in genetic makeup. The genetic model provided the justification for propagating Black inferiority and inequality. The authors argued the genetic medical model bases its theories on three false postulations: (1) race can be used to categorize biology, (2) genetic determinants which define race can also define health, and (3) overall health is determined by its racial makeup.

Johnson et al. (1995) incorporated a meta- analysis using keywords: minority, socioeconomic, Asian, Hispanic, African American, Native American, health status environment, and ethnic. Additionally, the authors interviewed a panel of experts from multiple ethnic backgrounds and centers.

Johnson et al. (1995) found that race is a marker of distinct experiences, and comprehending minority disparities in health is dependent upon understanding these unique lived experiences as determinants of health. SES was a strong predictor of health outcomes, but SES alone does not contain all the dimensions of living as a minority ethnic group in the U.S. Factors such as health behaviors and environmental and psychosocial stresses all play interrelated roles in the outcomes of health within these groups.

Ackerman, Kogos, Youngstrom, Schoff, and Izard (1999) researched the connection between family instability and problem behaviors of children from low SES. The purpose of the study was to illustrate the fact that children from low SES environments suffer from an assortment of social and academic issues. Additionally, the authors attempted to define more precise factors which lead to these poor behaviors and outcomes, particularly instability.

Ackerman et al. (1999) sampled 169 preschool children and their primary caregivers recruited from Head Start centers. Additionally, 151 first care givers and children were sampled. No information was given on how the particular children were enrolled in the study. Economic status in the first grade group attained was self-reported in which 27% of the sample group reported 0 family earnings. Additionally, the percentage of families reporting less than \$20,000 in earning was 30%, with half of these families reporting earning less than \$10,000. Roughly one- third of all the families in the study reported having one adult caregiver.

Ackerman et al. (1999) assessed family instability as a single index derived from questions from five categories: number of residences, number of intimate adult relationships, number of families the child has lived with, significant illnesses, and negative life events. The information used was gathered using an adapted version of the Life Events Survey, which is a 33 item scale. There was a dropout rate of 9% primarily due to childcare providers moving or not willing to complete assessment over the 6 month time period.

Ackerman et al. (1999) found a strong connection between family instability and children's ability to adjust. They also found the ability for children to regulate behavior was diminished when combined with family instability. Furthermore, they found family instability to be a certain variable in need of further investigation within low SES disadvantaged families.

Finally, there is insufficient research on the diversity with low SES families and the prior theories may need updated to reflect this need.

House (2001) examined the progress made in understanding inequalities in health in low SES populations, and how determinants of health have become social factors in health. The research examined the development of social epidemiology and how it changed the perception of the development of illness from a biological model to that of a task of psychological, social, and behavioral one. The study based much of the progression and current health disparity model on those constructs of Leo Reeder's model of psychosocial risk factors. The study reviewed the literature of the progression of psychosocial risk factors of illness and the disparity of health in the U.S. from a chronological order beginning in the 1950's.

House (2001) found that chronic stress is highly correlated to poor health outcomes, specifically coronary vascular disease. Specifically, stress, anger, hostility, mistrust, control, and hopelessness were risk factors found to be associated with low SES and minority groups. The author stated these risk factors need to be understood from a conceptual framework in order to understand their mechanisms of action. Additionally, the integration of psychosocial risk factors with racial and ethnic health disparity must be addressed because racial experience and SES position forms their contact and impression of all risk factors. Additionally, risk behaviors such as stress, smoking, drinking, and sedentary lifestyle and excessive BMI are found at much higher prevalence within lower SES levels. Finally, it was concluded reducing health disparity by decreasing social inequality is crucial for the overall health of the U.S.

Additionally, the author cautioned that there remained those who believe that health is the fundamental cause of socioeconomic position, and not the opposite. The author urged more

research needed to be conducted to fully elucidate SES position and health in order to discover ways to modify them. In addition, broader political forces are needed to re- shape the social and economical environment.

Haukkala (2002) researched the effects of socioeconomic status on five hostility measures, which included cynical distrust, anger traits, anger suppression, anger expression, and control. It was anticipated the association between SES and hostility to be inversely related, in other words as SES increases, hostility scores will diminish. The author based this premise on the findings in the Cook- Medley (1954) study which utilized the MMPI aggression inventory scores of aggression, as cynicism and hostility which were inversely related to SES. In comparison, Marmot et al. (1991) found similar findings when the Cook-Medley scale scores were assessed from British civil servants.

Methodologically, Haukkala (2002) collected data using a stratified random sample of multiple provinces within Helsinki, Finland. The study population was stratified by age group, gender, and region and the population was drawn from the population register. Additionally, there were 1,547 men and 1,856 women who returned the two questionnaires; the response rate was 67%.

Haukkala (2002) incorporated multiple measures to capture the five hostility components. Each component of hostility had its own corresponding scale. The scales ranged from 8-10 items each, they made statements which the participants rated from 1-4 with 1 representing (not agree) and 4 representing (agree exactly). Additionally, each scale was measured for internal consistency by use of Cronbach's alpha, where results ranged from an alpha = 0.76-0.89.

Similarly, social economic status (SES) was assessed using years of education, household income, and occupational status.

Haukkala (2002) measured the associations between the five hostility components and socioeconomic variables using the Pearson correlation coefficients. Additionally, MANCOVA was used to test differences in hostility variables among the different socio economic groups, and found participants from lower SES groups had elevated scores in cynical distrust, trait anger, and anger suppression compared to groups from higher SES, but among higher SES groups, anger expression scores were found to be higher than groups from lower SES groups. These findings conflicted with previous findings, but it is believed that the higher anger expression and lower anger suppression scores seen in the higher SES groups imitated the increased self efficacy and /or control those in higher SES groups are afforded. Interestingly, participants from both high and low SES groups demonstrated trait anger, in contrast, participants from the lower SES groups demonstrated higher levels of repressed anger.

Consequently, Haukkala (2002) argued hostility may be conceptually difficult to define and apply, and that more investigation needs to be conducted to better analyze more specific attributes of hostility. Also, he guided researchers to be careful making causal connections between attributes such as cynical distrust, and expression and heart disease, as well as labeling anger control as being protective, as social context must be taken into consideration.

McEwen (2003) researched how early childhood instability negatively affects long term physical and psychological health. The purpose of the study was to better understand the biological pathways involved in the prevalence of anxiety, depression, and other disorders seen in early childhood instability. Additionally, it was important to characterize landmarks in early

childhood development and to design intervention strategies to inhibit the effects of early negative childhood experiences. The study rationale stemmed from earlier research by Wilbrecht and Nottebohm, whom established animal models, which predicted long term behavior development from early life stress.

Methodologically, McEwen (2003) reviewed research based on search parameters of stress, depression, anxiety, amygdala, hippocampus, and early life trauma. All of the research reviewed was from peer reviewed articles from leading journals in their respective fields of research. The findings suggested stressful family environments during key early development periods are associated with immediate and long term health risk. Many of these effects are due to allostatic load and the stress responses which over activate glucocorticoid, cortisol, and catecholamine production. The downstream effects of these overproduced chemicals are psychological and physical morbidity leading to depression, anxiety, and maladaptive behavior responses. Furthermore, possible interventions which may alleviate early childhood stress responses should be directed at neonatal handling, or increased affection, along with home visits from professionals to support mothers in high risk environments. Also, increasing caring relationships with other family members may prove effective in reducing the negative effects of stressful family environments. Finally, the need for more research was proposed, to discover interventions which buffer young children from stressful family environments.

Pulkki, Kivimaki, Elovainio, Viikari, and Keltikangas-Jarvinen (2003) researched the effects of childhood and adult social economic status (SES) on cynical hostility and cardiovascular risk factors (behaviors) in a population based sample of young Finns. The basis of the study was the Psychosocial vulnerability model which states individuals with hostile

attributes suffer from psychosocial difficulties, which leads to undeveloped support systems, and interpersonal complications and increased cardiovascular risk.

Participants for the prospective cohort study were selected randomly from cities with medical schools by their personal Social Security number from Finland's Institution population registry in 1983. Parents' educational attainment were assessed at that time. In a 1992 follow-up, participants' SES, risk behavior, and cynical hostility were attained from 1,219 participants, which accounted for 62% of the original study pool (Pulkki et al. 2003).

According to Pulkki et al. (2003) cynical hostility was assessed using scales originating from the MMPI, which was then altered from a true/false format to a 5 point Likert scale; this was done to produce more variance in responses. The scale produced a Cronbach's alpha of internal reliability of 0.76. Additionally, SES was calculated by educational level, which was grouped as university, high school, or middle school. Also, social mobility was assessed, comparing parental social attainment and participant attainment. Finally, behaviors associated with cardiovascular risk were assessed; smoking, physical activity, diet, and alcohol usage was attained.

Variances in gender differences of SES were analyzed by chi-square, and gender differences in risk behavior and hostility were assessed using univariate analysis. Furthermore, the associations between cardiovascular risk behaviors and cynical hostilities were analyzed using linear regression, which took into account age, parental SES, and social mobility (Pulkki et al., 2002).

Pulkki et al. (2002) found that men reach appreciably higher hostility scores, consume more alcohol, and smoke more cigarettes in comparison to women. Additionally, compared to

men and women from higher parental SES levels, those from lower parental SES scored higher on cynical hostility scores and had a higher affinity for butter. Also found, was that increases in participants' own SES had a mediating effect on cynical hostility scores when parental SES was low.

Overall, cynical hostility scores were inversely related to SES scores, and as hostility scores increased, so did the number of cigarettes smoked, and amount of butter used. In addition, physical activity was inversely correlated with SES as well. Education had some mediating effects on cynical hostility and smoking in women, where the effects were borderline, adding education changed the p value from 0.019 to 0.103. But adding parental SES and social mobility to the model failed to attenuate the association between risk behaviors and SES.

Pulkki et al. (2002) concluded that the study failed to establish SES as a primary factor leading to hostility and cardiovascular risk behaviors. The author suggested unhealthy habits which are formed in childhood are challenging to change in adulthood. As a result, unhealthy diet attained in a low SES household while young, may be long lasting as an adult, even when the individual has ascended the social economic ladder. Consequently, childhood SES may prove to be more predictive as a health indicator and to understanding health inequalities.

Zimmerman and Bell (2006) tested proposed ecological causal pathways linking income inequality, to physical and mental health. The purpose of the study was to test suggested mediators such as education, crime, wages, cost of living, racial affect, and social capital linked to income inequality and health outcomes. The research was based on the conceptual model of Kawachi and Kennedy, which proposed three causal pathways in which income inequality could act to negatively influence health outcomes. The model proposed income inequality reduces

social capital and thus reduced health, income inequality leads to reduced public economic support by the wealthy, and income inequality leads to psychological stress.

Zimmerman and Bell (2006) utilized data from the National Longitudinal Survey of Youth (NLSY) in 2000. Data was completed for 4,817 persons with a mean age of 42.1, and an age range of 40-45. Blacks and Latinos were oversampled to insure comparable data. Health was determined from a self reported assessment which utilized a 5 point Likert scale, with 1 being excellent and 5 representing poor health. Following data collection, individual results were compared against a number of controlled ecological variables gathered at the county level according to the participants' residence.

Zimmerman and Bell (2006) concluded excluding state spending and unemployment rates, that the majority of ecological factors were not significantly associated with poor health. Additionally, the authors found income inequality, defined as the percentage of household income over \$150,000, was strongly associated with reporting poor health (O.R. 1.98). Furthermore, education was strongly associated with reported poor health as well (O.R. 0.59). Other findings included higher social capital being associated with less reported depression in Black and Hispanics, but this effect was not seen in the White subgroup. Interestingly, the study reported increased education attainment associated with less stress reported in the White subgroup but not for Blacks or Hispanics.

Zimmerman and Bell (2006) concluded ecological variables only modestly mediated poor health and income inequality; conversely, levels of state spending were associated with better health outcomes. In addition, there is an inverse association between the level of income inequality and health outcomes, but this association is much stronger among White people. The

authors theorized the effects of SES may be more subjective and that self definitions may play a more important role than previously thought, and that how one compares oneself to others they interact with regularly is an important consideration in better understanding SES and health outcomes.

Seeman, Epel, Gruenewald, Karlamangla, and McEwen (2010) researched the mechanisms which connect SES disparities and physiological dysregulation. The purpose of the research was to provide evidence which more concisely links gradients of social economic status to physiological outcomes. Additionally, the authors searched for ways to better operationalize the effects of allostatic load (AL). The research was based on the findings of McEwen and the theory that proposes the impact of SES is cumulative, and thus creates dysregulation of all biological systems. The results produced patterns of poor physiological aging. All material utilized originated from refereed, peer-reviewed literature, published in leading journal in their respective fields.

Seeman et al. (2010) found that allostatic load originating from gradients in SES can affect all biological systems beginning within the dysregulation of cortico-limbic system. The authors reported over activation of the hypothalamic-pituitary-adrenal (HPA) along with the sympathetic nervous system as being mainly responsible for the dysregulation. Ultimately, normal peripheral biological processes are disrupted because few biological processes are under local regulation. As a result, dysregulation of the cortico-limbic system speeds up the aging process of all major biological systems. In addition, reported level of stress may be an important mediator of SES and poor health leading to over activation of the HPA. The authors found stress responses to be a very unique human phenomenon. Therefore, perception of circumstances becomes threatening to some and extracts stress responses based on their own sensitivity to their

particular scenario. This finding may define why those from lower gradients of SES may present patterns of more severe chronic physiological response over time.

Seeman et al. (2010) found operationalizing allostatic load challenging, and reported the use of the MacArthur Study of Successful Aging group of 10 biometric measurements is a good assessment of a person's allostatic load. Utilizing the biometric measures allowed researchers a comprehensive way to standardize an index to make comparison more consistent. Increased AL was connected with an increased risk of poor health outcomes from a number of disease states when these measures were utilized.

Seeman et al. (2010) concluded that decreased SES is clearly associated with more severe deterioration of all biological systems. The authors additionally call recommended interventions which act on the current disparity of health in the U.S., they recommended interventions which can be tested and include those which can provide timely results. Furthermore, the authors warned against a too narrow focus, that all biological systems need evaluated, as AL acts on multifaceted levels.

Cohen, Deverts, Chen, Mathews (2010) researched how socioeconomic status in childhood affects adult health. The purpose of this study was to explain when childhood exposure to low SES is most impactful, how duration of exposure changes the effect and the pathways in which childhood exposure to low SES affect adult health outcomes. Cohen et al. (2010) conducted a literature review using keywords socioeconomic status, health, and childhood health. The authors reviewed three models which postulate the age in which children are the most vulnerable to the effects of low SES exposure, in addition to the consequence of exposure duration.

Cohen et al. (2010) found encounters with low SES during childhood are clearly associated with adult cardiovascular morbidity and mortality, as well as all cause mortality in general. In addition, they found there is not a preponderance of evidence which pinpoints a specific time point in which exposure mediation would be most beneficial. As a result, living in low SES environments creates a multi-faceted and complex matrix of covariates effects, both physical and psychosocial environments. Hence, the authors proposed additional defined environmental, behavioral, psychological, and biological attributes and pathways to better design interventions. Finally, they conceded the impracticality of controlling for all the measures which exist when researching the pathways as to how low SES factors work, and then comparing outcomes to differing time points along a timeline of childhood ages.

Mathews, Gallo, and Taylor (2010) reviewed the literature regarding the role psychosocial factors play as mediators of health and low SES. The purpose of the review was to examine the role of psychosocial stress, cognitions, and negative emotions as mediators of SES and health. The research was based on reserve capacity models originated from Cohen which states resources tend to act like a bank, thus they are deposited or withdrawn as necessary to protect against daily losses or harm which may arise. Moreover, excessive stressful settings diminished the reserve faster than they can be replenished, thus leaving a deficit of protection. Consequently, a diminished reserve capacity exacerbated any stressful situation which may be encountered, which led to depression and anxiety. Accordingly, unfettered stress activated the hypothalamic-pituitary-adrenocortical pathways and affects downstream biological systems. The researchers reviewed literature using keywords socioeconomic status, emotion, stress, resources, and development. Research used in this review was peer reviewed from leading journals.

The Mathews et al. (2010) findings suggested inadequate evidence existed to make strong associations between psychosocial factors and health disparities associated with SES.

Nevertheless, there was an abundance of support for the hypothesis that exposure to stress is a mediating factor for SES related health. However, there were only a limited number of studies which have examined the role of psychosocial factors as mediators of SES and health, and none have researched them directly. Consequently, the authors reported the need to explore all of the psychosocial pathways more thoroughly as there are many forms of psychosocial factors; for example, negative emotion, control and coping styles, etc. This is especially true regarding minority populations, where there are distinctive stressors, such as racism, segregation and less return from increased SES as compared to White populations.

Mental Stress and Cardiovascular Reactivity

This section will discuss the connection between mental stress and cardiovascular reactivity. Additionally, the challenges of how to operationalize stress, which is a complex and broad variable, objectively will be explored.

Kivimaki, Virtanen, Vartia, Elovainio, Vahtera, & Keltikangas-Javinen (2003) researched the effects of stress from workplace bullying and its association with cardiovascular disease. The researchers discussed how the effects of bullying are comparable to social isolation, where the self value of the subject is devalued and fatigued. The researchers argued that these subjects are prone to much higher rates of depression, anxiety, and stress, which they concluded may increase rates of cardiovascular disease through over activity of metabolic regulation and increased behavioral risk.

Study questionnaires were sent to the entire 10,969 employees of a hospital in Finland in 1998. Their ages ranged from 18-63 years, 1,712 were men compared to 9,257 women. Participants, who were employed at the hospital after two years, received a second questionnaire as a follow up. The initial response rate to the first questionnaire was 74% or 8,104 persons, 88% were women, and the participants' mean age was 43.3. Two years later, the number of participants who were still employed at the hospital was 6,674. The researchers reported the majority of those participants lost were temporary workers who were as a group younger than the mean age of the group who continued to be employed. The response rate of the 6,674 remaining participants who were eligible to receive to second questionnaire was 81%. The researchers state high income women, who were non-depressive, were overrepresented (Kivimaki et al., 2003).

Bullying was measured by a yes/no response to a question about the characteristics of workplace bullying. Whereas, depression and cardiovascular disease incidence was measured using a list of frequent disease states, where the participant would check the ones he/she had been diagnosed with during the two year period between questionnaires (Kivimaki et al., 2003)

Kivimaki et al. (2003) utilized logistical regression to assess the relationship between bullying, depression, and cardiovascular disease. In addition, odds ratios at a 95% C.I. were compiled for incidence of cardiovascular disease and depression which were age and sex adjusted. Meanwhile, the associations between risk behavior and levels of bullying were adjusted additionally with the association between bullying and cardiovascular disease.

Overall, Kivimaki et al. (2003) reported a solid association between bullying and ensuing depression, and has a strong dose response and an odds ratio of 4.2. Similarly, the researchers found a relationship between bullying and cardiovascular disease incidence defined by an odds

ratio of 2.3 versus non-bullied participants. Interestingly, this relationship was attenuated by obesity, which reduced the O.R. from 2.3 to 1.6. In contrast, risk behaviors of smoking and alcohol consumption showed little increase as a result of bullying. Alternatively, smoking and alcohol consumption were shown to increase the risk of depression. Also, the researchers found depression was predictive of increased incidence of bullying.

Jennings, Kamarck, Rose, Kaplan, Manuck, and Salonen (2004) researched blood pressure responses associated with mental stress in Finnish men. The purpose of the study was to assess how hemodynamic responses to psychological stress contribute to cardiovascular disease, specifically atherosclerosis in middle aged men from Finland. The study was based on the Kuopio Ischemic Heart Disease (KIHD) study, which reviewed cross-sectional association between blood pressure reactivity from a battery of stress tests and carotid thickness.

Jennings et al. (2004) recruited 756 men from the 2,682 participants from the KIHD study. The participants were placed into 4 age cohorts ranging from 42 to 60 at the onset of the study. Moreover, the participants were confronted with a standardized battery of mental stress tests, where heart rates and blood pressure were monitored. Concurrently, ultrasound was utilized to measure carotid intima-media thickness (c-IMT). Afterward, a follow up 7 years later was conducted, where the experimental process was repeated and differences in measurement were considered markers for atherosclerosis.

Jennings et al. (2004) discovered at the first experiment, systolic blood pressure reactivity to the stress batteries were positively and prospectively correlated to the mean c-IMT at the 7 year follow up experiment ($B=0.035$, $p=0.001$). Similarly, the correlations for IMT max and plaque thicknesses were closely related. In contrast, heart rate was not related to IMT outcomes

in this study. Additionally, when controlled for known risk factors, the statistical significance of the relationship between systolic blood pressure reactivity to psychological stress and c-IMT thickness were unchanged.

Jennings et al. (2004) concluded cardiovascular reactivity is connected to the development of atherosclerosis independent of other known risk factors, but recommended continued research should focus on assessing the connection between hemodynamic reactivity and cardiovascular disease. Additionally, psychological stress exaggerated and activated hemodynamic responses, which damage the endothelium linings of the coronary system. Furthermore, psychological stress over activated the hypothalamic-pituitary-adrenal (HPA) which may cause vasoconstriction, lipid, and blood platelet accumulation.

Hamer, Gibson, Vuononvirta, Williams, and Steptoe (2006) researched inflammatory and hemostatic response consistency to multiple psychological stress tests. The purpose of the study was to assess if inflammatory and blood platelet reactivity are consistent and reliable over time with repeated psychological stress test. The study was based on findings from Jennings that concluded psychological stress was associated with the increased development of c- IMT and was consistent and predictable over time.

Hamer et al. (2006) recruited 91 healthy, tobacco free men, whose mean age was 33 years old. They were mostly White, married, and educated. Participants were subjected to two identical psychological stress test batteries during identical times four weeks apart. Body mass index (BMI), as well as baseline heart rate, blood pressure and levels of serum cortisol, C-reactive protein, and platelet aggregates were assessed prior to stress testing. Subsequently, participants completed two stressful tasks which had been used in previous studies. The participants were

asked to rate their level of stress on a seven point Likert scale (1=not at all, 7=very).

Simultaneously, a second blood draw was administered 10 minutes after the task was completed, and saliva samples were taken at intervals of 1, 15, 30, and 50 minutes post testing.

Hamer et al. (2006) discovered no differences in the cardiovascular outcomes between the two sessions; cardiovascular reactivity was similarly elevated during both test sessions as well. Furthermore, blood platelet activation was elevated similarly during both tests, but CRP, interestingly, increased with the onset of the stress test and did not lower between test sessions. Cortisol levels acted similarly to cardiovascular reactivity, increasing during the test, and then diminishing between test sessions.

Hamer et al. (2006) concluded biological reactivity to stress was relatively constant, and is characteristic and predictable of responses which occur in real world circumstances. These findings are consistent with other research which has examined similar biological effects of psychological stress. These findings illustrated inflammatory responses due to psychological stress, are immune to habituation and are constant over time. The authors suggest limitations of this study are a lack of ethnic minority and women participants.

Mathews, Zhu, Tucker, and Whooley (2006) researched the connection between psychological stress, coronary calcification, and coronary artery risk. The purpose of the study was to assess the connection between coronary reactivity to a psychological task, to predict the existence of coronary calcification (CaC) in otherwise healthy young adults. The study was based on the hypothesis which proposed persons who demonstrate major increases in cardiovascular reactivity during psychological stress were at higher risk for atherosclerosis and subsequent cardiovascular disease.

Mathews et al. (2006) assessed 2,816 young, healthy black and White women, with ages ranging from 20-35 years. The participants, prior to the study, were evaluated for clinical symptoms of hypertension or diabetes and those enrolled in the Coronary Artery Risk Development in Young Adults Study (CARDIA) were without either. The study protocol required the participants to engage in video games and star tracing while blood pressure readings were recorded. Subsequently, thirteen years later, they were assessed for levels of CaC by cardio CT techniques.

Mathews et al. (2006) found 9.3% or (261 of 2,816) of the participants had CaC present at the conclusion of the study. In addition, they reported for every 10mm Hg increase in systolic pressure recorded during the stress tests was associated with a 24% increased odds ratio (1.25; $P=0.008$). Furthermore, they reported these findings were present after controlling for various confounders such as race, gender, education, smoking, alcohol consumption, BMI, family history, and age. Interestingly, blood pressure changes associated with the star tracing task were not linked to the presence of CaC. It was concluded cardiovascular reactivity may be beneficial in assessing potential CVD risk as adjunct to known risk factors.

Knepp and Friedman (2008) researched the connection between prolongation of stress recovery and cardiovascular disease risk. The purpose of the study was to assess the effects of worry and anxiety on the prolongation of stress recovery and thus an increase in risk from cardiovascular disease. The study was based on that proposed by Rosenman, which states Type A personality and anxiety through mediators of cardiovascular reactivity, are connected to the development of cardiovascular disease.

Knepp and Friedman (2008) recruited 41 female college students from the Virginia Tech Department Psychology's list server. There were 472 students who completed the online screening questionnaire, which measured levels of worry, as well as assessed and excluded those with a history of hypertension, diabetes, respiratory issues, who use tobacco, or those currently taking anxiolytic or depression medication. Ultimately, 22 women, who were assessed as having high worry, and 19 assessed as low worry was chosen to participate. The experiment conducted was double blind and randomized. The experimenters applied six different stressor protocols to measure stress response; these tests consisted of Orthostatic stress experiment, cold hand pressor, and imagery task patterns. The primary hypothesis, cardiovascular reactivity (baseline, task, recovery) were assessed by using ANOVAs for the (2) groups (high vs. low worry). Additionally, a t-test was used to evaluate differences within each group.

Knepp and Friedman (2008) found only one statistically significant finding, which was a greater propensity for high worriers to demonstrate greater heart rate versus those from the low worry group. The finding was consistent across all of the stress protocols, but no connection was observed with respect to heart rate variation (an important marker for parasympathetic control).

Hamer, Molloy, and Stamatakis (2008) researched for mediating factors between psychosocial stress and cardiovascular disease. The purpose of the study was to investigate the mediating factors which may connect psychosocial stress and cardiovascular disease. Behaviors such as alcohol consumption, smoking, and sedentary lifestyle may be mediating factors, or alternatively, they could also be coping or adaptations from stress. Additionally, increased sympathetic nervous system activations may also play an integral part in discovering mediating factors. The study was based on theories by Robinson, who demonstrated the existence of

psychosocial stress is associated with increased risk of cardiovascular disease comparable to other known risk factors such as sedentary lifestyle, obesity, and hypertension.

Hamer et al. (2008) enrolled 6,575 participants which were chosen from the Scottish Health Survey (SHS), an intermittent sample (every 3-5 years) that creates a nationwide representative sample of households. The sample was compiled using a multistage stratified probability technique. In addition, participants were recruited from two SHS pools, 1998 and 2003. Participation criteria included those 30 and older, who consented to a biometric blood screening. The study excluded those who had reported a prior CVD or refused the nurse visit or declined to have blood drawn. The overall rate of response was 75%. Of the 11,352 participants recruited, 6,576 finished the study. Interviewers made contact with each eligible household, where demographic as well as health related behavioral information was attained. In addition, mental health was assessed by means of a 12 item scale, which measured psychological distress. Cox Hazard Models were utilized to approximate cardiovascular risk along a psychological stress continuum. Hamer et al. (2008) integrated multivariate models controlled for confounders such as sex and age. Also, the investigators added health risk behaviors such as smoking, inactivity, and alcohol consumption as variables, and then included biological markers such as cholesterol level, hypertension, C-reactive protein, and fibrinogen.

Hamer et al. (2008) found that participants who were assessed to have high psycho-social stress were at higher risk of cardiovascular events compared to those who were not. They also engaged in high risk behavior such as alcohol consumption, smoking and sedentary lifestyle. There was a consistent demonstrated dose response relationship represented by a 7% increase in the risk of CVD for each 2.7 points increase in the stress assessment score. Behaviors such as

smoking and physical activity accounted for 83% of the effects of psycho-social stress. In comparison, hypertension only accounted for 13% when compared separately.

The Hamer et al. (2008) was a prospective study with an N=6576, which solidly established a temporal sequence. The researchers attempted to explain the association of stress and CVD by showing that behavioral risk is a more important factor than physiological risk, since it is more closely linked as a coping mechanism to stress. The researchers reported it is problematic to assume behaviors such as smoking, drinking, or physical inactivity are a manifestation of stress and anxiety, which in fact may be vice versa. In addition, stress and behavioral activities were measured simultaneously at the inception of the study further exacerbating the temporal sequence dilemma. Additionally, exposure to stress was not reassessed during follow-up, which they admit could have changed the outcome. Furthermore, stress may be considered common enough that a prospective study may create exposure misclassification.

McEwen (2008) researched the damaging effects of stress, and stress mediators as they contribute to disease. The purpose of the research was to delineate the differences between acute and chronic stress and how the body adapts to them differently. In addition, the research explored the concept of how the burden of chronic and allostatic overload leads to biological and personal alteration. The study was based on the model hypothesized by Stellar and McEwen, which states chronic allostatic overload leads to a breakdown or dysregulation of biological systems from an inability to shut down the biological responses when they are no longer necessary. Similarly, the incapability of orientation to reappearance of a stressor increases the allostatic load. McEwen (2008) reviewed literature based on keywords of stress, allostasis, chronic disease and the brain. Additionally, literature was chosen based on the quality of work, and their journals they were published, which originated from peer reviewed refereed journals.

McEwen (2008) reported there are two sides of the equation to the effects of stress: protection and damage. The traditional fight or flight response to stress and danger had adaptive value, and was protective, but is typically short in duration and protective in theory. The responses to danger or to stress from daily life are the same. The same stress which triggered bears to gorge and accumulate weight before winter for survival, is the same response to boredom bears experience when kept in zoos. Stress from boredom enacts sympathetic nervous system activation, in which bears then respond by overeating. Similarly, humans adapt to chronic stress the same way from sympathetic activity by overeating, drinking, smoking, physical inactivity, sleep deprivation, and depression and anxiety.

McEwen (2008) reported the importance of how one interprets what is a stressor, because the interpretation controls the physiological responses and downstream behavior. Consequently, depending on a person's unique life experiences, the variances in response to stress can greatly fluctuate. For example, a bad experience early in school can bias a person's response to a new circumstance. In the same way, an ethnic minority who experiences discrimination could approach society based on previous experiences, and could interpret similar situations more stressful than a White person in the same situation. Likewise, early life sexual abuse for example, could lead to long-term behavioral problems.

McEwen (2008) stated that social support, positive outlook, and good self esteem are important factors in buffering allostatic load. Positive outlook accumulating from positive experiences early in life, were connected to reduced cortisol levels as well as increased parasympathetic reactivity. In contrast, those with lower levels of self esteem have difficulty in habituating to repetitious events such as public speaking, where those with higher levels of self esteem are able to control their stress response to additional speeches given.

Guerrero and Palmero (2010) researched the influence of defensive hostility on the development of cardiovascular disease. The purpose of the study was to assess the association between defensive hostility and the cardiovascular reaction to stress, and the role hostility plays as a likely psychosocial risk factor to the reaction of stress. The study was based on research by Chesney which concluded 80% of cardiovascular deaths are associated with those who were relatively poor. Additionally, the research revealed traditional risk factors such as hyperlipidemia, hypertension, smoking, alcoholism, and sedentary lifestyle only account for 50% of cardiovascular deaths.

Guerrero and Palmero (2010) utilized the Cook-Medley Hostility Inventory and the Marlowe-Crowne Social Desirability Scale and assessed defensive hostility in 130 University of Castellon female students in Spain. All the students were volunteers, and were not taking prescription medication or ill with any disease which could alter the results of the test. As a result of assessment, there were four groups of participants classified as DH-high hostility/high defensiveness, HH-high hostility/low defensiveness, DEF-low hostility/high defensiveness, and LH-low hostility/low defensiveness. All participants were given real exams for the experiment, and cardiovascular biometrics were recorded through three phases (adaptation, task, and recovery). Hypothesized was those participants from in the high hostility and high defensive groups would demonstrate higher cardiovascular reactivity throughout the three phases.

Guerrero and Palmero (2010) found participants within the high hostility/high defensive group displayed the greatest elevation of cardiovascular reactivity, and thus supported the premise of defensive hostility as a predictive association with cardiovascular reactivity levels and function in stressful circumstances. Additionally, it was concluded participants who responded with high cardiovascular reactivity, reproduced identical patterns of response to repeated stress

stimuli. Consequently, those who display elevated cardiovascular reactivity to stress consistently face increased risk for cardiovascular disease.

Racial Stress and Cardiovascular Reactivity

This section explores the need to assess racial stress as a risk factor which may account for the high rates of hypertension which exists within the African American population. Historically, research has failed to firmly establish an etiology for the disparity in hypertension rates experienced by African Americans; this section will attempt to establish racially based stress as a potential cause.

Ong, Rowell, and Burrow (2009) researched the distinctive effects of chronic racial discrimination on psychosocial stress. The purpose of the study was to discover mediators of chronic racial discrimination which lead to poor mental health outcomes in African Americans. The study was based on research by Bolger, which developed the Stress Process Framework: five models which measure direct, moderated, and mediated factors linking racial discrimination and psychological morbidity. The five models are (1) Chronic exposure to discrimination increases distress. (2) The connection between racial discrimination and stress is mediated by daily discrimination. (3) The existence of chronic discrimination produces increased physiological stress responses. (4) The connection between chronic discrimination and stress is mediated by every day negative events. (5) The existence of chronic discrimination will produce increased physiological response to day to day negative events.

Ong et al. (2009) recruited 174 African American graduate students (141 women, and 33 men) from a national fellowship program, and other Black graduates associations. The response

rate was 81.3%, and 40 participants not completing the required number of diary reports. The omitted participants revealed no differences of demographic makeup than those who completed the tasks. Data was collected using a secure website, and instructions were given on how to access and provide responses. Participants completed a baseline assessment on their experience with discrimination prior to beginning the diary portion of the research. Afterward, participants accessed the website and completed measures which assessed daily events, moods, and stressors for 14 consecutive days. The measures included multiple Likert scales which measured racial discrimination (chronic and daily), negative events and effects, and anxiety and depression by frequency, and their reaction. The participants were compensated \$25 for their time.

Ong et al. (2009) found participants experienced discrimination on 26% of the 14 days of the study. Additionally, the daily reports of discrimination were highly correlated to reports of chronic discrimination. Lastly, the effects of discrimination such as negative outlook, depression, anxiety, and negative events were all strongly correlated together. It was concluded racial discrimination contributes greatly to the negative mental and physical outcomes of African Americans. In addition, stress proliferation resulting from racial discrimination led to additional stressors, which is often an overlooked result of stress accumulation. Finally, daily racial discrimination and negative events were discovered to provide the connection for chronic discrimination and physiological distress.

Morell, Myers, Shapiro, Goldstein, and Armstrong (1988) researched cardiovascular reactivity in African American men compared to White men. The purpose of the study was to assess and compare cardiovascular reactivity in Black and White men to behavioral stress. The study was based on the Krantz's hypothesis which proposed the differences in hypertension rates between Black and White men are attributed to CNS dysregulation of cardiac and vasculature

control. Additionally, proposed is that dysregulation is partly due to chronic exposure to elevated cardiac reactivity.

The Morell et al. (1988) study participants were recruited by newspaper and public group marketing. Volunteers were required to complete a telephone health assessment, those who met the research criteria were enrolled in the study. Included in the study were 34 Black and 42 White participants, who were assessed and scored on variables such as current health status, psychosocial and behavioral risk factors. Again, a series of blood pressure readings were compiled and then averaged to establish a baseline reading. The study excluded those men who were not normotensive $>140/90$ BP. The Demographics of the study demonstrated no differences in the use of alcohol or tobacco, and ages were similar between the Black and White participants. Whites in the study had an average family income \$8,000 higher than the Black participants. Additionally, Whites in the study had significantly higher levels of education than the Black participants.

Morell et al. (1988) required the participants to complete a mathematical test, which consisted of various timed examinations. Blood pressure readings were continuously captured during the battery of 5 1-minute tests. The results were analyzed which resulted in higher baseline diastolic blood pressure readings within the black participants compared to the White participants, that comparison remained constant through the testing sessions as well, but there were no covaried differences, and there were no task effect differences found in the study. It was concluded no evidence existed which linked past family hypertension history to elevated CV responses to mathematical stress compared to those without family history. Additionally, there were no task effect differences between racial groups which were connected to the educational and income disparities between the racial groups.

Armstead, Lawler, Gordon, Cross, and Gibbons (1989) researched the connection of racial stress to anger, and blood pressure response in Black college students. The purpose of the study was to establish etiological variances of hypertension rates between Black and White persons in the U.S. The study was based on the theory by Garrison which proposes the differences in essential hypertension rates between African Americans and Whites are rooted in racism and poverty, which led to chronic stress, even though African Americans display historically lower BMI, less dyslipidymia, and lower heart rates compared to Whites.

Armstead et al. (1989) enrolled twenty-seven African American college students, out of fifty-one students, who were originally invited by personal letter. The participants viewed nine film selections, each of them were 1.5 minutes long, which expressed either anger provoking, racist, or neutral content. A mood checklist was used to self-report the existence of anger; the 5 options given were depressed, angry, happy, indifferent, and amused. At that point, stable BP measurements were obtained, and the film excerpts were started. Blood pressure readings were conducted after each 1.5 minute film was finished, and the mood checklist was completed.

Armstead et al. (1989) reported that BP considerably increased in connection to racial stimuli, but anger provoking and neutral scenes did not provide similar task effect. Additionally, self assessments of anger states, captured by the mood checklist, were positively correlated to both the racist and anger provoking film excerpts. The researchers concluded CV reactivity underlies the etiological differences African Americans hypertension rates, and that racial stimulus incites a greater CV response compared to anger. Hypothesized is African Americans' sensitivity to racism may be culturally constructed, and the increased CV reactivity demonstrated may be a cognitive reaction to "symbolic" threat.

Anderson (1989) researched the differences in cardiovascular reactivity arising from induced stress. The purpose of the article was to more clearly delineate the reasons for the apparent disparity in cardiovascular reactivity in African Americans compared to Whites. The research was modeled from Krantz, which conceptualized chronic hypertension in African Americans has its etiology based on psychosocial factors such as crowding, conflict, and racism. As a consequence, over activation of the sympathetic nervous system leads to elevated cardiac response, such as increased heart rate and blood pressure, and when this is sustained leads to chronic essential hypertension. Consequently, the author hypothesizes, if elevated CV reactivity is connected to an increased vulnerability to hypertension, then the increased disparity in hypertension rates among blacks implies the high level of CV reactivity reported to stress may be a factor in development of hypertension in Blacks.

Anderson (1989) collected data on areas of research which focused on variances in resting CV activity of adults, adolescents, and children. Also, research was sought on the variances of CV reactivity by racial groups, as well as variations within African Americans. Lastly, data was collected which addresses to recognize individuals' characteristics that may lead to greater risk for hypertension stemming from increased levels of CV reactivity.

Anderson (1989) discovered resting heart rates of Black children younger than 6 months were faster compared to White children; this result was independent of SES and body weight. In contrast, the heart rate finding of younger children reverses with age, with Black adolescent and adult rates being lower compared to White adolescent and adult rates. In comparison, resting blood pressure differences between Black and White infants appear to be negligible, but as both racial groups age, blood pressure increases in both groups. As for adults, blood pressure was found to increase in both groups as they age, when in adulthood, blood pressure levels increase

more within the Black population. In addition, blood pressure variation is greater in African Americans compared to Whites, where SES, stress, coping style, and social support have been demonstrated to mediate the differences found within in blood pressure with Blacks.

Anderson (1989) reviewed literature conducted on the cardiovascular reactivity differences observed in different racial groups, when challenged by physical stress tests. In 6-15 year old boys, there were no differences in resting systolic blood pressure between White and Black subjects. However, when stress-tested on a stationary bike, black youth demonstrated larger increases in systolic blood pressure than White subjects, although there were no differences recorded in overall heart rate. The systolic blood pressure variances may have been due to greater differences in vasculature of the Black subjects. In addition, 10-15 year old Black youths who presented with a family history of hypertension, demonstrated higher blood pressure overall than those Black subjects without, including pre tested and during exercise. Alternatively, White subjects demonstrated no variation in blood pressure dependant on familial hypertension history. Finally, when presented without familial history of hypertension, no difference in Black and White youth blood pressure was demonstrated.

Anderson (1989) reported among normotensive adults during a physical stress test, Black participants aged 18-22 demonstrated greater CV reactivity to a cold pack stimulus test than did Whites. A similar finding was found when Black and White young adults were sodium loaded; Black participants demonstrated a lower sodium excretion rate than did White participants. Also, when blood pressure was measured after different levels of sodium were infused; Black participants demonstrated higher blood pressure responses than Whites to doses of sodium 600 mg and above. Suggested, is that Blacks may be at increased risk for hypertension based upon

familial history, and interaction with other risk factors may exacerbate this tendency for cardiovascular reactivity.

Anderson (1989) reported the data has not clearly illustrated a difference racially in cardiovascular reactivity to psychosocial stress. Even though sporadic results have demonstrated some increased blood pressure responses, in contrast, some results have demonstrated less reactivity to task in Whites than Blacks. This test effect has been seen especially in heart rate, where White participants have demonstrated increased heart rate to study tasks.

Anderson (1989) concluded while there has been racial variances found in resting heart rates, in Blacks the difference disappears over time. Additionally, it has been demonstrated that resting blood pressure overall is higher in Black populations than that of Whites. The study showed CV reactivity to exercise among young Blacks to be higher than that of Whites in the same study, but results were not unequivocal when related to psychosocial stress and CV reactivity. Thus, the role of excessive beta-adrenergic function as a mediator of the development of chronic hypertension among African Americans remained unclear.

Strogatz, Croft, James, Keenan, Browning, Garrett, and Curtis (1997) researched psychosocial factors rooted in low social economic status, and factored in the disproportionately high rates of hypertension and cardiovascular disease within African Americans. The purpose of the study was to measure any connection between social support and perceived psychosocial stress along a SES gradient, with the burden of hypertension in African American adults in Pitt County, North Carolina. The study was based on the psychosocial constructs of Gentry that relates the variances of hypertension across race are linked to the differences in lived experiences

of African Americans. These lived experiences are interlaced with stress, discrimination, disparity of economic resources, lack of support, and reduction in control.

Strogatz et al. (1997) recruited a community based sample of 1,784 Black adults, which were stratified and randomized to construct an economically diverse sample. Eligibility requirements were that they were Black, between the ages of 25-50 years old, and willing to be interviewed. Interviews were performed at participants' homes where baseline blood pressure was captured. A four point Likert scale provided scoring for questions answered (never, sometimes, most of the time, and always) regarding social support. Perceived stress was assessed by the use of the Perceived Stress Scale, and it used the same 4 point scale and answer choices utilized for social support.

Strogatz et al. (1997) reported finding for both emotional and economic support to be predictably inversely related to blood pressure measurements, and in contrast, stress was predictably directly related. In addition to the separate indices association found, there was also a large cumulative effect when stress and low support effects were combined. Furthermore, three SES strata were constructed based on educational attainment and income level. Found was women in the highest SES levels reported the greatest social support and the least amount of stress. In contrast, this finding only held true for men for social support. Reduced perceived stress for men was found in the middle levels of SES only, and was elevated in men from the highest levels of SES.

Clark, Anderson, Clark, and Williams (1999) investigated how racism as a possible stressor is connected to the high rates of overall morbidity and mortality. The purpose of the article was to chart a biological psychosocial model of the mechanism of action of perceived

racism, to direct research in the future. The article is based on the model by Lazarus, which states that perception of a particular stimulus such as racism produces an elevated psycho-physiological response to that stressor, and the responses are filtered through a complex variety of environmental, psychological, socio-economic factors. Consequently, when activated over time these stress responses are unequivocally attributed to poor health outcomes.

Clark et al. (1999) suggested that the perception of racism is ultimately subjective, and is complexly constructed through filters of life experience such as socioeconomic position, constitutional and environmental, and psychological and behavioral factors. Socioeconomic position was found to be an important factor regarding the perception of racism. SES position was found to be inversely connected with experiences of racism, though some studies reported direct associations with SES and experiences of discrimination. It was thought this may be due to the nature of definition of discrimination. For example, African Americans from higher levels of SES with higher levels of education perceive more covert types of discrimination, where those from lower levels may experience more overt types and report higher levels as well. Additionally, constitutional factors such as skin tone have been reported as having an impact on the level of perceived discrimination within African Americans. Also, environmental factors have been shown to dampen or elevate the perception of discrimination. Lower wages, high unemployment, and substandard housing all affect the perception, and thus the associated physiological and psychological responses to stress. Lastly, determining the amount of perceived racism and stress was dependent on many social factors, which act as lenses which focused and/or distorted the environment in which African Americans live.

Alba, Logan, and Stults (2000) researched the segregation of middle class African Americans. The purpose of the study was to assess if increases in SES status of African

Americans decreases segregation, which is the case for low SES African Americans. The study is based on the theory by Massey, which stated an increase in SES position of African Americans does not diminish segregation.

Alba (2000) utilized the location-attainment model which was pinpointed at the individual; the independent variables used were educational level and household family income. The dependent variables were the attributes of the neighborhoods where individuals live. Additional models were used to assess the percentage of Whites, the zip code tract median household income, and the median income of Whites, which defines which Whites are residing in a particular tract.

Alba (2000) found in the most segregated cities in the country, middle class Blacks who live in suburban areas, have many more White neighbors than the poorer Black who live in the inner city. Nevertheless, these White neighbors have lower household income from the Blacks who live beside them. In contrast to Whites who have similar SES affluence, Blacks reside in less affluent residential areas than Whites with equal SES attainment. Furthermore, they found home ownership for Blacks equates to living in residential areas with fewer White neighbors. Thus, home-ownership has been shown to be associated with more segregation than renting. These findings then may be extrapolated in terms of the advantages gained by increased SES for Blacks, which do not appear to keep up with the pace of advantages enjoyed by Whites with the same SES attainment.

Williams and Morris (2000) researched the mechanism of action of racism and detrimental mental health outcomes. The purpose of the research was to better understand how institutionalized racism attributes reduced SES growth and leads to chronic poverty, as well as

understand how discrimination disrupts normal physiological and psychological pathways which are detrimental to mental health. Lastly, they described how societal approval of negative minority stereotypes lead to poor self evaluations, and have negative effects on overall well-being. The study was based on the findings in Catchment Area Study, and the National Comorbidity Study, which found that increased rates of psychiatric disorder were inversely related to SES attainment, with those from the lowest SES quartiles having the highest rates. Williams and Morris reviewed population based, peer reviewed and refereed research, from well known journals in their respective fields of study.

Williams and Morris (2000) concluded that in the United States, historical beliefs of Black inferiority, residential segregation policies were created and support by the government. Additionally, cooperation from banking and real estate institutions, have been the primary mechanism employed to propagate racial inequality. Furthermore, in addition to institutionalized segregation, restrictions to equal educational and employment opportunities have also contributed to the racial disparity in health which currently exists. Institutionalized racism has been the pathway which underlies the condition of chronic poverty and poor health outcomes for African Americans.

William and Morris (2000) cited instructional racism has had a dramatic effect on SES outcomes for African Americans. African American rates for unemployment are more than twice that of Whites; even when educational levels are equal, unemployment rates for African Americans are dramatically higher. Consequently, low chronic economic attainment has been associated with increased rates of psychiatric disorders, all-cause morbidity and mortality rates among African Americans. Also, mental health can be affected by racism from an individual's subjective experience. Experiences of discrimination elicited stress from a myriad of contexts

which people filter discrimination, but the effects of contact to discrimination have been shown to elicit cardiovascular and physiological hyperactivity from the downstream effects of stress.

Foreman (2003) researched the social psychological effects of racial segmentation in the workplace. The purpose of the study was to examine the association of racial segmentation in workplace and the effects on psychological impairment. The study was hypothesized from research conducted by Kohn and Schooner on the connection between social stratification and health outcomes. The author reported little research existed which focused on measurements of race segmentation in the workplace.

Foreman (2003) utilized data from two sample surveys, the National Survey of Black Americans and the Detroit Area Study. These studies assessed perception of racial segmentation in addition to assessments of psychological wellness. The National Survey of Black Americans was a nationally conducted probability sample of African American adults, where every African American household had the same chance of being selected for inclusion in the study. The study had a 67% response rate where 1,199 household out of 2,107 responded. The Detroit Area Study sampled residents from the greater Detroit area, where in-person interviews of 1,139 African Americans and Whites were conducted, (586 African American, and 520 White) with a response rate of 70%. Dependent variable items measured were personal efficacy, perceived life quality, and psychological distress. Independent items selected were perceived racial segmentation, as well as individual and occupational characteristics, perceived discrimination, substantive complexity, physical demands, and job quality. Each of these items of measurement was assessed by Likert scale responses to questions which required participants to answer yes or no, or strongly agree or disagree etc and treated as ordinal level variables.

Foreman (2003) discovered nearly half of African Americans perceive racism at work, and were limited to more menial types of work compared to Whites, and that Whites were hired for more highly sought-after positions. This perceived segmentation for African Americans was associated with negative psychological outcomes, and reported less control over their lives. Reduced life quality, efficacy, and psychological distress also provided evidence to support the hypothesis that workplace racial segmentation is associated with lower levels of well being.

Arthur, Katkin, and Mezzacappa (2004) researched cardiovascular (CV) reactivity to mental stress of study participants of African Americans, Caribbean Americans, and White Americans. The purpose of the study was to assess CV reactivity of Black Americans of different ancestry (Africa and Caribbean) as there has been no published research differentiating different Black ethnicity and CV reactivity to stress. As a result, ethnicity may be important due to the historical differences of the two groups, and as a consequence, may alter their perceptions of discrimination, values, control, family structure, identity, and social trust. These results may affect CV reactivity and outcomes. The study was based on research by Sloan which proposes increased CV variability to stress may be a risk factor for cardiovascular disease, the study showed African Americans demonstrated higher levels of CV variability than White Americans. As a consequence, higher CV reactivity in conjunction with higher levels of stress due to discrimination, may lead to higher levels of CV morbidity and mortality.

Arthur et al. (2004) recruited 43 men and 45 women from an undergraduate psychology course. Thirty of the participants had previously self indicated as being from Caribbean descent, an additional 15 male and 15 female African Americans were selected from a completed questionnaire, along with 30 ethnic White participants. The participants were sex and age matched for this study. Study procedures included a health assessment which included elements

regarding psychosocial, SES, and ethnicity. Following the assessment, participants performed a 4 minute math task, and a 2 minute cold pressor task, where a number of CV metabolic measurements were taken to assess reactivity to the task.

Arthur et al. (2004) reported differences existed in SES, as the White participants demonstrated the highest SES backgrounds, with the Caribbean American group having the lowest. Differences also existed for task effect between all three groups tested. Blacks demonstrated less parasympathetic activation than Whites in the study; in contrast, the Caribbean group demonstrated parasympathetic activity similar to those of the White group's levels. Prior studies found decreased parasympathetic activation increased the risk for CVD. In addition, Whites demonstrated greater heart rate increases to stress than Blacks, which has been found to be consistent with prior research. There were no differences in blood pressure or peripheral resistance measurements between any of the test groups, which are atypical in comparison to prior research. Consequently, Black subjects typically demonstrate increased peripheral resistance in stress tests. The researcher also found Whites had the largest systolic blood pressure changes of the three test groups, which are not typically found in previous literature. As a result, illustrated blood pressure measurement alone does not provide enough information to make definitive extrapolation on the effects of stress.

The researchers recommend ethnicity variations should be taken into account when conducting research on CV reactivity. Additionally, there are distinct differences in perceptions of health and illness which may alter the psychological filter in which experiences are processed, and thus reactions to stress are expressed (Arthur et al., 2004).

Lepore, Revenson, Weinberger, Weston, Frisina, Robertson,...and Cross (2006) researched cardiovascular (CV) reactivity effects in Black and White women subjected to social stressors. The purpose of the study was to determine and compare the CV reactivity to social stress exposures in Black and White women. Additionally, specific types of stress, specifically racial stress stimuli, were tested to determine if Black and White women respond similarly. The research was based on the findings of Fang, that both White and Black men responded similarly to racially themed events and no difference exists. Lepore et al. (2006) pointed out that different stimuli were utilized in the study, and thus made outcome comparability difficult.

Lepore et al. (2006) assessed the CV reactivity response in 40 Black and 40 White women in a quasi- experimental design. The participants were recruited through campus flyers or given extra credit for participation. Participants were excluded if history or treatment for hypertension were disclosed. The participants all gave three speeches, one was a control speech, the other two speeches were meant to invoke stress by the participant. One of the speeches was an airport scenario which depicted a flight delay, and was non racial in nature. The third, a racially charged scene at a department store where the person was charged with shoplifting. Speech orders were randomized to eliminate effects of order. Blood pressure and heart rate were monitored and recorded during the process. The participants were asked to self assess their own level of stress using a 7 point Likert scale with 0 (none) to 6 (extremely).

Lepore et al. (2006) found in comparison to White women, Black women demonstrated much greater increases of diastolic blood pressure in response to the racial stimulus compared to responses to the non racial speech. In addition, Black women who openly mentioned race as an attribute of the speech, had much greater CV reactivity than those who did not. The researchers

concluded that perception of racism is a very important variable and may be tied to the ability to predict how stress responses may be evoked from racism and thus CV reactivity response.

James, Van Hoewyk, Belli, Strogatz, and Williams (2006) researched the effects of relative socioeconomic deficit across the lifespan in African American men. The purpose of the study was to test the three major constructs associated with SES and the development of early onset morbidity and mortality. First of the three models tested was the Latency Effect model, which proposes the health effects associated with impoverished conditions in early life occur independent of changes to SES in later life stages. Second the Social Chains of Risk model proposes, in contrast to Latency Effect model, changes in adult SES can affect health risk trajectories established in early childhood. Last, the Cumulative Burden model proposes that health risk is an aggregated process occurring over time in response to changing environmental changes.

James et al. (2006) utilized data from a 2001 follow up survey of the participants of the 1988 Pitt County Study, which assessed risk factors for hypertension in African Americans from age 25-50 in 1988. Selected for this study were 379 of 418 African American men who agreed to be interviewed. The participants were first stratified on their parents' SES attainment; furthermore, on their own adult SES which included educational, employment, education, and home ownership status. The produced classification groups (4) included low childhood SES/ low adulthood SES, high childhood SES/low adulthood SES, high childhood SES/high adulthood SES.

James et al. (2006) found a moderate latency effect (OR=1.60) in hypertension rate disparity between the two groups from low childhood SES/high childhood SES alone.

Alternatively, a stronger association was observed in hypertension rates between the low adulthood SES/high adulthood SES, with low adulthood SES group demonstrating an doubling in risk for hypertension (OR=2.25) versus the high adulthood SES group. Additionally, when the high/high SES group was compared to low/low SES group, the low/low SES demonstrated a 7 times greater odds of developing hypertension than the group from both high childhood/high adulthood SES. Finally, when both high childhood SES groups were compared, the group with low adulthood SES attainment demonstrated a higher risk for hypertension 6 times greater than the group who maintained their high childhood economic status (OR=5.87).

James et al. (2006) concluded the greatest evidence provided by the study supported the cumulative burden model, which demonstrated an Odds ratio of 7.27. Those who were disadvantaged throughout their lifespan, revealed the highest risk versus the participants who have always enjoyed less burden in their life. Additionally, there is evidence supporting the theory that early lifespan hardship contributes to long term health effects, but may be moderated by increasing SES attainment. Upward mobility reduced rates of hypertension by 47% in the low childhood SES participants who attained higher level SES status, demonstrating a buffering effect.

Stereotype Threat and Cardiovascular Disease

This section will discuss the phenomena of stereotype threat, the mechanisms, and the implications to long-term health. African Americans dissimilarly to White Americans are constantly being judged by the color of their skin. Their skin color connotes many negative attributes and stereotypes about the group in general, and when a Black person engages in an

activity which has negative stereotypes attached to it, the member of that group develops fear, anxiety, and threat. The threat occurs when the individual fears they are being judged under a particular stereotype, and fear they may confirm the stereotype they are judged against to be true. The additional stress created from this situation causes performance issues and avoidance, and leads to the development of chronic over activation of the cardiovascular system. The totality of this process may lead to the disparity in rates of chronic hypertension, and cardiovascular disease.

Steele (1997) researched how stereotype threat can disrupt the domain identification in academics in African Americans, which results in underperformance in academic settings. The purpose of the study was to examine the role stereotype threat plays as a mediator for domain identification malfunction, which creates underperformance in academic settings for African Americans.

Steele (1997) based the research on the domain identification theory, which proposes success in any particular domain, such as academics, necessitates a person first to positively identify to that particular domain. Self definition is needed in order to achieve positive results and thus continued motivation to reproduce those positive outcomes. In addition, a person must perceive positive outcomes from the domain, which requires attention, proficiency, opportunities, and resources to do well. Consequently, if there is a failure for a connection to be made or the association gets fractured for any particular domain, then a failure to perform within that domain will exist.

Steele (1997) found social structural limitations of access, SES disparity, segregation, and historical patterns of discrimination have long lasting effects on the ability to identify with

scholastic domains. Additionally, barriers exist for those who survived these obstructions and have self identified with academic domains, which is stereotype threat. Stereotype threat takes place when someone is performing a task or in a domain where a negative stereotype is associated with a particular group and the members belonging to it. A threat develops from being negatively stereotyped, judged, and treated in a stereotypical way; this is especially true where a group has been historically marginalized and discriminated against based on stereotypes. Members of these groups are especially susceptible to stereotype threat, as bad long held stereotypes typically exist, which increase the fear of being classified or labeled under a negative stereotype. Consequently, when a member of a group is involved in a domain where a negative stereotype exists, the situation or task can become self threatening.

Steele (1997) suggested when stereotype threats exists, anxiety occurs affecting performance, such as when African Americans are tasked with taking a math test, the stereotype which lowers performance expectations creates stress and anxiety which hampers performance. When stereotype threat becomes a chronic condition, then re-identification can occur. This phenomenon is described as a person who once self identified with a particular domain, and in the presence of chronic stereotype threat, re- evaluates their own identity about the domain, and disassociates with the domain.

Steele (1997) discovered the collegiate drop- out rate for African Americans is 62% compared to the national rate of 41%. In addition, the G.P.A. of those African Americans who do graduate is almost .6 points lower than other graduates. Steele found SES differences do not explain the disparity in academic performance found between African Americans and Whites. Otherwise, academic performance should be dissimilar between African Americans from higher SES and those from lower SES, which is not supported in the literature. Additionally, African

American academic skill level measured by the SAT, consistently over-predicts academic performance, but for some reason African American academic performance does not correlate with their measured ability, and there must be other factors which reduce grades of African Americans in college.

Steele (1997) concluded chronic and frequent exposure to discrimination, oppression and stereotype threat has predictable outcomes on health. Additionally, prejudiced group members will internalize stereotypes, and will develop a loss of self efficacy, and inadequacy which integrates into their personality, and can lead to disassociation with a domain which is associated with threats of stereotypes. Steele recommended it would be much more feasible to eliminate the stereotypes than to focus on amending individual internal psychology.

Claire and Croizet (1998) researched the effects of SES and stereotype threat on poor academic performance with undergraduate students in low SES levels. The purpose of the study was to test the theory of stereotype threat and its effects on academic performance; also assessed was the ability to generalize stereotype threat effects to social class. In addition, the ability to improve academic performance with a lexical change in directions on standardized tests was assessed. The study was based on Steele's model of stereotype threat which states negative stereotypes about groups of people create threats for the members of groups. Stereotype threat occurred in participate who enter into domains such as African Americans in academic settings where negative stereotypes exist. The threat of self confirming the stereotype can negatively affect performance on its own, often pushing members to disassociate with the domain altogether.

Claire and Croizet (1998) performed diagnostic tasks on 128 undergraduate students, who were randomly placed into four (salience x diagnosticity) study experiment categories. Participants were either told the test was diagnostic, and measured their intellectual ability, or non-diagnostic and being used to test some random hypothesis. Then participants' SES levels were assessed by student number and records from the university. Then the researchers manipulated salience by randomly asking participants to identify their parents' employment status and educational attainment or non salient information was obtained about participants' home town. Participants then completed a verbal battery similar to the GRE in difficulty and graded according the GRE guidelines.

Claire and Croizet (1998) found that when tests were presented as an intellectual measure, participants from high level SES performed better than those from low level SES. In contrary, when test were not presented as non-intellectual measure, test scores were virtually identical between the low and high level SES participants. However, the scores of low level SES participants in the non intellectual group markedly outperformed the low level SES group in the intellectual measurement group. The results support the extension of Steele's theory of stereotype. Threat to groups from low socioeconomic status can exist as a result of negative stereotypes applied to other groups similarly to the effects of performance demonstrated African Americans where they are relevant to the domain being tasked. Results suggested prior beliefs of genetic or other structural causation associated with low academic performance and low SES may not exist, and that stereotype threat may contribute largely to the disparity in academic performance measured between students segmented by SES.

Spencer, Steele, and Quinn (1999) researched the effects of stereotype threat on math performance in women at the University of Michigan. The purpose of the study was to test the

theory that the outcomes of stress, anxiety, and pressure from stereotype threat, not genetic or gender differences, contribute to the suppression of math performance in women. The study was conducted to test Steele's theory of stereotype threat which states where a particular stereotype is associated with a particular group; its members face threat of being appraised according to the stereotype. To guard against this threat of appraisal, or threat to one's self image, a person may choose to disassociate from the event or domain in question. Alternatively, if a person must participate in activities which are associated with a negative stereotype, he/she may succumb to the psychological activation and perform at a level not indicative of their ability.

Spencer et al. (1999) utilized two separate studies, the first utilized two sample math GRE exams as the instrument in the study. One was the advanced version, which incorporated advanced calculus; the other was from the general exam which required geometry, algebra, and trigonometry ability. Twenty eight women and men from an intro to psychology class were chosen to participate based on having no more than one year of calculus, and achieving higher than the 85th percentile on the math section of the SAT. In addition, those chosen were identified through an assessment of mathematical self efficacy. Those participants included in the study must have answered they strongly agree that they were good at math, and that being good at math was important to them. The design protocol matched female with male participant X advanced and easier math exam. Measurements included scores on the exam as well as time necessary to complete exam. The instructions given were the standard GRE instructions which were available on the screen to be read by the participants. The first study resulted in a positive effect for both gender and difficulty, and overall, men outperformed women on the difficult test, and both men and women scored equally well on the easier exam. This is consistent with the finding of current literature.

Spencer et al. (1999) conducted a second study which attempted to explain the causes of the differences in scores found between genders found in the first study. The researchers hypothesized when taking math exams, women encounter stereotype threat which threatens to confer the stereotype that men are better at math than women. In comparison to the first study where two versions (basic and advanced) of GRE math section were administered, the second study included only the advanced version. Exposure to stereotype threat was randomized by relevance condition. One group was exposed by being informed gender differences had been measured on the test previously in order to raise awareness about women's math ability. The other group was informed the test results had never demonstrated gender bias previously. This attempted to eliminate gender threat in the female participants specifically. The same selection criteria were applied to this study as was used in the first. This study included 24 males, and 30 females from another introduction to psychology class at the University of Michigan. The test was conducted identically as the test in the first study with students taking the GRE based exam on computers.

The results suggested when women were unconcerned about gender bias; they performed equally as well as men. In contrast, when gender bias was introduced as in the second group, men dramatically outperformed women in the exam. It was concluded, if there were genetic difference associated with the test outcomes, this study did not demonstrate it. Conversely, the study provided evidence which supports stereotype threat and the relevance of the threat actually shaped performance in the study (Spencer et al., 1999).

Link and Phelan (2001) researched how people assemble cognitive categories which led to stereotype beliefs. The purpose of the study was to better define how stereotype beliefs are constructed socially and psychologically, as well as how the life chances are altered by stigma of

stereotypes. The study was based on the model by Goffman, which demonstrated how labels, stigmas, and stereotypes can have dramatic effects on life chances of those who are afflicted by such events. Link and Phelan utilized Medline and PsychInfo to reviewed articles from 1980-1999 to including the keywords: stereotypes, labeling, discrimination, status loss, deviance, and exclusion to define the concept of concept of stigma and stereotypes.

Link and Phelan (2001) found that stereotypes are based from a set of characteristics that run counter to norms which are established by the dominant culture. Those who possess an attribute which deviates from the norm are devalued in society. It was hypothesized that the mechanisms under which stereotyping takes place can be described in (4) categories: labeling, negative attributes, separation, status loss and discrimination.

Link and Phelan (2001) suggested the first step in the process of stereotyping is the need to label differences. They suggested human differences selected for differentiation are socially constructed and maintained by forces which are cultural, economic, and social. The next step identified in the creation of stereotype is the need to attach negative attributes to human differences. They stated in order to form stereotypes, a difference such as skin color must be associated with a label, such as “Black”, followed by a negative attribute attached to the label of Black, and thus lead to automatic cognitions. These cognitions are then utilized to make “pre-conscious” decisions about categories and stereotypes. Furthermore, the process of stereotyping takes on the mechanism of separating those from the dominant culture from those who are not seen as belonging, due to the negative attributes and stereotypes developed over time to aid in the process of separation.

Link and Phelan (2001) stated that historically in the U.S. examples of the separation process include groups such as African Americans and Native Americans, where their differences were deemed as negative, and were labeled accordingly as immoral or barbaric. This perceived difference was the rationale and the mechanism for separating those from the minority from the majority. Due to the process of stereotyping and ultimately segregating minorities from the majority, discrimination occurs. Those segregated groups through this process lose their status and are rejected, devalued, and excluded, which leads to a decrease in their life chances and health.

Link and Phelan (2001) recommended two interventions which might change the process of stereotypes and discrimination. One is to address the mechanisms which affect outcomes from being discriminated, which includes the individual as well as structural mechanisms of discrimination. Additionally, long term beliefs and attitudes of the politically and socially powerful must be addressed, which are the root and underlying mechanism which perpetuates stereotyping and discrimination. Consequently, without systematic structural change, all intervention targeted at individual mechanisms will fail. This failure will be due to the undermining forces of the politically powerful majority, who benefit from such discrimination.

Blascovich, Spencer, Quinn, and Steele (2001) researched the mediating effects of stereotype threat in the development of hypertension in African Americans. The purpose of the study was to investigate the effects of recurrent occurrences of cardiovascular reactivity as a result of experiencing the specific stressor of stereotype threat, which is hypothesized to contribute to the higher rates of chronic hypertension demonstrated in African Americans. The study was based on the theory proposed by Steele, which found African Americans and women have established negative stereotypes in certain domains such as math. As a consequence, groups

have members which incur stereotype threat when participating in this domain as a result of fear from being judged stereotypically when in a relevant situation and thus create performance anxiety as a result.

Blascovich et al. (2001) tested the theory of stereotype threat by recruiting 41 volunteers who received \$20 for their time. The researchers tested the mean arterial pressure of these volunteers of which there were 20 African American, and 19 European American undergraduate students. The participants were randomized into two groups, (high stereotype threat, and low stereotype threat) the high stereotype group viewed a video which informed participants of the argument of standardize test bias to African American subcultures, and that they would be taking such an exam of intelligence to establish a sample which is nationally representative. In contrast, the low stereotype group viewed a video which informed participants the exam they would take was developed to be culturally unbiased. Afterward, the participants were asked to complete (2) ten item samples of the Remote Associates Test, which test verbal ability. Concurrently, the mean arterial pressure of the participants were being assessed and recorded.

Blascovich et al. (2001) reported the test performance analysis demonstrated an effect for item difficulty, with lower scores as the tasks became more difficult, and African Americans in the high stereotype group performed the poorest of all groups tested. The results of the cardiovascular reactivity measurements demonstrated African Americans in the high stereotype threat group had the highest levels of mean arterial pressure in the study, and demonstrated higher levels than European Americans and continued after the end of the tasks. In contrast, African Americans in the low stereotype threat group demonstrated similar mean arterial pressures as the European participants. The results suggested stereotype threat is an important component in the development of hypertension in African Americans. Assumed is that African

Americans' exposure to situations in which stereotypes may be relevant, one could extrapolate these findings to real world experiences and recognize how chronic exposure to cardiovascular reactivity could have pathophysiological effects where hypertension could be developed.

Merritt, Bennett, Williams, Sollers, and Thayer (2004) researched the connection between low SES, stress, John Henryism, and cardiovascular reactivity. The purpose of the study was to assess if high levels of John Henryism (JH), (characterized by an active high effort coping style to psychosocial stressors) in combination with low SES attainment is connected with increased cardiovascular reactivity in African Americans. The study was based on the reactivity hypothesis which states that high levels of chronic stress are associated with a higher risk of hypertension.

Merritt et al. (2004) recruited 58 Black men ranging in age from 23 to 47, who were normotensive. Participants were recruited by use of public service announcements and other community based communications. Inclusion was based on the completion of a health and social assessment, which compiled information on SES and health history. Afterward, the participants took part in a three part battery of mental tasks. The first task was to establish a heart rate and blood pressure baseline, where the participants read a speech on how to wash clothing. The second task included a 10 minute speech, where participants were randomly selected to either an overt racist setting or a non-racist setting about a situation in a shopping store. The participants were then asked to reflect on the scene they played out, and were asked standardized questions in order to assess their coping styles they might use in that hypothetical situation. The third task involved an anger recall test, where the participant recounted an occurrence in the past which provoked the angriest response remembered. The researchers then asked about the way the participant had responded. Blood pressure and heart rate were assessed during every portion of

the tasks, and the participants were debriefed about the experiment and results obtained, additionally they were compensated \$30 for their participation.

Merritt (2004) study revealed low SES was inversely related to increases in blood pressure in the participants with high levels of JH. In addition, prolonged cardiovascular recovery was assessed in participants during the anger irritant portion of the study. Additionally, participants who presented with both high JH and low SES demonstrated elevated BP and HR compared with those from the low JH and high SES, and this trend extended into the recovery stage. The findings suggested there is prolonged recovery period from cardiovascular reactivity in persons who exhibit high JH and low SES in the racial stress task. Chronic exposure to sympathetic activation may disrupt the negative loop feedback system and differentiate those who are at higher risk for cardiovascular events from those who are not. Finally, higher levels of SES emerged as a buffer which protected participants from the effects of John Henryism, as they may have additional social and economic resources to deal with circumstances of living as an African American.

Merritt, Bennett, Williams, Edwards, and Sollers (2006) researched the cardiovascular reactivity effects in Black men to obvious discrimination in contrast to responses to non-discriminatory stimuli in speech tasks. The purpose of the study was to analyze differentials in cardiovascular activity in Black men to overt racism in contrast to racially neutral stimuli. In addition, the role of perception was also accounted for in how racism influences cardiovascular reactivity. The study is based on the premise of Armstead and others who propose African Americans face chronic exposure to racism, which leads to chronically elevated blood pressure and thus leads to increased morbidity and mortality as a result.

Merritt (2006) recruited 73 normotensive African American males, ages ranged from 18-47, and educational levels ranged from 9-21 years. Psychosocial and SES demographics were attained by questionnaire, thereafter; the participants completed a three part battery of tasks. First, baseline blood pressure was measured followed by the participants' first clinical task. The participants were randomly assigned to either viewing a racially charged movie scene or a racially neutral scene. Subsequently, they were asked to take 5 minutes to prepare a 5 minute speech to convey their personal feelings and actions they would engage in if they had been the target in the scene. Following the reaction speech, utilizing a 4 point Likert scale, the participants were asked to rate the level of racism they perceived (zero to all) as the cause of the poor treatment in the movie scene. Blood pressure and heart rate measurements were constantly assessed and recorded during the entire testing study session.

Merritt (2006) found participants who perceived high levels of racism, demonstrated higher levels of blood pressure in the non racial task as compared to participants from the low perception- high racial task. Also, participants demonstrating high racial perception recorded much higher blood pressure before they were asked to reflect on perceptions of race, eliminating the possibility the elevated blood pressure was provoked by questions about racism. The researchers proposed persons who perceive high levels of racism frequently demonstrate higher levels of cardiovascular reactivity. In contrast, people who perceived the same situation less racially charged and less overt, perceived lower levels of racism. Additionally, overt racism less pervasive in society today and less obvious forms are more prevalent, as a result Black persons may interpret behavior in cross racial environments as induced by racism. Consequently, high levels of racial perception may be a strong factor in the difference in rates of hypertension between Black and White Americans.

Rosengren et al. (2004) researched the connection of multiple psychosocial stressors to the risk of cardiovascular disease in a large and diverse population. The purpose of the study was to question the reliability of past research, which attempted to define psychosocial variables narrowly. They argued the majority of research had been conducted in North America and Europe, who share similar cultural characteristics; alternatively, the researchers argue stress and anger along with other psychosocial elements are difficult to objectively measure. Consequently, current constructs on psychosocial factors may not be applicable to other cultures, as meanings and context may affect how they are perceived.

Rosengren et al. (2004) reported on 12,461 cases, defined by clinical acute myocardial infarction. These cases were recruited from 52 countries which utilized 262 medical centers. In addition, 14,637 controls were recruited, including age, sex and site matched, of visitors and/or relatives of patients from non- cardiovascular wards. The controls were screened to rule out possible pre-existing cardiovascular infarction.

Rosengren et al. (2004) obtained demographic and health history information by trained personnel, which included socioeconomic, lifestyle, behavioral risk factors and psychosocial states. Moreover, stress was stratified into multiple categories of work, domestic, financial, adverse life events, control, and depression. Scores from all measurements were combined and totals were quartiled into 4 categories ranging from lowest to highest scores.

Rosengren et al. (2004) included stratifying the age distribution into 5 year increments. T-tests were used to assess continuous effects of stress, with linear regression utilized for continuous variables, and frequencies were tested using Cochran-Armitage test. Finally, odds ratios were calculated using 99% CI's as a benchmark using SAS software.

Rosengren et al. (2004) reported greater rates of stress were associated with cases than with controls. Overall, general stress (combination of home and work stress) was associated with an OR of 1.5. In comparison, stress which was defined as being more permanent in nature was associated with an OR of 2.17. Additionally, perceived internal locus of control was concluded to have a protected effect with an OR of 0.72.

The Rosengren et al. (2004) study demonstrated great statistical power with an $N = 24,767$ which aids in avoiding type II errors. Study participants were also age, sex and site matched which may assist in comparability between cases and controls and may aid in reducing elements of confounding. Additionally, this study also incorporated only incidence cases which remove the question of temporal sequence, firmly establishing exposure prior to outcome. This study also had its limitations, which were discussed by the investigators, one of these was the fact that stress is difficult to assess, and there is no hard set criteria for how to define it or how to calculate it. Furthermore, they admitted that stress is inherently subjective and no two people perceive it the same, which could lead itself to bias as well as confounding. In addition, a dose response was not discovered in this particular study, which leads to questions of causality.

Overall, the study was reasonably designed, but like all studies, there are limitations to the extension of association which can be taken from this particular study this study. Overall, this study sheds light first on the adverse health effects of stress, but additionally on the difficulties which exist in measuring how stress is embedded differently by individuals. Thus improved methods to define and operationalize stress need to be undertaken.

CHAPTER III

METHODS

This article review attempts to submit an inclusive view of the literature relating to effects of SES and the mediating role psycho-social factors have on the prevalence of cardiovascular disease of those who live in poverty. The articles for this review were located through Medline searches, accessing the University of Northern Iowa and the University of Iowa library search engines. Articles chosen were limited to peer-reviewed Journals from well known sources such as *The Lancet*, *The American Journal of Epidemiology*, *Journal of the American College of Cardiology*, and *Circulation*. Keywords used in search of these articles included African American, socio-economic status, cardiovascular disease, poverty, risk factors, psychosocial factors, myocardio-infarction, allostatic load, and stress.

Although the primary population of study was African Americans, many of the articles obtained in the section of SES were primarily generic concerning ethnicity. These articles were meant to establish the connection between SES and health status as a population in general, the assumption was that African Americans would undoubtedly suffer similarly from the conditions of low SES. The overall objective was to demonstrate African Americans regardless of SES, are subject to additional stressors which may contribute to the disparity of hypertension rates noted in the African American population. Articles were also chosen by date, with the most current available prioritized, and then searching backward by date.

CHAPTER IV

CONCLUSION

Cardiovascular disease continues to be the leading cause of death in the United States; this is especially true for African Americans who die from cardiovascular disease at rates much higher than Whites (Avis et al., 2005; Pearcy & Keppel, 2002). The literature in this project supported the connection between social economic status and all cause mortality, as well as cardiovascular disease. Additionally supported, is the inverse relationship between SES and overall health outcomes, but the literature is vague on the specific combination of risk factors which have the most impact on health, as the number of possible confounders are abundant. In contrast, it remains unclear if African Americans receive the same health benefits as Whites with increased SES. Several studies support the theory which argues African Americans' health does not improve at the same level as Whites when compared to Whites within the same SES level (House, 2001; Johnson et al., 1995; Marmot & Smith, 1991; Zimmerman and Bell, 2006).

The literature supported the belief that social economic position accounts for much of the exposure from biomedical, environmental, psychosocial, and behavioral risk factors. The literature clearly supported the cumulative burden model, which contends the length and duration of the exposure to the burden of low SES equated into highest risk of cardiovascular disease (Cohen et al., 2010; Seeman et al., 2010). Additionally, increasing SES provided some protection from the effects of living in a disadvantaged level. The literature provided evidence of a reduction of hypertension, when African Americans were able to obtain increased economic security, but increased income obtainment only mediated the occurrences of cardiovascular

disease marginally (~20-35%) (Guerro & Palmero, 2010; James et al., 2006; Thomas, 2005). The literature suggests that African Americans live with increased stress, which creates an environment where anger, hostility and cynicism are prevalent, and hopelessness leads to cardiovascular reactivity and thus cardiovascular disease.

The literature suggested the disparity in hypertension rates experienced by African Americans' can be partially contributed to the dysregulation resulting from chronic cardiovascular reactivity as a result of race based stress. Racial stress as described in the literature is a feature not experienced by most Whites. Multiple studies demonstrated how racial bias when integrated into mental tasks creates differences between Blacks and Whites in cardiovascular reactivity. Blacks demonstrated higher blood pressure excursions compared to Whites in mental and psychosocial stress tasks. Cardiovascular reactivity to racial stress is one of the important finding in the literature. Increased cardiovascular reactivity to racial stimuli in African Americans has led to the supported conclusion for the etiology of exaggerated blood pressure, may be due from peripheral vaso-constriction. Vaso-constriction differentiates African Americans from Whites, where Whites essential hypertension stems more from Beta Adrenergic etiology. The distinction is important because vaso constriction is more indicative of stress response than essential hypertension as a result of beta adrenergic- rennin excursions commonly seen in Whites (Anderson, 1989; Armstead et al., 1989; Clark, 1999; Strogatz et al., 1997).

The literature illustrates Blacks are much more susceptible to race related stress than Whites are, and frequently encounter racism at every level of society, such as: in school, the housing market, working environments, shopping environments, by their own members. The research has demonstrated Blacks are highly suspicious of Whites, and as a result may see even the most guiltless transgression as racist. It has been suggested in the literature the existence of a

wide variance of responses to the perception of racism, based on early experiences, which plays an important role on how much racism, is perceived, and thus the level of cardiovascular response excursion which occur as a result (Clark et al., 1999; Link & Phelan, 2001; Lepore et al., 2006; Ong et al., 2009; Seeman et al., 2010).

The literature has illustrated, increases in SES generally translate into increases in overall health. One of the primary indices of increasing SES has been education, but for Blacks that has not been as easy. Research has shown there are major obstacles leading to differences in academic achievement between Black and White students. The research indicates this stems from the stereotypes which produced misconceptions of the ability of Blacks to achieve. These stereotypes have been linked to educational discrimination of Black students in the U.S. Stereotypes inhibit the students' ability to perform at their best; and the literature provides evidence for this. Much of the research has reported individual math performance is reduced because of the stereotype that Blacks are bad at math, and the threat of validating the stereotype, creates a large amount of anxiety and stress. Stereotypes in African Americans historically constructed and reinforced by segregation, educational access, and historical myths to create and perpetuate these impediments (Alba et al., 2000; Arthur et al., 2004; Armstead et al., 1989; Claire & Croizet, 1998; Morrell et al., 1988; Kaplan & Keil, 1993; Spencer & Steele, 1998; Steele, 1997).

Research has revealed stereotypes go beyond the classroom, labels and negative attributes are woven in all aspects of African Americans lives. Stress is exacerbated by the increased exposure to the risk of judgment or confirmation of a particular stereotype. Consequently, chronic excursions of stress, cardiovascular reactivity and eventually cardiovascular disease then prevail. Blacks experience status loss, loss of control, hopelessness, low self esteem and anger.

As a result, African Americans spend all their resources for basic survival leaving little for other aspects of their lives (Blascovich et al., 2001; Spencer et al., 1999; Link & Phelan, 2001)

Overall, research has provided support connecting SES and overall health outcomes. In addition, poverty rates among Blacks are disproportionately greater than Whites, and as a result, suffer greater consequences from the overexposure to low SES. In contrast to Whites, Blacks from disadvantaged socioeconomic levels must deal with the additional plight of stereotypes, racism, and discrimination. Being born Black in the U.S., undoubtedly produces a chronic state of stress, cardiovascular reactivity, and increased rates of hypertension and cardiovascular disease in this population.

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Table 1: Summary of the Literature Review

Topics	Author	Year	Findings
SES AND HEALTH OUTCOMES	PERCY & KEPPEL	2002	OVERALL MEASURES OF HEALTH DISPARITY
	MARMOT & SMITH	1991	SES AND INVERSE RELATIONSHIP TO HEALTH IN ALL CAUSE MORTALITY
	KAPLAN & KEIL	1993	SES FACTORS AND CV DISEASE
	JOHNSON et al.	1995	MACROSOCIAL AND ENV. INFLUENCES ON MINORITY HEALTH
	ACKERMAN et al.	1999	SES, FAMILY INSTABILITY, AND PROBLEM BEHAVIORS
	HOUSE	2001	PSYCHOSOCIAL RISK FACTORS
	HAUKKALA	2002	SES DIFFERENCES IN CYNICAL DYSTRUST AND ANGER EXPRESSION AS A PSYCHOSOCIAL LINK TO POOR HEALTH
	MCEWEN	2003	CHILDHOOD SES AND BEHAVIORS AND LEADS TO STRESS RESPONSES GLUCOCORTICOIDS PRODUCTION
	PULKKI et al.	2003	SES HOSTILITY LEADS TO CV RISKY BEHAVIORS
	ZIMMERMAN & BELL	2006	INCOME INEQUALITY LEADS TO PHYSICAL AND MENTAL HEALTH CAUSAL PATHWAYS TO POOR HEALTH
	SEEMAN et al.	2009	SES LINKED TO POOR HEALTH
	COHEN et al.	2009	CHILDHOOD SES AND ADULT HEALTH CONNECTED
	MATHEWS et al.	2010	SES MEDIATORS SUCH AS EMOTIONS, COGNITIONS, STRESS, WHICH CONNECT SES AND HEALTH
MENTAL STRESS AND CV REACTIVITY	KIVIMAKI et al.	2003	CV DISEASE AND DEPRESSION IN WORKPLACE STRESS
	ROSENGREN et al.	2004	PSYCHOSOCIAL RISK FACTORS AND M.I.
	JENNINGS et al.	2004	MENTAL STRESS AND INCREASED BLOOD PRESSURE RESPONSES

HAMER et al.	2006	INFLAMMATORY RESPONSES TO REPEATED MENTAL STRESS
MATHEWS et al.	2006	PSYCHOLOGICAL STRESS AS A RISK FACTOR FOR CVD
KNEPP & FREIDMAN	2008	CV ACTIVITY IN WOMEN HIGH VS LOW WORRY (STRESS)
HAMER et al.	2008	PSYCHOLOGICAL STRESS AS A RISK FACTOR FOR CVD
MCEWEN	2008	EFFECTS OF STRESS HORMONES AND DISEASE "ALLOSTATIC OVERLOAD"
GUERRERO & PALMERO	2010	IMPACT OF HOSTILITY AS A RISK FACTOR FOR CV DISEASE DEV."HYPOTHESIS OF CV REACTIVITY"
RACIAL STRESS AND CV REACTIVITY		
MORELL et al.	1988	PSYCHOPHYSIOLOGICAL REACTIVITY TO MENTAL MATH IN BLACK & WHITE NORMOTENSIVE MEN
ARMSTEAD et al.	1989	RACIAL STRESSORS, ANGER, AND BLOOD PRESSURE RESPONSES
ANDERSON	1989	RACIAL DIFFERENCES IN STRESS INDUCED CV REACTIVITY AS CAUSE FOR INCREASED PREVELENCE OF HYPERTENSION
ARMSTEAD et al.	1989	CONNECTION OF RACIAL STRESS AND BLOOD PRESSURE RESPONSE AND ANGER EXPRESSION IN BLACK COLLEGE TUDENTS
STROGATZ et al.	1997	STRESS AND SOCIAL SUPPORT AND BLOOD PRESSURE IN BLACK ADULTS DUE TO RACISM AND LOW SES
CLARK et al.	1999	RACISM AS A STRESSOR FOR AFRICAN AMERICANS
ALBA et al.	2000	SEGREGATION OF MIDDLE CLASS AFRICAN AMERICANS
WILLIAMS & WILLIAMS	2000	RACISM AND MENTAL HEALTH...PERSISTENCE OF NEGATIVE RACIAL STEREOTYPES
FORMAN	2003	THE PSYCHOSOCIAL COST OF RACIAL SEGMENTATION AND WELL BEING
ARTHUR et al.	2004	CV REACTIVITY TO MENTAL ARITHMETIC IN BLACKS,CARRIBEAN, AND WHITE AMERICANS
LEPORE et al.	2006	SOCIAL STRESS AND CV

			REACTIVITY IN BLACKS VS WHITES
	JAMES et al.	2006	THREE MODELS OF HOW SES CONTRIBUTES TO HYPERTENSION IN AF.AM. MEN
	ONG et al.	2009	RACIAL DISCRIMINATION AND THE STRESS PROCESS
STEREOTYPE THREAT AND CVD			
	STEELE	1997	HOW STEREOTYPE SHAPE INTELLECTUAL IDENTITY AND PERFORMANCE
	CLAIRE & CROIZET	1998	EXTENDING THE CONCEPT OF STEREOTYPE THREAT TO SOCIAL CLASS LOW SES
	SPENCER & STEELE	1999	STREREOTYPE THREAT AND WOMENS MATH PERFORMANCE
	BLASCOVICH et al.	2001	AFRICAN AMERICANS AND STEREOTYPE THREAT
	LINK & PHELAN	2001	CONCEPTUALIZING STIGMA AND STEREOTYPES
	MERRITT et al.	2004	JOHN HENRYISM AND INCREASED STRESS
	MERRITT et al.	2006	PERCEIVED RACISM AND CV REACTIVITY AND ANGER RECALL TASK