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INDIVIDUAL VARIABILITY IN THE RELATIONSHIP AMONG STRESS, COPING, AND HEMODYNAMICS IN AFRICAN AMERICAN ADULTS

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ABSTRACT

The objective of this study was to examine sources of individual differences in measures of stress, coping, and hemodynamics, and the relationship among these factors in adult African Americans. The specific aims of the study were: 1. to decompose the genetic and environmental sources of variance in measures of stress, coping, and hemodynamic indices; 2. to examine the contribution of age to variability in measures of stress, coping, and hemodynamic indices among a sample of African American twins; 3. to account for sources of covariance in the relationship among stress, coping, and hemodynamic measures. African Americans experience one of highest rates of hypertension in the world. However, little is know about the sources of individual differences in blood pressure in this population. The proposed study analyzed measures of perceived stress, John Henryism (active coping), and hemodynamics (systolic and diastolic blood pressure, and pulse pressure) collected from participants of the Carolina African American Twin Study of Aging (CAATSA). Participants in CAATSA ranged in age from 22 to 89 years. The sample for the current study included 102 identical twin pairs and 110 same-sex fraternal twin pairs. Data analyses included phenotypic analyses of the relevant variables including correlation and regression, as well as biometrical model fitting procedures. Multivariate genetic models were used to examine genetic and environmental influences on measures of stress, coping, and blood pressure, and the interrelationship among these factors. The results of the analyses show genetic influences are a significant component of the variability in systolic and diastolic blood pressure, pulse pressure, and perceived stress. Age significantly contributes to variability in systolic pressure, pulse pressure, and perceived

stress. The findings indicate that additive genetic and common environmental influences, in combination, account for covariance among blood pressure, perceived stress, and active coping. Finally, the results indicate a significant correlation among unique environmental factors between blood pressure measures, but not between blood pressure and psychosocial measures. The results are discussed in terms of understanding the age, and genetic and environmental influences on individual differences in blood pressure and psychosocial variables in this population.

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Acknowledgments

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Chapter 1: Introduction

Introduction

Recent demographic trends show an increasing number and proportion of elders in the United States. This phenomenon is the result of relatively low fertility and declining mortality rates that have been common among Western nations over the past century (Kinsella & Velkoff, 2001; Hayward & Zhang, 2001). For the U. S., much of the demographic shift toward an older population has resulted from declines in mortality. Declines in mortality are of particular interest in studies of aging and health because such declines not only signal an increasing proportion of elders but also have implications for long term health care system demands and costs (Hayward & Zhang, 2001; Crimmins, Hayward, & Saito, 1994). However, declining mortality rates provide little information about specific conditions that result in morbidity or mortality within populations. Summary statistics, such as mortality rates, also provide little information concerning the relationship between such conditions and age (Kinsella & Velkoff, 2001).

As mortality rates decrease, and people live longer, health-related quality of life becomes an increasingly important issue (Kinsella & Velkoff, 2001;Serow, 2001). In fact, longer life doesn't necessarily equate to better health. Data show that while many people are living longer, they are also living with chronic health conditions (Kinsella & Velkoff, 2001; Serow, 2001; Crimmins, et al, 1994). However, the relationship between morbidity and mortality is also complicated by a number of factors. For example, differential allocation of research and health care resources results in more medical

progress for some conditions, such as heart disease, but not others(Serow, 2001). In addition, disparities in health care access could leave some patient groups untreated. Another important factor is the change in population composition. Moreover, the nature, prevalence, and treatment of chronic conditions may vary across ethnic groups (Kinsella & Velkoff, 2001;Serow, 2001; Jackson et al, 1990).

In the U.S., lower mortality rates have produced a longer life expectancy for all segments of the population, with larger life expectancy gains at later ages (Serow, 2001). As a result, it is expected that the population of people 65 and older will grow from about 13% in 1990 to about 20% in 2030 (U.S. Bureau of the Census, 1996). Similar demographic shifts are occurring in other nations as well. In fact, the elderly are the fastest growing segment of the population in most developing nations (Kinsella & Velkoff, 2001; Hayward & Zhang, 2001).

Aging is increasingly marked by demographic diversity both globally and within the U.S. (Kinsella & Velkoff, 2001;Jackson, Antonucci, & Gibson,1990). For example, it is estimated that within the U.S., minority elders are growing at a faster rate than their Caucasian counterparts (Williams & Wilson, 2001; Kinsella & Velkoff, 2001). The increase in the number and proportion of minority elders in the U.S. is in part due to declining mortality rates, although immigration has had some impact (Kinsella & Velkoff, 2001;Serow, 2001; Jackson et al, 1990).

The experience of minority elders in late life is often substantially different from their majority counterparts. Easy identification, differential treatment, relative deprivation among many ethnic minority groups, and ethnic and racial factors make the aging experience different for minority elders (Jackson et al, 1990). There are

significant differences in a number of areas such as social support, socioeconomic status, well-being, but the most striking differences are seen in the area of health (Kinsella & Velkoff, 2001;Serow, 2001; Jackson et al, 1990).

It is important to note that the burden of chronic morbidity and subsequent mortality is not equally distributed across racial groups (Williams & Wilson, 2001; Kinsella & Velkoff, 2001;Jackson et al, 1990), although data show that there are a few categories of illnesses that are responsible for most morbidity and mortality across all racial groups (Manton & Stallard, 1997; Kinsella & Velkoff, 2001). For example, cancers, diabetes, infections, lung disease, and renal and liver disease are responsible for much of the observed mortality for all groups, while osteoarthritis and related rheumatologic diseases, and hip fracture are responsible for much of the disability experienced in old age (Wong, Sharpiro, Boscardin, & Ettner, 2002; Manton & Stallard, 1997; Kinsella & Velkoff, 2001). However, cardiovascular disease as a class, is the primary source of mortality for all groups, especially with increasing age (Wong et al, 2002; Sowers, Ferdinand, Bakris, & Douglas, 2002; Stewart & Silverstein, 2002; Kinsella & Velkoff, 2001; JNC IV, 1997; Manton & Stallard, 1997).

Considerable attention has been focused on health and mortality disparities related to cardiovascular disease among ethnic minority groups (Wong et al, 2002; Sowers, et al, 2002; Hayward et al, 2000). While there have been declines in cardiovascular disease rates for all groups, there are still substantial differences between groups, with minority groups generally faring worse than their majority counter parts (Wong et al, 2002; Sowers, et al, 2002; Hayward et al, 2000). One of the most well-documented health disparities is the disproportionate rate of cardiovascular disease

experienced by African Americans (Wong et al, 2002; Sowers, et al, 2002; Hayward et al, 2000). In fact, cardiovascular diseases (including congestive heart failure, stroke, hypertension, coronary heart disease, and others) are responsible for most of the disparities in health between African Americans and Caucasians. Furthermore, hypertension is the largest contributor to lost years of life for African Americans (Wong et al, 2002; Sowers, et al, 2002; Hayward et al, 2000).

Hypertension is one of the most prevalent and significant chronic conditions experienced by African Americans. African Americans have one of the highest rates of hypertension in the world (Sowers et al, 2002; JNC VI, 1997) and are estimated to have twice the prevalence compared to Caucasians (Sowers et al, 2002; Anderson, McNeilly, & Meyers, 1991). Hypertension is associated with chronic and life threatening illnesses such as coronary heart disease, kidney failure, blood vessel damage, and stroke (Saab & Schniederman, 1993; American Heart Association, 2002; JNC VI, 1997). African Americans have an 80% higher rate of stroke mortality, 50% higher rate of heart disease mortality, and a 320% higher rate of hypertension related kidney disease than their Caucasian counterparts (JNC VI, 1997). Evidence indicates that the higher rates of morbidity and mortality among African Americans is related to prolonged hypertension (Hayward et al, 2000; JNC VI, 1997).

Age is also an important factor in the relationship among hypertension, morbidity and mortality among African Americans. For example, African Americans have higher blood pressure values, more severe associated morbidity, and higher hypertension-related mortality at younger ages than other groups (Hayward et al, 2000; Sowers et al, 2002; JNC VI, 1997). Moreover, the disparate rates of hypertension have severe

consequences with advancing age. For instance, beginning in middle age (between 50 and 65 years), the impact of cardiovascular morbidity results in greater disability, and widens the health and socioeconomic gap between African Americans and Caucasians which continues to grow until the oldest ages (Hayward et al, 2000). Hypertension affects approximately 65 percent of African-American elders between the ages of 65 and 74 and is predictive of functional decline (Wagner, Grothaus, Hect, & LaCroix, 1991). In addition, increasing age is related to important behavioral components related to health such as regular doctors visits, monitoring blood pressure and cholesterol levels, and other screening behaviors (Stewart & Silverstein, 2002).

While the exact origins of essential hypertension are unknown, several theories have been proposed to account for the disparate rates of hypertension among African Americans, which can categorized as focusing predominately on environmental and psychosocial factors, or on genetic aspects to account for individual differences in blood pressure within this group. For example, some theories have suggested that genetic factors related to sodium retention are dominant in determining blood pressure among African Americans (Wilson & Grim, 1991; Harshfield & Grim, 1997;Grim, Henry, & Meyers, 1995; Baker, Ireson, Carney, Markandu, & MacGregor, 2000; Su & Menon, 2001). In contrast, other theories posit that environmental and psychosocial factors, such as racism (Anderson, McNeilly & Myers, 1993*;* Clark, Anderson, Clark, & Williams, 1999) or socioeconomic stress (Pickering, 1999; Burke & Motulsky, 1992; Dressler,1996 & 1990) are important factors in determining blood pressure among African Americans. However, there has been little research on the relative influence of genetic and

environmental factors on blood pressure for African Americans (for exceptions see the review to follow).

The present study represents an important extension of the current knowledge about sources of individual variation in blood pressure among African Americans. Considerable attention has been focused on describing the nature of the disparities in hypertension and related sequelae within this group. Genetic factors, as in other populations, certainly play a role in determining blood pressure among African Americans, although relatively few twin and family studies have been conducted to estimate the proportion of genetic and environmental influences on blood pressure variability. The few twin studies that have been conducted have included small samples, with less than 40 pairs per study (Grim, Wilson, Nicholson, Hassell, Fraser, Grim, & Wilson, 1990; Harshfield, Grim, Hwang, Savage, & Anderson, 1990). With this in mind, the proposed study will use data from the Carolina African American Twin Study of Aging (CAATSA). To the best of the author's knowledge, this is the largest study of African American adult twins to date.

CAATSA has other advantages as well. For example, there is a considerable range of ages for the sample (22 to 89 years). This unique characteristic allows the effect of age as well as heritable influences on blood pressure among African American adults to be better estimated. Much of the research concerning blood pressure among African Americans has focused on environmental and psychosocial factors (Pickering, 1999; Dressler, 1996; Clark, 2001). However, these studies fail to account for genetic influences that affect blood pressure and which may account for some portion of the covariance observed between blood pressure and psychosocial factors. Another

advantage of the data set is the inclusion of psychosocial variables such as perceived stress and John Henryism. These variables have been previously associated with elevated blood pressure and hypertensive status among African Americans (Dressler, 1990; James, 1994; Pickering, 1999).

The proposed research has three specific aims:

1) To estimate the proportions of genetic and environmental influences for measures of perceived stress, active coping, and hemodynamic indices (systolic and diastolic blood pressure, and pulse pressure) among a sample of African American adult twins.

2) To examine the impact of age on measures of perceived stress, active coping, and hemodynamic indices (systolic and diastolic blood pressure, and pulse pressure) among a sample of African American adult twins.

3) To examine shared genetic and environmental influences that account for covariance among measures of perceived stress, active coping, blood pressure, and pulse pressure among a sample of African American adult twins.

The first aim of the proposed analyses involves the estimation of genetic and environmental influences on perceived stress, active coping (John Henryism), and hemodynamic indices (systolic and diastolic blood pressure, and pulse pressure) among African Americans. While previous studies have examined genetic influences on blood pressure in this group, very little is known about genetic and environmental influences on psychosocial factors among African Americans.

The second aim will focus on the impact of age on the variables of interest. This will assessed primarily by the use of an age-regression biometrical model. The use of this type of model yields estimates of genetic, environmental, and age influences on the

relevant variables. The use of this model provides and estimate of variability accounted for by age which may otherwise result in an inflated estimate of common environmental influence, particularly in sample with a wide range of ages (Snieder, 2000).

Finally, the third aim will address sources of covariance among blood pressure and previously correlated psychosocial factors. This analysis will be used to examine the existence of shared genetic and environmental influences that may account for the relationship among perceived stress, John Henryism, and hemodynamic indices among African American adults.

As noted earlier, considerable attention has been focused on the nature and consequences of disparities in rates of hypertension in African Americans. In addition, researchers have proposed a number of models to explain the etiology of hypertension in this group. However, little is known about the role of genetic and environmental influences on blood pressure in this group. The following section is a review of essential conceptual and empirical literature concerning the role of genetic and environmental influences, as well as the role of psychosocial factors on blood pressure among African Americans.

Chapter 2: Literature Review

Blood Pressure Regulation

Blood pressure in general, and hypertension in particular are multifaceted and result from both genetic and environmental influences (Luft, 2002; Adams, Aubert, & Clark, 1999; Vogler & Quirk, 1999). Blood pressure is determined by numerous interactive biological systems, with the kidney and related neuro-hormonal factors playing a central role in its regulation (Ferrario, 2003; Hall, 2003).

The kidney's role is regulated by series of interconnected pathways that are central to effective circulating volume (Hall, 2003; Stanton & Koeppen,1998). Effective circulating volume is the portion of extracellular fluid (ECF) that effectively perfuses the tissues, and reflects the demands on the vascular system pressure and volume. Effective circulating volume varies directly with the vascular system, arterial pressure, and cardiac output (CO) (Hall, 2003; Stanton & Koeppen,1998). A decrease or increase in ECF and vascular volume, arterial pressure, or CO is sensed as an increase or decrease in effective circulation volume. Reductions in effective circulating volume result in diminished renal NaCl excretion. The opposite occurs (natriuresis or increased NaCl excretion) for increased circulating volume. Sodium balance determines effective circulating volume, ECF volume, and vascular volume. Vascular volume in turn affects arterial BP and CO (Hall, 2003; Stanton & Koeppen,1998).

There are a number of processes and mechanisms that are essential to these changes that ultimately affect sodium retention or excretion and therefore blood pressure (Ferrario, 2003; Stanton & Koeppen,1998). For example, baroreceptors sense

changes in blood volume or pressure and signal the brain stem (via the vagus nerve) to stimulate sympathetic outflow and anti-diuretic hormone (ADH) release which affects blood pressure change by altering sodium retention in the kidneys (Ferrario, 2003; Stanton & Koeppen,1998).

There are several other neuro-hormonal factors that play an important role in renal regulation of blood pressure (Ferrario, 2003; Stanton & Koeppen,1998). For example, renin is produced in the renal arterioles and its secretion is stimulated by pressure decreases and inhibited by pressure increases. Its secretion is also affected by sympathetic activity and NaCl delivery to renal structures. Renin acts on circulating angiotensinogen, produced primarily in the liver. Renin cleaves angiotensinogen to form angiotensin I which in turn is cleaved by angiotensin converting enzyme (ACE) to form angiotensin II (Ang II). Ang II stimulates aldosterone release from the adrenal cortex, vaso-constriction (i.e. BP increases), and NaCl retention (Stanton & Koeppen,1998). In addition, aldosterone results in decreased NaCl excretion. This system, collectively called the renin, angiotensin, aldosterone system (RAAS) is activated during blood volume decreases (reduced NaCl secretion) and suppressed during blood volume increase (Ferrario, 2003; Stanton & Koeppen,1998).

The kidneys respond to sodium intake and concomitant fluid volume increases by adjusting sodium excretion (Ferrario, 2003; Hall, 2003; Stanton & Koeppen,1998). Euvolemia is maintained when sodium is balanced (NaCl ingestion is equal to its excretion). During this state small adjustments in sodium excretion are made at the collecting ducts of the nephron, and this is predominantly under the influence of aldosterone (Ferrario, 2003; Hall, 2003; Stanton & Koeppen,1998). During volume

expansion, the kidneys respond by increasing water and sodium excretion until euvolemia is restored and the opposite occurs during volume depletion (Ferrario, 2003; Hall, 2003; Stanton & Koeppen,1998). Depending on the sodium intake, restoration of euvolemia may take hours or days. Therefore, the kidneys and the RAAS are important to long term blood pressure regulation and have been implicated in the development of hypertension (Ferrario, 2003).

Hypertension is hypothesized to result from a dysfunction of the blood pressure regulatory systems (Luft, 2002; Vogler & Quirk, 1999). However, in the case of essential hypertension the exact set of affected regulatory systems is unknown. Much of the research concerning the genetics of essential hypertension has focused on components of the RAAS (Luft, 2002; Padmanabhan, Padmanabhan, & Connell, 2000). The RAAS is integral in the control of blood pressure, body fluids, and electrolyte balance and is often inhibited to effectively treat hypertension (Ferrario, 2003; Padmanabhan et al, 2000). In addition, evidence from animal studies as well as heritable, single gene forms of hypertension consistently show associations between alteration of RAAS components such as aldosterone and elevated blood pressure (Ferrario, 2003; Luft, 2002; Padmanabhan et al, 2000).

A number RAAS genetic polymorphisms have been implicated in the development of hypertension or cardiovascular disease and are therefore thought to be critical in the control of blood pressure. For example, considerable attention has been focused on the M235T and the T174M polymorphism of the angiotensinogen gene (Luft, 2002; Padmanabhan et al, 2000). Another important candidate gene polymorphism is the angiotensin converting enzyme (ACE) insertion / deletion variant which has been

linked to myocardial infarct as well as coronary heart disease (Padmanabhan, et al. 2000). Other candidate genes include A1166C variant of the angiotensin 2 receptor gene, and the G442 variant of the endothelial sodium channel gene (Luft, 2002; Padmanabhan et al, 2000).

A number of the genes have been implicated in racial differences in hypertension rates. For instance, G442 variant of the endothelial sodium channel gene has a much higher frequency among African Americans, and is associated with increased sodium retention (Luft, 2002; Su & Menon, 2001). Similar evidence exists for the M235T variant of the angiotensinogen gene. For example, this variant has a much higher frequency among Africans, African Americans, and Japanese compared to Caucasians. It has also been associated with increased sodium retention among Africans and African Americans but not consistently associated with hypertension (Corvel & Jeunemaitre, 1997). This suggests that having a genetic predisposition toward sodium retention is not sufficient for increased blood pressure.

The values that represent the hypertensive range are extremes of a distribution of blood pressure values (Vogler & Quirk, 1999). However, environmental and genetic factors play a role in determining blood pressure values at all points of the distribution (Vogler & Quirk, 1999). Furthermore, blood pressure values may be impacted by a number of factors that may differ between and within populations. Researchers have noted that sample characteristics such as age, gender, or ethnicity have been shown to affect heritability estimates (Harrap, 1994; Hong, deFaire, Heller, McClearn, & Pedersen, 1994). Therefore, understanding sources of variance in blood pressure has implications for comprehending the etiology of essential hypertension, as well its

treatment among African Americans.

Age, Blood Pressure, and Pulse Pressure

Previous research has established elevated systolic and diastolic blood pressure levels as risk factors for cardiovascular disease (JNC VI, 1997; Lee, Rosner, & Weiss, 1999; Franklin, Khan, Wong, Larson, & Levy, 1999; Glynn, Chae, Guralnik, Taylor, & Hennekens, 2000). For example, aberrant blood pressure patterns such as isolated systolic hypertension have been associated with adverse cardiovascular morbidity and mortality such as stroke, MI, and congestive heart failure in the elderly (JNC VI, 1997; Lee, Rosner, & Weiss, 1999).

Age has been consistently associated with increased blood pressure values and associated risk of cardiovascular events (Glynn, Chae, Guralnik, Taylor, and Hennekens, 2000; JNC VI, 1997; Benetos, 1999; Franklin, 1999). Among middle aged people under 65 years of age, blood pressure is consistently and positively associated with increased cardiovascular morbidity and mortality (Glynn, et al, 2000; Franklin, Khan, Wong, Larson, & Levy, 1999). For instance, evidence indicates those with a systolic blood pressure between 140 and 150, or a diastolic pressure between 90 and 104, are eight times more likely to develop cardiovascular disease relative to those with lower blood pressure values (Alderman, 1999). However, in populations over age 65 the relationship between blood pressure and mortality is less consistent. This is due to decreased aortic compliance, concomitant increases in systolic blood pressure, and age related declines in diastolic blood pressure. As a result, blood pressure maybe a less accurate predictor of morbidity and mortality with advancing age (Glynn et al., 2000; Franklin, Khan, Wong, Larson, & Levy, 1999; Lee et al, 1999).

Blood pressure is a well established, non-invasive index of cardiovascular function, although considerable attention has been focused on another non-invasive index of cardiovascular function, pulse pressure. Pulse pressure provides an index of large artery compliance and is calculated as the difference between systolic and diastolic blood pressure (Franklin, 1999; Glynn et al, 2000; Franklin et al, 1999). Increased arterial stiffness, represented by a wider pulse pressure, in turn conveys increased risk for cardiovascular events (Lee et al, 1999; Glynn et al, 2000; Franklin, 1999).

Both, systolic and diastolic blood pressures indicate the extreme values of blood pressure oscillation around the mean blood pressure, which represents a steady pressure state over the cardiac cycle. However, pulse pressure represents the pulsatile component of the same cycle (Van Bortel, Struijker-Boudier, & Safar, 2001; Asmar, Darne, el Assaad, & Topouchian, 2001). Pulse pressure and systolic blood pressure are the product of ventricular ejection and arterial compliance. Compliance, is in turn determined by arterial volume and elasticity (Van Bortel, et al., 2001).

Reduced arterial compliance results from a number of vascular changes that result in increased blood pressure and a wider pulse pressure (Van Bortel, et al., 2001; Asmar et al, 2001). For example, stiffening in the central arteries results in an increase in pulse wave velocity. The initial pulse wave is generated by left ventricular ejection, and reflected back toward the heart at points of vessel bifurcation. With arterial stiffness, this pulse wave is reflected quickly and results in increased ventricular and systolic pressure, and thus increasing pulse pressure (Van Bortel, et al., 2001; Asmar et al, 2001).

Arterial stiffness also results from changes in arterial vessel walls, particularly medial layer hypertrophy (Van Bortel,et al., 2001; Asmar et al, 2001). The medial layer is the primary site of smooth muscle cells in vascular walls and is integral determining vascular compliance (Van Bortel, et al., 2001). Medial layer hypertrophy, and therefore increased arterial stiffness is associated with hypertension and increasing age, independent of blood pressure level (Van Bortel, et al., 2001; Asmar et al, 2001).

Increased intima-medial thickness has also been associated with increased pulse pressure and systolic blood pressure values in a number of studies. For example, Khattar, Acharya, Kinsey, Senior and Lahiri (1997), examined the relationship among blood pressure, pulse pressure, left ventricular dimension and carotid artery structure. Twenty four hour, ambulatory, intra-arterial blood pressure data were initially collected from 723 untreated hypertensive participants. A random sample of 140 of these participants was reassessed approximately 10 years later (Khattar, et al., 1997). Outcome measures of interest were collected at follow up and included left ventricular mass index (via echocardiography), carotid intima-media thickness (via carotid ultrasonography), and carotid cross-sectional area (calculated from carotid intima-medial thickness). The results of the study show significant, positive correlations between mean pulse pressure and intima-media thickness (r=.45). Mean pulse pressure was also predictive of left ventricular mass index and carotid intima-media thickness, but not carotid artery cross-section area (Khattar, et al., 1997).

Zanchetti, Crepaldi, Bond, et al., (2001) reported similar results from preliminary analyses in a sample of hypertensives with hypercholesterolemia. Data were collected from 508 individuals who participated in a randomized trial designed to compare the

effects of ACE inhibiting drugs combined with statins, and diuretics combined with lipid lowering diet on carotid lesions (Zanchetti et al., 2001). Measures included 24 hour, ambulatory blood pressure, carotid artery sonography, and BMI. The results indicate consistent, positive correlations among systolic blood pressure, pulse pressure, and carotid intima-media thickness, even after controlling for the effects of age, gender, and smoking status (Zanchetti et al., 2001). The authors hypothesize the consistent correlations among pulse pressure, systolic pressure, and measures of carotid intima-media thickness are probably the result of arterial stiffness due to media smooth muscle hypertrophy. However, the authors also note the atherosclerotic plaques could also be responsible for the increased intima-media thickness (Zanchetti, et al., 2001).

Recent findings indicate that increased pulse pressure is also an independent risk factor for cardiovascular disease (Franklin et al, 1999; Glynn et al, 2000; Alderman, 1999). For example, Alderman (1999) examined longitudinal data from 8690 treated hypertensives, ranging in age from 30 to 84 years. Blood pressure was controlled by medication for the first year of the five year follow-up. Outcome events of interest included congestive heart failure, stroke, MI, and other CVD events. The results show that a wide pulse pressure (e.g. greater than 60 mmHg) at any level of systolic or diastolic blood pressure was significantly predictive of cardiovascular events (Alderman, 1999). In addition, systolic but not diastolic blood pressure was linearly associated with age and cardiovascular event risk. Finally, participants with the highest pulse pressure values showed a seven fold increase in risk of a CVD event compared to those with the lowest the pulse pressure values (Low PP$\leq$ 46; Middle PP 47 - 62;High PP $\geq$63). These results suggests that pulse pressure combined with elevated systolic blood pressure is

a better predictor of cardiovascular events than diastolic blood pressure (Alderman, 1999).

Lee et al. (1999) examined the use of pulse pressure compared to SBP or DBP alone as predictors for fatal cardiovascular events (stroke, MI, and others) in the elderly. Data were drawn from participants in the Normative Aging Study. Analyses were conducted on data from 1828 participants under 60 years old, and 897 participants over 60 years old (Lee et al 1999). The results show that fatal cardiovascular events were best predicted by pulse pressure in the elderly (aged 60 and older), compared to either systolic or diastolic pressure alone (Lee et al. 1999).

Franklin et al (1999), assessed the relationship among age, blood pressure, pulse pressure and CHD in a review of longitudinal data from the Framingham Heart study. The results show a linear increase in systolic and diastolic pressure, and mean arterial pressure from 30 to 49 years. This was followed by a near plateau of diastolic pressure from 50 to 59 years, and decreasing diastolic pressure after 60 years of age (Franklin, 1999). As a result of these age-related changes, elevated systolic pressure and a wide pulse pressure were the best predictors of increased risk for cardiovascular disease among middle-aged and elderly subjects. These findings suggest that age-associated risk for CHD events is primarily due to large artery stiffness, as indicated by pulse pressure (Franklin,et al., 1999).

Similar findings were reported among elderly participants in a study by Glynn and colleagues, (2000). Data were collected from 9431 subjects, ranging in age from 65 to 102 years who participated in the Established Populations for Epidemiologic Studies of the Elderly study. The results show the highest risk of cardiovascular related mortality

was among those with elevated systolic and low diastolic pressure (SBP ≥ 160 & DBP < 70), followed by those with elevated systolic and moderately elevated diastolic pressure (SBP ≥ 160 & DBP of 70 -79). However, the lowest mortality was observed in normotensives with a low pulse pressure (SBP < 130 & DBP < 89). They also found that pulse pressure was best single predictor of cardiovascular mortality when controlling for systolic pressure, diastolic, and mean arterial pressure, for older people over age 65 (Glynn, et al. , 2000).

The preponderance of evidence points to pulse pressure as a reliable predictor of cardiovascular morbidity and mortality, particularly for older adults. A number of studies have focused on understanding sources of variance in SBP and DBP because of their predictive relationship to cardiovascular disease (Alderman, 1999; Lee, Rosner, & Weiss, 1999; Palatini, & Julius, 1997). Pulse pressure as a hemodynamic index has also been shown to have important implications for cardiovascular disease. However, there are no data available (to the best of the author's knowledge) on genetic and environmental sources of variance for pulse pressure among African American adults. In addition, little is known about the relationship between pulse pressure and psychosocial variables in this population.

Genetic Influences on Blood Pressure: Familial Aggregation Studies

A number of studies provide evidence that genetic factors are significant in blood pressure control in humans (Katzmarzyk, Perusse, Rice, Gagnon, Skinner, Wilmore, Leon, Rao, & Bouchard, 2000 ; Katzmazyk, Rankinen, Perusse, Rao & Bouchard, 2001; Luft, 2002; Chen, Srinivasan, Bao, & Berenson, 2001). The results of such studies show considerable resemblance of blood pressure values and risk for hypertension

among family members (Katzmarzyk et al, 2001; Chen et al., 2001). For instance, Katzmarzyk et al. (2000) examined familial resemblance for a number of heart disease risk factors such as plasma cholesterol level, age, and blood pressure in 113 African American, and 99 Caucasian families. The results of this study show a greater resemblance for risk factors within African American families compared to Caucasian families (Katzmarzyk, et al, 2000). The results also indicated a higher heritability for risk factors (plasma cholesterol and blood pressure) in African American families (53%) than in Caucasian families (34%) (Katzmarzyk, et al. 2000). In a follow up study, Katzmarzyk and colleagues (2001), noted the greatest similarity in blood pressure values and the highest risk for hypertension among first degree relatives. However, spouses also showed considerable resemblance indicating a substantial environmental effect on blood pressure as well (Katzmarzyk et al, 2001).

In another study, family aggregation of systolic and diastolic blood pressure as well as heart rate was assessed in 522 Caucasian, sedentary, normotensive families (An, Rice, Gagnon, Borecki, Perusse, Leon, Skinner, Wilmore, Bouchard, & Rao, 1999). The results showed substantial resemblance between spouses and heritability estimates ranging from 42% to 51% for diastolic and systolic blood pressure, respectively, within families (An et al., 1999). Similar clustering of blood pressure and other cardiovascular risk factors have been demonstrated among African American families in the Bogalusa Heart Study (Chen, et al., 2001). The study examined age-specific associations of parent and off-spring cardiac risk factors among 830 African American and Caucasian parent, off-spring pairs. The results of this study show that offspring values for a number of factors including systolic and diastolic blood pressure

were significantly associated with and predicted by childhood parental values of the same variables (Chen et al. 2001).

While the results of family studies clearly indicate significant genetic influences on blood pressure there are some methodological issues that should be considered. One the most prominent issues is the role of cultural transmission from parents to off-spring. This component of shared environment may inflate heritability estimates in family studies (Plomin, Defries, & McClearn 1990a). Another issue is the impact of age on a phenotype. For blood pressure this may be particularly problematic. As a result the twin method offers an efficient alternate method to estimate the heritability for blood pressure while minimizing parent-offspring cultural transmission and the effects of age (Neale & Cardon, 1992).

Twin Studies of Blood Pressure

The “classic twin design” is a frequently used quantitative genetic design and involves the comparison of identical, or monozygotic (MZ) twin pairs with fraternal, or dizygotic (DZ) twin pairs on some measurement. Using this approach, assumptions can be made about the variance in a measurement due to genetic and environmental sources, since MZ twin pairs share all their genes and DZ twin pairs share half of their genes (Plomin, Defries, & McClearn, 1990a). The proportion of variance in a measure due to genetic influences (the heritability), can be calculated by taking twice the difference in between MZ and DZ twin correlations (Plomin et al. 1990a). Furthermore, variation in an observed measure (phenotype) can be decomposed into variance due to genetic, shared environmental, and non-shared environmental influences:

$$Pv = Av + Cv + Ev$$

where Pv is the total variance in the phenotype, Av is the additive genetic variance of the phenotype, Cv is environmental variance that arises from sharing the same environment, and Ev is variance from non-shared environment for each twin and measurement error (Plomin et al., 1990a).

Previously, twin and family studies have been employed to estimate the sources of individual variability in blood pressure among Caucasian populations (Harshfield, Grim, Hwang, Savage, & Anderson, 1990). The results of these studies show that among Caucasians, heritability estimates range from .44 to .64 for SBP, and .34 to .73 for DBP (Alderman, 1999; Lee, Rosner & Weiss, 1999; Palatini & Julius, 1997; Ditto, 1993).

The classic twin design has been used to estimated heritable components for systolic and diastolic blood pressure, and heart rate. For example, Ditto (1993) using a sample of 100 young twin pairs (mean age 20, SD =5) from Montreal Canada, reported heritabilities of .65, .63, and .58 for resting heart rate, systolic, and diastolic blood pressures, respectively. Another study by Fagard, Brguljan, Staessen, Thijs, Derom, Thomis, and Vlietinck (1995) employed the 53 young male twin pairs to examine heritable factors for conventional and ambulatory blood pressures. The results indicated heritabilities for conventional blood pressure measures were .64 for systolic, .73 for diastolic, and .41 for heart rate (Fagard, et al, 1995).

Hong, de Faire, Heller, McClearn, and Pedersen (1994) examined genetic and environmental influences on blood pressure in a sample of 289 elderly twin pairs (mean age 63 SD= 8) from the Swedish Adoption/ Twin Study of Aging. Heritability estimates were .44 for systolic and .34 for diastolic blood pressure (Hong et al, 1994).

Taken together, these studies indicate that the twin method is an exceptional approach for estimating the heritability of blood pressure while controlling or minimizing effects of age differences among participants. In addition, the results of these studies suggest there is a decline in the genetic influence on blood pressure with age. However, these studies did not investigate the relationship between psychosocial measures such as stress or coping that may have an impact on blood pressure. In addition, they provide no information on heritability of blood pressure among African Americans.

Environmental and Psychosocial Factors, and Blood Pressure

Previous researchers have also noted the considerable impact of environmental factors on blood pressure (Pickering, 1999). For example, in a recent review of literature Pickering (1999) notes that among Western societies there is a historic, consistent graded association between socioeconomic factors and cardiovascular risk factors, namely smoking, cholesterol levels, and blood pressure. This line of research shows that socioeconomic factors, such as education tend to be inversely related to smoking, as well as cholesterol level (Pickering, 1999). Similarly, blood pressure has shown an inverse graded relationship with education in a number of studies (Pickering, 1999).

One of the most compelling findings concerning the impact of environment on blood pressure is the comparison of rates of hypertension for African Americans and Africans (Pickering, 1999). African Americans experience high rates of hypertension while rates are significantly lower among other African groups, especially in rural areas. However, rates of hypertension increase among African and African Americans with

increasing urbanization, or migration to cities (Pickering, 1999). Such finding suggest that psychosocial stress and racism work to impact blood pressure among African Americans (Pickering, 1999).

Other researchers have noted a number of environmental and behavioral factors common among groups with low rates of hypertension and little age associated blood pressure increase. These factors include low levels of industrialization, low sodium intake, high physical activity, and low levels of stress (Burke & Motulsky, 1992; Dressler, 1990). Other environmental conditions, such as crowding, noise, frequent change, and uncertainty of negative events have also been linked to increases in blood pressure (Burke & Motulsky, 1992; Dressler, 1990). In addition, stressful social factors such as discrimination and unemployment have also been associated with elevated blood pressure values (Burke & Motulsky, 1992).

Similar results have been found for other cardiovascular risk factors. For example, Sundquist, Malmstrom, & Johansson (1999) examined the impact of neighborhood environments on various risk factors including smoking, obesity, and sedentary behavior. The sample which consisted of resident ages 25 to 74, was drawn from the Swedish Annual Level of Living Survey. Data were collected via interview. The results of the logistic regression show a higher proportion of smokers, obese individuals, and inactive individuals with lower education levels and increasing indices of neighborhood deprivation, such as unemployment, crowding, or home ownership (Sundquist et al, 1999). Furthermore, residents of the most deprived neighborhoods showed an increased risk of being obese, physically inactive, and smoking compared to those in less deprived neighborhoods after controlling for individual educational

attainment (Sundquist et al, 1999).

In a similar study Marmot, Fuhrer, Ettner, Marks, Bumpass, & Ryff (1998) examined impact of psychosocial factors on the socioeconomic health gradient using data from the Midlife Development in the United States survey. Data were initially collected via telephone from 3,032 respondents ages 25 to 74 years. Later, respondents provided additional information via mail (Marmot et al., 1998). Outcome variables included self-reported health, waist-hip ratio, and psychological well-being (a composite score representing autonomy, mastery, personal growth, positive relations, purpose, and self-acceptance). Explanatory variables included occupation, income, and education (college graduate, college attendance, high school graduate, and high school attendance). There also are a number of moderating variables including parental occupation, household income, area poverty index, and perceived social inequalities. The results of the analyses indicate there was an inverse socioeconomic gradient (by education level) for all outcome measures (Marmot et al, 1998). The logistic regression results also show that area level measures of poverty were predictive of poor/ fair health, independent of education. In addition, for men father's occupation (but not mother's) and for women, mother's occupation (but not father's) was predictive of fair/poor health controlling for education (Marmot et al, 1998). These results show a significant relationship between environmental factors and health. The results also suggest that early environmental factors affect mid life health status (Marmot et al, 1998).

While these studies demonstrate the importance of environmental factors on health indices, they provide little information about the relative importance of genetic

factors in determining individual differences in health. These studies also fail to explicitly account for the impact of age on the health outcomes of interest. In addition these studies provide little insight into the role of environmental influences on variability in health indices among African Americans.

A number of researchers have suggested that environmental effects on blood pressure are mediated by psychosocial factors, particularly stress (Dressler, 1996; Pickering, 1999). In fact, much of the previous research examining blood pressure variability among African Americans has focused on the impact of psychosocial factors, particularly stress on hypertension (Falkner, 1996). The relationship between adverse, chronic environmental stress and an increased incidence of hypertension among those exposed to such conditions is well recognized (Falkner, 1996; Dressler, 1996). For instance, factors such as low socioeconomic status, racism, or behaviors such as perceived stress, and active coping, have been linked to increased blood pressure, heart rate, and heart contractility among African Americans (Dressler, 1996; Anderson, Myers, Pickering, & Jackson,1989). In addition, a numerous studies have examined role of stress reactivity in relation to blood pressure elevations and the etiology of hypertension (Falkner, 1996). A number of other studies have focused on more specific aspects of stress related to the experience of African Americans such as racism (Clark, 2001). It is important to note that while specific stressors (e.g. low SES, racism, etc.) are associated with blood pressure among African Americans, there still remains considerable variability in blood pressure levels within this group (Dressler, 1996).

Perceived Stress

Previous research indicates that perceived stress is associated with increased blood pressure (Suter, Maire, Holtz, & Vetter, 1997). Much of this research has examined perceptions of stress specifically related to work and /or home (Fauvel, Quelin, Ducher, Rakotomalala, & Laville, 2001; James & Bovbjerg, 2001). For example, James & Bovbjerg (2001), examined the independent effects of age and perceived stress on blood pressure variation among women employed outside the home. The study sample consisted of 91 healthy women ranging in age from 18 to 50 years. Measures included self rated perceived stress at home and at work, as well as ambulatory blood pressure (James & Bovbjerg, 2001). The results of the analyses indicate higher systolic and diastolic blood pressure values for women reporting higher perceived stress at work compared to those with equal work and home stress, and those with greater reported stress at home (James & Bovbjerg, 2001). In addition, blood pressure values showed a U-shaped curve when plotted against age with higher values for younger and older women (James & Bovbjerg, 2001).

In a similar study, Fauvel and colleagues (2000), investigated the relationship among perceived job stress, cardiovascular reactivity, and blood pressure at work. The sample consisted of 303 normotensive laborers ranging in age from 18 to 55 years. Measures included a job demands and job decision survey, and a measure of job strain. Other measures included the Stroop test, and Finapres and ambulatory blood pressure measures (Fauvel, et al. 2000). The results indicate that high strain workers exhibited significantly higher diastolic blood pressure values at work compared to non-high strain workers (Fauvel, et al. 2000).

There are a few studies that have examined the impact of more global measures

of stress (Suter, Maire , Holtz, & Vetter, 1997; Heslop, Smith, Carroll, Macleod, Hyland, & Hart, 2001). For instance, Suter and colleagues (1997) examined the relationship between self-perceived stress and blood pressure in a sample of 1666 Swiss volunteers. Measures included self rated health, family health history, overall stress, BMI, and two blood pressure measures. The results of the regression analysis indicated that age, measures of body fat, self rated health, and perceived stress were significant predictors of systolic blood pressure (Suter et al, 1997). It is interesting to note that perceived stress was inversely related to systolic blood pressure. In addition, those with high normal blood pressure (SBP of 130 - 139 or DBP of 85 -89) had significantly lower stress scores than normotensive participants (Suter, er al., 1997).

In a more recent study, Heslop, and colleagues (2001) examined the relationship between measures of perceived stress and coronary risk factors. This study focused on the impact of socioeconomic position in determining the impact of stress on risk factors (Heslop, et al, 2001). The analysis was performed on secondary data collected from a sample of 6832 men and women from various working classes in Britain. Coronary risk factor measures included in the study were blood pressure, plasma cholesterol level, BMI, FEV, physical activity level, alcohol consumption, and smoking. Perceived stress was measured by the Reeder stress inventory, a five item likert scale designed to measure global daily perceived strain (Heslop, et al. 2001). The results of the study show an inverse relationship of perceived stress with diastolic blood pressure, BMI, FEV, physical activity. However, perceived stress scores were significantly and positively associated with plasma cholesterol, smoking, and alcohol consumption (Heslop, et al. 2001).

The relationship between psychosocial stress and blood pressure increase may be somewhat variable (Suter et al, 1997). Although there is evidence that specific environmentally mediated psychosocial stressors such as racism are associated with blood pressure elevations among African Americans (Anderson, McNeilly & Myers, 1993; Clark, Anderson, Clark, & Williams, 1999; Clark, 2001). However, data concerning the relationship between perceived stress and blood pressure among African Americans are limited (Dressler, 1990; Adams, Aubert, & Clark, 1999).

One exception is the work of Dressler (1990) who examined the relationship between lifestyle incongruity, as a measure of stress and blood pressure measures in a sample of volunteers from an African American community in the Southeastern U.S. In this study, lifestyle incongruity was defined as the degree to which a person's lifestyle (e.g. status reflected by material possessions), exceeds that person's occupational class (Dressler, 1990). In this conception, a person's social status is indicated by lifestyle. Therefore, a large discrepancy between the financial resources available through occupational class, and lifestyle may result in stress through financial strain, increased work, or lowered social esteem (Dressler, 1990).

The sample consisted of the heads of households or spouses, ages 25 to 55, for 186 African American households (Dressler, 1990). Data were collected via interview and included family and household composition, medical history, occupational class, style of life, chronic stressors (marital relations and financial strain), BMI, and systolic and diastolic blood pressure measures (Dressler, 1990). The results of the analysis indicated that age, BMI, lifestyle incongruity, and chronic stressors were significant

predictors of blood pressure.

There were also significant interactive effects for age, lifestyle incongruity, and chronic stressors for blood pressure (Dressler, 1990). For instance, in the total sample, lifestyle incongruity, and chronic stressors were significant predictors of systolic and diastolic blood pressure for older (40 -55 years), but not younger (25 -39 years) participants (Dressler, 1990). Hypertensive status also showed an interactive effect with age, chronic stressors, and lifestyle incongruity. For younger hypertensives, compared to normotensives, higher blood pressure was associated with lower socioeconomic status and fewer chronic stressor. However, for older participants lifestyle incongruity was associated with higher blood pressure values regardless of hypertensive status (Dressler, 1990).

The author interpreted the results as supportive of the notion that increased lifestyle incongruity and concomitant stress result in increased blood pressure values for older participants (Dressler, 1990). Furthermore, the results also show that age is an important factor affect the relationship between indicators of stress and blood pressure among African Americans (Dressler, 1990). While the results show an increase in blood pressure associated with higher levels of lifestyle incongruity in the sample, the study failed to explicitly address perceptions of stress in the sample. In addition, the study implicitly assessed the stress associated with occupation and income, which are relatively specific stressors.

In another study, Adams et al., (1999) examined the relationship between a global measure of perceived stress, John Henryism, education, and blood pressure among African Americans. The results of the analyses indicated that perceived stress

was negatively associated with systolic blood pressure in this population (Adams, Aubert, & Clark, 1999). This study is discussed in further detail below.

Overall, the results of these studies indicate an association between blood pressure and measures of stress. Interestingly, several of the studies cited above show an inverse relationship between these factors, with lower reported levels of stress being associated with increased blood pressure values. However, these studies also have several limitations. For example, most of these studies have no or very few African American participants, with the exception of Adam et al, (1999), and Dressler (1990). As a result, there is still little known about the relationship between measures of perceived stress and blood pressure in this population. In addition, these studies provide no information about genetic sources of variance in perceptions of stress, and little explanation concerning sources of covariance between measures of perceived stress and blood pressure.

John Henryism

Lower socioeconomic status and the concomitant stress associated with socioeconomic status have been consistently associated with increased blood pressure in previous research. This relationship has been implicated as essential in the etiology of the disparate rate of hypertension experienced by African Americans. While socioeconomic status (SES) may be indexed in a number of ways including education, income, or occupation, African Americans still show a greater risk of hypertension compared to Caucasians, for all measures and levels of measurement of SES (James, 1994). It has been hypothesized that such findings result from the unrelenting psychosocial stress and differential daily demands experienced by African Americans

(James, 1994).

Previous studies have indicated that sustained cognitive and emotional effort to cope with difficult psychosocial problems (high effort or active coping) was associated with increased heart rate and systolic blood pressure, for the duration of coping (James, 1994). This association was found in real life and laboratory settings. It has been hypothesized the abrupt shifts in sympathetic arousal, resulting in elevated blood pressure, and heart rate, may over time (i.e. years) lead to established essential hypertension (James, 1994). It was also proposed that prolonged, high effort coping associated with the difficult psychosocial environment imposed by low SES (especially for African Americans) may explain the inverse relationship between SES and hypertension as well as the race differential for this relationship. These ideas coalesced with the story of the legendary African American steel driver, John Henry, into the concept of John Henryism.

John Henryism (JH) is a synonym for prolonged, high effort coping with psychosocial environmental stressors. Moreover, this conception of JH suggests that behavioral stressors are met with physical vigor, tenacity, and a strong sense of personal efficacy (James, LaCroix, Kleinbaum, & Strogatz, 1984). However, individuals differ in their propensity toward JH, with some people showing less effort or a shorter duration of active coping. As a result, James (1983; 1994), hypothesized that the inverse relationship between lower SES and blood pressure would be much greater for people who frequently exhibit high effort coping (i.e. high in JH) compared to those who exhibit high effort coping much less often (low in JH). Based on this hypothesis, JH has been the focus of much of the previous research on the impact of psychosocial

factors on health among African Americans.

There have been a few studies that have examined the impact of the JH coping style on health. Based on the JH hypothesis, studies that have examined the relationship between JH and blood pressure have regularly paired the JH measure / construct with other variables such as education or income. For example, Williams and Lawler (2001), examined the relationship among JH, hardiness (resiliance), and self reported stress-related illness in a sample of 100 low-income women. Although the results showed that being high on hardiness, and being African American buffered the effects of stress on health, there was no relationship between JH and self reported illness. Another study by van Loon, Tijhuis, Surtees, and Ormel (2001) examined the relationship between psychosocial factors and cancer risk factors in a Finnish sample. The results indicate that JH among other factors was significant in predicting smoking cessation. This suggests an important link between active coping and health behaviors (van Loon, et al, 2001).

The studies mentioned above demonstrate that the construct on John Henryism has been examined in relation to various health indices, in a variety of populations with sometimes mixed results. However, most research involving the construct has focused on JH's relationship with blood pressure, particularly among African Americans. For example, James, Hartnett, and Kalsbeek, (1983), examined the relationship between JH and blood pressure in a sample of African American men from eastern North Carolina. It was hypothesized that those high on JH but low on education would show elevated blood pressure compared to other groups (James, et al, 1983). The population-based sample consisted of 132 men, ranging in age from 17 to 60. The sample was of

working class status and education levels ranged from 0 to 18 years (median of 11 years). JH was measured using an 8 item scale and scores ranged from 14 to 24 (median of 20). Median splits for JH and education were used to form 4 groups: High JH-Low Education; High JH-High Education; Low JH - Low Education; and Low JH-High Education. Data, including three BP measures, were collected during an in person interview. SBP and DBP were analyzed separately (James et al, 1983).

The results indicate those High JH - Low Education men had the higher DBP values compared to the High JH-High Education group. JH was also negatively associated with education. However, JH was best predicted by work related factors (James et al, 1983). These results are interpreted as indicative that low education (i.e. lack of skill and material resources) limit an individual's ability to effectively interpret and control stressors (James et al, 1983). This coupled with a propensity to meet demands with hard work and determination (i.e. active coping) result in increased autonomic activity, BP, and risk for hypertension. However, the results also suggest that for those with high education, high JH may have a protective effect on cardiovascular health against the demands imposed by behavioral stressors (James et al, 1983).

In a follow-up study, James, LaCroix, Kleinbaum, and Strogatz, (1984) more closely examined the role of occupational stressors in the relationship between JH and BP in African American men. The purpose of this study was to examine the effects of selected job stressors (unemployment, negative perceptions), inhibited anger, and on-the-job social support on resting BP, and the effect of JH on these relationships (James et al 1984). Using the same sample as the previous study, the analysis included employment status, job security, job success, the role of race in job success, and

unfairness of wages and concomitant anger variables. JH and on-the-job social support were considered effect modifiers (James et al, 1984).

The results showed higher BP for unemployed men, those with less job security, and those with a low job success rating. However, the results also indicate a significant interactive effect for JH and job success. Those low on JH showed significant decreases in DBP with increased job success rating compared to those high on JH for whom success was not associated with decreased DBP. In addition, DBP was higher for high JH men who felt being African American had hindered their job success compared to those who felt advantaged by race (James et al. 1984). These results are taken as indicative that success following prolonged struggle, under difficult conditions is related to increased blood pressure. In other words, attaining job success when being African American is considered a hindrance, for High JH males may result in increased risk for hypertension (James et al, 1984).

Results from the studies cited above indicate that among African American men high JH combined with limited educational resources and occupational stressors produced an in increased risk for elevated blood pressure (James et al, 1983; 1984). These results are taken as support for the JH hypothesis (James, 1994), however a number of other variables may be potentially important to understanding the relationship between JH and blood pressure among African Americans. For example, Dressler, Bindon, and Neggers (1998) investigated gender differences in the relationship between JH and BP. Evidence suggests that social contextual factors related to gender may differentially affect the relationship between the JH active coping style and health outcomes for African American men and women (Dressler et al, 1998). Dressler et al

(1998) suggested traditional culturally based gender expectations combined with an active coping style may moderate the psychophysiological effects of stress for women, but exaggerate those same effects for men. In other words, for women, high effort coping in the service of one's family is health protective. However, for men high effort coping for upward social mobility in the face of limited resources is deleterious (Dressler et al 1998). It was hypothesized that JH would be more strongly associated with blood pressure for men than for women. The sample consisted of 600 African American men and women ranging in age from 25 to 65 years with an average of approximately 12 years of education. Measures included SBP, DBP, hypertensive status and the 12 item JH scale (Dressler et al, 1998).

The results of the regression show a significant interactive effect for gender and JH, on SBP and hypertensive status (Dressler et al, 1998). The results indicate that high JH is associated with increasing SBP for men, but decreasing SBP for women. In addition, women low on JH were more likely to be hypertensive than women with moderate or high JH scores, and men with low JH scores. However, high JH men are more likely to be hypertensive than high JH women and low to moderate JH men. The results suggest that for African American women, low JH scores are indicative of cardiovascular health risk (Dressler et al 1998).

Researchers have suggested that other psychosocial factors may interact with JH to affect blood pressure. Factors such as perceived stress may be an important component in the interactive effect of JH, education, and SES on blood pressure (Adams, Aubert, & Clark, 1999). Adams and colleagues, (1999) examined the relationship of JH, hostility, perceived stress, social support, and elevated blood

pressure in a sample of African American college students. It was hypothesized that: 1. high levels of JH, and Hostility will each increase the risk of elevated blood pressure; 2. JH will be inversely related to hostility; 3. social support will have a buffering effect on blood pressure, especially for those high on perceived stress; 4. perceived stress will be positively related to blood pressure; 5. JH will buffer the effect of perceived stress on blood pressure (Adams, Aubert, & Clark, 1999).

Two hundred and forty five African American male and female, college juniors and seniors ranging in age from 19 to 26 years volunteered to participate (Adams, Aubert, & Clark, 1999). Blood pressure was measured using a Finapres monitor. The average of the second and third measurements were used for statistical analysis. Elevated blood pressure was defined as SBP over 140 mmHg and DBP over 90 mmHg (Adams, Aubert, & Clark, 1999). Psychosocial measures for the study included the 12 item John Henryism scale, the Cook and Medley Hostility scale, Cohen's Perceived Stress scale, and the Perceived Social Support From Friends and From Family. Median splits were used to create Hi and Low categories for the psychosocial variables (Adams, Aubert, & Clark, 1999). The data were analyzed using Chi-square, Pearson correlation, and logistic regression.

The results show no difference in risk of elevated blood pressure based on JH scores (Adams, Aubert, & Clark, 1999). Hostility was not significantly correlated with blood pressure. However, people high on hostility had elevated DBP (Adams, Aubert, & Clark, 1999). JH and hostility were not significantly correlated (Adams, Aubert, & Clark, 1999). The results also show that people high on family or friend support were less likely to report high levels of stress. There was no association among family or friend

support and elevated blood pressure (Adams, Aubert, & Clark, 1999). However, those reporting high family support and low friend support were at lower risk for elevated DBP. There was not a significant interaction among family support, stress and risk for elevated blood pressure (Adams, Aubert, & Clark, 1999). The analysis showed a significant negative association between perceived stress and SBP, but not a significant interactive effect among perceived stress, JH and blood pressure (Adams, Aubert, & Clark, 1999). Finally, the results showed there was not a significant interactive effect of JH and perceived stress on blood pressure (Adams, Aubert, & Clark, 1999).

Social support was associated with lower level of stress, and family support lowered the risk of elevated DBP (Adams, Aubert, & Clark, 1999). These findings were consistent with previous literature. However, JH did not interact with perceived stress or hostility to affect blood pressure in this sample (Adams, Aubert, & Clark, 1999). The present findings do not support the hypotheses that JH's effect on blood pressure is mediated by either hostility or perceived stress in this sample. However, the authors note the results may be in part due to the nature of the sample or the role of development factors. For instance, the sample was relatively young and healthy (Adams, Aubert, & Clark, 1999). The authors suggest the relationship among these factors may become evident only at later ages (Adams, Aubert, & Clark, 1999).

Although the results have been somewhat mixed, the previous research has generally indicated that high JH, in combination with limited resources (e.g. low education, low SES, etc.) is associated with increased blood pressure among African Americans (James, Hartnett, & Kalsbeek, 1983 ; James, LaCroix, Kleinbaum, & Strogatz, 1984; James, Strogatz, Wing, & Ramsey, 1987). The studies cited above fail

to answer some address important consideration concerning JH. For example, previous research has failed to account for genetic and environmental sources of variation in JH. Since JH is conceived of as affecting health outcomes in the presence of limited resources, it could be expected that unique environmental influences would be significant and predominant in determining variance in JH. This active coping style is also thought to encompass a trait like propensity that varies across individuals, as well as physical vigor in its expression(James, 1994), therefore genetic influences may also play a significant role in determining JH. In addition, previous studies concerning the relationship of JH and blood pressure in African Americans have not explicitly examined the impact of age on the construct.

Twin Studies of Stress and Coping

To the best of the author's knowledge there have been no twin studies performed to examine genetic and environmental influences on perceived stress alone. However, there have been a few twin studies that have examined the heritability of measures of stress and coping (Mellins, Gatz, & Baker, 1996; Kendler, Kessler, Heath, Neale, & Eaves, 1991).

Mellins, and colleagues (1996) examined sources of individual differences in coping following stressful events among children. Coping with stressful events is a vital strategy with implications for long-term psychological adjustment and well-being, especially in children (Mellins, et al., 1996). The purpose of this study was to examine the extent that differential coping strategies are influenced by genetic and environmental factors and to estimate the influences of shared environmental factors on coping among co-twins (Mellins, et al, 1996). The sample consisted of 74 same sex twin pairs (41

male pairs), ranging in age from 9 to 16 years. Data were collected during in-person interviews and consisted of open-ended questions about coping with situations, the perceived coping questionnaire, experiences of stressful events, and the sibling inventory of stressful events. These measures were used to form seven latent constructs for coping in the biometrical modeling. These constructs included distraction, use of parents, use of peers, problem solving, self-soothe, problem focused, and emotion focused coping.

The results of the modeling show that genetic and unique environmental influences were significant in several problem focused coping styles. Heritability estimates for five (distraction, use of parents, use of peers, self-soothe, and problem focused) of the seven coping strategies ranged from .18 to .99. Shared environmental factors, but not genetic factors were found to be significant for emotion focused coping strategies, while most of the variance in problem solving strategies was accounted for by unique environmental influences (Mellins, et al, 1996).

The authors did not examine the perceived stress measures using biometrical modeling, but noted higher correlations for identical twin pairs compared to fraternal twin pairs, suggesting some genetic influence (Mellins, et al, 1996). The authors interpret these findings as indicative that genetic and unique environmental factors play a much greater role in determining several aspects of effective coping in children. However, in this sample shared environmental factors play a lesser, but significant role in emotion focused coping (Mellins, et al, 1996).

It is interesting to note that problem focused coping showed a significant and substantial genetic component (genetic influence = .57; environmental influence = .37)

(Mellins, et al, 1996). The authors define this construct as “attempts to alter or control threatening conditions” (Mellins, et al, 1996). This definition of the construct indicates an active engagement with the environment as an effort to cope with stressors, which is similar to the conception of John Henryism. This type of coping shows a fairly strong genetic influence suggesting similar results may be found for John Henryism.

Kendler and colleagues, (1991) carried out a similar study of coping with a sample of adult, Caucasian, female twin pairs. The study was designed to examine the factorial structure of several coping strategies and their the relationship to psychiatric symptoms, as well as estimate the genetic and environmental influences on coping (Kendler et al, 1991). The sample consisted of same-sex female twin pairs enrolled in a study of risk factors for psychiatric disorders. Data from 827 pairs of twins were collected via mail survey. Measures included a modified Ways of Coping Checklist, the Center for Epidemiologic Studies Depression Scale, the anxiety subscale from the SCL-90, and self reported life-events (Kendler et al, 1991).

The factor analysis resulted in the development of three coping scales: ‘turning to others’, ‘problem solving’, and ‘denial’ (Kendler et al, 1991). ‘Turning to others’ and ‘problem solving’ were inversely related to self-reported depression and anxiety, while ‘denial’ was positively associated with these measures. The results of the biometrical modeling show heritabilities of 30%, 31%, and 0 for ‘turning to others’, ‘problem solving’, and ‘denial’, respectively (Kendler et al, 1991). However, shared environment estimates for ‘denial’ were 19%. The results indicate that genetic factors are significant for ‘turning to others’ and ‘problem solving’ coping strategies, but not ‘denial’. These results are interpreted as inconsistent with social learning theories for ‘turning to others’ or ‘problem

solving' and suggest genetic factors related to temperament are most important for these coping strategies (Kendler et al, 1991).

The results of these studies indicate that genetic factors play a significant role in the variability in coping. However, to date no studies (to the best of my knowledge) have examined sources of individual variability in JH, an active coping measure that have been associated with health outcomes among African Americans.

The Study of Individual Differences in Minority Populations

There is a general paucity of literature concerning the sources of variability in various aspects of aging and health among minority elders (Whitfield & Baker Thomas, 1999). Much of the previous research concerning aging minority populations has focused on between group comparisons. The results of such studies are often said to apply to the respective groups as a whole. This implies the conception of groups as single monolithic entities. This conception is exacerbated among minority elders by the dearth of information concerning variability within these groups, particularly in old age (Whitfield & Brandon, 2000).

The individual differences approach when applied to minority populations can help redress this issue. The individual differences approach allows for the examination of inter-individual factors, while simultaneously examining intra-individual change over time (Whitfield & Baker-Thomas, 1999). The individual differences perspective holds that groups are heterogenous populations with traits that differ between individuals and these traits also account for intra-individual change over time. Previously this approach has been applied predominantly in the explanation of developmental phenomena among Caucasians. As a result, there is little information concerning traits that produce

inter and intra individual variability, and therefore group variability among minority elders (Whitfield & Baker-Thomas, 1999).

An important extension of the individual differences perspective on racial and ethnic minority health and aging is the use of within group designs (Whitfield & Brandon, 2000). Much of the previous research concerning racial and ethnic minorities has focused on between groups comparisons, and consisted mostly of the examination of differences between African Americans and Caucasians (Whitfield & Baker-Thomas, 1999; Jackson et al 1990). One critical limitation of between group, race comparison studies is they offer little information about variability within the groups being compared. This loss of distinction limits the interpretation of between group differences (Whitfield & Baker, 1999). Examining heterogeneity within a group can provide important information concerning sources of the variability that result in between group differences (Whitfield & Baker, 1999). In other words within group studies are one means to establish the "why" behind group differences, to accompany the "what" and "how" provided by between group comparison studies.

Quantitative genetic designs represent an important first step in understanding the etiology of individual differences in a phenotype (Plomin & McClearn, 1990). These designs can provide important conceptual and methodological approaches to assess origins of individual differences within minority populations (Whitfield & Brandon, 2000). Genetically informative samples can be used to identify environmental as well as heritable factors that influences individual variation. This approach is important for the study of African Americans because the socio-cultural environment that usually accounts for a significant proportion of variability can be examined within the concept of

minority groups as heterogeneous populations (Whitfield & Miles, 1995).

Quantitative genetic methods are an approach that can be used to examine sources of individual differences within ethnic groups (Whitfield & Brandon, 2000). Minority populations are distinct due to unique cultural, social, environmental, and historical factors (James et al, 1990). Within each minority group there are represented a heterogeneous set of qualities and traits that are unique to group. These characteristics and traits vary in expression between individual members of the group. As a result of the unique interplay of factors in minority populations, the proportion of the sources of individual differences (genetic and environmental) may vary across different groups (Whitfield & Brandon, 2000). However, understanding sources of individual differences can help to identify factors that may undermine or optimize health differences observed between groups (Whitfield & Brandon, 2000).

African American Twin Studies and Blood Pressure

To date few studies have explored the relative contribution of genetic and environmental factors to hemodynamic indexes among African Americans using the twin design. These researchers have used the classic twin design to examine heritabilities of blood pressure and cardiovascular disease indices among African descendants in Barbados and African Americans (Grim, et al., 1990; Harshfield, Grim, Hwang, Savage, & Anderson, 1990). For example, Grim, et al., (1990) designed a study to explore the relative contribution of genes and environment to blood pressure among African descendants in Barbados. The authors note this study was extension of their research concerning the “survival hypothesis” (Grim et al., 1989; Grim, Henry, & Meyers, 1995). This hypothesis states the ability to retain sodium was inherited by

surviving descendants of slaves and is responsible for the increased rate of salt sensitivity among African descendants in the Western Hemisphere. This salt sensitivity in combination with high salt environments leads to increased risk of essential hypertension among African Americans (Grim et al., 1990; Grim, et al., 1995).

Data were collected and analyzed from 37 pairs (17 MZ pairs, 20 DZ pairs)of African American twins. Measures collected during the assessment included age, height, weight, skin fold thickness, six supine systolic blood pressure readings, and sodium and potassium levels from 24 hour urine collection. There were no data collected on diastolic blood pressure. Intraclass correlations were used to calculate heritability estimate for the total sample and for the male twin pairs separately. The results show that heritability estimates for SBP were .04 for the entire sample, and .70 for the male twin pairs (Grim, et al., 1990). The results are interpreted as supportive of the hypothesis that blood pressure among African descendants in the Western Hemisphere is strongly affected by genetic factors (Grim, et al., 1990).

Harshfield, and colleagues (1990) employed the twin design to examine sources of variance in Left Ventricular Mass (LVM) and its determinants among normotensive African American twin pairs. A number of factors such as African American race, age, recurring stressors, and elevated blood pressure have been associated with increased LVM. Study participants were 22 same sex twin pairs (7 MZ pairs, 15 DZ pairs) with a mean age of 25 years and no significant health problems (Harshfield et al, 1990). Measures included demographic information, M-Mode Echocardiography to measure ventricular structure, self reported physical activity to measure caloric expenditure, and six blood pressure measurement (Harshfield et al, 1990). The results of the study show

that gender, age and systolic blood pressure were significant determinants of LVM and Left Ventricular Mass Index (LVMI - LVM adjusted for body surface area). Heritability estimates were calculated from MZ and DZ intraclass correlations after adjusting for gender, systolic blood pressure, and age. The estimated heritability for LVMI was .70 after adjustment (Harshfield et al, 1990). The authors interpret these results as supportive that genetic factors play a substantial role in determining cardiac structure among African Americans (Harshfield et al, 1990). It should be noted that the authors did not estimate genetic and environmental influences on the determinants of LVM, such as systolic pressure, in the current study. This may have been due to the very small number of twins included in the sample.

Generally, these studies indicate that among African Americans and African descendants, genetic factors are a significant source of variability in systolic blood pressure and left ventricular mass. However, these previous studies calculated estimates of heritability with small numbers of twin pairs. This can produce unstable estimates, and limits the generalizability of the results. In addition these studies failed to estimate sources of variance for diastolic blood pressure and pulse pressure which have also been show to be predictive of cardiovascular morbidity and mortality.

Chapter 2 Summary

Studying variability among and within individuals, across adulthood can provide important insight into the relationship between health and aging. Using an individual differences perspective can also shed light on the impact of aging on different variables and the affect of aging on the relationship among variables. The individual differences approach provides an accurate identification of significant factors for minority groups

that may be overlooked in cross-group comparisons with samples from the majority population.

The quantitative genetics approach in general, and the classic twin design in particular, offer a unique methodology for identifying genetic and environmental sources of variance in psychosocial measures, blood pressure, and the inter-relationship among these measures in African American adults. This approach allows for the identification of patterns of heritability and the impact of the environment that may be unique to African American populations. The quantitative genetic approach also provides an opportunity to examine the relative impact of genetic and environmental influences as people age within this population.

There are a number of limitations in the previously cited studies that will be addressed in the proposed research. In general, these previous studies offer little information concerning African Americans. For example, there are few studies of perceived stress or genetic influences of on blood pressure in this population. Where they do exist, sample sizes are often small which limits power and generalizability. In addition, the studies cited above generally do not explicitly examine the impact of age.

The current study represents an important extension of previous work. The proposed research will be the first study to examine sources of individual differences in the co-variance of among perceived stress, JH, SBP, DBP, and Pulse pressure. The previous twin studies of blood pressure in African American adults share several limitations including: 1) small sample size; 2) blood pressure measures limited to systolic pressure; 3) limited age variability; and 4) failure to test the significance of parameter estimate for blood pressure. The proposed study will address these

limitations. This study will include a larger number of twin pairs, which allows for better, more stable estimates of genetic and environmental influences. The proposed study will also include measures of DBP, and Pulse pressure, in addition to SBP. Furthermore, the proposed study will estimate sources of individual variance for Pulse pressure in an African American sample. Finally, the present study employs model fitting to test the significance of parameter estimates for genetic and environmental components of variance for SBP, DBP, and Pulse pressure.

This proposal is designed to conduct in-depth and higher order analyses using biometrical modeling to examine if the decomposed variation in well-being and blood pressure will show common sources of variance for correlated phenotypes of well-being and blood pressure. This decomposition will help to explain the source of the relationships among correlated phenotypes. A common source of variance may result from shared genetic mechanisms or shared environmental mechanism. In this way the current approach will allow for the identification of genetic, and environmental influences affecting both biological (blood pressure) and behavioral (perceived stress and active coping) phenotypes.

Research Questions

Based on the literature cited above, there are three main research questions that were addressed:

1) What are the proportions of genetic and environmental influences for measures of perceived stress, active coping, blood pressure, and pulse pressure among a sample of African American adult twins?

2) What is the impact of age on intra-individual variability in measures of perceived

stress, active coping, blood pressure, and pulse pressure among a sample of African American adult twins?

3) Are there shared genetic and environmental influences that account for the covariance among measures of blood pressure, perceived stress, and active coping for African Americans.?

Hypotheses

The analyses outlined in this proposal will be used to address the following hypotheses:

1) There will be significant genetic influences on perceived stress, John Henryism, blood pressure measures and pulse pressure.

2) Age will be a significant component of the variance for measures of systolic and diastolic blood pressure, pulse pressure, perceived stress, and John Henryism.

3) There will be common latent factors that account for covariance in the measures of blood pressure, perceived stress, and John Henryism at the genetic level.

4) There will be a significant environmental correlation between blood pressure and perceived stress.

Chapter 3: Methods

Subjects

The sample for the proposed study consists of same sex monozygotic (MZ) and dizygotic (DZ) twin pairs from the Carolina African American Twin Study of Aging (CAATSA). CAATSA was designed to examine health and psychosocial factors among adult African Americans between the ages of 22 and 89 years. Twin studies are one methodological approach to decomposing genetic an environmental sources of variance for variables of interest. CAATSA provides a unique opportunity to examine genetic and environmental influences on and among measures of perceived stress, coping, and blood pressure in a population based sample of African American Adults.

The total sample for the analyses consisted of 566 [including opposite sex dizygotic (OSDZ); 424 without OSDZ] individuals. The sample for the analyses included 102 MZ twin pairs, 110 DZ twin pairs, and 71 OSDZ twin pairs (Table 1).

Table 1: The number of Twin Pairs by Zygosity in the Sample

	Pairs	Individuals	Percent
MZ	102	204	36
DZ	110	220	38.9
OSDZ	71	142	25.1
Total	283	566	100

Sampling

The 1990 census data for all counties in North Carolina was reviewed to establish the ethnic distribution of African Americans within our target age range for the

initial study. In addition vital statistics by county from 1920, 1930, 1940, and 1950 were searched to examine the ethnic distribution of births by county. The statistics for living births in each county, for each of the years of interest were combined, and the counties were rank ordered according to birth rates. Next, the counties were ranked according to 1990 census data for the population 50 years of age and older. As a result of this method a list of 23 counties was created which represented more than 50% of the population over age 50 and also represented more than 50% of the African American births for the state.

Once the sample counties were identified, information from the birth records of twins was collected from the Offices of Vital Records and Register of Deeds of each selected county. The information recorded included date of birth, gender, name, number of living siblings, and name, address, and occupation of parents. This process resulted in a total of 14,852 twin records. These records were stored electronically and later used in the process of locating twins.

The records for deceased people were eliminated prior to the search for twins. As a result, 1,573 records for stillborn twins were initially eliminated. In addition, birth records for 1,253 people were marked as deceased, indicating their death sometime between birth and examination of the birth records for the study. Next, the Social Security Death Index (SSDI) was searched for the years of 1913 to 1932 to identify deceased subjects who may have drawn social security prior to 1998. This method eliminated an additional 717 records. A total of 11,309 records remained after the elimination of records for deceased individuals.

Twins were located through two methods. The first method involved finding

possible addresses and phone numbers for twins using the white pages from telephone books and internet web sites. Then the names and addresses were used to access credit reports for prospective subjects, using the “DAC” credit service. This service provided no credit information on potential subjects nor did not adversely affect potential subject’s credit profile. The person’s name, current and prior addresses, and occasionally a year of birth were included in the report. The recruitment process continued for potential subjects whose credit report information and birth record information matched. However, a number a records were excluded from further recruitment when: 1) a potential subject’s address was entered into DAC and the verification attempt produced a record for a different individual based on their date of birth, or 2) no record was available for access.

Searching voter registration records was the second method for locating twins. Voter registries were obtained from the 23 counties of interest. The registries were in both paper and electronic form. The paper copies of voter registries were searched by hand for matches to our birth registry. For the counties with electronic records, we used the Version 6.12 of the Statistical Analysis System (SAS) program for Windows to compare birth dates and first names listed in the voter registries to birth record information from all of the sampled counties.

This search method provided contact information for 8,810 twin records in total. However, there was no information available for 2,499 twin records using this method. The information from DAC and voter registries, minus unavailable records and deaths reported from SSDI and birth records, resulted in the identification of 59.3% of the twin registry.

Recruitment

Although there are relatively few published articles that address subject recruitment among African Americans, a number of successful recruitment strategies have been proposed (Picot, Stuckey, Humphrey, Smyth, & Whitehouse, 1996; Young, Edevie, Young, & Peters, 1996; Prohaska, Walcott-McQuigg, 1966). The results of previous research show that the successful recruitment of African Americans requires attention to cultural issues. For instance, memories of poorly designed and/ or unethical research, such as the Tuskegee Experiment make recruitment from this population more difficult (Bowman, 1991; Gamble, 1993). Current safeguards against participant abuse have not eliminated the fear of mistreatment for sake of research experienced by many African Americans (Gamble, 1993). This fear of research has contributed to the low participation rates and subsequent under representation of African Americans in clinical trials (King, 1992; Sevensson, 1989; Smith, 1991). However, successful recruitment of African Americans into research has been done and is possible.

The procedures and methods used in the CAATSA were drawn from previous research experiences with African American seniors and from other aging twin studies (e.g., The Swedish Adoption/Twin Study of Aging; the Minnesota Twin Study of Adult Development and Aging) (McGue, Hirsch, & Lykken, 1993; Pedersen, McClearn, Plomin, Nesselroade, Berg, DeFaire, 1991). Initially, potential participants were mailed a letter which provided basic information about the study. Two weeks after the letter, potential subjects were called. The phone call had a number of functions including verification of twin status, to provide additional information about study, it's purpose, and the interview process. The phone call also provided potential participants the opportunity to ask

questions and clarify information. Upon initial agreement to participate, a time and place for the interview were scheduled at the subject's convenience.

Measures

Age: Age was recorded in years was assess via self report during the interview and confirmed by date of birth from birth record.

Gender: Gender was recorded as male (0) or female (1) and was assess via self report during the interview and confirmed using the birth record.

Zygosity: Zygosity for the present study was established using a physical similarity questionnaire (Nichols & Bilbro,1966). This questionnaire was derived from physical similarity criteria established by research from Nichols and Bilbro (1966). In their study, physical similarity criteria were used to predict zygosity with 93 percent accuracy compared to genetic markers from blood (Nichols & Bilbro,1966).

Blood pressure: Blood pressure was taken by using an oscillometric automated device (A & D model UA-767; Milpitas California). Three measurements were taken in a sitting position, from the same arm, using a cuff of appropriate size for the participant's arm (Beevers, Lip, & O'Brien, 2001). The average SBP and DBP values were used in the analysis.

Antihypertension Medication Use: The status of antihypertensive medication use was ascertained from subject self-report. Data included the total number of medications, type of medication, and the dose. However, these variables were not analyzed in the present study.

Pulse Pressure: Pulse pressure (PP) was calculated for each subject by subtracting the average sitting DBP value from the average sitting SBP value.

The Perceived Stress Scale *(PSS)*(Cohen, Kamarck, & Mermelstein, 1983) is a 14 item, global measure of perceived stress. This measure is designed to measure the degree to which situations in one's life are appraised as stressful. The PSS has been shown to correlate with life events scores, depressive and physical symptomatology, utilization of health services and social anxiety. (For an example of scale items see Appendix A)

John Henryism is a 12 item scale developed by James, Hartnett, and Kalsbeck (1996) to assess the degree to which people feel they can control their environment. However, this measure does not assess Type A behavior or hostility (James, 1994). This measure is included because of past findings of its relationship with hypertension in African Americans. Previous literature (James, et al., 1983, 1994) found that those African Americans diagnosed with high blood pressure had higher scores on the JH scale. (For an example scale items see Appendix A)

Procedures

Participants completed an informed consent and then completed a 2.5 hour interview in the participant's home. The information was read to the participant to reduce the effects of low education. Once the interview was completed, the participants received 40 dollars.

Blood pressure measurements were taken at the mid-point of the 2.5 hour interview following a 5 minute rest period. The measurement was taken at the mid-point of the testing session to allow the subject to become acclimated to the interviewer to reduce arousal and anxiety (Beevers, Lip, & O'Brien, 2001). The rest period was introduced to attenuate any minor psychological stress that may have been produced during the interview. The blood pressure cuff was placed on the participants' bare arm.

The measurements lasted approximately 5 minutes and were taken while the subject sat upright in a comfortable chair.

Statistical Analyses

The analyses outlined in this section were used to address the following hypotheses: 1) there will be significant genetic influences on perceived stress, John Henryism, blood pressure measures and pulse pressure; 2) Age will be a significant component of the variance for measures of systolic and diastolic blood pressure, pulse pressure, perceived stress, and John Henryism.; 3) there will be common latent factors that account for covariance in the measures of perceived stress, John Henryism, blood pressure, and pulse pressure at the genetic level; 4) there will be a significant environmental correlation between blood pressure and perceived stress.

There were 3 different types of analyses to be conducted in the present study. These analyses included: *a) phenotypic analyses*: These analyses are based on individual level data and do not involve variance decomposition. Phenotypic analyses were performed for the average systolic blood pressure, average diastolic blood pressure, perceived stress, and John Henryism; b) *univariate analyses*: These analyses are based on twin pair data and involve variance decomposition. Univariate analyses were performed for average systolic blood pressure, average diastolic blood pressure, pulse pressure, perceived stress, and John Henryism; and c) *multivariate analyses:* Similar to the univariate analyses, these analyses also used twin pair data and involve variance decomposition. Multivariate analyses were performed for average systolic blood pressure, average diastolic blood pressure, pulse pressure, perceived stress, and John Henryism. Missing all three blood pressure measures for one twin of a pair,

eliminated that pair from the analyses. Based on this criterion one pair of MZ twins was excluded from the analyses with the remaining pairs included in this study (see Table 1).

Phenotypic Analyses

Initially, means and standard deviations were calculated for the variables of interest using the total sample. This was followed by a series of regression analyses with the average values for the variables of interest as the dependent variable in each analysis. The regressions were performed using data from randomly selected individual members of twin pairs. This, in effect, provided a population based, random sample of African American adults.

For the hemodynamic indices, factors previously identified as affecting blood pressure such as smoking, obesity (BMI), and anti-hypertensive medication use were entered into the model as well as age, gender, and education. The regression analyses were also used to assess the predictive relationship of age, gender, and education on perceived stress and JH.

The cross-correlations among the measures of interest were calculated following the regression analyses. These data were adjusted for significant predictors based on the results of the regression analyses (see Tables 6-10 for significant predictors for each variable). However, values were not adjusted for age since its effects were accounted for in the ACE-age regression analyses. After the phenotypic correlations were calculated, intra-class correlations were also calculated separately for MZ and DZ twin pairs. The pattern of intra-class correlations was inspected as an initial indicator of genetic and environmental influences.

Overview of Model Fitting Procedures

After the phenotypic analyses, the data were analyzed using biometrical model fitting procedures (see, Neale & Cardon, 1992). The following is a general overview of the model fitting procedures employed in this study.

Model fitting involves solving a series of simultaneous equations to estimate genetic and environmental parameters that best fit the observed familial covariances (Eaves, Last, Young, & Martin, 1978; Jinks & Fulker, 1970; Loehlin, 1987). Model-fitting offers a number of advantages including: 1) models that make assumptions explicit; 2) testing the fit of a particular model given its set of assumptions; 3) enabling analyses of data for several different familial relationships simultaneously; 4) providing appropriate estimates of quantitative genetic parameters and errors of estimate given the assumptions of the model; 5) it enables comparison of the fit of alternative models.

In model fitting, parameters are dropped to see if a more parsimonious model (i.e., a model with fewer parameters or latent variables, but still fitting the data) is available to explain the data. Statistical significance of these parameters is then assessed by Maximum Likelihood-ratio comparisons of the models after the parameters have been dropped. Significance of parameters is evaluated by taking the difference between the χ^2 statistics of the full and modified models and using that difference as a χ^2 statistic (see, Neale & Cardon, 1992). For example, the impact of genetic effects are tested by dropping the genetic parameter from the model. The χ^2 of the full model is then compared with the χ^2 of the modified model. If there is a significant difference between the χ^2 for each model it indicates the genetic parameter that was dropped is significant. The degrees of freedom are calculated by taking the difference between

the degrees of freedom for the full and modified models.

In the basic twin structural model, the overall variance is explained using three components: A -additive genetic variation, C -common or shared family environmental variation, and E -unique environmental variation (Plomin, et al., 1990). Additive genetic variation is the sum of the effects of genes influencing a behavioral trait. Common environmental variation is the phenotypic variation due to the subjects living in the same family and thus sharing the same environment (Plomin, et al., 1990). Unique environmental variation is the component of phenotypic variance that can be attributed to environmental factors not shared by family members and thus making family members different from one another (Plomin, et al., 1990).

Univariate Analyses

Hypothesis 1 and 2 were addressed using an ACE-age regression model (see Figure 1) to estimate the additive genetic (A) common environmental (C) and unique environmental (E) variance for the variables of interest. In addition, significant confounding factors for blood pressure such as smoking, obesity (BMI), and medication use were residualized prior to analysis.

Age is an important factor to explore for the hemodynamic variables included in the project. In addition, the age variability of the sample is quite high, so an ACE-age regression model was used in the analysis of the data. In this ACE-age regression model, estimates of genetic and environmental influences are corrected for age. This correction strengthens the estimates of common environmental influences and provides estimates of the proportion of variation associated with age (Snieder,2000). An example of this model can be seen in Figure 1.

Multivariate Analyses

Hypotheses 3 and 4 were addressed using multivariate genetic analysis. The multivariate genetic analyses were addressed primarily with the Cholesky: Factorial Genetic and Environmental Estimates. The Cholesky decomposition model allows for a factorial examination of the variances, specific and shared, to the measures at the genetic and environmental levels. An example of this model can be seen in Figure 2. This factorial model was used to assess the relationship among genetic and environmental factors at the latent construct level. This model was estimated for systolic and diastolic blood pressure, and the measures of John Henryism and perceived stress.

Chapter 4: Results

The results of the phenotypic and biometrical analyses are presented in this section. A description of the sample characteristics is presented first and includes the mean, standard deviation, range, and or frequency of demographic variables as well as the hemodynamic measures and psychosocial variables of interest. Data are presented separately for males and females. The sample description is followed by the phenotypic analyses and include regression analyses for SBP, DBP, Pulse pressure, perceived stress, and JH as well as correlations among these variables. This is followed by intraclass correlations presented by zygosity.

Following the initial phenotypic analyses the results of a test for gender differences in genetic and environmental influences is presented. This is followed by a series of biometrical models to test hypotheses 1 - 4 and includes univariate ACE-age Regression models of each variable, and a cholesky decomposition model.

Descriptive statistics and the phenotypic analyses were calculated using SPSS 11.0. Gender differences in genetic and environmental influences were assessed using the structure equation modeling program Mx. The ACE-age regression and Cholesky models were analyzed using the structural equation modeling program Lisrel 8.51.

Description of the Sample

The descriptive statistics for the entire sample are shown in Table 2. The total sample consisted of 566 (including Opposite Sex DZ; 424 without Opposite Sex DZ) individuals. The mean age for the sample was 46.76 (range 22 to 88). The sample was approximately 40 percent male. The mean level of education was higher than expected

with a value of 15.39. The average body mass index (BMI) for the sample was 29.28. The average for the hemodynamic indicators were 132.34, 81.48, and 50.87 for SBP, DBP, and Pulse pressure respectively. The mean perceived stress score was 19.76, while the average score for JH was 41.42 for the total sample. In the present sample 37.3 percent (Table 3) reported having been diagnosed with hypertension with 27.6 percent taking antihypertensive medication as directed, 2.1 percent taking it as needed, 1.4 percent reported no longer requiring antihypertensive medication, while 7.4 percent report no longer taking prescribed antihypertension medication (Table 4). In addition 21.8 percent of the sample reported smoking currently with 78.2 percent reported not smoking currently (Table 5).

Table 2: Descriptive Statistics for the Total Sample

	Mean	S.D	Range
Age (years)	46.76	13.44	22 - 88
Gender (% male)	40.5		
Education(years)	15.39	3.19	0 - 31
BMI ($mmkg^2$)	29.28	6.64	14.78 - 65.76
SBP (mmHg)	132.34	19.44	89.67 - 215
DBP (mmHg)	81.48	12.07	48.33 - 125.33
PP (mmHg)	50.87	14.13	21.33 - 125.33
Pstress	19.76	8.05	0 - 58
JH	41.42	4.5	10 - 48

BMI indicates body mass index; SBP indicates systolic blood pressure; DBP indicates diastolic blood pressure; PP indicates pulse pressure; Pstress indicates perceived stress; and JH indicates John Henryism

Table 3: Frequency of Self Reported Doctor's Diagnosis of Hypertension

	Percent
Not Diagnosed	62.7
Diagnosed	37.3

Table 4: Frequency of Self Reported Antihypertensive Medication Use among Hypertensives

	Percent
Not taking Medication	7.4
Discontinued	1.4
As Needed	2.1
As Directed	27.6

Table 5: Frequency of Self Reported Smoking

	Percent
Current Smoker	21.8
Not a Current Smoker	78.2

Regression Analyses

A series of regression analyses were performed on SBP, DBP, Pulse pressure, Perceived stress, and JH. The regression models for the hemodynamic variables included demographic variables such as age, gender, education and health variables previously found to be related to blood pressure values including BMI, antihypertensive medication use, and smoking. The regression models for perceived stress and JH included age, gender, and education as a continuous variable. Gender was entered as

‘0' for males and ‘1' for females. In addition, anti-hypertension medication use as well as smoking status were re-coded into dichotomous variables (0 = not taking anti-hypertensive medication, or not currently smoking; 1 = currently taking antihypertensive, medication or currently smoking).

SBP

Table 6 shows the results of the regression analysis for SBP. The overall model was significant (F = 10.742; p = <.001) and explained almost 20 percent of variance (R^2 = .18). Age (B=.335; p= <.001), gender (B= -.133; p=.02), medication use (B= -.111; p=.05), and BMI (B=.189; p=.001) were significant predictors in the model.

Table 6: Regression Analyses results for SBP

	Beta	t-value	p-value
Age	.335	5.72	<.001
Gender	-.133	-2.39	.02
Education	-.034	-.580	.56
Medication	-.111	-1.99	.05
Smoking	.024	.431	.67
BMI	.189	3.40	.001

DBP

The results of regression are presented in Table 7. The overall model for DBP was significant (F=3.42; p=.003) and accounted for 5 percent of the variance (R^2 = .05). The only significant predicator in the model was BMI (B=.210; p=<.001).

Table 7: Regression Analyses results for DBP

	Beta	t-value	p-value
Age	.107	1.70	.09
Gender	-.095	-1.58	.115
Education	.011	.18	.860
Medication	-.074	-1.23	.220
Smoking	.070	1.19	.236
BMI	.210	3.54	<.001

Pulse Pressure

Table 8 shows the results of the Pulse pressure regression analysis. For Pulse pressure, age (B=.379; p=<.001) was the only significant predictor. However, the overall model was significant (F=10.94; p=<.001) and explained nearly 20 percent of the variance (R^2 =.18).

Table 8: Regression Analyses results for Pulse pressure

	Beta	t-value	p-value
Age	.379	6.48	<.001
Gender	-.103	-1.85	.065
Education	-.058	-1.00	.317
Medication	-.091	-1.635	.103
Smoking	-.030	-.552	.581
BMI	.076	1.378	.169

Perceived Stress

The overall regression model for perceived stress was significant (F=7.05;

p=<.001) and explained 6 percent of the variance (R^2 = .06). For perceived stress, age (B= -.199; p=.001), gender (B=.171; p=.004), and education (B= -.186; p=.003) were significant predictors. These results are shown in Table 9.

Table 9: Regression Analyses results for perceived stress

	Beta	t-value	p-value
Age	-.199	-3.26	.001
Gender	.171	2.92	.004
Education	-.186	-3.03	.003

John Henryism

For JH the overall model was not significant (F=.33; p=.81). As the results reported in Table 10 show, neither age, gender, nor education were significant predictors in the present model.

Table 10: Regression Analyses results for John Henryism

	Beta	t-value	p-value
Age	.001	.018	.98
Gender	.056	.925	.36
Education	-.026	-.410	.68

Correlation Analyses

Correlations Between Indicators

After controlling for significant predictors, correlation analyses were used to assess the strength of the interrelationship between the factors of interest. The results of these analyses are shown in Table 11. With the exception of gender and JH, all the

variables showed significant correlations with age. In the data gender was coded as 0 for males and 1 for females. The results show significant positive correlations for BMI and perceived stress, but negatives correlations for SBP and Pulse pressure. Neither DBP, nor JH were significantly correlated with gender.

Table 11: Phenotypic Correlations for the Total Sample

	age	SBP	DBP	PP	Stress	JH
age	1					
SBP	.350**	1				
DBP	.123*	.695**	1			
PP	.414**	.745**	.096	1		
Stress	-.183**	-.182**	-.076	-.144*	1	
JH	.012	.058	-.006	.075	-.188**	1

* significant at p<.05; ** significant at p<.01

Intraclass Correlations

The results of the intraclass correlations are shown in Tables 12 and 13. The bolded values represent the correlations between Twin 1 and Twin 2. The pattern of the intraclass correlations shows higher correlations among MZ twin pairs compared to DZ twin pairs for SBP, DBP, Pulse pressure, perceived stress, and JH. This pattern of results suggests additive genetic influences for all the variables of interest.

Table 12: MZ Intraclass Correlations

	1	2	3	4	5	6	7	8	9	10
1-SBP 1	1									
2-DBP 1	.604	1								
3-PP 1	.729	-.003	1							
4-Stress1	-.281	-.175	-.175	1						
5-JH 1	.195	.117	.170	-.233	1					
6-SBP2	**.490**	.202	.494	-.148	.221	1				
7-DBP2	.301	**.408**	.124	-.082	.106	.636	1			
8-PP2	.441	.044	**.597**	-.123	.158	.762	.059	1		
9-Stress2	-.093	.118	-.188	**.312**	-.146	-.128	-.098	-.065	1	
10-JH2	.164	.047	.178	-.124	**.357**	.106	.114	.052	-.299	1

The MZ intra-class correlations appear in bold

Table 13: DZ Intraclass Correlations

	1	2	3	4	5	6	7	8	9	10
1-SBP 1	1									
2-DBP 1	.718	1								
3-PP 1	.712	.106	1							
4-Stress 1	-.116	.056	-.124	1						
5-JH 1	-.045	-.107	-.007	-.139	1					
6-SBP2	**.164**	-.108	.337	-.016	-.024	1				
7-DBP2	.067	**.102**	.037	.090	-.067	.625	1			
8-PP2	.139	-.200	**.442**	-.095	-.014	.758	.034	1		
9-Stress2	-.053	-.089	.011	**.143**	-.121	-.167	-.133	-.085	1	
10-JH2	.067	.046	.044	.043	**.138**	.140	.148	.035	-.197	1

The DZ intra-class correlations appear in bold

Gender Differences in the Factoral Structure of Genetic and Environmental Influences

Tables 14 and 15 show descriptive statistics for males and females in the total sample for SBP, DBP, Pulse pressure, perceived stress and JH. Examination of the descriptive statistics by gender indicates differences in male and female mean level values for SBP, DBP, and Perceived stress. In addition, the regression analyses indicate that for SBP and perceived stress, gender was a significant predictor. This raises an interesting question: Are there significant gender differences in the factoral structure of genetic and environmental influences on the variables of interest in the present sample?

Table 14: Descriptive Statistics for Males

	Mean	S.D.	Range
SBP	135.24	18.66	100.33 - 215
DBP	82.37	11.96	51.67 - 120.67
Stress	18.43	7.86	0 - 58
JH	41.25	4.64	10 - 48

Table 15: Descriptive Statistics for Females

	Mean	S.D.	Range
SBP	130.38	19.74	89.67 - 197.67
DBP	80.87	12.13	48.33 - 125.33
Stress	20.66	8.06	1 - 43
JH	41.54	4.40	29 - 48

To test for possible gender differences in genetic and environmental influences

among twins in the present sample, a Cholesky model was created to estimate the latent ACE factorial structure for SBP, DBP, perceived stress and JH for same sex male and female, MZ and DZ twin pairs, as well as OSDZ twin pairs.

Initially, a model was created in which additive genetic, common and unique environmental influences were estimated separately for five groups consisting of : male MZ and female MZ twin pairs, male DZ and female DZ twin pairs, and opposite sex DZ twin pairs. Next, estimates for males and females were constrained to be equal within zygosity. The change in -2 log likelihood was employed as a chi-square test statistic of the difference between the models. The difference in degrees of freedom (df) between the models was used as a the df for the chi-square statistic.

The results of this test are shown in Table 16. The results of the Mx analysis produced a -2 log likelihood of 17,121.490 with 2198 degrees of freedom. This model was compared to a reduced model in which matrices for calculating the covariance structure for same sex male and female twins pairs was constrained to be equal. The results for the reduced model produced a -2 log likelihood of 17185.363 with 2228 degrees of freedom. Subtracting the log likelihood and degrees of freedom values results in a χ^2 - delta test of the difference between the models. The χ^2 - delta was 36.873 with 30 degrees of freedom. This value did not exceed the critical chi-square value of 43.773 at p-value = .05. The results of this test indicate there are no significant gender differences in the factorial structure for genetic and environmental influences in the sample. Therefore, the male and female same sex twin pairs were pooled together within zygosity for the subsequent analyses.

Table 16: Test of Gender differences in Factoral Structure of Genetic and Environmental influences on SBP, DBP, Perceived Stress, and John Henryism.

Model	-2 Log Likelihood	df	-2 Log Likelihood$_{\Delta}$	df$_{\Delta}$	p-value
Full	17121.490	2198			
Reduced	17158.363	2228	36.873	30	N.S.

ACE - Age Regression Models

The analyses in this section will address the following hypotheses:

1) There will be significant genetic influences on perceived stress, John Henryism, blood pressure measures and pulse pressure.

2) Age will be a significant component of the variance for measures of systolic and diastolic blood pressure, pulse pressure, perceived stress, and John Henryism.

The ACE-Age regression analyses were employed to test the significance of genetic and environmental influences as well as age in accounting for similarity between twins in the variability of the measures of interest. Univariate ACE-age regression models were run for SBP, DBP, pulse pressure, perceived stress, and John Henryism. An example of the ACE-age regression model is depicted in Figure 1. The results of these analyses are presented separately by variable in this section. For each variable, parameter estimates based on the full model will be presented first. These estimates are squared to provide an estimate of the proportion of variance accounted for by the latent factors in the model. These values are presented with the parameter estimates.

The model fitting results for each variable are presented after the table of

parameter estimates. For each variable, the fit of the full model is presented by chi-square statistic and two indices of practical fit: the goodness of fit index (GFI) and the root-mean-square-residual (RMR). For GFI, adequate model fit is suggested by increasing values at or near 1.0. By contrast, for RMR adequate fit is suggested by decreasing values that approach zero.

The significance of a parameter is tested by dropping it from the model and assessing the resulting change in overall model fit. Therefore, only the chi-square statistic is reported for each reduced model. The difference between the full model chi-square and each reduced model chi-square was used as a chi-square statistic (chi-square delta), with the difference in degrees of freedom (df delta) used as the degrees of freedom for the statistic.

For each model age was tested first. This was followed by tests of common environmental influences (C) and additive genetic influences (A), respectively. However, each of these parameters was initially tested in the full model to assess a familial effect. For example, if C was tested and found to be non-significant initially it was placed back in the model for the test of A. This was done because it was possible for A and C to be non-significant singularly, but significant in combination, indicating a familial effect composed of genetic and common environmental influences.

After A and C were tested individually in the full model, any non-significant parameters were dropped to produce a more parsimonious model. Subsequently, the remaining significant parameter(s) were tested against the reduced model.

SBP

The parameter estimates and proportions of variance are shown in Table 17. Approximately 11 % of the variability in SBP was accounted for by age. Additive genetic factors accounted for about 34 % of the variability in this measure, with the remainder made up of unique environmental influences.

Table17: Parameter Estimates and Proportions of Variance for SBP

SBP	parameter estimate	percent variance
Age	.33	11
A	.58	34
C	0.0	0
E	.74	55

The model fitting results for SBP are shown in Table 18. The results show there was adequate fit for the full model which included parameter estimates for age, additive genetic, and common and unique environmental influences. The chi-square value was 3.66 with 7 df, and a p-value of .82. In addition, the practical fit indices also show adequate fit with a GFI of .99 and RMR of .05.

The first model tested was one for which the age parameter was dropped ($Age^{D}ACE$). The results show a substantial change in model fit with a 36.1 unit change in chi-square value. This change was significant at a p-value of .001 with 1 df. This change indicates that age is significant in the model for SBP. Therefore age was included in all subsequent models. Next, the common environmental influence parameter was tested ($AC^{D}E$). Dropping the C parameter from the model resulted in no change in chi-square. However, to test for a familial effect this parameter was placed

back in the model. Leaving the A parameter out of the model ($A^{D}CE$) resulted in a significant reduction in the fit of model at p-value .05, as indicated by the 6.32 unit change in chi-square. Next, C was removed from the model and additive genetic influences were tested in an AE model. Dropping A from this model resulted in a 22.41 unit change in chi-square. This reduction in fit was significant at the .001 level of significance. The results indicate a model that includes age, additive genetic influences, and unique environmental influences best explains the data for SBP.

Table 18: Univariate Age Regression Structural Equation Model for SBP

Model	χ^2	df	pvalue	GFI	RMR	χ^2_{Δ}	df_{Δ}	pvalue
Full	3.66	7	.82	.99	.05			
$Age^{D}ACE$	39.76	8	<.001			36.1	1	.001
$AC^{D}E$	3.66	8	.89			0.0	1	NS
$A^{D}CE$	9.98	8	.27			6.32	1	.05
AE	3.66	8	.89	.99	.05			
$A^{D}E$	22.41	9	.007			18.75	1	.001

Full: indicates ACE and Age parameters have been estimated
χ^2_{Δ}: χ^2 difference test of the full and reduced models
$Age^{D}ACE$: Reduced model, with the Age parameter dropped
$AC^{D}E$: Reduced model, with the C parameter dropped
$A^{D}CE$: Reduced model, with the A parameter dropped
AE: Reduced model, with estimates for the A and E parameters
$A^{D}E$: Reduced model, with the A parameter dropped- compared to AE model

DBP

Table 19 shows that most of the variability in DBP (63 %) is accounted for by unique environmental factors. There is also a substantial contribution from genetic influences (36 %) while age provides a small contribution to variability in DBP (1%).

Table 19: Parameter estimates of the full model for DBP

DBP	parameter estimate	percent variance
Age	.10	1
A	.60	36
C	0.0	0
E	.79	63

The model fitting results for DBP are shown in Table 20. The fit indices for the full model for DBP show exceptional fit. The chi-square was non-significant with a value of 1.99, at 7 df, resulting in a p-value of .96. A good fit was also indicated in the fit indices with a GFI of .99 and a RMR was .04.

The significance of age was tested in the first model ($Age^{D}ACE$). The results show that dropping age from the model resulted in a 3.11 unit change in chi-square. This was not a significant change in the fit of the model. Next, the common environmental parameter was tested. Leaving it out of the model resulted in no change in chi-square. There was a significant decrease in the fit of the model after dropping the additive genetic parameter (χ^2_{Δ}= 5.04;df_{Δ}=1; p=.05). Based on these results a reduced model was estimated that included additive genetic influences and unique environmental influences. The model showed adequate fit (χ^2 = 5.10; df= 9; p=.83; GFI=.99; RMR=.06). Dropping the additive genetic parameter from this model resulted in a 18.50 unit change which was significant at the .001 level of significance. Taken together, these results indicate the best fitting model for DBP is one that includes additive genetic and unique environmental influences.

Table 20: Univariate Age Regression Structural Equation Model for DBP

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df_Δ	pvalue
Full	1.99	7	.96	.99	.04			
Age^DACE	5.10	8	.75			3.11	1	NS
AC^DE	5.10	9	.83			0	1	NS
A^DCE	10.14	9	.34			5.04	1	.05
AE	5.10	9	.83	.99	.06			
A^DE	23.60	10	.009			18.50	1	.001

Full: indicates ACE and Age parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
Age^DACE: Reduced model, with the Age parameter dropped
AC^DE: Reduced model, with the C parameter dropped
A^DCE: Reduced model, with the A parameter dropped
AE: Reduced model, with estimates for the A and E parameters
A^DE: Reduced model, with the A parameter dropped- compared to AE model

Pulse pressure

Table 21 shows the variability in Pulse pressure is distributed across all the parameters of the full model. Age accounted for approximately 16% of the variability with the remaining 34%, 10%, and 40% of the variability distributed among additive genetic, common environmental, and unique environmental influences, respectively.

Table 21: Parameter estimates of the full model for Pulse pressure

PP	parameter estimate	percent variance
Age	.41	16
A	.58	34
C	.32	10
E	.63	40

The model fitting results for Pulse pressure are shown in Table 22. Model fitting

results indicate adequate fit for the full Pulse pressure model. The chi-square for the full model was non-significant with a value of 7.36 at 7 df, resulting in a p-value of .39. The GFI value was .98 and RMR was .04, also indicating adequate fit of the model.

The contribution of Age was tested in the first model ($Age^{D}ACE$). The results show that age is significant in the model. Dropping this parameter from the model resulted in a 52.38 unit change in chi-square which is significant the p=.001 level. Next, the common environmental parameter was tested ($AC^{D}E$). Dropping this parameter from the model resulted in a non-significant change in models fit (χ^2_{Δ}= .44;df_{Δ}=1; p=NS). Similarly, dropping the additive genetic parameter from the model did not significantly alter the fit of the model (χ^2_{Δ}=3.67;df_{Δ}=1; p=NS). However, dropping the additive genetic and common environmental parameters from the model resulted in a significant decrease in the fit of the model (χ^2_{Δ}= 7.82;df_{Δ}=2; p=.01). The results indicate the model that best explains the data for Pulse pressure is the full model. These results suggest a familial effect for Pulse pressure.

Table 22: Univariate Age Regression Structural Equation Model for Pulse Pressure

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df_Δ	pvalue
Full	7.36	7	.39	.98	.04			
$Age^D ACE$	59.38	8	<.001			52.38	1	.001
$AC^D E$	7.80	8	.45			.44	1	NS
$A^D CE$	11.03	8	.20			3.67	1	NS
$A^D C^D E$	51.92	9	<.001			44.56	2	.001

Full: indicates ACE and Age parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
$Age^D ACE$: Reduced model, with the Age parameter dropped
$AC^D E$: Reduced model, with the C parameter dropped
$A^D CE$: Reduced model, with the A parameter dropped
$A^D C^D E$: Reduced model, with the A and C parameters dropped

Perceived Stress

The parameter estimate presented in Table 23 show that age accounted for approximately 4% of the variability in perceived stress. The remaining proportions of variance are accounted for by additive genetic influences (25%) and unique environmental influences (71%).

Table 23: Parameter estimates of the full model for Perceived Stress

Stress	parameter estimate	percent variance
Age	-.21	4
A	.50	25
C	0.0	0
E	.84	71

Table 24 shows the model fitting results for perceived stress. The full model for

perceived stress showed excellent fit. The chi-square for this model was non-significant (p=1.0) with a value of .49 at 7 df. Similarly, the GFI and RMR show good fit with values of 1.0 and .02, respectively.

Age was initially assessed and found to be significant at the .001 level. There was a 16.21 unit change in chi-square after dropping this parameter from the model. Dropping the common environmental parameter resulted in no change in chi-square. Dropping the additive genetic parameter resulted in a non-significant change in chi-square of 1.49 units. However, dropping the additive genetic and common environmental parameters resulted in a significant reduction in the model fit (χ^2_Δ= 8.95;df_Δ=2; p=.05). These results suggest a familial effect, and indicate the best fitting model of perceived stress is one that includes age, additive genetic influences, and common and unique environmental influences.

Table 24: Univariate Age Regression Structural Equation Model for Perceived Stress

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df_Δ	pvalue
Full	.49	7	1.0	1.0	.02			
Age^DACE	16.70	8	.03			16.21	1	.001
AC^DE	.49	8	1.0			0	1	NS
A^DCE	1.98	8	.98			1.49	1	NS
A^DC^DE	9.44	9	.40			8.95	2	.05

Full: indicates ACE and Age parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
Age^DACE: Reduced model, with the Age parameter dropped
AC^DE: Reduced model, with the C parameter dropped
A^DCE: Reduced model, with the A parameter dropped
A^DC^DE: Reduced model, with the A and C parameters dropped

John Henryism

Table 25 shows the parameter estimates for JH. The results indicate that age accounted for approximately six-one thousandths of variability in JH. Approximately 34% of the variability was accounted for by additive genetic influences with remaining variability attributable to unique environmental influences.

Table 25: Parameter estimates of the full model for John Henryism

JH	parameter estimate	percent variance
Age	.08	006
A	.58	34
C	0.0	0
E	.81	65

Table 26 shows the model fitting results for JH. The chi-square and fit indices indicate an adequate fit for the full JH model. The chi-square value was 5.61 with 7 df, and a p-value of .59. The GFI and RMR also indicate adequate fit with values of .99 and .06, respectively.

The significance of the age parameter was assessed first. Dropping this parameter from the model ($Age^{D}ACE$) resulted in no significant change in the chi-square value (χ^2_{Δ}= 1.93;df_{Δ}=1; p=NS). Similarly, there were non-significant changes in the fit of the model after dropping the common environmental (χ^2_{Δ}= 0.0;df_{Δ}=1; p=NS) and additive genetic (χ^2_{Δ}= 2.88;df_{Δ}=1; p=NS) parameters. However, dropping both parameters from the model resulted in a significant decrease in the fit of the model (χ^2_{Δ}= 17.63;df_{Δ}=3; p=.001). These results indicate the data for JH are best explained by a

model that includes additive genetic influences, and common and unique environmental influences.

Table 26: Univariate Age Regression Structural Equation Model for JH

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df_Δ	pvalue
Full	5.61	7	.59	.99	.06			
Age^DACE	7.54	8	.48			1.93	1	NS
AC^DE	7.54	9	.58			0	1	NS
A^DCE	10.42	9	.32			2.88	1	NS
A^DC^DE	23.24	10	.009			15.70	2	.001

Full: indicates ACE and Age parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
Age^DACE: Reduced model, with the Age parameter dropped
AC^DE: Reduced model, with the C parameter dropped
A^DCE: Reduced model, with the A parameter dropped
A^DC^DE: Reduced model, with the A and C parameters dropped

Cholesky Model for Common Genetic, Shared and Unique Environmental Factors

The analyses presented in this section are used to test the following hypothesis:

3) There will be common latent factors that account for covariance in the measures of perceived stress, John Henryism, blood pressure, and pulse pressure at the genetic level.

A Cholesky decomposition model was created to investigate the genetic and environmental influences on the covariance among the SBP, DBP, perceive stress, and JH in the sample. An example of this model is depicted in Figure 2. In this model, latent additive genetic, common environmental, and unique environmental factors are estimated for SBP, DBP, perceived stress, and JH. This model results in four main latent factors for each source of variance (A, C,& E). Variable loadings are estimated

for each latent factor. As a result, the observed covariance among variables may be explained by a reduced number of shared, latent additive genetic, common environmental, and unique environmental factors.

The parameter estimates of factor loadings are presented in Table 27. The results show that SBP (element 1,1 of Table 27), DBP (element 2,1 of Table 27), and JH (element 4,1 of Table 27) load on the first genetic factor, although the loadings for perceived stress and JH are small. Perceived stress shows a substantial loading on the second genetic factor (element 3,2 of Table 27). The results also show that DBP, perceived stress, and JH load on the second genetic factor, with a substantial loading for perceived stress (element 3,2 of Table 27). Only JH showed a substantial loading on the on the third factor (element 4,3 of Table 27). JH showed a small loading on the fourth genetic factor (element 4,4 of Table 27). Overall, this structure suggests there are three latent additive factors that account for the covariance among the variables of interest.

DBP has a moderate loadings on the first common environmental factor (element 2,5 of Table 27). SBP (element 1,5 of Table 27), perceived stress (element 3,5 of Table 27), and JH (element 4,5 of Table 27) show small loadings on the first common environmental latent factor. None of the variables loaded on the second, third, or fourth common environmental factors. This suggest a single, latent common environmental factor affecting the covariance among the variables.

Each variable loaded substantially on it's own individual unique environmental factor (see elements 1,9 ; 2,10 ; 3,11 ; and 4,12 of Table 27). However, DBP, perceived stress and JH also loaded on the first factor as well as the second unique

environmental factor. Perceived stress and JH also loaded on the third unique environmental factor. With the exception of DBP (element 2,9 of Table 28) loading on the first factor, all loadings were fairly small. This structure suggest some overlap among the latent unique environmental factors accounts for the covariance among the variables of interest.

Table27: Parameter Estimates of Loading on Shared Latent Components

	A1	A2	A3	A4	C1	C2	C3	C4	E1	E2	E3	E4
SBP	.63 (1,1)				.14 (1,5)				.76 (1,9)			
DBP	.41 (2,1)	.27 (2,2)			-.31 (2,5)	0.0 (2,6)			.60 (2,9)	.55 (2,10)		
Stress	-.04 (3,1)	-.50 (3,2)	-.02 (3,3)		-.17 (3,5)	0.0 (3,6)	0.0 (3,7)		-.10 (3,9)	-.12 (3,10)	.83 (3,11)	
JH	.17 (4,1)	.13 (4,2)	.49 (4,3)	-.06 (4,4)	.02 (4,5)	0.0 (4,6)	0.0 (4,7)	0.0 (4,8)	-.04 (4,9)	.10 (4,10)	-.13 (4,11)	.83 (4,12)

The matrix designation for each estimate appears in parentheses with row number first, followed by the column number

The model fitting procedures for the Cholesky decomposition model are shown in Table 28. The full model shows adequate fit with a chi-square of 36.74, 42 df and a p-value of .74. The GFI and RMR also show adequate fit with values of .95 and .08, respectively.

Initially, a model was tested in which all shared unique environmental components were dropped. This resulted in a significant reduction in the fit of the model (χ^2_{Δ}= 131.01;df $_{\Delta}$=6; p=.001). Next, all common environmental parameters were dropped. This resulted in a non significant 7.7 unit change in chi-square. Similarly, there was not a significant change in the fit of the model after dropping all additive genetic parameters from the model (χ^2_{Δ}= 15.48;df $_{\Delta}$=10; p=NS). Although this change

in chi-square approached significance (the critical value is 18.31). Dropping all additive genetic parameters and all common environmental parameters resulted in a large and significant decrease in the fit of the model (χ^2_Δ= 115.5;df $_\Delta$=20; p=.001). Therefore, results indicate the data are best explained by a model that includes shared additive genetic influences, and shared common and unique environmental influences. These results suggest a familial effect for the covariance among blood pressure measures, perceived stress, and JH in the present sample.

Table 28: Cholesky Model fitting results for Shared Genetic, Common, and Unique Environmental influences for SBP, DBP, John Henryism, and Perceived Stress

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df $_\Delta$	pvalue
Full	36.74	42	.7	.95	.08			
ACE^{SD}	167.75	48	<.001			131.01	6	.001
AC^DE	44.44	52	.76			7.7	10	NS
A^DCE	52.22	52	.47			15.48	10	NS
A^DC^DE	152.24	62	<.001			115.5	20	.001

Full: indicates all latent factor loadings for ACE parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
ACE^{SD}: Reduced model, with the all shared unique environmental parameters dropped
AC^DE: Reduced model, with all of the C parameters dropped
A^DCE: Reduced model, with all of the A parameters dropped
A^DC^DE: Reduced model, with all A and C parameters dropped

Correlations among the Latent Factors

The following analyses address the following hypothesis:

4) There will be a significant latent environmental correlations between blood pressure and perceived stress.

The same cholesky model described above was used to test the significance of shared environmental parameters between SBP, DBP, perceived stress, and JH. In the

previous assessment the shared unique environmental parameters taken together, (i.e. all cross-factor loadings) were significant in the model. The parameter estimates from individual cross factor loadings in the cholesky model (see Table 27) were used to calculate the correlation among the latent factors. The correlations between the latent additive genetic, common environmental, and unique environmental factors are reported in Table 29.

The results of the cholesky model fitting indicate that the genetic parameter estimates when tested together approached, but failed to reach, significance. Therefore, the correlations among the latent genetic factors for blood pressure and the psychosocial measures are not significant. Although, it is interesting to note there are substantial correlations between SBP and DBP for the latent genetic and unique environmental factors, suggesting the failure to reach significance may be related to statistical power.

The shared unique environmental parameters, taken together were significant in the model. Therefore, the significance of the cross factor loadings (and corresponding latent factor correlations) between the blood pressure factors and the psychosocial factors were tested. In addition, the substantial correlation between the unique environmental factors for SBP and DBP begs the conceptual question of whether shared unique environmental factors significantly account for the covariance between these variables. Therefore, this correlation was tested in an exploratory analysis.

There are substantial correlations between the latent common environmental factors. However, the factor loadings are quite small (see Table 27). Using such small values to calculate the variance-covariance matrices resulted in variance-covariance

values of almost identical scale. For example, the variances ranged from .0004 to .10, while the covariances ranged from -.003 to .04. Using values that are so similar in the correlation formula [COV_{XY} / / (σ^2_X *σ^2_Y)] results in fairly high correlations. However, because that factor loadings are so small, there is actually very little variance accounted for in the parameter estimates for the latent common environmental factors.

Table 29: Latent Factor Correlations for BP, Stress, and JH

A		SBP	DBP	Stress	JH
	SBP	1			
	DBP	.84	1		
	Stress	-.09	.49	1	
	JH	.32	.11	-.02	1
C		SBP	DBP	Stress	JH
	SBP	1			
	DBP	-.89	1		
	Stress	.83	-.92	1	
	JH	(-)	(-)	(-)	1
E		SBP	DBP	Stress	JH
	SBP	1			
	DBP	.74	1		
	Stress	-.12	-.19	1	
	JH	-.05	.04	-.17	1

(-) indicates the variance / covariance values were too small to accurately calculate

The following analyses assessed the significance of the factor loadings for perceived stress and JH on the unique environmental factors for SBP and DBP (the unique environmental factors 1 & 2) from the cholesky model. The results of the tests of significance are reported in Table 30. Dropping the factor loadings for perceived stress

(element 3,9 of Table 27) and JH (element 4,9 of Table 27) for the first unique environmental factor did not significantly alter the fit of the model (χ^2_Δ= 1.58;df $_\Delta$=2; p=NS). Similarly, there was not a significant change in the model fit after dropping the factor loadings for perceived stress (element 3,10 of Table 27) and JH (element 4,10 of Table 27) on the second unique environmental factor (χ^2_Δ= 2.11;df $_\Delta$=2; p=NS). These results indicate the cross factor loading between the blood pressure factors and psychosocial factors are not significant. As a result, the correlations between the latent, unique environmental factors for blood pressure and the psychsocial variables are not significant.

Table 30: Model Fitting Results for Shared Unique Environmental Parameters for BP, Stress, and JH

Model	χ^2	df	pvalue	GFI	RMR	χ^2_Δ	df $_\Delta$	pvalue
Full	36.74	42	.70	.95	.08			
E-1^{SD}	38.32	44				1.58	2	NS
E-2^{SD}	38.85	44				2.11	2	NS

Full: indicates all latent factor loadings for the ACE parameters have been estimated
χ^2_Δ: χ^2 difference test of the full and reduced models
E-1^{SD}: Reduced model, with the cross factor loadings for Stress and JH on first (SBP) Unique Environmental factor dropped
E-2^{SD}: Reduced model, with the cross factor loadings for Stress and JH on second(DBP) Unique Environmental factor dropped

Latent Unique Environmental Factor Correlation between SBP and DBP

This section provides the results of an exploratory analysis designed to test the significance of the shared Unique Environmental latent factor correlation between SBP and DBP. The results of the model fitting procedures are shown in Table 31. Dropping the factor loading for DBP (element 2,9 of Table 27) from the first unique environmental

factor significantly reduced the fit of the model (χ^2_{Δ}= 121.01;df_{Δ}=1; p= <.001). This indicates the cross factor loading between SBP and DBP is significant and therefore indicates the latent unique environmental factor correlation is also significant.

Table 31: Model Fitting Results for Shared Unique Environmental Parameters for SBP and DBP

Model	χ^2	df	pvalue	GFI	RMR	χ^2_{Δ}	df_{Δ}	pvalue
Full	36.74	42	.70	.95	.08			
E-1^{SD}	157.75	43				121.01	1	<.001

Full: indicates all latent factor loadings for the ACE parameters have been estimated
χ^2_{Δ}: χ^2 difference test of the full and reduced models
E-1^{SD}: Reduced model, with the cross factor loadings for DBP on first (SBP) Unique Environmental factor dropped

Chapter 5: Discussion

The analyses presented in the previous section were completed to address the proposed research questions which were based on the prior literature. In the following section, an overview of the results will be presented. This overview will include a brief summary of the results of the phenotypic and biometrical analyses. In addition, the research questions and hypotheses will be addressed based on the results of the present study and previous research presented in the literature.

For some phenotypes, age is an important factor that should be accounted for when estimating sources of individual variability. This was especially important for the hemodynamic variables included in the present study. However, the contribution of age to the variability in measures of stress and coping among African Americans has not been well studied. The assessment of the influence of age on these variables represents an expansion of our knowledge about sources of variability in stress and coping for this group.

Phenotypic Analyses

Overall, the results of the phenotypic correlation analyses revealed that age was significantly related to all the variables of interest, with exception of JH and DBP. In addition, the results of the regression analyses show that age was a significant predictor for SBP, pulse pressure, and perceived stress. This is consistent with previous research, which has shown age to be consistently associated with increased blood pressure and pulse pressure values (AHA, 2002; Glynn, et al., 2000; JNC VI, 1997; Benetos, 1999; Franklin, 1999). There are essentially no studies available concerning

the relationship between age, perceived stress, and health among African Americans. The exception is Dressler's, (1990) work, which shows an interactive effect for age, stress (as measured by lifestyle incongruity), and blood pressure in African American adults. In this study, stress was associated with higher blood pressure values for all older participants, but only among hypertensives for younger participants (Dressler, 1990). Adams et al., (2001), suggested that JH may, and its relationship with blood pressure may, strengthen with age. However, the results of the phenotypic analyses do not support this contention.

Examination of the intra-class correlations showed a pattern of higher correlations for MZ twin pairs compared to DZ twin pairs for all the variables of interest. This pattern is consistent with the presence of genetic influences. Following this brief assessment of genetic influences, potential gender effects were tested using biometrical model fitting. This examination showed there were no significant gender differences in the overall genetic and environmental influences. This finding suggests sources of variability for the phenotypes are similar for both men and women in this population. After these analyses, male and female same-sex twin pairs were analyzed together by zygosity.

In the following sections, the results for each analysis will be addressed as well as the relevant research questions and hypotheses.

Univariate Analyses: ACE-Age Regression Model

The ACE-Age regression model was used for the univariate analyses. This model was employed to address the following Research Questions:

1) What are the proportions of genetic and environmental influences for measures of

perceived stress, active coping, blood pressure, and pulse pressure among a sample of African American adult twins?

2) What is the impact of age on intra-individual variability in measures of perceived stress, active coping, blood pressure, and pulse pressure among a sample of African American adult twins?

Th ACE-Age regression model was employed to test the following Hypotheses:

1) There will be significant genetic influences on perceived stress, John Henryism, blood pressure measures and pulse pressure.

2) Age will be a significant component of the variance for measures of systolic and diastolic blood pressure, pulse pressure, perceived stress, and John Henryism.

SBP and DBP

For SBP, results of the ACE-Age regression analyses show that age accounted for approximately 11% of the variance in SBP, while 34% and 55% of the variance was attributable to additive genetic and unique environmental influences, respectively. The biometrical model fitting analyses show that the influence of age, additive genetic factors, and unique environmental factors are significant in explaining the variability in SBP. For DBP, 1% of the variability in this measure was attributable to age. Additive genetic factors accounted for 36% of the variability in DBP, with the remaining 63% of the variance accounted for by unique environmental factors. The biometrical model fitting results indicate that additive genetic and unique environmental influences are significant in determining variability in DBP. These findings are within the range of previous heritability estimates in other populations. For example, previous researchers have estimated heritabilities that ranged between .44 and .64 for SBP, and between .34

and .73 for DBP in Caucasian populations (Alderman, 1999; Lee et al., 1999; Palatini & Julius, 1997; Ditto, 1993).

It is interesting to note that for SBP and DBP, unique environmental factors are quite substantial and account for 55% and 63% of the variance in the measures, respectively. This is somewhat different from the results of studies by Ditto (1993) and Fagard et al.,(1995). Both of these studies employed samples of young twins and based on the heritability estimates for each, unique environmental influences ranged between 36% and 37% for SBP, and between 27% and 42% for DBP. However, Hong et al., (1994) estimated that among older twins, unique environmental influences accounted for 56% and 66% of the variance in SBP and DBP, respectively. This difference suggests that unique environmental influences increase, while genetic influences decrease with age among Caucasians. However, the results of the present study suggest that for African Americans, unique environmental factors exert substantial influence on SBP and DBP across adulthood for African Americans. For instance, social forces related to racism or the struggle for improved socioeconomic status may explain the relatively large unique environmental component observed in this sample. These factors will be further explored in future research in this area.

Previous research has noted that age is an important factor associated with SBP. For example, researchers have consistently found age-associated increases in SBP and concomitant risk for cardiovascular morbidity and mortality (Glynn, et al., 2000; JNC VI, 1997; Benetos, 1999; Franklin, 1999). The results of the present study add to these findings by estimating the proportion of variability in SBP due to age. This provides information about the contribution of age to SBP variability, relative to genetic

and environmental influences.

These results confirm the findings of previous researchers (Glynn, et al., 2000; JNC VI, 1997; Benetos, 1999; Franklin, 1999) and show that age is a critical factor related to blood pressure variability in this population. The relevant factors may be associated with biological phenomena. For instance, the contribution of age to variability in blood pressure may also reflect age-associated biological changes in vascular structure and function, such as decreased aortic compliance (Glynn et al., 2000; Franklin et al., 1999; Lee et al., 1999). Age's contribution to blood pressure variability among African Americans may also reflect social phenomena with health implications. For example, the findings may represent a cohort effect. Given the substantial variability in age in the sample, broader social factors related to age, such as medical access or changes in nutrition may produce age associated changes in vascular structures (e.g. stiffness) and thus contribute to variability in blood pressure. Similarly, shifts in social structures, such as the move from a segregated to a more integrated society, may result in age associated changes in the vascular system. For example, greater and more frequent exposure to noxious events for older adults may manifest as age associated variability in blood pressure (Anderson, et al, 1993; Clark, 2001).

The present results are somewhat different from those reported in previous twin studies with samples of African descendants. For example, Grim et al., (1990) estimated the heritability for SBP to be .04 for their entire sample of 37 twins, and .70 for the male twins. These results represent a substantial departure from the results of the present analyses and the heritability estimates for blood pressure in other

populations. This is probably attributable to differences in sample size. In addition, the present study did not find significant gender differences in additive genetic influences, therefore separate heritability estimates were not calculated for men and women. Given the larger sample size, the present work represents an improvement over heritability estimates from these previous studies of African American twins.

The results for SBP support hypotheses 1 and 2. The results indicate that genetic influences as well as the influences of age are significant in determining the variability of SBP. The results support Hypothesis 1, but not Hypothesis 2 for DBP. The results indicate a significant genetic, but not age, component for DBP. These results are interpreted as representative of evidence of a multi-factoral combination of genetic and environmental influences affecting blood pressure regulation in this population.

Pulse Pressure

Pulse pressure provides an index of large artery compliance and is calculated as the difference between systolic and diastolic blood pressure (Franklin, 1999; Glynn et al, 2000; Franklin et al, 1999). Pulse pressure has been associated with increased carotid intima-media thickness (Khattar, et al., 1997) and is an independent predictor of cardiovascular morbidity and mortality, especially with advancing age (Franklin, et al., 1999; Glynn et al., 2000).

The estimation of sources of variability in pulse pressure among African American twins is a unique feature of the present study. The results show that 16% of the variance in pulse pressure was accounted for by age. Additive genetic influences, common environmental influences, and unique environmental influence accounted for

34%, 10%, and 40% respectively.

The results indicate the full model best explained the data. The model fitting analyses indicated that neither additive genetic, nor common environmental influences alone were significant in the full model. However, dropping both sets of parameter estimates, simultaneously from the model resulted in a significant change in the model's fit. These results suggest a significant familial effect on variability in pulse pressure. Therefore, heritable and common environmental effects are significant in determining variability in the measure.

The finding of a familial effect may be explained by the association of pulse pressure with increased intimal-media thickness or other vascular structural changes (Khattar, et al., 1997; Zanchetti, et al., 2001). For example, Zanchetti et al., (2001) suggest the association between pulse pressure and increased measures of intima-media thickness is due primarily to medial smooth muscle hypertrophy. However, this group's results also suggests that atherosclerotic processes may responsible for the observed intima-media thickness (Zanchetti, et al., 2001). The familial effect for pulse pressure observed here may reflect genetic influences related to vascular structure, as well as early family influences, such as dietary fat consumption, that contribute to arterial stiffness as indicated by pulse pressure.

The present results may also reflect the influence of social factors on hemodynamic factors among African Americans. These factors may represent social phenomena that affect cohorts as well as individual factors (e.g. social mobility, occupational stress, individual level racism) and may have a cumulative effect. As an example, inadequate access to medical care early in life, combined with high fat diets,

and repeated exposure to noxious social events may contribute to the age related variability in pulse pressure. Anderson et al., (1993) have suggested that repeated exposure to psychosocial stressors related to SES and race result in greater and earlier vascular hypertrophy and peripheral resistance among African Americans. This hypothesis is supported by the present findings, especially when one considers the substantial contribution of age and unique environmental influences to variability in pulse pressure.

The proportion of variability due to age was largest for pulse pressure. This is interesting, and somewhat consistent with the previous research indicating pulse pressure is predictive of cardiovascular events in older adults (Franklin et al., 1999; Glynn, et al., 2000). In addition this may also reflect the importance of age related changes in arterial stiffness and concomitant increases in pulse pressure and SBP (Van Bortel, et al., 2001; Asmar et al, 2001).

Together, the results for pulse pressure are interpreted as indicative of early environmental influences, present in families with lasting effects over time. For example, nutritional factors such as fat consumption may have contributed to atherosclerotic factors in vascular walls. In a fashion similar to blood pressure, these results may also reflect broad time / age related social factors that affect vascular health.

The findings partially support Hypothesis 1 for pulse pressure. The results indicate that some combination of genetic and common environmental influences is significant in determining variability in pulse pressure. The results support Hypothesis 2 for pulse pressure. Age is a significant component of the variability in pulse pressure.

Perceived Stress

The analyses for perceived stress show that age, additive genetic influences, and unique environmental influences accounted for 4%, 25%, and 71% of the variability in measure, respectively. The results also indicate a familial effect is present for perceived stress, suggesting both genetic and common environmental effects are significant in determining variability in perceived stress.

To the best of the author's knowledge, no previous studies have examined genetic and environmental influences on perceived stress. The present findings suggest that additive genetic and common environmental influences work in concert to affect perceptions of stress. This may be reflective of social learning factors, which have been suggested to account for significant common environmental effects on coping with stress (Kendler et al., 2001). However, the results also indicate that genetic factors play a role.

The findings suggest that an individual's propensity to perceive situations as stressful is influenced by heritable as well as family environmental factors. For example, the inclination to perceive certain events as stressful may be determined by some early biologically based factors related to temperament. These factors may be reinforced by modeling among family members. However, the results can also be interpreted as reflective of substantial unique environmental influences. This in combination with the presence of a significant age component is indicative of the array of psychosocial stressors confronted by African Americans across adulthood. The age component may reflect some changes in societal structures (economic mobility, segregation, etc.) that may affect cohorts. However, unique environmental experiences

account for most of the variability in perceived stress in this group. These findings are supportive of previous results that indicate factors such as occupation demands, socioeconomic status, or perceived racism are most important in the perceptions of stress in this group (Dressler, 1996; Clark, 2001).

The findings partially support Hypothesis 1 for perceived stress. The results suggest that some combination of genetic and common environmental influences is significant in determining variability in perceived stress. The results support hypothesis 2 for perceived stress and indicate that age is a significant component of variability in perceived stress.

John Henryism

Approximately 34% and 65% of the variability in JH was attributable to additive genetic, and unique environmental influences, respectively. Age was non-significant in the model and accounted for approximately six-one thousandths of the variability in the measure. The results also suggest a familial effect for JH. The model fitting results indicate that dropping either the additive genetic influences or the common environmental influences individually, failed to significantly alter the model's fit. However, dropping both parameters simultaneously significantly reduced the fit of the model.

Previous research on genetic and environmental influences on coping style have noted significant genetic influences on coping (Mellins, et al., 1996; Kendler et al., 2001). For example, Mellins et al., (1996) noted that problem-focused coping, which is somewhat similar to JH in conception, had an estimated heritability of .57. However, Kendler, et al., noted that heritability estimates, as well as the sources of variability,

differed depending on the measure of coping. For instance, denial showed a significant common environmental influence (19%), while "problem solving" showed a significant genetic influence (30%). The authors interpreted these results as suggesting that shared environmental influences, consistent with social learning in childhood are predominant in some forms of coping (Kendler, et al, 2001). The results for JH indicate some combination of genetic influences and shared environmental effects exert significant influence on this variable. This familial effect suggests there are heritable and learned factors at work in determining one's propensity to cope actively with environmental challenges. These findings are consistent with James' (1994) conception of JH as including a trait like propensity for active coping. However, the findings suggest there is a learned component as well.

The results also show that unique environmental factors are quite substantial in determining variability in JH. This suggests that factors related to SES, occupation, education, etc. may elicit active coping, or partially determine the extent to which the propensity to cope actively is fulfilled. For example, increased efforts to overcome occupational demands, improve individual socio-economic status, or alter unremitting social stress related to racism, may result in state-like increases in JH active coping. This notion is supported by recent literature that shows for African Americans, increased job demands are associated with significant blunting of waking cortisol response compared to Caucasians (Bennet, Merritt, Edwards, Sollers, & Williams, 2002).

The findings partially support Hypothesis 1 for JH. The findings indicate that some combination of genetic and unique environmental influences is significant in determining variability in JH. However, the findings do not support Hypothesis 2 for JH.

The model fitting results indicate that age does not significantly contribute to the variability of JH.

Multivariate Analyses: Cholesky Model

The Cholesky decomposition model was applied in the multivariate analyses. This model was employed to answer the following Research Question:

3) Are there shared genetic and environmental influences that account for the covariance among measures of blood pressure, perceived stress, and active coping for African Americans?

The Cholesky decomposition model was also employed to test the following Hypotheses:

3) There will be common latent factors that account for covariance in the measures of blood pressure, perceived stress, and John Henryism at the genetic level.

4) There will be a significant environmental correlation between blood pressure and perceived stress.

Taken together, the results of the Cholesky model also indicate that a familial effect accounts for the observed covariance among blood pressure measures, stress, and coping in the present sample. In other words, the covariance is the result of some combination of shared additive genetic, and common and unique environmental influences, although the results of the model fitting indicate the shared genetic components, in total approached significance. Dropping all genetic parameter estimates from the model resulted in a 15.48 unit change in chi-square, but the critical value is 18.31. Therefore, the results may be attributable to insufficient power as a result of sample size.

The findings suggest that family transmission is significant in determining the covariance of blood pressure and psychosocial factors, and reflect a synthesis of previous findings. For instance, the work of Katzmarzyk and colleagues (2000 & 2001) indicates that spouses show similarity in blood pressure values, which clearly represents a shared environmental effect. Similarly, common environmental influences have been shown to be significant for certain types of coping for Caucasian women (Kendler et al., 2001). The present study's results suggest a similar effect for the covariance of blood pressure and psychosocial factors in African Americans. This may be the result of a combination of heritable factors related to stress reactivity and inefficient stress and coping responses acquired through social learning.

It was surprising to note that the latent unique or common environmental correlations between the psychosocial factors and the blood pressure factors were non-significant. Previous research has noted the considerable impact of environmental factors on blood pressure values (Pickering, 1999; Sundquist, et al., 1999) as well as psychosocial stress (Dressler, 1996) and coping (James, 1994). Therefore, it was expected that latent unique or common environmental factors for blood pressure and the psychosocial measures would correlate. The phenotypic analyses show that these variables are related. However, the results of the biometrical modeling analyses indicate that neither shared unique, nor common environmental factors account directly for this relationship, although these data suggests an indirect pathway. For example, high levels of perceived stress, or ineffective coping related to environmental events may result in blood pressure changes only over time. This temporal component may be detected longitudinally, or by examining the data using specific age cohorts. The results

may also represent separate, unique environmental factors, occurring in temporal proximity but affecting blood pressure and the psychosocial factors separately. For example, high occupational demands and inadequate resource availability may occur during the same period, with each affecting blood pressure or stress separately. Therefore, the lack of a significant latent unique environmental correlation between blood pressure and psychosocial factors may reflect a lack of power to detect such a relationship due to sample size, rather than the absence of an association.

One of the most interesting findings was a significant shared unique environmental correlation between SBP and DBP. The correlation between the latent unique environmental factors is quite high (r=.74). This indicates environmental factors, unique to the individual are significant in the covariation between SBP and DBP in this population. These shared unique environmental factors are most likely related to specific stressors, not included in the present study, such as racism (Clark, 2001) or neighborhood level SES conditions (Pickering, 1999; Sundquist et al., 1999). These findings warrant further exploration of these powerful, and interrelated phenomena that affect cardiovascular health among African Americans.

The findings of the multivariate model analyses partially support Hypothesis 3. The results indicate that some combination of heritable and common environmental factors account for the covariance among blood pressure and psychosocial measures. The results of the multivariate analyses do not support Hypothesis 4. The findings show moderate latent factor correlations between the blood pressure and psychosocial factors, but these correlations failed to reach significance.

Summary

The results of the ACE-age regression analyses partially support **Hypothesis 1) There will be significant genetic influences on perceived stress, John Henryism, blood pressure measures and pulse pressure**. The results indicate that genetic influences are significant in determining variability in SBP, DBP, pulse pressure, and perceived stress. However, these results do not support significant additive genetic influence for JH.

The findings of the ACE-Age regression analyses partially support **Hypothesis 2) Age will be a significant component of the variance for measures of systolic and diastolic blood pressure, pulse pressure, perceived stress, and John Henryism.** The results indicate that age significantly contributed to the variability of SBP, Pulse pressure, and perceived stress. However, for age the results do not support significant age contribution to variability in DBP, or JH. The analyses indicate that while age may be significant for SBP, pulse pressure, and perceived stress it is not necessarily substantial, and ranged form 4% to 16% of the variability in the measures of interest.

The results of the Cholesky decomposition analyses partially support **Hypothesis 3) There will be common latent factors that account for covariance in the measures of blood pressure, perceived stress, and John Henryism at the genetic level.** The findings show that shared latent additive genetic and common environmental influences, in combination are significant in the model. The results suggest a familial effect accounts for covariance of psychosocial factors and blood pressure measures in the sample. This is an interesting finding and represents a

unique contribution to our understanding of the relationship between psychosocial factors and blood pressure among African Americans.

The Cholesky decomposition analyses failed to support **Hypothesis 4) There will be a significant latent environmental correlations between blood pressure and perceived stress**. Testing the parameter estimates for the cross-factor loadings of the psychosocial variables on the blood pressure factors did not result in a significant change in the model fit. As a result, the correlations calculated from these parameter estimates are not significant. However, it is interesting to note that exploratory analyses show a significant correlation between unique environmental influences for SBP and DBP.

Limitations

The primary limitation of this study is the sample size. The current sample represents (to the best of the author's knowledge) the largest study of adult African American twins to date. However, the estimates of shared influences and the calculation of latent factor correlations would be improved with a larger sample size. A larger sample size would, provide more variability, strengthen correlation estimates, and reduce error estimates. Moreover, the Cholesky model required the estimation of a large number of parameters. A larger sample would provide more stable parameter estimates for this model. In addition, there was a familial effect for several of the variables of interest including pulse pressure, perceived stress, and JH. There was a similar effect for shared influences among blood pressure and the psychosocial measures. A larger sample size may allow further decomposition of this effect and produce more definitive estimates of additive genetic and common environmental

influences.

Another limitation of this study is the number of stress and coping measures employed. The present study included one measure for each construct. The PSS is a 14 item scale and provides a global measure of stress. However, more specific measures of stress, such as occupational stress, job demands, or family strain may provide additional information not included in a global measure of stress. Similarly, JH was the only coping measure included. Other measures of coping style, such as emotion-focused or problem- focused coping may provide more information on coping and health related outcomes in this population.

The present research may be improved by inclusion of measures of perceived racism, which is a significant stressor for the population of interest. For African Americans, race and racism profoundly shape health and the aging experience. The inclusion of perceived racism may provide additional information about conditions, environments, and experiences that impact health in this population.

Conclusions

This study represents an important extension of research designed to understand individual differences in hemodynamic indices, stress, and coping using the classical twin approach. Moreover, the present study improves and expands our understanding of the relationship among hemodynamic indices, stress, and coping for African American adults.

The study's findings indicate that for African Americans heritable factors and familial influences are important in explaining variability in stress, coping, and hemodynamic indices, separately and in relations to one another, among African

Americans. These data support the notion of blood pressure regulation as a complex trait, determined by multiple genetic and environmental factors. The present work also adds considerably to our understanding of stress and active coping among African Americans. The results indicate these constructs also represent complex traits in this population, with heritable as well as learned components.

The findings indicate that understanding hemodynamics, stress, and coping in this population requires an integrative approach that accounts for psychosocial, behavioral, and heritable factors. This has implications for ameliorating CV health disparities. This study indicates that addressing the disproportionate burden of hypertension, and concomitant cardiovascular disease among African Americans will require addressing multiple causal pathways that include biological, psychological, and sociological components. Finally, the results indicate that age and aging are important to consider in understanding variability in hemodynamic indices as well as stress among African Americans. Age may reflect societal and institutional phenomena such as race and its meaning, that have direct and indirect effects on health indices as well as behaviors that may contribute to ethnic minority health disparities.

Future Directions

Future directions of this line of research will focus on environmental decomposition. This will involve an examination of the role of specific environmental factors that influence hemodynamics and hypertensive status in this population. This research will focus on socioeconomic factors for the individual and the immediate context, such as one's neighborhood. For instance, the CAATSA data-set includes a number of self-reported (i.e. subjective) neighborhood characteristics such as safety,

crowding, noise, educational composition, and adequacy of financial resources. This information will be combined with objective information such as census data and urban/rural influence information. This combination of factors will then be used to test mediational models of the effect of perceived stress on blood pressure and hypertensive status in the sample.

There is evidence that biochemical mechanisms linking DNA variation in the RAAS with hypertension. The focus of much of this work has been on angiotensin I converting enzyme (ACE) and variants of angiotensinogen (AGT) genes. Given the present findings, these genes represent excellent starting points for investigations of genetic influences on blood pressure and hypertension in African Americans in future studies with the same sample.

Given the considerable variability in age in the sample and the study findings, there is impetus to further explore the relationship among the components of variance in future research. For example, future work will address changes in heritability in hemodynamic factors as a function of age in this group. There may be age-related "critical periods" at which genetic and environmental mechanisms culminate to produce important increases in age-related variability in blood pressure. This suggests that there may be important sources of genetic and environmental variance correlated with age that were not included in the present analysis. This possibility will be further explored in future studies.

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Figure 1: Example of the ACE-Age Regression Structural Equation Model

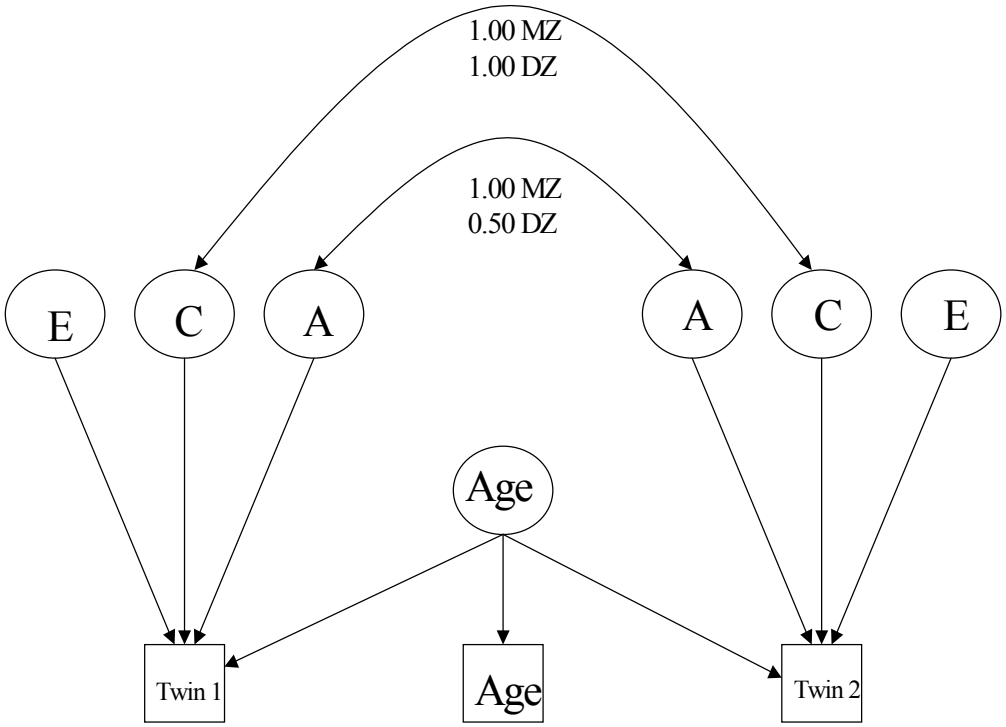
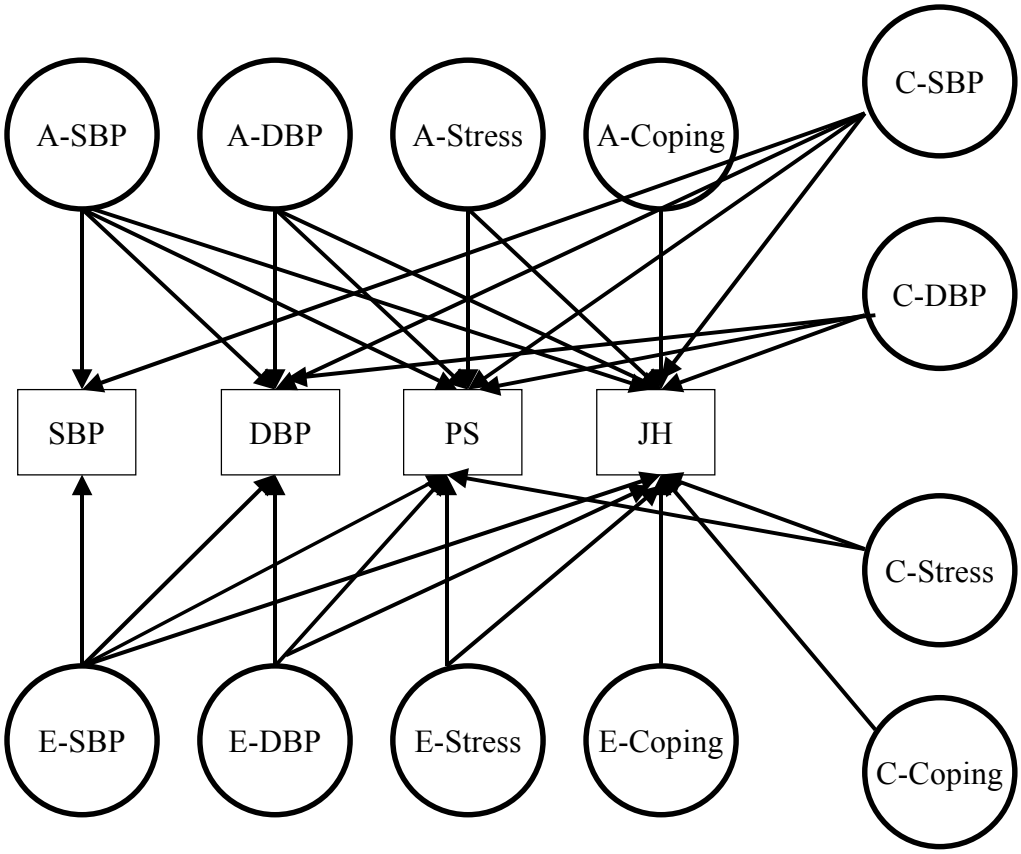


Figure 2: Example of the Cholesky Decomposition Structural Equation Model



Appendix: Instruments

Perceived Stress

The questions in this scale ask you about your feelings and thoughts during the **last month**. In each case, you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.
For each question choose from the following alternatives:

Never almost never sometimes fairly often very often

1. In the last month, how often have you been upset because of something that happened unexpectedly?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

2. In the last month, how often have you felt that you were unable to control the important things in your life?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

3. In the last month, how often have you felt nervous and "stressed"?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

4. In the last month, how often have you dealt successfully with irritating life hassles?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

6. In the last month, how often have you felt confident about your life ability to handle your personal problems?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

7. In the last month, how often have you felt that things were going your way?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

8. In the last month, how often have you found that you could not cope with all the things that you had to do?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

9. In the last month, how often have you been able to control irritations in your life?
( )never ( )almost never ( )sometimes ( )fairly often ( )very often

10. In the last month, how often have you felt that you were on top of things?

( )never ( )almost never ( )sometimes ( )fairly often ( )very often

11. In the last month, how often have you been angered because of things that happened that were outside of your control?

( )never ( )almost never ( )sometimes ( )fairly often ( )very often

12. In the last month, how often have you found yourself thinking about things that you have to accomplish?

( )never ( )almost never ( )sometimes ( )fairly often ( )very often

13. In the last month, how often have you been able to control the way you spend your time?

( )never ( )almost never ( )sometimes ( )fairly often ( )very often

14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

( )never ( )almost never ( )sometimes ( )fairly often ( )very often

John Henryism Scale for Active Coping

Personal Style: The following questions concern how you see your self, today as a person living and doing things in the real world. Each person is different so there is no "right" or "wrong" answers. Simply give us an honest appraisal of how you generally see yourself.

1) I've always felt that I could make of my life pretty much what I wanted to make of it

Completely True Somewhat True Somewhat False Completely False DK

2) Once I make up my mind to do something, I stay with it until the job is completely done.

Completely True Somewhat True Somewhat False Completely False DK

3) I don't let my personal feelings get in the way of getting a job done.

Completely True Somewhat True Somewhat False Completely False DK

4) It's important for me to be able to do things in the way I want to do them rather than in the way other people want me to do them.

Completely True Somewhat True Somewhat False Completely False DK

5) Sometimes I feel that if anything is going to be done right, I have to do it myself.

Completely True Somewhat True Somewhat False Completely False DK

6) I like doing things that other people thought could not be done.

Completely True Somewhat True Somewhat False Completely False DK

7) I feel that I am the kind of person who stands up for what he/she believes in, <u>regardless of the consequences</u>.

Completely True Somewhat True Somewhat False Completely False DK

8) Hard work has really helped me to get ahead in life.

Completely True Somewhat True Somewhat False Completely False DK

9) When things don't go the way I want them to, that just makes me work even harder.

Completely True Somewhat True Somewhat False Completely False DK

10) It's not always easy, but I manage to find a way to do the things I really need to get done.

Completely True Somewhat True Somewhat False Completely False DK

11) Very seldom have I been disappointed by the results of my hard work.

Completely True Somewhat True Somewhat False Completely False DK

12) In the past, even when things got really tough, I never lost sight of my goals.

Completely True Somewhat True Somewhat False Completely False DK

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