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# PSYCHOSOCIAL RISK FACTORS FOR ESSENTIAL HYPERTENSION AMONG BLACK WOMEN

by

Stephanie L. Livingston

A Dissertation Submitted to the Faculty of the Graduate School

of Loyola University of Chicago in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

December

1989

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# VITA

The author, Stephanie Lennore Livingston, is the daughter of Horace and Osceen Livingston. She was born September 2, 1952 in Decatur, Illinois.

Her elementary and secondary education was completed in Decatur, Illinois. She graduated from Stephen Decatur High School.

She received a Bachelor of Arts Degree in Administration of Justice from Southern Illinois University, Carbondale, Illinois in May, 1975. In May, 1982 she completed the requirements for a Master of Arts degree in Psychology from Sangamon State University, Springfield, Illinois.

Ms. Livingston was awarded the Illinois Minority Graduate Incentive Program Fellowship, while attending Loyola University of Chicago. She completed a one year pre-doctoral, clinical psychology internship in Behavioral Medicine at The University of Chicago Hospitals beginning July, 1988. She currently holds a clinical appointment in the Department of Psychiatry--Behavioral Medicine Section and serves on the faculty of The Pritzker School of Medicine at The University of Chicago and affiliated hospitals.

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# CHAPTER I

## INTRODUCTION TO THE PROBLEM

The field of behavioral medicine, including health psychology, has grown substantially in recent years. However, very little research on psychological factors affecting physical health and illness (e.g., stress, cardiovascular disorders, health damaging behaviors, compliance, prevention and psychoneuroimmunology) has been focused on racial and ethnic minorities. This is disturbing in view of the fact that in this society, racial and ethnic minorities have higher incidence and prevalence rates for a large number of diseases and physical illness (Stone, Weiss, Matarazzo, Miller, Rodin, Follick, and Singer, 1987). Essential hypertension is one of the more widely studied disorders that appears to have racial delineations. However, despite the alleged racial differences in hypertension rates, the research has not clearly demonstrated that the variation is along racial lines.

In addition, some research indicates that there are sociocultural and socioeconomic differences in behavior and reactions to illness, both among and within racial and ethnic groups, that have implications for physical health and illness. Since the preponderance of medical literature has focused on differential biological mechanisms between Blacks and Whites, while rendering inconclusive evidence to support a biological hypothesis, little emphasis has been placed on psychological and socioecological factors that might play essential roles in the development and maintenance of this disorder.

## ETIOLOGY OF ESSENTIAL HYPERTENSION

Essential hypertension, or primary hypertension, is defined slightly differently by various researchers. A common definition is sustained blood pressure that cannot be attributed to any particular organic cause and that exceeds 140 mm Hg systolic and 90 mm Hg diastolic. Approximately 85 percent of all hypertension cases fit this category. The

other 15 percent fall into the category of secondary hypertension which is caused by disorders such as ateriosclerosis, kidney disease, and adrenal hypersecretion. Both primary and secondary hypertension are the result of a constriction of the blood vessel walls, although via different mechanisms. Secondary hypertension is usually attributed to biolgical processes alone while primary hypertension often has psychosocial precursors as well as physical ones. Evidence strongly suggests that essential hypertension, the focus of this study, is the product of multiple interactingmechanisms rather than from a single source (Anderson, 1988; Anderson & Jackson, 1987). The ambiguity of the exact pathophysiology of the disease has presented problems in terms of understanding possible racial differences in the development of hypertension and differential responsivity to pharmacologic intervention. Hypotheses that have been generated to explain this disorder have included biological (Grim, Luft, Miller, Meneely, Battarbee, Hames & Dahl, 1980; Hastrup, Light, & Obrist, 1982), psychological (James, 1983, 1984), and sociological (Harburg, 1973; James, 1984, 1987) aspects, since each of these domains appears to be contributory.

One major hypothesis regarding the course of hypertension in biobehavioral research is that essential hypertension is mediated by the sympathetic nervous system (SNS) branch of the autonomic nervous system (ANS). This research (Henry & Cassel, 1969) has focused on short-term increases in SNS-mediated cardiovascular activity during behavioral or environmental stressors in the development of cardiovascular disorders, including essential hypertension. Animal research (Henry & Cassel, 1969) has also focused on the various aspects of the impact of the fight/flight response (beta-adrenergically mediated sympathetic outflow). This area of investigation has revealed that the fight/flight response that produces increased heart rate (HR), blood pressure (BP), catecholomines and renin release, and muscle blood flow precedes sustained high blood pressure. However, the underlying assumption that a beta-adrenergic response mechanism

necessarily mediates the development of essential hypertension in Blacks has been called in to question by investigations that fail to support this hypothesis (Anderson, 1989). Specifically, the correlates of beta-adrenergic response mechanisms, such as plasma renin levels, and cardiovascular activity (i.e., heart rate) have not been found as would expected in a beta adrenergic response in Blacks. Blacks tend to have lower plasma renin levels and heart rates than whites, rather than higher (Anderson, 1989). As a result, some researchers are investigating the possibility of an alpha-adrenergic response mechanism in Blacks that might underlie the development of essential hypertension.

## INTER-RACIAL DIFFERENCES

Essential hypertension is a problem that affects 18 percent of adults between 25 and 74 years of age. The incidence rate for Blacks, however, is twice as high compared to whites for both males and females (Roberts & Rowland, 1981). Furthermore, between the ages of 33 and 54, Blacks are ten times more likely than Whites to suffer from the hypertensive vascular diseases (National Center for Health Statistics, 1984). As a result, Blacks experience correspondingly high rates of hypertension-related morbidity and mortality from coronary heart disease (Myers, 1984), stroke (Hypertension Detection & Follow-up Program Cooperative Group, 1977), renal disease, and renal failure (Rotstand, Kirk, Rutsky, & Pate, 1982). Consequently, hypertension is often considered the number one health problem among Blacks (Saunders & Williams, 1975). Despite the scientific evidence that indicates greater vulnerability to cardiovascular disease in Blacks, research efforts continue to neglect this understudied and clinically significant population.

Some of the major factors that have been hypothesized to explain racial group differences in prevalence of essential hypertension are: 1) biological (i.e., sodium retention, plasma renin levels), 2) genetic predisposition (i.e., family history of essential hypertension) and 3) differential cardiovascular reactivity to stressors (i.e., physical and

psychosocial).

## Biological Mechanisms

There have been several studies that have investigated biological explanations of racial differences in hypertension. Some of the main areas of concern have been sodium retention, plasma renin levels, heart rate, and beta versus alpha-adrenergic response mechanisms.

### Sodium Rentention

One biological factor that has shown some promise in assessing racial/ethnic differences in the development of essential hypertension is sodium retention. Luft, Grim, & Weinberger (1985) and his colleagues, in a sodium loading study, found that black subjects excreted less sodium in urine and subsequently exhibited greater blood pressure increases than their white counterparts (Barnett, Biener, & Baruch, 1987). Consequently, they concluded that there could be a heritability factor between racial groups that might predispose Blacks to developing hypertension.

Sodium excretion has also been shown to be inhibited by psychological stress in animals (Koepke, Light, Grignolo, & Obrist, 1983) and humans (Light, Koepke, Obrist, Grignolo, & Willis, 1983). To the degree that Blacks, particularly low-income Blacks, experience more psychological distress than do Whites or higher-income Blacks (Kessler & Neighbors, 1986), these lower-income Blacks may subsequently be more susceptible to inhibited sodium excretion that could augment reactivity. Furthermore, these findings further support the notion that essential hypertension cannot be viewed solely as a biological phenomenon without consideration of social factors.

Sympathetic Nervous System (SNS) Hypothesis

Most of the hypertension research has focused on the role of the sympathetic branch of the ANS. The role of the SNS has been evaluated primarily through the measurement of plasma norepinephrine (NE) which is released via sympathetic nerve endings and from the adrenal medulla. The action of NE results in a vasoconstriction of the blood vessels creating greater resistance against circulation, resulting in increased blood pressure. Studies have demonstrated however, that Blacks do not have higher resting SNS activity levels than do Whites (Jones, Hamilton, & Reid, 1978; Rowlands, Giovanni, McLeary, Watson, Stotland, & Littler, 1982).

Studies that have investigated black/white differences have also failed to provide support for the hypothesis that Blacks might have higher resting SNS activity as measured by plasma renin levels which aids in the production of angiotensin II--a vasoconstrictor that increases blood pressure. In fact, it has been demonstrated that 36 to 62 percent of black hypertensives have suppressed renin levels as compared to 19 to 55 percent of white hypertensives (Vick, 1984). The Hypertension Detection and Follow-up Program (HDFP, 1977) provided further evidence for the lack of racial differences. This study revealed that blood pressure differences between black and white females disappear when education and obesity are controlled for. Since research has failed to adequately demonstrate that systematic variation in susceptibility to hypertension falls along racial lines, it seems appropriate to focus on intra-racial individual differences rather than inter-racial differences.

Parental History of Hypertension

One of the more consistent findings in the medical literature on hypertension is that parental history of hypertension is a strong predictor of hypertension in the offspring. However, it should be noted that this research has been based upon the exaggerated

cardiovascular reactivity hypothesis which may not be applicable as an underlying mechanism in Blacks.

Hastrup, Light, & Obrist (1982) investigated the relationship between parental hypertension and the heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) levels of 103 healthy college-age men (98 Whites and 5 Blacks), during two resting conditions, a stressful cold pressor, and a reaction time task. The findings revealed that sons of hypertensive parents showed higher HR and SBP than sons of normotensive parents during both rest and stress, but these differences were greatest during the stressful reaction time task. Since the incidence of high blood pressure is known to be greater among the offspring of hypertensive parents, these findings suggest that cardiovascular responses to certain types of stress (e.g., active coping tasks) may help to predict future risk of hypertension.

Anderson, Lane, Taguchi, and Williams (in press) examined cardiovascular responses of black and white normotensive women selected for parental history of hypertension, in response to two stressors: mental arithmetic and the cold face stimulus. Racial differences were found in diastolic blood pressure recovery from the mental arithmetic task; black women having a slower recovery rate. Black women also showed a greater systolic blood pressure response to the cold face stimulus. Furthermore, black women demonstrated increases in emotional responses (i.e., anxiety, guilt, fear, restlessness) to the math task. In a similar study, Anderson, Lane, and Taguchi (1988) found similar trends among black men although race nor parental hypertension was significantly related to cardiovascular responses to either of the two stressors. Black men exhibited slower diastolic blood pressure recovery following arithmetic and had significantly higher systolic and diastolic blood pressure levels before, during, and after the cold stimulus than white men.

These findings suggest that individuals with hypertensive parents are more

cardiovascular hyperreactive and that Blacks are more reactive than Whites. Although black men did not exhibit racial differences in reactivity to the same extent as black women, they too showed racial differences. This evidence helps to establish the importance of genetic predisposition in the development of essential hypertension.

Cardiovascular Reactivity

Racial differences in stress-induced cardiovascular reactivity and hypertension has been another focus of concern when assessing inter-racial differences. According to Anderson (1989) the data thus far on racial differences in response to psychosocial stressors among hypertensive individuals have not clearly dmonstrated a propensity toward hyperactivity in Blacks in relation to Whites. In fact, in some cases Blacks have been shown to have lower cardiovascular responses than white hypertensives, especially in HR (Fredrikson, 1986). These findings suggest that beta-adrenergic (i.e., SNS) influences may not underlie hypertension mechanisms in Blacks.

Light, Sherwood, Obrist, James, Strogatz, & Willis (1986) compared cardiovascular and renal responses to stress in black and white normotensive and borderline hypertensive men and found that stress produced a significantly larger decrease in total peripheral resistance in Whites than in Blacks. This finding suggests a different pathophysiology of essential hypertension than was originally proposed. These findings suggest an alpha-adrenergic response mechanism in Blacks in contrast to a beta-adrenergic response that is typically implicated in SNS activation. This is further supported by the superior effect of Labetolol (alpha/beta blocker) over Propanolol (beta blocker) in reducing blood pressure in black hypertensives (Flamenbaum, Weber, McMahon, Materson, Albert, & Poland, 1985).

Anderson (1989), reviewed research that examined racial differences in resting and stress induced cardiovascular reactivity and concluded that most studies in this area have

focused on inter-racial differences rather than intra-racial differences. Based on Anderson's review, the evidence is contradictory and inconclusive regarding racial differences. He found that although the research data on adult samples are inconclusive, findings to date are suggestive of a possbile decreased cardiac reactivity (i.e., heart rate, cardiac output) and increased peripheral vascular reactivity among Blacks compared to Whites. Methodological considerations are also of importance (e.g., experimenter race, genetic predisposition, laboratory environment) when interpreting data on inter-racial differences. Based on these findings, Anderson suggests that future research consider the possiblity of different mechanisms underlying hypertension in Blacks and Whites. Specifically, less emphasis should be placed on the SNS (beta-adrenergic response) as the primary source of this disorder; and more often should be directed toward alpha-adrenergic responses in Blacks for explanation. Lastly, Anderson provides evidence to support the notion that intra-racial differences, that consider variablity among Blacks, is a more appropriate avenue to pursue in the study of risk factors for essential hypertension.

## Psychosocial Mechanisms

### Socio-ecological Factors

Harburg, Erfurt, Chape, Hauenstein, Schull, and Schork (1973) conducted one of the more important studies that has investigated socioecological variables and their impact on hypertension by studying the effects of environmental differences between black and white urban populations in relation to blood pressure. The major hypothesis, that blood pressure levels will vary with extremes of stressor conditions in socioecological areas was partially supported. The findings revealed that black males and females residing in high stress areas had significantly higher blood pressures than individuals from low stress areas. These findings also have implications in support of the findings that propose that

hypertension is related to sodium excretion efficiency as a result of psychological distress . Blacks only make up 12 percent of the U.S. population yet comprise 30 percent of the population below the poverty line and one third of those considered to fall in the low-income bracket. Given that Blacks tend to live more often in high stress areas due to poverty and are likely to experience more psychological distress, resulting in sodium excretion inhibition, environmental factors may appear to play a major role in the mediation of essential hypertension.

## INTRA-RACIAL DIFFERENCES

### Gender Differences

Recent research suggests that there are differences in mortality and morbidity rates between men and women (Stone et al., 1987), but the reason for this differential is still unclear. What is known is that women tend to have lower mortality rates pre-menopause but higher morbidity rates because they generally live longer than men. In addition to biological explanations to account for these differences, many psychological and behavioral variables have been proposed as potential influencing factors in illness and mortality. Because of the disparity between morbidity and mortality rates between the sexes that is left unexplained, their is a need to further investigate these differences. An increased focus on women's health issues in particular is important since a disproportionate amount of research dollars is allocated to the study of predominantly male disorders (i.e., heart disease, respiratory disease, AIDS). Even though heart disease is a major concern of both men and women, it is unclear why women respond differentially to prevention efforts. Consequently, research needs to be taken a step further in this area, not only to address disorders that afflict women specifically, but also to provide understanding about differential responsivity to prevention and treatment efforts as well as gender specific risk

factors of disorders common to both sexes.

## Psychosocial Factors

### John Henryism

The research investigating psychosocial markers of essential hypertension in Blacks has been minimal. Moreover, studies that have investigated psychological variables have primarily focused on black men. Three studies conducted by Sherman James on John Henryism as a predictor of hypertension, provided evidence for James' theory that personality characteristics (e.g., personal competence and environmental mastery characterized by efficacious mental and physical vigor, commitment to hard work, and a single-minded determination to achieve one's goals) in interaction with an individual's coping resources (i.e., education, income) have mediating effects on blood pressure among Blacks. Furthermore, James proposed that John Henryism is indicative of an active coping style that is characterized by coping with environmental stressors via behavioral responses, and has been shown in the psychophysiologic literature to lead to sustained elevations in blood pressure (Obrist, 1981).

Despite the fact that some black women were included in James' three major studies on John Henryism, all 836 females were part of only one of these studies; 50 percent of this total were white females. Consequently, only 23 percent of the total subjects in these three studies were black women (James, 1983, 1984, 1987). One of the purposes of this study will be to provide additional information on the construct validity of the John Henryism Active Coping Scale, a scale developed by James to measure Johns Henryism with black urban women as opposed to rural black men on which the scale was standardized.

Type A Behavior Pattern

Another personality characteristic that has been well documented in the literature regarding the relationship to coronary heart disease (CHD) is the Type A Behavior Pattern (TABP). Research investigating this pattern has been largely restricted to white males. Studies such as the Western Collaborative Group Study (Matthews, Glass, Rosenman, & Bortner, 1977) and the Framingham Study (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978) are both large scale prospective research endeavors that have focused primarily on the incidence of CHD, as predicted by TABP and other psychological factors (e.g., anger, suppressed hostility) among white males. Booth-Kewley & Friedman (1987) conducted a meta-analysis to integrate and organize the results of studies that investigated certain personality variables in relation to CHD. The personality variables included were anger, hostility, aggression, depression, extroversion, anxiety, TABP, and the major components of TABP. The results indicate that modest but reliable associations exist between some of the personality variables and CHD. These findings suggest that these emotions (i.e. anger, hostility, depression, etc.) , rather than the hurried, impatient, workaholic profile that had previously been proposed in earlier research, are the strongest predictors. Since no real effort has been made to test the TABP hypothesis on Blacks, it is unclear whether or not the same conclusions can be applied to Blacks.

Social Mobility

Social mobility is another concept that has been investigated in an attempt to identify psychosocial risk factors that may contribute to cardiovascular disorders. Whether it is cultural, occupational, or geographical mobility, the common denominator in susceptibility to subsequent development of CHD appears to be the necessity to adapt to an unfamiliar environment. Oftentimes this adaptation results in a physiological change over

time.

In a study on intergenrational mobility, Gillum and Paffenbarger (1978) investigated the impact of intergenerational mobility on hypertension, myocardial infarction, and angina pectoris in Harvard graduates. They found that among white males, occupational status of the offspring's father was inversely related to subsequent incidence of CHD and MI but not to hypertension. Since it is unclear if Blacks were included in this study, (although it can assumed that there were few black Harvard graduates at the initiation of the study) it is unclear if this inverse relationship would hold true for this group or if intergenerational mobility might be predictive of hypertension in Blacks, even though this relationship was not established for whites.

In summary, biological explanations of the etiology of essential hypertension have superceded other explanations in the medical literature. However, the findings of these various studies have been inconsistent and inconclusive in establishing a specific biological etiology that is linked to this disorder. It has been even more difficult to establish racial differences in the development of hypertension. However, some research has produced evidence to suggest that there are different underlying mechanisms of the disorder for Blacks and Whites--an alpha-adrenergic response in Blacks and a beta-adrenergic (SNS) response in Whites. Consequently, the literature on the primacy of the SNS in essential hypertension must be viewed with suspicion when applying these same principles to Blacks. Furthermore, some of the other biological explanations of this disorder can also be explained psychosocially (i.e., sodium retention hypothesis). As a result, a multifactorial approach that considers biological, psychological, and socioecological factors will be utilized in this investigation to more adequately address the complexity of the disorder as it pertains to urban adult black women.

## PURPOSE OF THIS STUDY

The purpose of this study is to continue the line of research that has demonstrated the importance of intra-racial differences among Blacks rather than inter-racial differences between Blacks and Whites when attempting to explain the etiology and development of essential hypertension. The direction of this investigation is timely given the failure of medical research to conclusively establish a biological, and therefore racial differential, to explain the disparity of incidence and prevalence of hypertension between Blacks and Whites.

Even if racial differences could conclusively be established, this aspect alone is only one dimension of a multidimensional phenomenon. Much evidence has been provided to strongly suggest that psychological and sociological factors play a major role in this disorder. In view of this evidence, this study will attempt to identify psychosocial risk factors that might interact with biological and other standard risk factors (i.e., age, diet, obesity, family history of hypertension, smoking, excercise) to contribute to the development of essential hypertension in a sample of black urban women.

Another aspect of this study will be to investigate the impact of psychosocial risk factors on black women in particular. Since black women have been understudied in this area, it will be interesting to note if the same predictors of essential hypertension in black men (James, 1984), are also predictors of essential hypertension in black women in an urban setting.

Questions that will be discussed in addressing these issues are: 1) How do personality characteristics relate to blood pressure? 2) Do current measures of psychological constructs (i.e., John Henryism Active Coping Scale to measure John Henryism have external validity to urban black women? 3) Do environmental factors, specifically social mobility, relate to blood pressure? 4) What mediating effects, if any, do other variables

such as age, family history of essential hypertension, smoking, medication usage, and education have on blood pressure levels? 5) What other factors might be unique to black women in an urban setting that are related to blood pressure levels, but have not yet been considered? and 6) Based on light shed on the foregoing, what direction should future research and clinical applications take.

## Chapter II

## LITERATURE REVIEW

Research on the etiology of essential hypertension has provided converging evidence that the disorder is not a unidimensional phenomenon. Strong empirical evidence points to a more comprehensive, multifactorial means of understanding this disorder, including biological, psychological, and socio-ecological factors. In addition, not only is the etiology unclear, but the exact pathophysiology that underlies the disorder is also ambiguous. Contradictory hypotheses have been proposed that fall along racial lines. However, categorization by race has inherent problems of its own, when considering the views of some that race is a socio-economic concept rather than a biological one. The questionable nature of race as a legitimate categorization principle, coupled with the inconsistent findings in the medical, psychological, and sociologic literature when looking at between group racial differences, has guided research toward investigating intra-racial differences as a potential source of variability.

## THE CONCEPT OF RACE

Ferreting out the contribution of race is made more difficult by the neglect on the part of researchers to cite the racial composition of their studies, especially in the medical literature. According to Svensson (1989), because black people are under-represented in studies of new medications, researchers may miss racial variations in the effects of certain drugs. In a meta analysis of 50 published studies on the efficacy and safety of new drugs, Svensson found that in 15 studies the researchers failed to indicate racial composition and in 20 studies where it was listed, Blacks were under-represented. More specifically he states,

> Even in clinical trials that involved antihypertensive agents, where racial differences havebeen clearly demonstrated, only about 50 percent of investigators reported racial data. This indicates a lack of consideration on the part of most investigators of the potential contributionof race to variability in drug response (p. 263). In trying to understand the neglect of Blacks, where the risk of hypertension is disproportionately high compared to other racial groups, yet the research is disporoportionately low, Bristow (1989,) responds to these findings by stating, "Racism wasn't killed by the civil rights struggle of 20 years ago. Wounded, it retreated to more subtle expressions from its most deeply entrenched bunker--the arena of economics (p. 284)."

This line of reasoning has been supported by others in the field who uphold the notion that economics plays a major role in the concept of race and its subsequent ramifications in epidemiological research. Cooper (1984) indicates that the concept of race has served an economic function in the U.S. since the advent of slavery. He states,

> Use of the category of race in epidemiological research presupposes scientific validity for a system that divides man into subspecies. Although the significance of race may be clear cut in many practical situations, an adequate theoretical construct based on biologic principles does not exist. Anthropologists have in large measure abandoned the biologic concept of race, and its persistent widespread use in epidemiology is a scientfic anachronism.The assumption that race designates important genetic factors in a population is in most casesfalse. Racial definitions should be seen as primarily social in origin and should be clues to environmental rather than genetic causes of disease. An understanding of the social forces leading to racial differentials in health will give further direction to preventive campaigns (p. 715).

Cooper further indicates that all humans, in terms of susceptibility to all diseases except those that are quite rare, are genetically similar. Thus far, systematic variation in susceptibility has not been adequately demonstrated to fall along racial lines for any common diseases. However, he qualifies this observation by indicating that hypertension "might" be considered the one exception to the proposition that racial differences in common diseases are social in origin. He further proposes that although it is assumed in the medical domain that Blacks are genetically predisposed to hypertension, an environmental hypothesis is equally tenable based upon hypotheses regarding sodium retention and plasma renin levels.

Cooper cites the Hypertension Detection and Follow-up Program (HDFP,1977) data as evidence. These findings revealed that blood pressure differences between black and white females disappeared when education and obesity were controlled for. It was further established that rates of hypertension for Blacks with a college education were similar to those for Whites who did not finish high school. This finding was explained by data suggesting that the earning capacity of black college graduates is almost identical to that of white high school graduates (HDFP, 1977). Consequently, if each of these variables could be more adequately measured and controlled for, the differences attributed to race might disappear altogether. With this in mind, exploring a non-genetic explanation of hypertension is needed. In reviewing the literature on biologic hypotheses of essential hypertension, these factors should be kept in mind.

# ETIOLOGY OF ESSENTIAL HYPERTENSION

## Biological Factors

### Sympathetic Nervous System (SNS)

The commonly held view of how essential hypertension is mediated is via the SNS. Sustained blood pressure elevations are thought to be preceded by fight/flight response mechanisms. Contrary to the parasympathetic division of the Autonomic Nervous System (ANS) which is a rest-response system, the SNS is concerned with the processes involving the expenditure of energy. When in homeostatis, the SNS is mainly concerned with counteracting the effects of the parasympathetic division in order to carry out normal processes that require energy. However, if the body is stressed in any way, the SNS takes over. Activation of the SNS triggers a fight/flight response that is characterized by 1) dilation of the pupils, 2) increased heart rate, 3) constriction of blood vessels of the skin and viscera, and 4) dilation of the remaining blood vessels. This results in a rise in blood pressure and a faster flow of blood into the dilated blood vessels of skeletal muscles, cardiac muscle, lungs, and brain--organs that are useful in protecting one from physical danger. Other effects include; rapid breathing, increase in blood sugar levels, and production of epinephrine and norepinephrine (NE) that prolong this response. The SNS is innervated by adrenergic fibers that produce the neurotransmitter NE, resulting in a beta-adrenergic response mechanism.

From an evolutionary standpoint, the fight/flight response provided an adaptive means of coping with potential danger in one's environment during prehistoric times. If a caveman determined that his survival was being threatened by a wild animal, the fight/flight response was useful in providing a means of either fighting or fleeing from the animal (i.e.,

increasing blood supply to the skeletal muscles for added strength). However today, human beings do not have to be concerned with being attacked by vicious animals; rather their stressors tend to be those that are not easily acted upon. Job, interpersonal relationship, and environmental factors are today's sources of stress that might elicit the fight or flight response, despite the fact that they are not directly life threatening compared to being attacked by vicious animals. However, unlike the scenario of the caveman who can resolve this response by fighting or fleeing the animal, the twentieth century human oftentimes has no recourse to either option; although the physiological response is the same (i.e., increased heart rate and blood pressure). Today's situations are ones that do not customarily provide a means of coping by physical retaliation (fight response) or escape (flight response). Consequently, people today might make the mistake of inappropriately labeling the situation as being "dangerous", and respond maladaptively with repeated and sustained activation of the SNS. This sustained activation over time can have a deleterious physiological effect on the body (Obrist, Black, Brener, & DiCara 1974).

It is not completely clear why archaic mechanisms such as the fight/flight, the immune system, and other internal regulatory systems have not evolved to more closely respond to current environmental and internal factors that compromise homeostatis. We do know, however, that the laws of natural selection by "survival of the fittest" do not always hold true in terms of defense reactions. Modern medicine has provided means of improving upon nature in this regard by developing the ability to suppress responses which were apparently developed for defense, but which are not necessarily useful under all circumstances. In today's society "fittest" does not always mean "strongest". Although neurochemistry provides a means of understanding the role of various substances in the maintenance of homeostatis, much is still unknown. For instance, substantially more is known about syntoxic substances (i.e., those that produce peaceful coexistance--

endorphins) than catatoxic substances (i.e., those with destructive properties--cortisol) within the organism. Until more is known about the brain's function, why our brains have not developed in accordance with our needs will continue to be a mystery.

General Adaptation Syndrome (GAS)

According to Selye (1974), the body responds similarly to various stressors, whether physical or psychological. He described the General Adaptation Syndrome (GAS) in 1936 in defense of his hypothesis that there is a uniform or non-specific way in which the body reacts to stressors. This process consists of three stages, 1) the alarm reaction; 2) the stage of resistance; and 3) the stage of exhaustion. One important factor of this model is that the body's adaptability is finite. He likens these stages to that of human developmental stages--childhood, adulthood, and senility whereby as one ages, the body's ability to resist stressors gradually declines resulting in inevitable death. In view of the fight/flight response, if an individual continues to elicit the "alarm reaction", the body's ability to resist will eventually result in exhaustion, which in turn results in illness and ultimately death. Selye further points out that it is emotional arousal that is characteristic of a stress response, not whether the stressor is aversive or pleasant. The identification of the GAS has implicatons for the understanding of the development of essential hypertension via the SNS. The fight/flight response is triggered by some stressor (alarm stage), the body repeatedly attempts to adapt to the sustained blood pressure elevations (adaptation phase), and eventually the body is unable to adapt to the repeated elevations and eventually exhausts its adaptive mechanisms and results in a disease process (e.g., essential hypertension, stroke). This traditional conceptualization of stress views it as a specific biological syndrome that is a response to nonspecific damaging agents (stressors). The response has a particular time-frame (the GAS), and its activation by one

stressor may have implications for the organisms capacity to resist other stressors. Although this is the commonly accepted view of stress, other explanations have been proposed. Mason (in Selye, 1974 p.18) challenges the idea that stress is a purely biological response. He suggests that a single biological response to a wide variety of stimuli is difficult to explain on a physiological basis.

Mason conducted a series of experiments that investigated the impact of psychological parameters surrounding the stressors on the general stress response. He found that certain aspects of the stressful situation (e.g. degree of discomfort, pleasantness of stressor, sudden versus gradual appearance of stressors) could account for the presence or absence of the biological stress response even if the actual stressors remain unchanged. In Mason's view, the stress concept should be viewed as a behavioral rather than a purely physiological one. Furthermore, the initial response an organism
makes to a stressor is first at the behavioral level and may have a subsequent physiological impact.

Specificity Theory

Another major challenge to Selye's GAS (nonspecificity theory) which implies a universal response to stressors is the concept of specificity theory, which proposes that different types of illnesses have different precursors. Psychosomatic medicine, which is concerned with the influence of psychological factors on illness and health, was based on the idea that specific mental factors (e.g., anger, dependency) are associated with specific physiological expressions (e.g., hypertension, asthma). The most classical formulation of emotional specificity was developed in 1959 by Franz Alexander (in Freedman, Kaplan, & Sadock, 1976) . He believed that if a specific stimulus or stress occurred, it was expressed in the specific response of a predetermined organ due to a constitutional

vulnerablity. Using the available physiology of his day, he developed a set of hypotheses, characterizing seven classical psychosomatic disorders; essential hypertension, bronchial asthma, neurodermatitis, peptic ulcer, ulcerative colitis, rheumatoid arthritis, and thyrotoxicosis. He viewed each of these disorders as a physiologic manifestiation of chronic dammed-up emotions that are the result of unresolved childhood conflicts. Real or anticipated life crises stirred up these fixated, unconscious conflicts, setting in motion both the person's immature psychological defenses and activation of the physiological responses that had been associated with these conflicts in childhood. He explained essential hypertension as the chronic partial emergence of aggressive tendencies never sufficiently repressed. The conflict over expression and concealment and a compromise formation in partial, distorted expression catergorizes many of these disorders as primitive forms of a conversion disorder described by Freud. Alexander viewed conflict as a stress and suggested that when conflict arises an individual might suppress this stress and produce through the voluntary nervous system, a conversion reaction. Or, after suppressing the stress, the individual might cope via the autonomic nervous system by keeping sympathetic responses alert for heightened aggression or flight by keeping parasympathetic responses alerted for heightened vegetative activity. This prolonged alertness and tension can produce physiological disorders and eventual pathology of the organs of the viscera.

Although empirical evidence is lacking to clearly substantiate a specificity theory, some research has supported the "weak link" theory that suggests that individuals who have historically experienced difficulties with one organ system tend to respond to stressors with signs and symptoms in that system. For example, (Wolf and Goodell, 1968) found that patients with vascular headache, cardiovascular problems, and duodenal ulcers showed a stress-related hyperactivity in those particular organs.

Parental History.

One important indicator of elevated risk for subsequent development of essential hypertension is the presence of the disorder in either or both parents. Hastrup, Light, & Obrist (1982) examined the relationship between parental hypertension and Heart Rate (HR), systolic (SBP), and diastolic (DBP) blood pressure levels of 103 healthy college-age men, during two resting conditions and stressful cold pressor and reaction time (RT) tasks. The purposes of these comparisons were: 1) to replicate the finding of an association between parental hypertension and high cardiovascular reactivity to an active coping task; 2) to determine whether parental hypertension is more strongly related to the cardiovascular responses to the active coping RT task (e.g., one that requires a behavioral response) than to responses to passive cold pressor test; and 3) to assess the differences between subjects with or without a hypertensive parent during "baseline" measurement periods.

Of the 103 subjects, who ranged in age from 18-27, 98 were white and 5 black. The parents of the subjects ranged in age from 38-71 years. A total of 34 of the 206 were classified as hypertensive using a self-report measure of physician diagnosis and/or use of antihypertensive medication. For all subjects, physiological measures (i.e., SBP, DBP, & HR) were monitored during the last three minutes of a five minute prestress waiting period and during a 14 minute RT task involving threat of shock. Forty-five of the 103 subjects formed a subsample who were also monitored during a cold pressor test. Fifty-eight of the 103 subjects were also administered the student version of the Jenkins Activity Survey. All subjects were additionally monitored during two 15 minute relaxation sessions on later days when subjects were told that they would not be exposed to any stressful events.

The results indicated that offspring of hypertensive parents had higher HR and SBP than offspring of normotensives under all conditions, but cardiovascular reactivity was

noticeably more pronounced at the onset of the unsignaled RT task. These findings confirm previous research conclusions that offspring of hypertensive parents reach higher HR and BP levels during stressful active coping tasks. In addition, this study suggests that high cardiovascular response (especially HR), during tasks with strong incentives for active coping may indicate an increased risk for development of hypertension. This study also supports the notion of cardiovascular hyperreactivity and the underlying mechanism of beta-adrenergic SNS activation.

Gender Differences

Some research indicates that there are marked gender differences in cardiovascular disorder incidence rates, despite the fact that coronary heart disease accounts for the greatest proportion of all deaths occurring in both men and women among the industrialized nations (Kannel, 1982). In addition, age-related increases in the incidence of essential hypertension are significantly delayed in women compared to men (Roberts & Rowland, 1981). Although premenopausal years tend to provide protection against the development of cardiovascular disorders in women this does not hold true postmenopause. It is still unclear why these gender differences exist even when traditional risk factors (e.g., serum lipid concentrations & cigarette smoking) are taken into account (Kannel, Hjortland, McNamara, & Gordon, 1976). One possibility is that men and women differ with respect to cardiovascular reactivity to specific stressors in addition to the influence of female reproductive hormones on psychophysiolgic reactivity.

Hormonal Influences. The body of literature on neuroendocrine reactions to stressors reveal that females show a less pronounced elevation in urinary excretion of epinephrine than do males. Frankenhaeuser, Dunne, and Lundberg (1976) found that when

young adult males and females were exposed to two experimental stimuli--repeated venipuncture and a frustrating cognitive task--only the males showed a significant rise in urinary epinephrine. Epinephrine has been found to be more reactive to psychological stressors (Dimsdale & Moss, 1980; Ward, Mefford, Parker, Chesney, Taylor, Keegan & Barchas, 1983). Catecholomines (i.e., epinephrine, norepinephrine) contribute to cardiovascular disorders through a number of mehcanisms including elicitation of the fight/flight response which in turn results in blood pressure elevation.

## Psychosocial Factors

Looking at the fear phenomenon (fight/flight response) from a psychological standpoint, it might be argued that the basic concept of fear has not changed over time, although the feared object has changed. Consequently, if the fight/flight response were to drop out altogether, humankind would be left unarmed in situations where this mechanism might prove beneficial. The resolution of the misutilization of this response seems to be in the area of cognitive processes of appraisal and labelling of a situation as dangerous. No doubt cavemen on occasion mislabeled a situation as dangerous thereby eliciting the fight/flight response in non-theatening situations.

### Syncrony/Desynchrony

Current research supports the notion of a multiple response system to fear, not just physiological (Eysenck, 1979; Lang, 1978; Rachman & Hodgson, 1974). Synchrony takes place when all response systems (i.e., physiological, behavioral, cognitive) vary together and desynchrony occurs when they do not vary together. Lang and Lazovik (1963), using automated desensitization of snake phobias, found that while some subjects

showed rapid change in overt behavior (e.g., decreased avoidance), they continued to label themselves as fearful. Other subjects showed a decrease in fear assessed by fear questionnaires, yet exhibited marked cardiovascular response. Based on this study and subsequent ones (Lang 1968, 1971, 1978) Lang concluded that different behavioral systems (e.g., cognitive/behavioral, physiological, and motoric) to some extent are capable of independent change through the shaping by environmental demands. As a result he developed the 'Three-Systems Model' of fear. The implications of this model, as well as Rachman's (1974) model of 'Synchrony and Desynchrony in Fear and Avoidance' , is that fear can no longer be conceptualized as a unitary phenomenon as would be suggested by non-specificity theory. In addition, individual differences also contribute to the subsequent differential reponses to fear as well as various emotions evoking specific responses. The burden of illness and disease must lie with modern man's failure to fully utilize the knowledge in psychology and medicine to educate individuals about the interacting role of biological, sociologic, and psychologic factors. This failure may be a more appropriate explanation for incongruence between environmental demands and physiologic response than to assume that the brain's evolutionary development has failed to provide adaptive means of coping.

Controllability and Predictability

The degree to which environmental stimulation is perceived to be predictable and controllable influences the extent to which it induces a stress response in the individual (Cohen, Glass, Phillips, 1979). More recently other factors have been demonstrated to have an impact on the stress response such as social situations, emotions, and coping abilities. Lazarus, Cohen, Folkman, Kanner, and Schaefer (1980) have proposed that the essential mediator of the stress response is psychological and that the cognitive appraisal of

threat is crucial to the elicitation of the response. What one person views as a fearful situation or negative experience may not be viewed in the same way by another individual. Furthermore, the success or failure of the coping process will determine whether the stress response will be relaxed or maintained. Coping abilities of an individual can be determined by the availability of resources such as finances, education, social support, control over the stressor, and current health status. How well an individual copes with a stressor is largely dependent upon how much control one has in the stressful situation and the types of coping strategies used.

In 1970 Weiss (1977) demonstrated that control and predictability of a stressor has physiological implications in laboratory experiments. Weiss yoked two rats together, neither one having control over the stressor (shock). Although neither rat knew when the shock would occur, one rat was given a warning signal before the shock and the other rat was not given a signal. The rats who were not warned of the oncoming shock developed six times the number of stomach lesions found in the rats who were warned. Consequently, Weiss concluded that predictability of an imminent stressor was a significant variable in illness sypmtoms associated with a stressor.

Two years later, Weiss (1977) examined the effects of a stressor when the subject could control the stressor onset. Similar to his first study, rats were yoked together, although this time one rat was able to avoid shock when a signal light came on by rotating a wheel. The rat who could not control the shock also had access to a wheel but could do nothing to avoid the shock. A third rat was used as a control, receiving the same warning signal but no shocks. The findings revealed that the rat that received no shocks had the fewest lesions; the rat that was able to control the shocks had more; but the greatest number of lesions appeared in the rat that received shocks but was unable to control their occurrence. Control over the stressor was shown to be an important variable in coping.

Based on the research by Lazarus and others on coping, evidence is provided to support a non-specific theory of stress and illness. Not all individuals have a generalized response to stressful situations neither at the level of appraisal (GAS alarm stage), coping (GAS adaptation stage), or illness/death (GAS exhaustion stage). In terms of essential hypertension, non-specificity theory suggests that individual differences in appraisal of a situation as stressful and one's ability to cope with the stressor, influences one's physiologic response (i.e., blood pressure elevation).

## Racial Differences

Most of the hypertension research that has investigated racial differences has focused on the role of the sympathetic branch of the autonomic nervous system (ANS) as well as sodium regulatory mechanisms and plasma renin levels. Studies that have investigated black/white differences have failed to provide support for the hypothesis that Blacks might have higher resting SNS activity as measured by plasma norepinephrine (NE) levels (Jones, Hamilton, and Reid, 1978; Rowlands, Giovanni, McLeary, Watson, Scotland, and Littler, 1982; Sever, Peart, Meade, Davies, Turnbridge, and Gordon, 1979).

Rowlands et al. (1982) conducted a study of sixteen untreated black patients with mild-to-moderate hypertension and no evidence of target organ damage. These subjects were matched for age, sex, casual blood pressure (BP), and socioeconomic status (SES) with sixteen white hypertensives. The purpose of the study was to compare the responses of matched black and white hypertensive patients, measured under standardized conditions using intraarterial ambulatory BP monitoring, and to assess the cardiovascular reflex responses to pressor stimuli. None of the subjects had secondary hypertension nor were any on antihypertensive medication. All patients were admitted to the hospital for approximately 36 hours during which time a 24-hour collection of urine was made for

estimation of urea, electrolytes, and creatinine. Additional data were collected on the following; ambulatory intraarterial blood pressure, cardiovascular reflexes, baroreflex sensitivity, dynamic exercise, cold pressor test, catecholamine, plasma renin activity, plasma cholesterol and triglyceride levels. The results indicated that significant black/white differences were only found with respect to plasma renin activity (PRA). The median resting PRA of black hypertensives was significantly lower than that of white subjects. The median plasma norepinephrine of Blacks was not significantly different from that of Whites. These data provide evidence to refute the hypothesis that Blacks might have higher resting SNS activity and thus higher blood pressures. In fact the lower PRA in Blacks suggest diminished sympathetic tone. These findings suggest that the pattern of BP responses does not account for observed differences in morbidity and mortality between black and white hypertensives.

Evidence has been provided that indicates that there are racial differences in the distribution of cardiovascular diseases. Heyman, Fields, and Keating (1972) revealed that autopsy data comparing black and white cadavers showed a difference in the distribution of atherosclerosis. Similar findings were demonstrated by Solberg and McGarry (1972). Blacks had a higher incidence of atherosclerosis in the intracranial arteries while whites had a greater deposits in the aorta an coronary arteries. However, the etiology of this differential distribution has not yet been determined.

Based upon the idea that the fight/flight response is an SNS reaction to a stressor, most clinical treatment of hypertension is now accomplished by interfering with SNS transmission, concomitant with treatment with a diuretic which depletes salt from the body, thus lowering blood volume. This combined treatment lowers blood pressure by reducing cardiac output. Since the SNS is characterized by a beta-adrenergic response, one treatment of choice is a drug that has beta-blocker properties. However, pharmacologic research has

demonstrated that beta blockers have a differential effect on Blacks and Whites (Flamenbaum, McMahon, Materson, Albert, & Poland, 1985). Specifically, an alpha/beta blocker (Labetolol) had a superior effect over a beta blocker (Propranolol) in reducing blood pressure in black hypertensives. These findings suggest that the SNS mechanism thought to underlie the development of essential hypertension, may not be applicable to Blacks.

## INTER-RACIAL DIFFERENCES

### Biological Mechanisms

#### Sodium Retention Hypothesis

The sodium retention hypothesis postulates that when sodium intake surpasses regulatory possibilities, the extracellular distribution of sodium results in an increase in extracellular fluid volume. This in turn increases cardiac output. In the beginning stages, the increased cardiac output appears to be responsible for the increased arterial pressure as the peripheral resitances are normal or diminished. As hypertension develops, the cardiac output gradually returns to normal, while the peripheral vascular resistance increases. Arteries and arterioles begin to contract with increased pressure within the blood vessels and relax with decreased pressure. Furthermore, it is proposed that it is this constriction of the renal arterioles that most likely accounts for the lack of sodium excretion efficiency which would normally otherwise result with increased arterial pressure. Hypertension due to sodium retention results in an increased reactivity of smooth muscle (i.e., organs, glands). However, despite the evidence in support of this hypothesis, excess sodium retention alone does not result in hypertension. A genetic factor is also thought to be an

important marker.

Investigators at Indiana University conducted a series of sodium loading studies on the differential effects on sodium loading in black and white adult subjects (Grim, Luft, Weinberger, Miller, Rose, & Christian, 1984; Luft, Grim, & Weinberger, 1985). Grim, Luft, Miller, Meneely, Battarbee, Hames, and Dahl (1980), conducted an investigation to test the hypothesis that the higher prevalence rates of hypertension in Blacks may be related to a greater dietary intake of sodium. Subjects were randomly selected from a survey of approximately 25% of the households in Evans County, Georgia. The sample consisted of 226 white and 89 black subjects. A physician and dietician made a joint visit to each household where they obtained sitting blood pressure, 24 hour urine specimen, height, and weight. Subjects were also asked to provide an equivalent amount of liquids and solids that they had consumed in this 24 hour period, in order to more accurately assess their food intake. None of these subjects were taking antihypertensive medication.

The findings were as follows: 1) Black men and women had greater systolic blood pressures than white men and women; 2) Black men and women consistently had a greater percentage of those with diastolic pressures greater than 90 mmHg than their white counterparts ($p<.05$); 3) Dietary potassium intakes were consistently less for black men and women than for white men and women ($p<.05$); 4) The 24 hour urinary sodium excretion of black men and women was less than that of white men and women ($p<.05$); and 5) Differences in urinary potassium excretion persisted ($p<.05$). These findings suggest that blood pressure differences between Blacks and Whites may be due to sodium excretion inefficiency among Blacks as a result of insufficient potassium intake.

The role that potassium plays in the maintenance of blood pressure is unclear since it has not been studied extensively. However, studies conducted by Langford, Watson, & Douglas, (1968) suggested that the sodium-potassium ratio is an important

relationship in blood pressure elevation. Sodium and potassium play a complementary role in maintaining homeostatic fluid volume. Sodium is an extracellular ion and potassium is an intracellular ion. When sodium ions move out of the cell they are replaced by potassium ions. When this ratio becomes disproportionate blood volume is altered, which can in turn result in elevated blood pressure. Specifically, potassium is thought to attenuate the hypertensinogenic effects of sodium.

These results indicate that the level of racial physiologic departure, if a differential does exist, is not at the level of sodium intake. However, the decreased efficiency by which sodium is excreted due to sodium sensitivity and decreased potassium intake among blacks suggests a racial difference at these levels. This sodium sensitivity is thought, by some, to be due to evolutionary considerations. Afro-Americans, having originated in a warmer climate such as Africa, may have adaptively developed sodium sensitivity. However, having been displaced to a colder climate, this response no longer remains adaptive.

Other researchers (Grim, Luft, Miller, Brown, & Weinberger, 1979; Grim, Luft, Weinberger, Miller Rose, & Christian, 1984), have proposed that the origin of this problem is social and not biological. Koepke, Light, Grignolo, & Obrist, (1983), in a study investigating the effects of pyschological stress on sodium excretion in animals, found that the latter can be inhibited by the former. The renal and neural mechanisms underlying the excretory response to behavioral stress (aversive conditioning) were examined in conscious dogs. Of thirty healthy mongrel dogs used in the study, twenty-one dogs decreased urine flow more than 20% during stress, while only nine dogs showed less than a 10% decrease. Of the 21 renal-reactive dogs, 11 demonstrated decreases in urine flow and sodium excretion that were associated with unchanged glomerular filtration rate (GFR) and effective renal blood flow (RBF). Similar decreases in urine flow and sodium excretion that occurred

with GRF and RBF were seen in the other 10 renal-reactive dogs. Cardiovascular activity (i.e., increased heart rate) during stress was also associated with renal excretion. Specifically, greater increases in heart rate were associated with greater decreasees in renal excretion. It was also found that when surgical renal denervation took place, this procedure abolished the excretory response to stress in four of five dogs. The implications for these findings are that excretory responses in most dogs are mediated; 1) primarily by increased tubular reabsorption rather than decreased GFR, 2) via central integration with cardiovascular responses, and 3) via the renal nerves.

In humans, the evidence that psychological stress may induce sodium and fluid retention is primarily indirect. Light, Koepke, Obrist, Grignolo and Willis (1983) demonstrated the impact of psychological stress on sodium and fluid retention in men at high risk for hypertension. These findings revealed that exposure to competitive mental tasks significantly reduced the urinary sodium and fluid excreted by young men (18-22 years old) with at least one hypertensive parent or with borderline hypertension.

Forty college male students participated in the study, 24 of them were selected for the "stress" condition and 16 for the "nonstress" condition. All subjects had resting diastolic blood pressures less than 90 mm HG and no clinical signs of any cardiovascular or renal disorder. Subjects were required to maintain high rates of fluid excretion by drinking one liter of water during the first hour of the five hour experiment and 200 ml every 30 minutes thereafter. The expectation was for voluntary voiding to occur every 60 minutes during which time urine collections were obtained. Sodium excretion rate was determined by multiplying fluid excretion rate by sodium concentration. Cardiovascular measures were also taken during each of the last three hours. Both heart rate (five minute sample) and blood pressure (BP) were (four to six samples) obtained from each sample. Subjects were then divided into high risk (HR) and low risk (LR) groups determined by the

presence or absence of borderline systolic hypertension or parental history of hypertension. During the nonstress conditon, nine LR and seven HR subjects rested or read while seated in a quiet room throughout the experiment. Repeated measures analysis of variance showed that no significant changes had occurred over hours three, four, and five in sodium or fluid excretion rates or in BP levels for either HR or LR subjects. For the stress condition, hour three was designated at the baseline period, hour four the stress period, and hour five the post-stress period. All aspects were the same in the stresss period as the non-stress period except that during the stress period subjects were exposed in pairs to competitive tasks in which the subject who recognized a target stimulus and pressed a telegraph key faster than his competitor won small money incentives. Groups were further divided into high (>13 beats/min.) and low (<13 beats/min.) heart rate reactors to assess the effects of stress.

The results indicated that substantial stress-induced reductions in sodium and fluid excretion were shown only by HR subjects who were high heart rate reactors to stress.These reductions persisted into the post-stress period as well. All other groups showed slight increases in sodium excretion and no consistent changes in fluid excretion during stress. These findings suggest either an alteration in GFR or tubular reabsorption of sodium or both. The sympathetic nervous system is also implicated due to the relation between heart rate response and decreased sodium excretion in HR subjects.

This interpretation is further supported by animal studies that found that surgical destruction of the renal sympathetic nerves or infusion of Propanolol results in stress-induced sodium retention being abolished. These animal and human studies provide some converging evidence that to the degree that Blacks experience more psychological distress than whites, this socio-ecological variable interacting with biological factors, may be the more crucial mediating factors in hypertension rather than genetic predisposition alone.

Plasma-renin hypothesis.

In addition to sodium retention, the kidneys also regulate blood pressure levels through the release of the hormone renin, especially in response to reduced blood flow. Factors which lead to renin secretion are: lowered blood pressure, lowered salt level, lowered level of circulating fluid, increased activity of renal sympathetic nerves, decreased concentration of urinary sodium, and a fall in the plasma concentration of angiotensin II. Renin converts to angiotensin I, which is then converted to angiotensin II by other enzymes in the body. The latter is a powerful vasoconstrictor. It also has two additional effects on the Central Nervous System (CNS) by increasing fluids and increasing blood pressure. Any situation leading to a decrease in extracellular volume and/or cardiac output stimulates renin secretion to counteract the effects of hypotension. Consequently, high levels of plasma renin are thought to produce hypertension. Racial differences in this aspect of human physiology have also been investigated. However, the research actually shows that Blacks have lower plasma renin levels than do Whites (Gillum 1979). It has been found that approximately 36-62% of black hypertensives have relatively suppressed renin levels in comparison with 19-55% of white hypertensives. The importance of renin has to do with its purported relationship to SNS overactivity. Again, no clear racial distinction of blood pressure elevation can be made based upon the plasma renin levels.

Cardiovascular Activity/Reactivity

Heart Rate. Racial differences in both resting cardiovascular activity and reactivity have been investigated. Both heart rate and blood pressure are physiological indices that have been used to assess potential differences. In a study conducted by Persky, Dyer, Stamler, Shekelle, and Schoenberger (1979) that examined mean heart rate in a sample of

30,786 adults (approximately 12% black), racial differences were found. The sample consisted of individuals aged 18-64 who were screened as part of the Chicago Heart Association Detection Project in Industry. The findings indicated that among subjects aged 18-35 years, black men and women had lower heart rates than did white men and women. However, these differences tended to disappear by age 35 and older. In addition, between the ages of 18-24, heart rates were lower in Blacks diagnosed as hypertensive. The finding that Blacks tend to have lower resting heart rates contradicts the explanation of a beta-adrenergic (SNS) influence on blood pressure and supports the notion of an alpha-adrenergic influence in Blacks since one would expect an increased heart rate rather than decreased heart rate if the SNS were involved.

Blood Pressure. According to Roberts and Rowland (1981), systolic and diastolic blood pressure rose with age in both Blacks and Whites; although the mean pressures were generally higher in Blacks. Despite the fact that Blacks tend to have higher blood pressures than whites, there remains a high degree of within group variation as a function of age (Roberts & Rowland, 1981), obesity (Neser, Thomas, Semenya, Thomas, & Gillum, 1986), socioeconomic status (James, 1984), socioecological stress (Harburg, Erfurt, Hauenstein, Chape, Schull, & Schork, 1973); coping style (James, Hartnett, & Kalsbeek, 1983); and social support (Dressler, Dos Santos, & Viteri, 1986).

Parental History. Anderson, Lane Taguchi, Williams, and Houseworth (1989) examined the interaction of race and parental history of hypertension on patterns of cardiovascular responses among black and white women. Two different types of stressors were used to produce different patterns of cardiovascular responses. Mental arithmetic was used to assess beta-adrenergic (i.e., SNS) responsivity and the cold face stimulus to assess alpha-adrenergic responsivity (i.e., peripheral vascular resistance). Physiological measures

included systolic blood pressure, diastolic blood pressure, heart rate, and forearm blood flow. No differences were found as a function of parental history of hypertension in either racial group. In addition, no heart rate differences were found between black and white women. The results did reveal however that black women had a slower diastolic blood pressure recovery from arithmetic and exhibited somewhat greater stystolic blood pressure responses to the cold face stimulus. Furthermore, black women showed higher systolic and diastolic blood pressure and forearm bloodflow levels throughout the experimental periods. Although forearm vascular differences were found as a function of race (black women exhibiting greater peripheral vascular responses than white women), it was not to the same degree as a previous study (Anderson, Lane, Muranaka, Williams, and Houseworth, 1988) using black and white males. This may be indicative of a sex difference between black women and black men in alpha-adrenergic reactivity.

Physical Stressors. The alpha-adrenergic versus beta-adrenergic mediation hypothesis as differential explanations of underlying mechanims in Blacks and Whites respectively, has been substantiated in some studies. Anderson et al. (1988), found that compared to white subjects, black subjects exhibited significantly greater increases in sytolic and diastolic blood pressure, as well as increases in peripheral vascular resistance, in response to the cold face stimulus. Light, Sherwood, Obrist, James, Strogatz, and Willis (1986), found that black borderline hypertensives demonstrated significantly greater increases in total peripheral resistance than did Whites during a competive task following beta-adrenergic blockade. These results suggest the possiblity of the unmasking of an alpha-adrenergic effect in Blacks. These findings provide converging evidence for the hypothesis that racial differences in hypertension prevalence may be due in part to physiological differences in SNS reactivity.

Psychosocial Stressors. As previously mentioned, mechanisms by which hypertension is mediated is still under considerable scrutiny. However, one hypothesis is that the development of this disorder in Blacks is associated with exaggerated blood pressure reactivity, especially those mediated by vasoconstriction. The peripheral vasoconstriction is thought to be the result of an increased alpha-adrenergic response rather than a beta-adrenergic response which is primarily characteristic of SNS activity. It is surmised that this cardiovascular reactivity is evidenced with elevated blood pressure levels both at rest and during physical or psychosocial stressors.

Light, Obrist Sherwood, James, and Strogatz, (1987) conducted a comprehensive investigation of racial differences in stress reactivity over three separate studies. Both physical stressors (e.g., cold pressor) and psychosocial stressors (e.g., three reaction time tasks--noncompetitive, competitive, and competitive plus money incentive) were incorporated into the study. One hundred ten black college men and 120 white college men were included in the study. Subjects were tested in pairs; 74 black and 84 white subjects were tested in same-race pairs, while the 72 remaining subjects were tested in different-race pairs. Analyses were performed to determine possible differences in cardiovascular responses to stressors as a function of race and pairing type. Subjects were grouped for the purpose of data analyses according to race and presence or absence of marginally elevated blood systolic blood pressure (SBP=135-154 mm Hg--high casual blood pressure). The experimental procedure consisted of first seating the pair side by side in two armchairs. After ten minutes, initial blood pressure (BP) readings were made. Subjects were then asked to rest quietly for 10 minutes, during which time their heart rate (HR) was being recorded. The average HR of the last five minutes served as the pretask resting levels. The second step included the cold pressor test, which involved immersing the subject's foot in a pan of crushed ice and water at an average temperature of 4 degrees centigrade. Two blood

pressure readings were taken during this phase. A five minute rest period followed this test. The third step involved three reaction-time (RT) tasks, two without and one with monetary incentive, each six minutes long involving 20 response stimuli. The first stressor was a noncompetitive RT task and the final two were competitive RT tasks with and without monetary incentive. After the last stressor, a 30-minute relaxation period was used to record BP and HR in minutes 1 to 3, 7 to 9, 14 to 16, 21 to 23, and 28 to 30. Subsequent to the 30 minute relaxation period, subjects were assigned to separate rooms and asked to complete a series of questionnaires that included a comparison of the reaction-time tasks in terms of perceived stresfulness and how hard they were trying, information on parental socioeconomic status, family health history, weekly aerobic exercise, and personality traits.

The results of this investigation revealed that subjects with marginally elevated blood pressures demonstrated greater BP and HR responses to challenging psychological tasks than normotensive subjects ($p<0.0001$). However, this did not hold true for the cold pressor task test. It was hypothesized that this difference may be due to the fact that challenging tasks may tend to elicit beta-adrenergic receptor activity, while the cold pressor test elicits alpha-adrenergic activity. Another possible explanation of the differences in reactivity across tasks is related to behavioral factors. Obrist (1981) demonstrated that tasks that require active coping, such as those included in this study, tend to result in beta-adrenergic responses. Similar evidence was produced by Steptoe, Melville, & Ross (1984) who found that borderline hypertensives showed cardiovascular responses to two active coping tasks, a video game and the Stroop Color Word Test, but not to a passive task, viewing a stressful film.

Light et al. (1987) found that men with marginally elevated BP showed increased cardiovascular responses to the initial stethoscopic BP readings as compared to normotensive men. These elevations were present even after statistically controlling for

group differences under relaxed baseline conditions. Black men, especially those with marginally elevated BP, also demonstrated greater SBP responses during the stressors than did their white counterparts. This finding supports the hypothesis that increased reactivity is predictive of the later development of essential hypertension and that inter-racial differences may be due primarily to subjects with an initial elevated blood pressure. The increased SBP responses to the stressors shown by black subjects with marginal BP elevations were thought to be due primarily to total peripheral resistance rather than higher cardiac output responses. The conclusion was reached based on the fact that black subjects had lower HR responses than did white subjects at rest ($p<0.006$) and no differences in HR were found across stressors ($p>0.10$). A follow-up study was conducted using 40 of the same subjects (20 Blacks and 20 Whites) using impedance cardiography, which assesses changes in cardiac output and total peripheral resistance during two repetitions of the competitive task with money incentive, one before and one after beta-blockade with propanolol (unpublished observations, Light et al., 1987). These findings replicated the original findings of higher SBP responses among Blacks with marginally elevated BP. This increased BP response seemed to be due to higher peripheral resistance in Blacks than in Whites during the task. This effect was further enhanced following beta-blockade, providing additional evidence for an alpha-adrenergic mediation explanation. The studies to date have not consistently revealed racial differences in cardiovascular reactivity. Specifically, Blacks do not seem to exhibit a hyperreactivity to stressors. Although, in some cases Blacks have had greater blood pressure elevations, in other cases they have been shown to have lower cardiovascular responses, particularly heart rate. Consequently, it can not be surmised, based on these inconsistent and inconclusive findings, that Blacks have a higher incidence and prevalence of essential hypertension because of their cardiovascular reactivity and/or hyperreactivity. As a result, this converging evidence

continues to point to the need for a more comprehensive understanding of essential hypertension that includes factors other than biological racial differences.

## Psychosocial Mechanisms

### Psychological Distress

The socio-ecological hypothesis of psychological distress among Blacks has been supported by research on the relationships among race, social class, and psychological distress (Kessler & Neighbors, 1986). It has been consistently demonstrated over the last few decades that Blacks experience higher rates of psychological distress than do Whites (Warheit, George, Holzer, & Arey, 1975). Kessler and Neighbors conducted an analysis of eight different epidemiologic surveys (22,000 respondents) that investigated the effects of race, social class, and pyschological distress. Since Blacks tend to comprise only a small proportion of samples in most surveys, using several different surveys and pooling the results, provided an opportunity to increase the overall sample size. Initially, racial discrimination was thought to be the primary factor that resulted in a positive association between race and psychological distress. However, more recently, socioeconomic explanations have become more prevalent. A number of studies conducted between 1973 and 1984 demonstrated that initially higher levels of distress among Blacks were attenuated when controls for social class were instituted (Carr & Krause, 1978; Eaton & Kessler, 1981; Neff, 1984; and Warheit, Holzer, & Schwab, 1973). Based upon these findings, race does not appear to be the sole determinant of psychological stress, but rather serves as a proxy for socioeconomic position. In terms of the sodium excretion hypothesis that postulates that sodium excretion is inhibited by psychological distress in animals and humans, these findings appear to also support a psychosocial explanation.

Kessler & Neighbors (1986) used a linear additive regression equation with and without the interactive term Race X Social Class. The outcome variable, psychological distress, was measured using scales that rendered a subjective measure of depressed mood and somatic complaints associated with anxiety and depression. Social class was determined using a multidimensional measure, including family income and respondent's education which were entered as separate indicators. The pair of equations (i.e., one for each social class index) was estimated separately within each survey, and the results were then pooled (scaled to common metrics) across surveys to arrive at an overall significance test. The analyses were then repeated on subgroups classified by sex, age (over 40 versus under 40), and residence (urban versus non-urban). The results consistently suggested that Blacks have significantly higher gross distress levels than do Whites, but that this association could be explained away with controls for social class (i.e., all 18 of the replications showed that the race-distress association reduced when social class was controlled).

Further, interaction anayses of race-by-income and race-by-education provided evidence for a negative interaction between race and income in predicting depression and somatization. Seven of nine interactions were significant. The negative $r$ value of the interaction term suggests that racial differences in distress are greater among people with low, rather than high, incomes. These results have since been cross-validated in other demographic subsamples. When this interaction is taken into account the data tend to show that race, possibly due more to environmental (i.e., minority status) rather than biological factors, has a substantial effect on psychological functioning among lower-class people but not among upper class persons.

At least two explanations have been proposed to account for the effects of social class and race on pyschological distress. One possibility is that pronounced distress among

lower-class Blacks as compared to that of lower-class Whites is due to a greater proportion of Blacks than Whites with stifled mobility aspirations. Parker and Kleiner (1966) reported data consistent with this view among Blacks in Philadelphia. They found that high distress among lower-class Blacks was associated with high goal striving-stress. Other plausible explanations include the synergistic effects of poverty and discrimination on lower-class Blacks, or the unavailability of resources for coping with stress. Although no data exist to substantiate these hypotheses, future research in these areas might prove enlightening. Again, these findings point to the investigation of individual differences among Blacks, rather than racial differences, when attempting to understand the complexity of essential hypertension.

Socio-ecological stressors

Harburg et al. (1973) investigated the socio-environmental differences between black and white urban populations in relation to blood pressure. Their major hypothesis was that urban socioecologic areas which vary in rates of stressor conditions may have populations which vary in blood pressure levels. The underlying assumption was that socially disorganized life areas tend to generate problem situations that require adaption more frequently and with fewer resources than more organized areas. Scores for various census tracts in the city were computed by considering rates of economic deprivation, residential instability, family instability, crime and density. The rates were then factor analyzed and each of the 382 census tracts was assigned a factor score for two emerging oblique factors: socioeconomic status and instability. Within each ethnic group High Stress areas were established by determining if census tracts for each factor score list had both the upper range for the instability score and the lower range for the SES scores; and the converse for Low Stress areas. As a result, four groups were established; Black High

Stress, White High Stress, Black Low Stress, White Low Stress.

A sample of Blacks, Whites, males and females who were between the ages of 25 and 60, who were married and living with their spouse, and who had relatives living in the metropolitan area were selected to participate in the study. The sampling took place in two stages. Stage 1 consisted of a "door to door" census being taken in each of the four stress areas to screen and classify potential subjects. Persons identified as potential subjects were then interviewed again by a trained interviewer to verify that they met the criteria to be included in the study. Stage 2 consisted of randomly assigning same race nurses to interview verified potential subjects. Blood pressure (BP) readings were taken at the beginning of the interview, 5-10 minutes later, and again 10 minutes later during the first half hour of the medical history.

Chi-square analyses were performed with four dependent variables: Mean Systolic BP (SBP=mean average of the first three systolic readings), Mean Diastolic BP (DBP=mean average of the first three diastolic readings), 4 Category Systolic BP; a) ≤119 mm, b)120-139 mm, c)140-159 mm, d)160+ mm; and 4 Category Diastolic BP; a) ≤83 mm, b) 84-89 mm, c) 90-94 mm, and d) 95+mm classified as Low Normal, Normal, Borderline, and Hypertensive respectively, based on established blood pressure level criteria. Data were presented that tested the link between objective stressors as previously stated and the subject's perception of threat and report of desirability of living in the area. In this study 77% in the Black High Stress area and 50% in the White High Stress area desired to move to another neighborhood compared to 26% and 29% respectively in the black and white Low Stress areas.

The findings for males revealed that Black High Stress males have the highest percent of higher readings (140+/90+) compared to other male race-area groups, which are similar to each other. This trend was more apparent for DBP rather than SBP. Intra-racial

differences revealed that High Stress black males had higher proportions of Borderline and Hypertensive diastolic categories (38%) than Low Stress black males (19%, $p<.01$). No differences were found between white High and Low Stress areas. When blood pressure was adjusted using seven covariates: age, overweight, ponderal index, season of year, time of interview, hours since last meal, and rated tension at readings, the differences remained the same but were less for SBP. T-tests on the adjusted means showed no difference between white males by stress area, nor between Low Stress males and the two white male groups. Although, Low Stress black males had slightly higher DBP than High Stress white males, this difference disappeared when the variance due to age, overweight, and socioeconomic status were controlled.

For females the trends were similar but smaller for both SBP and DBP. Black High Stress females had the highest percent of Diastolic Borderline and Hypertensive (30%) categories than black Low Stress areas (22%) or white High (17%) or White Low (15%) ($p<.01$). These trends were similar for systolic blood presure, but were not significant. No differences were found between High and Low white females. When means were adjusted for age, overweight, ponderal index, season, time of day, time since last meal and tension, the rank order of levels remained the same but the differences between the groups changed. When controlling for these covariates, there were no significant differences between High and Low Stress black females and differences in Systolic BP appeared at the 0.05 level between High and Low Stress white females. These researchers concluded that this new effect was largely due to a greater percent overweight among High Stress black females.

Thus, the major hypothesis that blood pressure levels will vary with stressor conditions in socioecological areas, was partially supported. The major findings were as follows: 1) Black High Stress males had higher blood pressure levels than Black Low

Stress males; 2) Black Low Stress males did not differ in blood pressure levels from White Low Stress males; 3) White High Stress females showed higher systolic blood pressure levels than White Low Stress women; and 4) Black High Stress females have significantly higher blood pressure levels than Black Low Stress females. These findings suggest both inter-racial and intra-racial group differences in blood pressure. These results, indicate the need for additional investigation in the area of intra-racial blood pressure differences among Blacks.

## INTRA-RACIAL DIFFERENCES IN ESSENTIAL HYPERTENSION

### Biologic Risk Factors

#### Parental History

Johnson (1989) investigated intra-racial differences in cardiovascular reactivity, emotional factors, and home blood pressure in black males with and without a parental history of hypertension. Twenty-four black male undergraduates between the ages of 19 and 25, who had never been diagnosed as hypertensive were used in the study. Information on the subjects' and the subjects' parental health history was obtained on a health inventory that was administered during the screening process. Two classifications were made based on health history; Positive Family History (PFH)--if at least one parent had hypertension, and Negative Family History (NFH)--if subject had no first degree relatives with hypertension. Exclusion criteria for potential subjects included: 1) not having heart disease and/or diabetes; 2) not having hypertension; 3) not taking a prescription medication; and 4) not frequently using a relaxation technique. Fifteen students met the criteria for the PFH group and 12 for the NFH group.

Subjects were asked to complete the following questionnaires: 1) State-Trait Personality Inventory (STPI); 2) Anger Expression Scale (AX); 3) State Anger Reaction Scale (S-Anger/RX); 4) Jenkins Activity Survey (JAS); and 5) Cattell 16PF, Submissiveness-Dominance (S-D). Cardiovascular reactivity data (systolic blood pressure--SBP, diastolic blood pressure--DBP, heart rate--HR) were then collected in response to the following mental challenge tasks and resting periods that were presented in the same order to each subject. The conditions were as follows: baseline, anagram stressor, anagram recovery-resting period, mental arithmetic, arithmetic recovery-resting period. At the end of the final recovery period, subject's post level of state anxiety and anger were again measured using the STPI. Subjects were then instructed on how to take their own blood pressure. There were required to record their sitting SBP and diastolic DBP each morning upon awakening and before betime for 28 days.

Two tailed T-tests on cardiovascular data (SBP, DBP, HR) revealed that individuals in the PFH group had a significantly ($p < 0.05$) higher baseline SBP than subjects in the NFH group. DBP differences were in the same direction as SBP but did not reach statistical significance. SBP, DBP, and HR were analyzed separately using analysis of covariance using each experimental condition as repeated dependent variables and the pretask baseline as a covariate. Further findings revealed that there was a signifcant ($F=4.67$, $p<0.05$) main effect for DBP, the main effect for Family History Groups approached significance ($F=2.85$, $p<0.10$), and there was not a significant main effect for Family History Groups for HR. A significant main effect was found for experimental conditions for SBP ($F=24.49$, $p<.001$) DBP ($F=15.83$, $p<.001$), and HR ($F=9.44$, $p<.001$). However, the Family History Groups by Experimental Conditions interaction was not significant for SBP or DBP.

Multiple *t* test revealed significant differences between PFH and NFH groups on

psychological test scores, with the PFH group scoring higher on Trait Anger ($p<.06$), Trait-Anger/Temperament ($p<.05$), Anger-Out ($p<.01$), and the Submissiveness scale of the Cattell 16PF ($p<.01$). Stepwise discriminant function results indicated that Submissivesness was the only significant ($F=7.52$, $p<0.01$) independent predictor of family history of hypertension.

Self-monitored home blood pressure findings revealed significant main effect for Family History on morning SBP ($F=6.54$, $p<.01$) and evening SBP ($F=4.81$, $p<.05$) indicating that PFH groups had significantly higher SBP than NFH groups over the four weeks following the laboratory assessment. Analyses of DBP revealed that again there was a significant main effect for Family History on morning DBP ($F=5.83$, $p<.05$) and evening DBP ($F=4.43$, $p<.05$).

Forward stepwise multiple regression was used to determine whether cardiovascular responsiveness to laboratory stressors and personality characteristics contribute to the production of home monitored blood pressure. Analyses were conducted separately for SBP and DBP. The analysis for SBP showed that baseline SBP, weight, and family history accounted for a significant amount of the variance (67-73% for morning; 64-70% for evening), with SBP accounting for most of the variance. Similarly, baseline DBP, weight, and family history accounted for most of the variance in home DBP; although the amount of variance explained was substantially lower than the percentage for SBP. Psychological measures (i.e., Trait-Anger/Temperament, Anger-Out, and Submissiveness) contributed significantly to the predicition of SBP and DBP.

## Psychologic Risk Factors

Because the medical literature has not been able to successfully establish racial differences in essential hypertension along biological lines, biobehavioral researchers have

turned their efforts toward psychosocial variables for explanation. One of the more commonly studied psychological constructs has been Type A Behavior Pattern (TABP) as a risk factor for the development cardiovascular heart disease (CHD). Although hypertension is also a risk factor for CHD, very little research has investigated the relationship between TABP and essential hypertension. Even though, the components of Type A and of essential hypertension have ben shown to include similar emotional aspects (i.e., anger, hostility) the patterns for each is thought to be different. Hypertensives are often characterized as chronically hostile, resentful, conflicted about anger expression, and anxious when provoked by anger. On the other hand, Type As are thought to be aggressive, channel emotional arousal into action, and experience decreased anxiety when provoked. More research is needed to determine the aspects of emotional behavior mediating cardiovascular disorders to help differentiate been the relationship between Type A on CHD and essential hypertension and CHD.

Type A Behavior Pattern (TABP).

In an attempt to investigate the relationship between psychological factors and coronary heart disease (CHD), Friedman and Rosenman (1959) identified TABP as a characteristic of an individual's incessant struggle to achieve more and more in less and less time. Some of the major components of TABP are a sense of time urgency, aggressiveness, competitive achievement striving, and easily aroused hostility.

Booth-Kewley and Friedman (1987) conducted an extensive meta-analysis of both the medical and psychological literature from 1945 to 1984 on the relationship between personality factors and CHD from 1945 to 1984. Eighty-three of 150 studies that were located met the criteria to be included in the review. The criteria included: 1) the study had to have used at least one of the following personality traits as an independent variable, Type

A or one of the components of Type A (e.g., job involvement, time urgency), anger, hostility, aggression, depression, extraversion, or anxiety; 2) the study had to have used some manifestation of CHD or atherosclerosis as a dependent variable; 3) the study had to have used quantifiable variables and could not have been purely descriptive or anecdotal; and 4) the study had to have contained sufficient information to allow estimation of effect size and significance level.

To determine whether the various personality factors were associated with different clinical manifestations of CHD, results were analyzed separately for various disease outcomes (e.g., myocardial infarction, angina pectoris and for TABP measures) and were also combined for an overall analysis. The overall analysis revealed that the effect size between Type A and disease was .136, ($p<.001$). Based on Cohen's (1977) criteria this effect size (ES) is small yet reliable.

It was also found that Structured Interview (SI) (ES=.221) related more strongly to disease outcome than did the Jenkins Activity Survey (JAS) (ES=.067), as measures of TABP. In addition, of the JAS factors, Hard-Driving Competitiveness was most strongly associated with disease (r=.153). JAS Job Involvement was not found to be reliably associated with disease. Of the SI factors only Time Urgency was included in at least two or more studies. The combined effect size was (ES=.095, $p< .001$). This effect size is similar to the combined *r* for JAS Speed and Impatience (ES=.058, $p< .001$), indicating that speed and impatience/time urgency related reliably to disease to a minor degree. Lastly, a combined effect size of .272 ($p<.001$) between Type A and women suggests that the Type A-disease relation is as strong or stronger for women as it is for men.

In looking at the results of other personality variables, depression related most strongly to disease with a combined effect size of .205 ($p<.001$). This effect size is greater than all measures of Type A, suggesting that depression is a crucial factor in relation to

CHD. Modest effect sizes were found for anger/hostility/aggression (.121) and anger/hostility (.138). Independent effect sizes for anger (.077), hostility (.160), and aggression (.071) indicate that hostility was most reliably related to disease.

Results for different disease outcomes used the following classifications as outcome measures; global, myocardial infarction (MI), angina, and cardiac death. In general the findings were similar across disease classifications. However, one interesting deviation was that the combined effect size of JAS Type A and MI (.133) was considerably larger than the effect size (.067) in the overall analysis. This finding suggests that JAS may have differential clinical manifestations of CHD.

The results for cross-sectional versus prospective studies revealed that many more cross-sectional than prospective studies were included in the meta-analysis. Because of the minimal number of prospective studies, weaker evidence is provided that personality variables are predictive of disease. One major finding in this category was that the combined effect size for all measures of Type A was substantially higher in cross-sectional ($r$=.156) than in prospective ($r$=.045). studies. Since the combined effect sizes of JAS Type A and the SI are larger in cross-sectional than in prospective studies, the possibility of artificial inflation in these studies exists.

The results for pre-1977 versus post-1977 studies provided evidence that the combined effect size for all measures of Type A was considerable higher for pre-1977 studies ($r$=.204) than for those studies conducted during or since 1977 ($r$=.108). When SI Type A is considered separately this trend is still apparent, suggesting that the relation between SI Type A and CHD has been increasingly hard to demonstrate in recent years. This issue will be discussed in Matthews (1988) rebuttal of this study.

When interpreting these findings, Booth-Kewley and Friedman suggested that perhaps their results were skewed due to the preponderance of cross-sectional studies rather

than prospective studies. This was true for each of the variable categories, some of which had no prospective studies included. Although cross-sectional research is less expensive and less time consuming, it provides weaker evidence that personality variables are predictive of disease. This in part could be due to two factors, the fact that one is ill might have an impact on the way in which the subject responds, and the experimenter's awareness that the subject is ill could bias his/her assessment. Based on these possibilities, it has been suggested by the researchers that the combined effect sizes for Type A, all measures ($r$=.156) in cross-sectional studies, compared to ($r$=.045)) in prospective studies might be artificially inflated. This differential was also true for the combined effect size for JAS Type A cross-sectional ($r$=.102) versus prospective ($r$=.009); as well as for SI cross-sectional ($r$=.238) versus prospective ($r$=.062). However, although prospective studies minimize the possibility of providing evidence that the behavior is a product of the disease rather than the disease being a product of the behavior, this type of research is vulnerable to various threats to internal validity because subjects cannot be randomly assigned to be Type A or Type B. Consequently, both types of studies can provide useful information.

Another important finding of this review was that the average strength of the observed relation between Type A and cardiovascular disease has decreased over time. Several explanations have been proposed: 1) instrument decay (SI) due to the subjective ratings of interviewers whose conceptualizations of Type A may have changed over time (e.g., decreased emphasis on speed and impatience); 2) the advent and widespread use of the JAS Type A scale which is easier to administer, although less strongly related to CHD than the SI; and 3) the greater liklihood of having a study published that fails to reject the null hypothesis.

Contrary to the Booth-Kewley and Friedman findings, Matthews (1988) also conducted a meta-analysis of the association of Type A behaviors and CHD which revealed

that Type A behavior is not a reliable predictor of CHD incidence. These results held true across all measures of Type A and across prospective study designs when the number of independent studies and number of participants were weighted. Based on these findings, Matthews calls in to question Booth-Kewley and Friedman's study on conceptual and methodological grounds. The point of departure with these two studies lies in the rationale and decision to include or exclude studies and how they were subsequently weighted.

The inclusion criteria for this meta-analysis required that studies, 1) test for the significance of the associaton between CHD and Type A, hostility, depression, or anxiety, 2) be prospective in design; and 3) report sample sizes in the analysis. Estimates were calculated for the following: 1) Type A, combining all mesures and studies; 2) JAS Type A, combining all studies; 3) SI Type A, combining all studies; 4) Type A in population studies, combining all measures; 5) hostility; 6) depression; and 7) anxiety. The findings provided evidence to contradict the Booth-Kewley and Friedman study, namely that if the number of participants as well as the number of independent studies are weighted, then Type A behavior is not a significant predictor of CHD. However, when not weighting studies, Type A behavior combining all studies and measures is a significant predictor of CHD. When assessing the impact of various measures of Type A in predicting CHD, it was found that the SI was related significantly to CHD incidence across studies in the weighted analysis; but when measured with the JAS was unrelated. Evidence was also provided that supported the notion that high-risk and population studies should be analyzed separately. These analyses showed that Type A is not a significant predictor of CHD in studies that examine exclusively high-risk individuals.

Matthews offered several possible explanations for the failure to find an association in high risk populations including: 1) a higher prevalence of Type As in high-risk studies resulting in less variance in the predictor variable and thereby reducing the probability of

finding an association; 2) intervention undertaken by high-risk subjects (i.e., pharmacologic behavioral treatment) thereby making the Type A assessment less reliable; and 3) high-risk studies typically reported mortality data in contrast to population-based studies that generally reported morbidity data. As a result, one possible hypothesis is that Type A is more related to nonfatal events than to fatal events and/or that Type A may influence initial but not later events.

The hypothesis that emotions are associated with CHD was also supported. Hostility, depression, and anxiety were found to be associated with CHD, and significant predictors of CHD. One of the major differences between the two studies was that Matthews used only prospective studies because of the aforementioned methodological and interpretive difficulties that are inherent in cross-sectional research. Whereas cross-sectional research addresses the association between a risk factor and prevalence of disease, propspective studies examine the association between a risk factor and incidence of disease.

Matthews also criticized the fact that the previous meta-analysis did not divide studies according to high-risk versus population studies. A review conducted by Matthews and Haynes (1986) showed that population studies yielded significant Type A CHD-associations, whereas high-risk studies yielded few. Since the development of CHD depends on the extent of atherosclerosis and acute precipitating events, risk factors associated with initial events might differ from risk factors associated with later events. Further criticisms include failure to weight studies for number of participants in a given study, failure to obtain consensus on an adequate measure of Type A, and failure to assess negative emotions associated with CHD independently (e.g., depression, hostility, anxiety).

The results in this meta-analysis provide evidence indicating that the decision rules used to determine inclusion of specific studies and guidelines on how the data shall be analyzed and interpreted, contributes to the overall findings. In light of this, Matthews

suggests that previous reviews that have found that Type A is a reliable predictor of CHD have failed to consider these important aspects.

In general Friedman and Booth-Kewley's defended their use of cross-sectional studies that excluded certain high risk subjects (e.g., people with fatal myocardial infarctions) indicating that omission of these individuals actually attenuated their findings, thereby strengthening rather than weakening the associations that they did find. In addition, the fact that an association was found in those individuals whose Type A behavior may not have been as apparent because of pharmacologic or behavioral treatment also provides evidence that the findings were not artificially inflated but possibly suppressed.

Freidman and Booth-Kewley, as well as Matthews provide interesting arguments to substantiate their findings. Even though prospective studies might prove to be more illuminating over time, it is not always practical or convenient to use these type of studies. However, despite the fact that cross-sectional studies do not demonstrate predictability of the development of disease as well as prospective studies, useful information is provided on prevalence of the disorder in the population. This limitation is similar to that of the SI versus JAS usage. Although the SI is considered to have a stronger predictability to CHD than JAS, the latter is more commonly used because it is convenient and less expensive to utilize. Time and money factors are important research considerations that must be taken into account even though the ideal study might theoretically ignore these issues to maintain the purity of the research.

The Type A literature has provided valuable information in our understanding of the relationship between pychological factors and heart disease. However, there have been many valid criticisms of this research which have resulted in continued refinement of the construct and more adequately designed studies. Moreover, much of this research over the last fifteen years has been conducted on white, college educated, middle-class males. This

poses a threat to external validity of the studies, particularly with reference to women and minorities. Two of the more prominent prospective studies, the Framingham Study and the Western Collaborative Group Study excluded minorities from their samples. It is clear that further research is needed on Type A Behavior Pattern as well as other psychological constructs as they influence heart disease among women and minority subjects.

John Henryism

Sherman James is one of the few researchers to attempt to investigate psychosocial risk factors of essential hypertension (EH), often considered a corollary of CHD, among Blacks. In doing so he developed a measure of a psychological construct coined "John Henryism" to assess personality characteristics (e.g., personal competence and environmental mastery characterized by efficacious mental and physical vigor , commitment to hard work, and a single-minded determination to achieve one's goals) in conjunction with one's coping resources (e.g., income and education) that might subsequently lead to the development of EH. He specifically observed racial differences, and found that this measure was more predictive of EH in Blacks than Whites (James, Strogatz, Wing, & Ramsey, 1987). Similar to the utility of the JAS and the SI used to assess Type A Behavior Pattern (TABP) and subsequently predict CHD among Whites, James has developed the John Henryism Active Coping Scale that measures an individual's intense struggle to demonstrate personal competence and to achieve a sense of environmental mastery, while subsequently predicting the potential for the development of hypertension in Blacks. Most of James' research has focused primarily on black males in the rural South. Although it is noteworthy that James has initiated research that has focused on Blacks, it is unfortunate that black women continue to be understudied. Considering that a significant number of black women are the sole heads of households and earn less than their white

counterparts, the hardships that these women experience might be manifested by TABP or John Henryism coping styles that may in turn influence their susceptibilty to disease.

Tyroler and James (1978) further expanded this line of reasoning by proposing that high rates of hypertension among younger, low-income, black males were due to, in part, to their chronic and frequently intense struggle to demonstrate personal competence and achieve a sense of environmental mastery. These characteristics were exemplified in an acquaintance of Sherman James', John Henry Martin. He characterizes this elderly, hypertensive, black man in much the same way as the legendary steel driver who paid a high cost (death) for his victory over the mechanical steam drill (Johnson, 1929). Similarly, James observed that John Henry Martin was a poor, illiterate sharecropper who by age 21 had taught himself to read and write. Through hard work and determination, by age 40 he owned 75 acres of farm land in North Carolina. Unfortunately however, John Henry Martin's success cost him a great deal in terms of his physical health by age 60. At this time in his life, he was forced to give up farming due to severe medical problems; foremost among which was hypertension. John Henry Martin later attributed his poor health to his single minded relentless pursuit of economic self-sufficiency during his youth and middle adult years. He further reported that being black and having no formal education added significantly to the difficulties he experienced in reaching his life goals.

In an attempt to test the John Henryism concept, James et al. (1983) conducted a social epidemiologic investigation of hypertension among 132 semirural, low-income, black men between the ages of 17 and 60. These men were administered the John Henryism scale in order to measure the degree to which they felt they could control their environment through hard work and determination. It was hypothesized that men scoring below the median on education but above the median on John Henryism would have higher blood pressures than any other group. The research question was tested using a 2 X 2

analysis of covariance design with the first factor consisting of the two levels of John Henryism (high/low) and the second factor consisting of the two levels of education (high/low). The covariates included age, Quetelet Index (wt/ht), time of day, and number of cigarettes smoked daily. The data were in line with the prediction, in that men who scored low on education and high on John Henryism had significantly higher diastolic blood pressures than men who scored above the median on both measures.

Controllability and Predictability.

Ostefeld & Shekelle (1967), conducted a review of the literature providing evidence linking psychosocial factors and blood pressure elevation. Despite the limitations of external validity from the laboratory to the general population, they still concluded that psychological appraisal of stimulus threat and perceived ability to cope with the threat were probable long term mediators of blood pressure levels. They suggested that there are four characteristics of ordinary life situations which seem to be associated with pressor responses: 1) the outcome of the event is uncertain; 2) the possibility of psychological harm exists; 3) although running away or physical resistance may be considered, they are not appropriate behavior; and 4) the person involved commonly feels compelled to maintain a vigilant mental attitude until the situation is clarified or resolved.

Studies that have further substantiated Ostfeld's and Shekelle's framework have investigated the effects of unemployment (Kasl & Cobb, 1970); high vigilance on the job (Cobb & Rose, 1973); crowding (D'Atri & Ostfeld, 1975) and their relationship to high blood pressure. The factor that each of these field studies had in common is that they support the postulation that sustained blood pressure increases are most likely to occur when the behavioral stressor seems uncontrollable (e.g., the outcome is not clearly dependent upon one's actions), yet neither fight nor flight is possible.

Obrist (1978) provides support for Ostfeld's & Shekelle's framework as well as for James' "active coping" concept. In a series of three experiments involving young adult males who were recruited from the introductory psychology course at the University of North Carolina, beta-adrenergic influences on heart rate and carotid dP/dt were evaluated as a function of the degree of individual control over stressful events. The second experiment in this series on active coping provides a more direct test of the relationship between blood pressure elevation and perceived controllability of behavioral stressors. In this study, two different procedures were used. One procedure involved a comparison of the effects of two stressors over which the subject knowingly had no control over, to a stressor in which the subject was given actual control. The former included a painful event (e.g., cold pressor) and a sexually arousing event (e.g. the viewing of a pornographic movie). This was done to see whether differences in affect influenced the pattern and extent of myocardial and blood pressure changes under conditions where the subject had no control over events or was only passively involved. A shock avoidance vigilance task in which avoidance was contingent upon performance was used as the control condition. Another aspect of this condition incorporated the manipulation of the criterion required for successful avoidance so as to evaluate the effects of the degree of control of the aversive stimuli.

The physiological indicators that were measured as a function of the challenge to complete an unsignaled reaction time task to avoid intermittent mild shocks, were heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP). Three experimental conditions were established: 1) an "easy" condition, wherein the reaction time was comfortably mastered within the alloted time; 2) an "impossible" condition, whereby the alloted time was so brief that no one could master the task; and 3) a "hard" condition, where with real effort and concentration, faster reaction times could be occasionally achieved.

A repeated measures analysis of variance was used to analyze the data. In examining the effects of task difficulty, all three conditions showed similar increases in HR, SBP, and carotid dP/dt; however, these beta-adrenergic influences on the myocardium were more strongly evoked and sustained in the "hard" condition in which subjects believed that they could control the stressor and actively coped by attempting to do so. These results as well as others by Obrist, Lawler, Sutterrer, & Martin, (1974) indicated that providing an individual with some control in coping with stressful events, evoked more appreciable sympathetic influences on the heart than conditions in which control was minimal or not possible.

This study has implications for both Ostfeld's and Shekelle's four postulates as well as James' John Henryism concept. It is of particular relevance for Blacks and the poor who are continually subjected to many behavioral stressors in everyday life, yet have few coping resources (e.g., education, income). Furthermore, in congruence with the previously cited studies, those who think they can control their environment through active coping, may have higher blood pressures than similar others who are more resigned about the issues of environmental mastery and rely on more passive coping styles.

In another important study in this area, James et al. (1987) investigated socioeconomic status (SES), John Henryism (JH), and hypertension in Blacks and Whites. The study was conducted in a biracial community sample of 820 adults, aged 21-50 years, who resided in Edgecombe County, North Carolina, and had been interviewed in a previous study done in 1983. SES was determined in the same way as in the pilot study in 1983, using completion of high school versus non-completion. However, based on this criteria, the expected inverse relationship between education and blood pressure was not observed in any race-sex group. This led James and his colleagues to examine whether education had the same connotations in the Edgecombe County survey compared to the

pilot study, which consisted of poor and predominantly black subjects from the Coastal Plains region of North Carolina.

Analyses of construct validity were undertaken and revealed inter-sample differences in the ability of subjects from each study to use their high school diplomas to obtain skilled blue-collar or white collar jobs (33 % versus 56 %). Consequently, James and his colleagues decided to develop measures of SES for both Blacks and Whites that combined information on education and/or occupation. In addition, because of the minimal overlap of occupation scores for Blacks and Whites, separate SES criteria were established for each racial group. For Whites, persons with less than nine years of formal education or persons employed in blue-collar jobs (Hollingshead job status scores=1-4) and Blacks with less than nine years of formal education or employed in low blue-collar jobs (Hollingshead=2) were assigned to the low SES group. Conversely, Whites with a high school diploma or more and employed in white collar jobs (Hollingshead=5 or greater) and Blacks with nine years or more of formal education in at least high blue-collar jobs (Hollingshead=3 or greater) were assigned to the high SES group.

Analysis of covariance was used to estimate the relation between SES and blood pressure and its potential modification by JH. Regression analysis was used to estimate the relationship between SES, JH, and prevalence of hypertension. The results indicated that mean blood pressures differed by SES for Blacks but not for Whites. In addition, group differences for blacks were in the hypothesized direction; at low levels of John Henryism, SES differences in blood pressure were small (1 mm Hg for SBP and DBP); however, for persons scoring high on JH, these differences increased to 5.2 mm Hg for SBP and to 3.8 mm Hg for DBP. Even though the findings were in the expected direction, there was <u>not</u> a statistically significant SES X JH interaction. Multiple regression analyses revealed signifcant correlations between race, sex (Whites only--men scoring higher than women),

age (Blacks only), and SES (Blacks only--inverse relationship).

Similar to James' pilot study in 1983, the results of this study indicated that a high JH score alone did not increase the risk for hypertension. Although, when combined with inadequate coping resources, as determined by low formal educaton and/or unskilled, low paying jobs, there is an increased risk. It has been hypothesized then that when coping resources are high, the strong sense of personal efficacy that in part defines JH may attenuate sympathetic arousal through physiologic processes as yet undetermined, that in turn lowers risk of hypertension. Furthermore, James hypothesizes that the inverse relationship between education/SES and blood pressure almost always exists because the complexity of industrialized societies require more sophisticated problem solving abilities and communication skills which are usually attained through formal education. As a result, those individuals who have limited formal education may perceive a broader range of routine tasks as difficult and will adapt to their environment less efficiently. JH is thought to potentiate autonomic arousal in these individuals who attempt to actively cope with life's uncertainties.

Self-Efficacy and John Henryism. James points out that although John Henryism represents an aspect of self-efficacy, it differs from the latter in that the former usually results in a negative physiologic outcome (i.e., essential hypertension). Although both are characteristic of having a sense of control over the environment John Henryism goes beyond mere attribution of control (e.g., internal or external) and includes a behavioral mechanism active coping, that over time can have deleterious effects on the body. It should also be noted that despite the fact that both involve environmental mastery and personal competence, John Henryism, unlike self-efficacy, is derived from the psychophysiology literature which has empirically demonstrated in the laboratory that active coping with

stressor results in a negative consequence, increased blood pressure (Obrist, 1978). These same physiologic ramifications have not been demonstrated in relation to self-efficacy.

Learned Helplessness and John Henryism. John Henryism can be differentiated from Learned Helplessness in that the former is viewed as an active coping measure, whereas the latter is considered by some as a passive coping mechanism. In a more general sense the two are similar in that they both represent ineffective coping; John Henryism representing more direct confrontation with one's environment and Learned Helplessness being representative of an escape/avoidance coping process. Also, in terms of controllability John Henryism typifies a perception of having control over one's environment, whereas Learned Helplessness represents a perception of having no control over stressful events.

## Socio-ecologic Risk Factors

### Social Mobility

Social mobility is another concept that has been investigated in an attempt to identify psychosocial risk factors that may contribute to cardiovascular disorders. Whether it is cultural (Marmot, Syme, Kagan, Kato, Cohen, & Belsky, 1975), occupational (Kaplan, Cassel, Tyroler et al., 1971), religious (Lehr, Messinger, & Rosenman, 1973) or geographical mobility (Rhoads, Gulbrandsen, & Kagan, 1976) the common denominator in susceptibility to subsequent heart disease is the necessity to adapt to an unfamiliar environment. Oftentimes this adaptation results in a physiologic change over time.

The relationship between sociocultural mobility and subsequent CHD and essential hypertnesion was studied in a cohort of 13, 728 male former Harvard University students examined in 1939-1950. A total of 8852 subjects returned self-administered mail

questionnaires in 1962, 1966, and 1972 which inquired about doctor-diagnosed myocardial infarction (MI), angina pectoris (AP), and hypertension (HT) (Gillum & Paffenbarger, 1978).

Based on univariate analysis, there were significant inverse relationships between father's occupational status and risk of combined fatal CHD and MI and between geographic mobility and risk of HT. Intergenerational mobility, as indicated by occupational status of the father, was associated with 1.5 times increased risk of fatal CHD and MI. The socioeconomic class of parents was determined by occupations of the father (upper class=professional/managerial, lower class=clerical and skilled/unskilled laborers), level of education of the father, income of the parents, and absence of an automobile and servants in the household at the time of college entrance. Subjects were considered to be at least middle class considering they were Harvard graduates.

In this study, it is noteworthy that although father's level of occupation was inversely related to MI, it only reached statistical significance in sons of unskilled laborers. However, there was not an excess risk for HT among this sample. Also, in terms of intragenerational mobility, it was hypothesized that some degree of adaptation might have a positive effect in reducing blood pressure especially among higher socioeconomic groups. Whereas lower socioeconomic and less educated individuals might view mobility as stressful because it requires a degree of adaptation to an unfamiliar environment, higher SES and better educated individuals might view this same mobility as non-threatening and possibly gratifying because of the challenge. Although this study only considered "upward mobility", some research has suggested that "downward mobility" is also associated with higher blood pressure levels (Borhani, Borkman, Laughlin, & Slansky, 1968). Borhani and colleagues found that when comparing father's occupational status with the subject's, upwardly mobile men below age 55 had lower blood pressure levels than non-mobile or

downwardly mobile men in their Alameda County Study.

Racial composition was not reported in Gillum's & Paffenbarger's study on sociocultural mobility, however it can be assumed that there were few, if any Blacks included, considering that these were Harvard graduates who were examined between 1939-1950 at a time when black enrollment in Ivy League schools was probably miniscule. Consequently, it is yet undetermined if these findings can be generalized to a black population. Despite the exclusion of Blacks in this study, important information can be obtained from this sudy, namely that intergernerational social and/or occupational mobility can potentially have a significant impact on health and illness, particularly as they relate to cardiovascular disorders.

## SUMMARY AND CONCLUSIONS

In conclusion, based upon the empirical research conducted in this area, it is apparent that the exact etiology of essential hypertension remains unknown. However, some evidence has been provided for a comprehensive explanation of this disorder that includes biological, psychological, and socio-ecological factors; aside from standard risk factors including age, diet, smoking, weight, cholesterol, and exercise. More recently, an increased focus on lifestyle factors and their contribution to the development of cardiovascular disorders has become an area of interest. Although standard risk factors are most widely publicized, a increased emphasis is being placed on psychological (e.g., personality characteristics) and socioecological factors (e.g. stress) that might influence the development of cardiovascular disorders.

Issues of the concept of race have added to the difficulty in ascertaining alleged racial differences in morbidity and mortality rates of cardiovascular disorders. Studies produced in the medical literature for the most part have failed to empirically establish the

existence of biological racial differences in prodromal signs of the disorder (i.e., plasma renin levels, heart rate). Contrary to what would be expected, Blacks exhibit lower plasma renin levels and heart rates than Whites, as well as an increased peripheral vascular resistance under stressor conditions, failing to support a beta-adrenergic underlying mechanism among Blacks and suggesting an alpha-adrenergic reponse. These findings tend to negate the applicability of sympathetic nervous system hypothesis of essential hypertension to Blacks.

Although some evidence has been provided that demonstrates that sodium retention is an established marker for the development of hypertension in Blacks, it is unclear at what level this difference appears. Contrary to previous thought that racial differences of sodium intake was and important factor, research has revealed that Blacks consume similar amounts of sodium as Whites. Subsequently, some have proposed that Blacks excrete sodium less efficiently than do Whites, although it is unclear why this is the case. Evidence has been produced that suggests that potassium, an important ion in the regulation of blood volume and therefore blood pressure, is deficient among Blacks and therefore upsets the ideal sodium/potassium ratio, resulting in higher blood pressure. Other studies have shown that sodium excretion inefficiency may be more a function of psychological distress than inherent biological differences. Additional evidence suggests that other psychosocial factors such as coping styles, socioeconomic status, education, and cognitive appraisal to threat, to name a few, are important influencing factors in the development and maintenance of essential hypertension.

It has been repeatedly established that genetic predisposition, based on parental history of hypertension, is predictive of cardiovascular hyperreactivity and increased risk for the development of essential hypertension; especially under conditions where individuals are required to actively cope with stressors in the environment. Studies on

cardiovascular reactivity demonstrate that individuals who have initial baseline blood pressure elevations, due to parental history, tended to have higher blood pressure levels during psychosocial stressors than individuals whose baseline blood pressure were normal. This evidence promotes an interactional model of biological and psychosocial factors in the conceptualization of this disorder.

Since biological hypotheses have failed to provide conclusive evidence of inter-racial differences in the development of essential hypertension, attention has turned to the investigation of intra-racial differences. It has been established that even though Blacks do tend to have disproportionately greater prevalence and incidence rates of hypertension than Whites, risk factors vary within this group. Psychosocial risk factors among Whites primarily has become an area of concern. Although Type A Behavior Pattern has been extensively studied among middle class white males, as a potential predictor of cardiovascular heart disease, it has not been investigated among Blacks, especially black women. Although the findings on TABP have changed in recent years from implicating a personality type of one who is an impatient workaholic to one who exhibits a signifcant amount of maladaptive emotions (i.e., hostility, depression) it is unclear if these results generalize to Blacks. Furthermore, it is unclear if TABP is predictive of hypertension in Whites or Blacks since each is considered a separate risk factor. Research on Blacks in general and black women in particular, is clearly need in this area.

Some research has provided evidence on the effect of personality characteristics on essential hypertension in Blacks. Sherman James' research on John Henryism, using an active coping model of the development of essential hypertension, suggests that psychological factors (i.e., increased mental vigor) and socioeconomic factors (i.e., decreased income and education) interact to have a mediating impact on blood pressure. Based on these findings, James suggests that future research focus on the role of family

history of cardiovascular disorders and environmental factors such as social mobility on blood pressure level.

Studies on social mobility have provided evidence that suggest that socioeconomic, cultural, occupational mobility each can have deleterious effects on the body as they relate to the development of CHD. This study incorporates John Henryism, Social Mobility and their independent as well as interactive effects on blood pressure, while controlling for family history of essential hypertension.

## Hypotheses

Based upon the previously cited studies that support a biopsychosocial approach to understanding the eitology of essential hypertension, the data will be statistically analyzed to test the following hypotheses:

1) There will not be a significant main effect of John Henryism on mean systolic blood pressure.

2) There will not be a significant main effect of John Henryism on mean diastolic blood pressure.

3) There will not be a significant main effect of social mobility on mean systolic blood pressure.

4) There will not be a significant main effect of social mobility on mean diastolic blood pressure.

5) There will be a significant interaction between John Henryism and social mobility on mean systolic blood pressure.

6) There will be a significant interaction between John Henryism and Social Mobility on mean diastolic blood pressure.

# CHAPTER III

## METHOD

### Subjects

Prior to recruiting subjects, a power analysis was conducted to obtain the optimum sample size necessary for achieving .80 power to detect a moderate effect size (.25) with an alpha level of .05 (.24 was estimated to be the approximate effect size of previous research on John Henryism and hypertension). The results of this analysis, using analysis of covariance (ANCOVA) tables provided in Cohen (1977), revealed an appropriate sample size of 120 subjects.

Potential subjects, (black women between 21 and 60 years of age) were recruited individually and in groups from the staff and patients of The University of Chicago Hospitals Hypertension Clinic, and Nutrition and Weight Control Clinic groups. Staff as well as patients were recruited in order to obtain a sufficient number of normotensives to produce a range of hypertension "scores" adequate for sufficiently powerful statistical tests (i.e., so that a failure to support the stated hypotheses could not be explained by a restriction of range on the major independent and dependent variables).

A total of 119 subjects volunteered to participate after an explanation of the study was given and after signing a consent form (see Appendix A). The mean age for all subjects was 41 years. Twenty-nine percent were single, 40 % were married, 20% divorced/separated, and 5 % widowed. They had an average of one child and four siblings. Forty-seven percent of the sample was classified as hypertensive and 45 percent normotensive; the remainder was unclassifed. Of the hypertensive subjects, 30 percent were on medication and 65 percent were not. Medication status could not be determined for

five percent of the subjects.

Design

The major design for the study was a 2 (John Henryism) X 2 (Social Mobility) analysis of covariance on systolic and diastolic blood pressure. Covariates in this and supplementary multiple regression analyses were subject's age, education (number of years completed in school), body mass index (BMI=weight in kilograms/height in centimeters$^2$), smoking rate (number of cigarettes smoked per day), medication usage (use or nonuse of antihypertensive medication), family history of hypertension in either parent, and head of the household educational level when the subject was 16 years of age. The set of covariates was chosen in order to control for the effects of variables that prior research has shown to be related to hypertension.

Instrumentation

The following instruments were used to collect the data on the primary independent and dependent variables and covariates: Demographic Questionnaire (DQ), John Henrysim Active Coping Scale-12 (JHAC-12; James, 1987), Duncan Socioeconomic Index Revised (MSEI2; Powers, 1982). A copy of these instruments is included in appendices B,C, and D respectively.

Demographic Questionnaire (DQ). The DQ was designed to provide demographic information as well as information on subject's stress level, health risk behaviors (i.e.. smoking, exercise), and current job title. The questionnaire contains 13 items and was completed in an interview format with each subject. One hundred fourteen subjects were interviewed face to face on the hospital premises. Telephone interviews were conducted

with five individuals who were unable to be interviewed in person.

John Henryism Active Coping Scale-12 (JHAC-12). The JHAC-12 is a 12 item scale that provides measures of overall John Henryism and three subcomponents of John Henryism: 1) efficacious mental and physical vigor; 2) a comittment to hard work; and 3) a single-minded determinaton to achieve one's goals. Each item is rated on a five point (1=completely false, 5=completely true) Likert scale.

Internal consistency estimates for total scale scores obtained in prior studies (James, 1987) have ranged from .67 for black men to .74 for white women. Internal consistency calculated by Chronbach alpha in the present sample (.72) was nearly identical to the coefficient obtained for black women (.71) by James (1987). Prior validity studies (James, 1982) have shown theory-consistent positive relationships between total JHAC-12 scores and blood pressure levels, especially for subjects of low educational background.

Duncan Socioeconomic Index Revised (MSEI2). The MSEI2 is a revision of the original Duncan SEI (socioeconomic index). The MSEI2 scale rank orders 426 occupations on an ordinal scale from 1 to 100 based upon education and income criteria that are required for each job. Occupations requiring more education and paying higher salaries receive higher rankings and lower paying jobs requiring less education receive lower rankings. According to Stevens (1987) quantitative criteria (i.e., education and income) as opposed to qualitative criteria (i.e., prestige ratings), are better estimations of socioeconomic status (SES) even though most of these scales are highly intercorrelated. Thus, Stevens recommends that studies of intergenerational social mobility use scales that employ quantitative criteria (i.e., Duncan SEI and revised scales).

Featherman and Stevens (in Powers, 1982) recommend the MSEI2 as the best SES index for most research despite the fact that it was based upon characteristics of the male

labor force, using the 1970 census. Although attempts have been made to develop a scale that uses the total labor force of both men and women (e.g., Duncan TSEI2), the MSEI2 has been shown to be more sensitive to making a blue-collar/white-collar distinction among occupations (Powers, 1982). Validity evidence published on the MSEI2 indicates that it correlates highly with other frequently used quantitative and qualitative measures of SES (e.g., .94 with the original Duncan SEI and .89 with Siegel prestige rating respectively) (Powers,1982).

Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP). SBP and DBP were measured using a Bauamanometer Calibrated V-Lok Cuff while subjects remained in a standing position. SBP was recorded when the first Korotkoff sound was heard. DBP was recorded using the phase five (that period when all sounds disappeared). Two cuff sizes were used including large and regular. Proper cuff size was determined by assessing the width of the inflatable bladder inside the cuff, which should be at least 20% wider than the diameter of the screenee's arm. The cuff was then placed at least one inch above the natural crease across the inner aspect of the elbow. The inflatable rubber bladder was then centered directly over the brachial artery.

## Procedure

Immediately prior to data collection, subjects were given a brief explanation of the study and an opportunity to ask any questions. Then a structured interview was conducted using the Demographic Questionnaire. The interviews took place in private office space, or if necessary on the telephone. Following the interview, subjects were then given the JHAC-12 and the JAS to complete and return immediately to the researcher. On occasions in which a subject did not have her glasses or was lying on an examination table awaiting

medical attention, the JHAC-12 was administered orally by the experimenter. Blood pressure and weight were obtained immediately before or after the interview by a registered nurse.

Information obtained from the DQ was cross checked with information in the medical chart of subjects who were patients in the Hypertension, and Nutrition and Weight Control Clinics. This cross check could not be completed for employees since they had no available medical record in the clinics.

The researcher conducted an individual debriefing interview immediately after data collection to answer any questions regarding the study and to provide the subject with the name and phone number of the researcher if information was needed at a later date. All subjects were offered a summary of the research findings by mail (see Appendix E).

Data Analysis

The primary hypotheses of this study were tested by two separate (diastolic blood pressure and systolic blood pressure), 2 (High, Low John Henryism) X 2 (concordant, discordant social mobility) analyses of covariance. As indicated previously, covariates in both analyses included subject's age, education, body mass index, smoking rate, medication usage,family history of hypertension, and head of the household educational level when the subject was 16 years of age. High and Low John Henryism groups were created by a median split procedure (high $> 50$, low $\leq 50$).

Concordance and discordance on intergenerational social mobility was determined by a multistep procedure. First, the subject;s current occupation was classified as blue or white collar on the basis of a previously established (Hauser & Featherman, 1977) cut off score on the MSEI2 (blue collar $<31.99$, white collar $\geq 31.99$). Second, the occupation of the head of the household when the subject was 16 was classified as blue or white collar

using the same cut-off score. Third, the subject's current level of intergenerational social mobility was classified as discordant if her current occupation received a different classification at step 1 than did the head of the household occupation at step 2. A concordant classification was made if the occupations were classified identically at step 1 and 2. All codings were carried out by the experimenter. A second, independent rater classified the occupations of 20 randomly selected subjects as concordant or discordant. Interrater agreement on these 20 subjects, calculated by the formula number of agreements/number of agreements + number of disagreements X 100 was 100 %.

The analysis of covariance results were replicated by two hierarchical multiple regression analyses because of the potential for reduced power associated with the dichotomous classifications on the independent variables. In these analyses, diastolic and systolic blood pressure were regressed in separate analyses on (in order) the covariates (entered as a single block), JHAC-12 scores, MSEI2 scores, and a John Henryism X social mobility interaction term. In both analyses, JHAC-12 and MSEI2 scores were entered as continuous variables. In order to create a continuous scale from MSEI2 data, difference scores were computed between the numerical rating of the subject's and head of household occupations. Positive numbers on this index represent a condition of upward mobility and negative numbers signify downward mobility. Finally, raw scores on all variables were standardized before being entered into the multiple regression analyses in order to reduce potential multicollinearity among the independent variables.

# CHAPTER IV

# RESULTS

## Treatment of Missing Data

The treatment of missing data varied according to the variables used in the particular analysis. Subjects who did not know if either of their parents had a family history of hypertension were coded 3 (don't know). Subjects who did not know how much education their parents/head of household completed in school, but knew only that they did not finish high school, were given the mean of all subjects' heads of households who also did not complete high school (mean=7.00). Those subjects who had no idea what grade their head of household completed were coded as missing.

Two demographic variables (age of onset of hypertension and cholesterol level) were relevant only for hypertensives. Information was obtained on age of onset for 33 hypertensive subjects and on cholesterol for 40 hypertensives.

## Sample Description

An analysis of Table 1 demonstrates that this sample of urban black women had a mean age of 41 years (sd=9.64, range=21-65) and 13 years (sd=2.63, range=3-20) of education. Their mean body mass index was 30 (sd=7.54, range=19-55) and they smoked an average of four (sd=8.46, range=0-40) cigarettes per day. Sixty-seven percent of subjects grew up in a large city and only 20% were married. Table 2 shows that 70% of subjects had a positive family history of hypertension, while 51% of the participants were classified as hypertensive. Thirty-two percent of the total sample were taking antihypertensive medication. Tables 3 and 4 show a comparison of normotensives to two

groups of hypertensives (those taking medication and those not taking medication).

## Intercorrelations among Variables

The intercorrelations among traditional demographic risk factors (age, smoking rate, body mass index, education, and cholesterol level), independent, and dependent variables are summarized in Table 5. Analyses of risk factors--blood pressure relationships revealed some expected and unexpected results. As expected, age correlated positively with both SBP ($r=.51$, $p<.001$) and DBP ($r=.44$, $p<.001$). Both indices of education correlated inversely with SBP (subject's education, $r=-.42$, $p<.001$; head of household education, $r=-.37$, $p<.001$ ) and DBP (subject education, $r=-.48$, $p<.001$; head of household education, $r=-.39$, $p<.001$). Smoking rate correlated significantly as expected, with DBP ($r=.20$, $p<.01$), but, contrary to expectations, did not show a significant relationship with SBP ($r=.14$, $p<.10$). Also, contrary to expectations, body mass index did not correlate significantly with either index of blood pressure (SBP: $r=.10$, $p<.15$; DBP: $r=.13$, $p<.08$).

Table 1

Full Sample Characteristics on Continuous Variables

| Variable | Mean | Stan Dev | n |
|---|---|---|---|
| Age | 40.90 | 9.64 | 118 |
| John Henryism | 49.22 | 6.22 | 116 |
| Smoking | 4.39 | 8.45 | 115 |
| Subject's Education | 13.09 | 2.63 | 114 |
| Head of HH Education | 9.71 | 3.78 | 111 |
| MSEI2 Difference Score | 14.67 | 18.06 | 113 |
| Systolic Blood Pressure | 131.82 | 24.42 | 109 |
| Diastolic Blood Pressure | 87.73 | 13.91 | 109 |
| Age of Onset | 31.97 | 11.61 | 33 |
| Exercise | 2.06 | 9.35 | 115 |
| Cholesterol | 209.20 | 41.10 | 40 |
| Stress Level * | 7.38 | 2.75 | 113 |
| # of Children | 1.47 | 1.40 | 114 |
| # of Siblings | 4.05 | 3.31 | 114 |
| Body Mass Index | 29.59 | 7.53 | 112 |

Note* MSEI2 range= 13.35 minimum-88.49 maximum
* Stress range=1minimum-10 maximum

Table 2

Full Sample Characteristics on Discrete Variables

| Variables | Frequency | Percent |
|---|---|---|
| John Henryism Median Split | | |
| High | 58 | 50.0 |
| Low | 58 | 50.0 |
| Missing | 4 | 00 |
| Family History of Hypertension | | |
| Positive | 83 | 69.7 |
| Negative | 25 | 21.0 |
| Don't Know | 11 | 9.2 |
| Missing | 1 | .8 |
| Diagnosis | | |
| Hypertensive | 57 | 51.4 |
| Normotensive | 54 | 48.6 |
| Missing | 9 | 00 |
| Medication Usage | | |
| With Medication | 37 | 31.9 |
| Without Medication | 79 | 68.1 |
| Missing | 4 | 00 |
| City Size | | |
| Open country | 00 | 00 |
| Farm | 13 | 11.4 |
| Population < 50,000 | 10 | 8.8 |
| Population 50,000-250,000 | 10 | 8.8 |
| Suburb of a large city | 5 | 4.4 |
| Population > 250,000 | 76 | 66.7 |
| Missing | 6 | 0 |
| Marital Status | | |
| Single | 35 | 30.4 |
| Married | 49 | 42.6 |
| Divorced | 24 | 20.9 |
| Widowed | 7 | 6.1 |
| Missing | 5 | 0 |
| Social Mobility | | |
| Concordant | 54 | 47.8 |
| Discordant | 59 | 52.2 |
| Missing | 7 | 0 |

Table 3

Comparison of Normotensives and Hypertensives (with & without meds) on Continuous Variables

| | Normotensive | | Hypertensive (meds) | | Hypertensive (no meds) | |
|---|---|---|---|---|---|---|
| Variable | m | sd | m | sd | m | sd |
| JH | 49.13 | 5.77 | 49.55 | 7.36 | 48.95 | 5.97 |
| Mobility | 20.81 | 18.2 | 8.03 | 15.79 | 9.50 | 16.35 |
| Age | 36.46 | 7.77 | 47.58 | 8.90 | 43.66 | 8.17 |
| Smoking | 2.07 | 5.49 | 7.33 | 11.70 | 4.57 | 6.69 |
| $S_s$ Ed | 14.26 | 2.24 | 12.02 | 2.03 | 11.45 | 2.72 |
| HH Ed | 11.36 | 3.53 | 8.34 | 3.34 | 7.83 | 2.87 |
| Exercise [a] | 1.51 | 2.16 | .52 | 1.13 | 2.00 | 0.00 |
| Stress [b] | 7.34 | 2.44 | 7.05 | 1.34 | 7.90 | 2.63 |
| # Children | 1.53 | 1.05 | 1.55 | 2.00 | 1.25 | 1.07 |
| # Siblings | 3.51 | 3.15 | 4.25 | 3.33 | 4.55 | 3.64 |
| BMI | 8.02 | 5.98 | 30.72 | 8.86 | .33 | 8.10 |

Note. JH=John Henryism; Mobility=social mobility; $S_s$ Ed=subject'seducation level; HHED=head of household's education level; BMI=Body Mass Index; meds=medication

[a] hours of exercise per week;
[b] stress level range--1= minimum-10= maximum

Table 4

Comparison of Normotensives and Hypertensives (with & without meds) on Categorical Variables

| | Normotensive | | Hypertensive* | | Hypertensive** | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| John Henryism | | | | | | |
| Low | 27 | 51.9 | 4 | 38.9 | 12 | 60.0 |
| High | 25 | 48.1 | 22 | 61.1 | 8 | 40.0 |
| Social Mobility | | | | | | |
| Concordance | 16 | 30.8 | 23 | 65.7 | 12 | 60.0 |
| Discordance | 36 | 69.2 | 12 | 34.3 | 8 | 40.0 |
| City Size | | | | | | |
| country | 0 | 00.0 | 00 | 00.0 | 00 | 00.0 |
| farm | 4 | 7.7 | 4 | 11.1 | 5 | 25.0 |
| <50,000 | 4 | 7.7 | 2 | 5.6 | 3 | 15.0 |
| 50,000-25,00 | 6 | 11.5 | 3 | 8.3 | 0 | 0.0 |
| suburb | 3 | 5.8 | 1 | 2.8 | 1 | 5.0 |
| >250,000 | 35 | 67.3 | 26 | 72.2 | 11 | 55.0 |
| Marital Status | | | | | | |
| Single | 12 | 23.1 | 1 | 38.9 | 7 | 33.3 |
| Married | 28 | 53.8 | 11 | 30.6 | 9 | 42.9 |
| Divorced | 10 | 19.2 | 7 | 19.4 | 4 | 19.0 |
| Widowed | 2 | 3.8 | 4 | 11.1 | 1 | 4.8 |
| Family History of Hypertension | | | | | | |
| Positive | 32 | 61.5 | 30 | 83.3 | 18 | 85.7 |
| Negative | 19 | 36.5 | 2 | 5.6 | 1 | 4.8 |
| Missing | 1 | 1.9 | 4 | 11.1 | 2 | 9.5 |

Note: City Size=population

*with medication **without medication

Table 5 reveals that John Henryism, consistent with the hypotheses of this study, was not significantly related to either SBP (r=-.01, p <.45) or DBP (r=.09, p <.016). Intergenerational social mobility, as measured by the MSEI2 difference score procedure, was unexpectedly related inversely to SBP (r=-.26, p <.01) as well as to DBP (r=-.23, p <.01).

Lastly, it is important to note that there was not a strong correlation (r=-.01, p<.44) between the two independent variables, John Henryism and MSEI2 difference scores, which is important when considering their "independent" effects on blood pressure. The assumption is that these two variables are not themselves related and therefore have separate and distinct influences on the dependent variable.

## Tests of Hypotheses

It was predicted that no significant main effects on SBP of John Henryism (Hypothesis 1) or social mobility (Hypothesis 3), but a significant John Henryism X Social Mobility interaction (Hypothesis 5) would be found. Similarly, for DBP, a significant interaction (Hypothesis 6) but no significant main effects of John Henryism (Hypothesis 2) or social mobility (Hypothesis 4) were revealed.

Results of hypotheses tests were consistent across both primary analyses. Specifically, the 2 (John Henryism) X 2 (social mobility) ANCOVA revealed no significant main effects of social mobility on either SBP (see Table 6) nor DBP (see Table 7). Neither the main effect of John Henryism nor the John Henryism X Social Mobility interaction were significant on SBP and DBP analyses. Hierarchical regressions of SBP and DBP on the covariates, John Henryism, social mobility, and interaction terms revealed that only social mobility (see Table 8) contributed significantly to increments in SBP and DBP variance after the influence of the covariates had been controlled by entering them in the

first step. The interaction term also did not account for significant increments in SBP or DBP variance over and above that accounted for by the covariates, John Henryism and social mobility. However, when medication was entered as an additional covariate into the regression model, there were no significant main effects of John Henryism or social mobility on SBP or DBP. Although, this model yielded significant results for the independent contribution of medication to the variance in SBP, the John Henryism X Social Mobility interaction term also accounted for a significant ($\Delta R^2$=.03, $p < .05$) increment in SBP variance over an above that accounted for by family history, medication, John Henryism, and social mobility. The same did not hold true for the effects on DBP analyses.

Finally, supplementary 2 (John Henryism) X 2 (Social Mobility) analyses of variance revealed a significant main effects only for social mobility on SBP, but not DBP (see Appendix F, Tables 14 and 15). Thus, it does not appear that the results were a function of the statistical controls used in the ANCOVA and multiple regression. In addition, the regression model that entered medication as one of the predictor variables, rendered a significant ($\Delta R^2$=.03, $p < .05$) interaction between John Henryism and social mobility on SBP. The same did not hold true for the effects on DBP ($\Delta R^2$=.01, $p < .24$) analyses.

Covariates that contributed to a significant amount of the variance in blood pressure included age on SBP ($F(1)=9.84$, $p < .01$) and DBP ($F(1)=4.11$, $p < .05$) in the ANCOVA and medication on SBP ($R^2$=.13, $p < .01$) and DBP ($R^2$=.14, $p < .05$) in the hierarchical multiple regression that included the entire sample. There was also a significant relationship between subject's educational level and blood pressure (SBP, r=-.42, $p < .001$; DBP, r=-.44, $p < .001$); head of household's educational level and blood pressure (SBP, r=-.37, $p < .001$; DBP, r=-.39, $p < .001$); cholesterol and blood pressure (SBP, r=.31, $p < .05$;

DBP, r=.34, p <.01), and smoking (DBP, r=.20, p <.01); although these variables did not contribute to a significant amount of the variance in the ANCOVA. Age was the only standard risk factor that contributed to a significant amount of the variance across analyses. Contrary to the literature, no significant relationships were found for body mass index and blood pressure.

Table 5

Intercorrelations Among Selected Demographic Risk Factors and Blood Pressure

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Age | | -.44*** | -.38*** | .14 | .19 | -.04 | .04 | -.16* | .51*** | .44*** |
| 2. $S_s$ Ed | -.44*** | | .45*** | -.21** | -.28* | -.09 | .03 | .37*** | -.42*** | .44*** |
| 3. HHED | -.38*** | .45*** | | -.13 | -.37*** | -.12 | -.08 | .00 | -.37 | -.39*** |
| 4. BMI | .14 | -.21** | -.13 | | -.05 | .10 | .09 | -.12 | .10 | .13 |
| 5. CHOL | .19 | -.28* | -.37*** | -.05 | | .39*** | -.08 | -.08 | .31* | .34*** |
| 6. Smoking | -.04 | -.09 | -.12 | .10 | .39*** | | -.13 | -.09 | .14 | .20** |
| 7. JH | .04 | .03 | -.08 | -.09 | -.08 | -.13 | | -.01 | -.01 | .10 |
| 8. SM | -.16* | .37*** | .00 | .12 | -.08 | -.09 | -.01 | | -.26** | .23** |
| 9. SBP | .51*** | -.42*** | -.37 | .10 | .31* | .14 | -.01 | -.26** | | .80*** |
| 10. DBP | .44*** | -.44*** | -.39** | .13 | .38** | .20** | .10 | -.23** | .80*** | |

Note: $S_s$ Ed=subject's educational level; HHED=head of household's educational level; CHOL=cholesterol; JH=John Henryism; SM=social mobility

* p<.05 **p<.01 ***p <.001

Table 6

2 (John Henryism) X 2 (Social Mobility) ANCOVA Summary Table for SBP

| Source of Variation | DF | F | p |
|---|---|---|---|
| Covariates | 7 | 6.52 | .00 ** |
| Family History | 1 | .91 | .34 |
| Age | 1 | 9.84 | .00 ** |
| Medication | 1 | .51 | .47 |
| $S_s$ Education | 1 | 2.62 | .10 |
| Body Mass Index | 1 | .42 | .51 |
| Smoking | 1 | 2.18 | .14 |
| HH Education | 1 | 1.65 | .20 |
| Main Effects (Overall) | 2 | .82 | .44 |
| JH Median Split | 1 | 1.00 | .32 |
| CONDIS | 1 | .59 | .44 |
| 2-Way Interaction | 1 | .94 | .33 |
| JHMS X CONDIS | 1 | .94 | .33 |

Note: $S_s$=Subject's Education Level
HH=Head of Household's Education Level
JH=John Henryism
CONDIS=Concordant/Discordant Social Mobility

* p <.05 **p <.01 ***p <.001

Table 7

2 (John Henryism) X 2 (Social Mobility) ANCOVA Summary Table for DBP

| Source of Variation | DF | F | p |
|---|---|---|---|
| Covariates | 7 | 5.93 | .00 ** |
| Family History | 1 | .82 | .36 |
| Age | 1 | 4.11 | .04 * |
| Medication | 1 | .64 | .42 |
| Subject's Education | 1 | 3.35 | .07 |
| Body Mass Index | 1 | .02 | .88 |
| Smoking | 1 | 3.47 | .06 |
| Head of Household's Education | 1 | 2.91 | .09 |
| Main Effects (Overall) | 2 | .01 | .98 |
| John Henryism Median Split | 1 | .00 | .95 |
| Concordant/Discordant Social Mobility | 1 | .03 | .85 |
| 2-Way Interactions | 1 | .02 | .87 |
| JH Median Split X | 1 | .02 | .87 |
| CONDIS | 1 | .02 | .87 |

Note: JH=John Henryism
CONDIS=Concordant/Discordant Social Mobility

* $p < .05$ **$p < .01$ ***$p < .001$

In another hierarchical regression model that excluded downwardly mobile subjects (see Table 10), the only significant finding was for medication and its contribution to the variance in systolic ($\Delta R^2$=.11, p <.01) and diastolic ($\Delta R^2$=.11, p <.01) blood pressure. However, there was not a significant interaction between John Henryism and social mobility on either systolic or diastolic blood pressure.

## Summary

In summary, strong evidence was provided for the implication of some standard risk factors and their contribution to the variance in systolic and diastolic blood pressure, but not others. The relationship of age to blood pressure was consistently demonstrated throughout various analyses. Significant relationships between age and blood pressure were found in both the bivariate and multivariate analyses. Smoking and cholesterol level were also related to diastolic blood pressure as the literature indicates. However, contrary to the literature there was not a significant relationship between body mass index or family history of hypertension and blood pressure.

Table 8

Hierarchical Multiple Regression Summary Table (without medication entered)

| Variable | r | R | $R^2$ | $\Delta R^2$ | F$\Delta$ | p |
|---|---|---|---|---|---|---|
| | | | **SBP** | | | |
| Family History | -.13 | .13 | .02 | .02 | 1.64 | .20 |
| John Henryism | -.01 | .13 | .02 | .00 | .01 | .93 |
| Mobility | -.25 | .29 | .08 | .07 | 7.41 | .01** |
| John Henryism X Mobility | -.15 | .33 | .11 | .03 | 2.88 | .09 |
| | | | **DBP** | | | |
| Family History | -.15 | .15 | .02 | .02 | 2.29 | .13 |
| John Henryism | .09 | .17 | .03 | .01 | .85 | .35 |
| Mobility | -.22 | .29 | .08 | .05 | 5.62 | .02 * |
| John Henryism X Mobility | -.11 | .30 | .09 | .01 | .77 | .38 |

*p <.05 ** p <.001 ***p <.00

Table 9

Hierarchical Multiple Regression Summary Table (with medication entered)

| Variable | r | R | $R^2$ | $\Delta R^2$ | F$\Delta$ | p |
|---|---|---|---|---|---|---|
| | | **SBP** | | | | |
| Family History | -.13 | .13 | .02 | .02 | 1.65 | .20 |
| Medication | -.34 | .36 | .13 | .11 | 13.20 | .00** |
| John Henryism | -.01 | .36 | .13 | .00 | .08 | .77 |
| Mobility | -.25 | .40 | .16 | .03 | 3.65 | .06 |
| JH X Mobility | -.15 | .44 | .20 | .03 | 4.13 | .05* |
| | | **DBP** | | | | |
| Family History | -.15 | .15 | .02 | .02 | 2.29 | .13 |
| Medication | -.35 | .37 | .14 | .12 | 13.49 | .00** |
| John Henryism | .09 | .38 | .14 | .00 | .61 | .44 |
| Mobility | -.22 | .40 | .16 | .02 | 2.40 | .13 |
| JH X Mobility | -.11 | .42 | .17 | .01 | 1.38 | .24 |

Note: JH=John Henryism *p < .05 **p < .01 ***p <.001

Table 10

Hierarchical Multiple Regression Summary Table (without downwardly mobile subjects)

| Variable | r | R | $R^2$ | $\Delta R^2$ | F$\Delta$ | p |
|---|---|---|---|---|---|---|
| | | | **SBP** | | | |
| Family History | -.14 | .14 | .02 | .02 | 1.53 | .22 |
| Medication | -.35 | .37 | .14 | .11 | 10.73 | .00** |
| JH | -.08 | .38 | .14 | .00 | .70 | .40 |
| Mobility | -.27 | .42 | .18 | .03 | 3.01 | .00 |
| JH X Mobility | -.06 | .45 | .20 | .03 | 2.62 | .11 |
| | | | **DBP** | | | |
| Family History | -.20 | .20 | . 04 | .04 | 3.23 | .07 |
| Medication | -.35 | .39 | .15 | .11 | 10.43 | .00** |
| JH | .06 | .39 | .15 | .00 | .28 | .60 |
| Mobility | -.19 | .41 | .16 | .01 | .99 | .32 |
| JH X Mobility | -.07 | .42 | .17 | .01 | 1.19 | .28 |

Note: JH=John Henryism *p <.05 **p <.01 ***p <.001

Among psychosocial risk factors, only social mobility accounted for significant variance on systolic and diastolic blood pressure across the various analyses. Separate multiple regression anayses were most revealing in that they provided support for the independent effect of social mobility on blood systolic and diastolic blood pressure when medication was not controlled, as well as for the interactional hypothesis of John Henryism and social mobility on systolic blood pressure when medication was controlled. A separate regression analysis that excluded downwardly mobile subjects revealed that medication was the only significant predictor variable that contributed to a significant amount of the variance in both systolic and diastolic blood pressure.

No evidence was provided in any of the analyses to substantiate the hypothesis that family history of hypertension had any relationship to blood pressure. However, the incidence rate of family history of hypertension was higher in hypertensives than normotensives.

# CHAPTER V

# DISCUSSION

## Hypotheses 1 and 2.

*There will not be a significant main effect of John Henryism on mean systolic or diastolic blood pressure.*

The statistical tests used to test the effect of John Henryism on blood pressure revealed that there were no significant main effects for John Henryism on systolic or diastolic blood pressure in any of the analyses. This finding is consistent with the James et al. (1983) study on the effects of John Henryism and education on blood pressure. In James' study, a 2 X 2 ANCOVA was used, with two levels of John Henryism (high/low) and two levels of education (high/low). Covariates included age, Quetelet Index, time of day, and number of cigarettes smoked daily. These findings suggest that high levels of John Henryism alone are not associated with sustained blood pressure elevations. Rather, it appears to be the interaction of this personality characteristic with insufficient coping resources (i.e., education) that, according to James et al. (1983), tends to be related to blood pressure elevations. Thus, it may be that individuals who exhibit the characteristics that typify John Henryism, yet who have adquate coping resources, may not be as susceptible to the deleterious effects on blood pressure. Since the sample for this study had a mean educational level of 13 years and 58% were classified as white collar it is possible that adequate coping resources were available to offset potential blood pressure elevations.

Another interpretation for the results of the ANCOVA and ANOVA is that the median split procedure used to dichotomize the John Henryism variable may have r
educed the power of the analyses to detect a relationship between John Henryism and blood

pressure. Clearly, a significant amount of information is lost when categories are established using a median split. As a result, multiple regression analyses were used to alleviate this problem. In these analyses, John Henryism was used as a continuous variable.

Multiple regression analyses, however, also did not yield significant direct effects of John Henryism on systolic or diastolic blood pressure. The failure to do so may have been to the appropriateness of the JHAC12 in measuring John Henryism. It is possible that given the high educational level of this sample compared to James' sample, it may have had an impact on how subjects responded to the test items. It may be that people who have worked hard to get ahead educationally may exhibit John Henryism characteristics, but may in turn have more resources for coping. James argues that it is the lack of coping resources, that interacts with high John Henryism to produce increased levels of blood pressure. However, it is interesting to note that the mean John Henryism scores in James' study and this study were similar. The mean for James' study was 21 out of a possible total of 24, while the mean for this study was 50 out of 60. Also, the ranges obtained on John Henryism scores in this sample (26-60 out of a potential range of 12-60), appeared to be quite restricted and the distribution of scores skewed (i.e, most subjects scored in the upper range of John Henryism). Thus, it may be that our failure (as well as that of James et al., 1983) to obtain significant relationships between John Henryism and blood pressure is more methodological than substantive. It is, therefore, necessary that future research on John Henryism and blood pressure obtain subjects who are maximally heterogeneous with respect to John Henryism scores. Only then can range restriction be ruled out as a plausible rival hypothesis for "no significant relationships" results and only then can confidence be placed on the generalization that John Henryism and blood pressure are unrelated.

Hypotheses 3 and 4

*There will not be a signficant main effect of social mobility on mean systolic and diastolic blood pressure.*

The statistical tests used to assess the effect of social mobility on both systolic and diastolic blood pressure rendered a significant main effect for social mobility on systolic blood pressure only when medication was not controlled. Considering the importance of medication on blood pressure, this finding has little meaning in and of itself. As a result, even though the finding is not in accordance with the stated hypothesis, it does not carry much weight in terms of explanatory power without consideration of medication usage. In addition, the failure to demonstrate significant findings using ANCOVA is consistent with research findings (Gillum & Paffenbarger, 1978) that failed to reveal an increased incidence and prevalence of hypertension in male Harvard graduates who moved from a lower to a higher social stratum intergenerationally; even though the results indicated that intergenerational mobility was significantly related to other cardiovascular disorders (e.g., fatal coronary heart disease & myocardial infarction). Since the Harvard study probably consisted of white males, it is unclear if these findings would apply to Blacks. Also, with research findings (Light et al., 1987) that implicate a different underlying biological mechanism of essential hypertension in Blacks, the findings remain ambiguous as they apply to a black population.

Prior research on social mobility did not use cross sectional data, but instead have utilized prospective studies (Gillum and Paffenbarger,1978). The underlying assumption for using social mobility in this ANCOVA design was to continue James' line of thinking that John Henryism combined with other psychosocial variables (i.e., socioeconomic status, social mobility, education) may have an interaction effect on blood pressure

elevation. Although James et al. (1983) found that education had a significant main effect on blood pressure, after controlling for various covariates (i.e., age, body mass, time of day), the differences in systolic blood pressure were not longer significant. Thus, the findings in this study are consistent with the stated hypothesis and with previous research.

Contrary to the results of analyses of variance, multiple regression analysis revealed that social mobility contributed to a significant amount of the variance in systolic blood pressure after family history of hypertension and John Henryism were accounted for. However, this was not true in a separate regression that entered medication into the equation. In this latter regression model, medication attenuated the effects of social mobility on both systolic and diastolic blood pressure. In another regression model that considered only normotensives, none of the variables contributed to a significant amount of the variance. Of course, in this analysis medication was not a consideration for these subjects. This finding is surprising given the appreciable difference in mean MSEI2 difference scores for hypertensives versus normotensives as indicated in Table 3. The social mobility index for normotensives is more than twice that for hypertensives with and without medication. One explanation for the failure to obtain significant results in this analysis is that there was a restricted range of blood pressure among normotensives due to basement and ceiling effects. The mere definition of normotension (SBP≤120, DBP≤80) tends to restrict the range of blood pressure readings since average is approximately 120/80.

The partial regression that used only upwardly mobile subjects in the equation demonstrated the importance of including downwardly mobile individuals as well. There were no significant findings in this model of MSEI2 difference scores compared to the equation that included downwardly mobile subjects. The most impressive findings in this model were those found when medication was not entered into the equation, yielding a

significant main effect of social mobility on systolic and diastolic blood pressure. The negative *r* value indicates an inverse relationship between MSEI2 difference scores and systolic blood pressure. This relationship indicates that as MSEI2 scores increased, (i.e., increased social mobility), blood pressure levels decreased. However, when medication was entered into the analysis the significant findings for social mobility disappeared. Considering the importance of medication on blood pressure, the failure to find significant main effects after controlling for medication should be given more weight than the significant main effects when medication was not controlled.

Hypotheses 5 and 6

*There will be a significant interaction between John Henyism and social mobility on mean systolic and diastolic blood pressure.*

No significant interactions were found between John Henryism and social mobility on systolic or diastolic blood pressure in any of the analyses of variance designs. These findings, thus, do not support the stated hypothesis. However,
a separate regression model to predict systolic blood pressure was constructed entering medication second, as indicated in Table 9. The data show that the interaction between John Henryism and MSEI2 difference scores on systolic blood pressure was significant. These findings support the hypothesis that a John Henryism coping style, interacts with intergenerational mobility, in its relationship to blood pressure. The relationship in this case, if medication is controlled however, happens to be negative. However, when a regression model was used that excluded downwardly mobile subjects, the significant interaction between these two variables disappeared; leaving medication as the only variable that contributed to a significant amount of the variance.

Based on the data presented in Table 9, it can be construed that after family history of

hypertension and John Henryism were accounted for, social mobility contributed negatively to the variance in systolic blood pressure. The implicaton for this analysis is that greater differences between MSEI2 scores of subjects and their heads' of household scores was associated with lower, rather than higher, systolic and diastolic blood pressures. Based on the hypothesis that Blacks might tend to respond to the stress of social mobility via blood pressure elevations because of certain genetic predispositions (i.e., peripheral vasoconstriction), one would expect to see elevations with greater intergenerational mobility rather than decrements. Even though Gillum and Paffenbarger's (1978) study did not support this hypothesis in white males, it was unclear if the results held true for black males. Since there are possibly different underlying mechanisms of essential hypertension in Blacks and Whites, one could hypothesize that Blacks might indeed manifest the stress of social mobility via this particular disorder since it is a more common manifestation among Blacks than Whites. However, this study did not support this hypothesis. On the contrary, the findings in this study suggest that other factors (e.g., income, education) may be operating to influence the association of social mobility with systolic blood pressure.

The direction of mobility is another important consideration. Downward mobility also seems to be a strong component of social mobility as it relates to blood pressure in Blacks given that MSEI2 difference scores contributed to a significant amount of the variance when both upward and downwardly mobile subjects were included in the regression equation. However, when downwardly mobile subjects were exluded, the relationship between upward mobility and blood pressure disappeared. Even though this finding supports the hypothesis that social mobility alone would not be significantly related to blood pressure, it is questionable as to whether or not it is for the reason that was expected. Based on these findings, it appears that upward mobility has a paradoxical protective effect of decreasing blood pressure.

One possible explanation is for upward mobility being more strongly related to blood pressure than downward mobility is that approximately 50 percent of the total downwardly mobile subjects (n=25) had difference scores that did not exceed ten points. When comparing the means of MSEI2 difference scores for upward mobility versus downward mobility, it becomes apparent that the greatest mean difference in these scores is among upwardly mobile (mean=21.48) subjects compared to downwardly mobile (mean=7.67) subjects. This disparity suggests that downward mobility in this sample may not be representative of movement between social strata given the small mean difference, but merely a small decrement within the same social stratum. Thus, the relationship between downward mobility and blood pressure should be looked at more closely in future research. In so doing, a greater range of scores reflecting socioeconomic status would be desirable to more adequately ascertain the effects of moving from a higher to a lower socioeconomic status.

Another explanation for the finding that social mobility, specifically upward mobility, was associated with in decreased blood pressure is that since subjects in this study had a mean educational level of 13 years, this may have constituted a sample individuals who were more well informed about health risks than their heads of household. Subsequent changes in standard risk factors (i.e., diet, exercise, smoking) may have contributed to lower blood pressure levels, despite the potential stress of social mobility. The negative correlations between subject's educational level and sytolic blood pressure, as well as subject's educational level and diastolic blood pressure support this alternative explanation. Although there was not a significant correlation between subjects' education level and smoking or exercise, the association was in the expected direction, the former negative and the latter positive. James' research on John Henryism did not address the issue of socioeconomic status and/or educational level on lifestyle habits. Future research in this

area might reveal information about how education and income contribute to preventative health measures.

Since the findings on the interaction of social mobility and John Henryism on blood pressure were not in the expected direction (i.e., blood pressure decrease rather than increase) the findings only partially support James' hypothesis that John Henryism alone is not significantly related to blood pressure elevation. Furthermore, consistent with James et al. (1983) hypothesis, it is the availability of coping resources that is crucial in providing a protection against sustained blood pressure elevations. This study provides tentative support for this hypothesis that individuals who are more educated may tend to have better lifestyle habits that in turn have a positive influence on risk for cardiovascular disorders. Consequently, social mobility per se does not necessarily contribute to the development of essential hypertension, especially in Blacks, who as a people have made significant economic gains in the last twenty years, resulting in upward mobility. However, it appears that the stressors of moving up the socioeconomic ladder may be offset by the positive changes in lifestyle that impact on health.

Effects of Covariates

ANCOVA was used to test the effects of John Henryism and social mobility on systolic and diastolic blood pressure, after controlling for seven covariates. These covariates included family history, age, medication usage, subject's education, body mass index, smoking, and subject's head of houseld education. The findings indicated that these covariates together accounted for a significant amount of the variance in this anaysis.

Age. Using ANCOVA, age accounted for a significant proportion of the variance compared to the other covariates. Age was also highly correlated with both systolic and

diastolic blood pressure. These findings are consistent with those (Roberts & Rowlands, 1981) found in the medical literature that have established age as one of the standard risk factors for the potential development of cardiovascular disorders.

Medication Usage. Although medication usage did not appear to contribute to a significant amount of the variance in blood pressure in the analyses of variance, it did account for a substantial proportion of the variance in systolic and diastolic blood pressure in the multiple regression analyses (see Tables 9 & 10). However, when several covariates were considered in the analyses of covariance, medication was no longer a significant contributor, but was replaced by age which contributed to a substantial amount of the variance in systolic and diastolic blood pressure. This finding is not surprising given the fact that the relationship between age and blood pressure has been well established as a standard risk factor (Roberts & Rowlands, 1981). In the MANCOVA model, subject's education also contributed to a significant proportion of the variance in diastolic blood pressure, rather than medication. Based on the previously stated limitations of the analyses of variance the results of the multiple regression analyses are more meaningful, since the latter considers each of the variables simultaneously.

Subject's Educational Level. The MANCOVA (see Appendix F, Table 8a) also revealed that the subject's educational level contributed to a significant amount of the variance in diastolic, but not systolic blood pressure. Similarly, Pearson Product Moment Correlation Coefficients revealed an inverse relationship between subject's educational level and systolic blood pressure and diastolic blood pressure. Considering that this sample of black urban women had a mean educational level of thirteen years, it is not surprising to see a strong negative correlation between education and blood pressure in this sample. It is feasible that subjects who enjoy higher educational levels, may report lower blood pressure

levels because these individuals may be more aware of behavioral risk factors (e.g., smoking, diet, exercise) for essential hypertension and may therefore be less susceptible to the deleterious effects of their lifestyle on blood pressure. This hypothesis is at least partially supported by the significant inverse correlations found between subject's educational level and cholesterol; as well as between subject's educational level and body mass index. James et al. (1983) also established a negative association between education and blood pressure.

Family History of Hypertension. Contrary to the researcher's expectations, family history of hypertension did not play a significant role in contributing to the variance in blood pressure. The failure to find significant results for family history of hypertension may have been due to the disproportionate number of subjects who reported a positive family history (70%) of hypertension compared to those who reported a negative family history (9%); the remainder (21%) were missing values or subjects who were unsure of their family history of hypertension. Future research might be well served to balance the ratio of postive to negative family history among subjects such that both categories are equally represented.

## Limitations of the Study

### Methodological Considerations

External Validity. Since the sample for this study was not selected randomly for representativeness, the external validity of the results may have been affected. In addition, the fact that many of the subjects not only were volunteers , which may result in sample bias, but also were possibly more health conscious than a random sample that may have

included individuals who either did not work in a hospital or did not seek medical attention. This sample may be characterized as persons who are educated and/or health conscious possibly because as staff they worked in the hospital where positive health behaviors are emphasized or as patients they sought medical expertise for their condition. In James et al. (1983, 1984, 1987) epidemiological studies, subjects were randomly selected from the community to participate. These volunteers included people who had not sought medical attention for essential hypertension. Just as the current study may be limited in generalizability to educated, health conscious, urban females, James et al. (1983, 1984) studies were limited to generalizing to uneducated, rural black males. Future research in this area could benefit from considering a broader spectrum of educational levels and socioeconomic position among potential subjects as well as people who would equally readily seek or not seek medical attention.

The external validity of this study may also be viewed as limiting considering that Whites and men were excluded. However, James' research that originally included only men was supported by subsequent research that included men and women. When he included Whites in his research (James et al., 1987) the relationship between John Henryism and blood pressure was not detected with this group, but was detected with Blacks. It is unclear if racial differences would have been established be if the current study had included white subjects. However, if the biological research that implicates different underlying mechanisms for essential hypertension for Blacks and Whites is accurate, the relationship between John Henryism, social mobility, and blood pressure might differ for these two groups.

Body Mass Index. The impact of using patients from two different clinics (Nutrition and Weight Control Clinic & Hypertension Clinic) as well as staff at The University of

Chicago Hospitals, on the results of this study is unclear. Individuals who were recruited from the Nutrition and Weight Control Clinic (n=16) may have differed in important ways (e.g., body mass index). Since hypertension is positively correlated with weight, these individuals may have tended to have higher blood pressures. However, since the body mass index was used as a covariate in the ANCOVA and MANCOVA analyses, the contribution of weight and height to the variance in blood pressure was taken into account. Also, given the small number of participants in the study who were recruited from this clinic, it is likely that the results were not skewed in any significant way, and therefore is not considered a limitation of the study.

It is possible however, that subjects who were on staff at The University of Chicago Hospitals may have differed in some important way (e.g., higher SES, higher educational level, healthier lifestyle habits) from patients seen in the clinics. These potential differences could have in turn affected scores on John Henryism, direction of social mobility, or standard risk factors (e.g., smoking behavior, exercise, diet). It is unclear if significant differences do exist and what relationship, if any, they have on the obtained results in this study.

Medication. Patients recruited in the Hypertension Clinic who were on antihypertensive medication were used in the study. The effect of such medication on blood pressure would serve to suppress blood pressure levels. Therefore, blood pressure levels of those taking antihypertensive medication would still be considered an underestimate of their actual levels, which in turn may have restricted the range of blood pressure variability. This problem was addressed in the various analyses by controlling for the effects of medication when possible. The multiple regression analyses were most sensitive to the effects of medication on blood pressure. When medication was not entered

into the equation significant MSEI2 difference scores main effects on systolic and diastolic blood pressure were obtained. However, when medication was entered into the model, much of the variance was attributed to medication rather than MSEI2 scores. (Also using the model that included medication, a significant interaction between John Henryism and MSEI2 difference scores on systolic blood pressure was found, as indicated in Table 9.) This latter model demonstrates the importance of the effects of medication in interpreting these results. Future research should focus on the use of individuals who are not on antihypertensive medication in order to maximize the possiblity of finding significant relationships between psychosocial variables and blood pressure. By using only subjects who are not on medication, two things can be accomplished: 1) a selection bias would be minimized by eliminating subjects who might tend to seek medical attention due to the fact that they are on medication and 2) the variability of blood pressure levels would be greater. Community based epidemiological research would be appropriate avenues for accomplishing these goals.

Internal Validity

John Henryism Active Coping Scale-12 (JHAC-12). The JHAC-12 may have limited utility for this sample of black, educated, urban women since the original scale was normed on a semirural, low income, uneducated sample of black men. It is unclear if the revised version was normed on a similar population since the information was not provided in the literature. However, James et al. (1987) computed a Chronbach alpha (.71) for their biracial sample that included men and women, that was similar to that in this study (.72). The failure to achieve a wider range of scores may be due to the inability of the scale to adequately assess John Henryism in black women. The subjects in this sample may have tended to score at the higher end of the scale because of certain characteristics (e.g., higher

income, socioeconomic status, educational level). Although James noted a modest direct relationship between age and John Henryism scores this was not the case for the current study. James et al. (1987) also noted an inverse relationship between socioeconomic status and and scores on John Henryism. This finding was not supported by the current study if education is used as an indicator of socioeconomic status.

A social desirability factor may also be a reason for individuals to tend to score higher on this scale. Possibly the race of the experimenter being the same as the subject and/or an increased social awareness among urban blacks to display John Henryism characteristics may have skewed the results to the higher end of the continuum on this measure to please the experimenter. Socially conscious indivduals may feel a need to highly endorse items that reflect positive attributes (e.g., hard work, mental vigor, & environmental mastery) for which many Blacks are striving; especially during a time when it appears that Blacks are regressing by many traditional standards of success (i.e., income, education), resulting in black leaders ecouraging hard work and determination as a solution.

Even though the JHAC-12 total score has been used to compute median splits and therefore establish a dichotomous variable, this may not be the most accurate way of differentiating between high and low John Henryism. Using this method, there could be a one point difference between scores that could result in different classification. The question then becomes does this demarcation result in a loss of information that in turn affects the results. This was true in each of the analyses of variance that used the John Henryism median split dichotomy. The failure to find any significant results may have been due in part to this limitation. However, since John Henryism results were replicated in multiple regression analyses, this may be ruled out as an alternative explanation in favor of a restriction of range explanation.

Social Mobility. The MSEI2 appears to be an acceptable quantitative way of measuring social/job status. Although the MSEI2 takes into account education and income when rendering a numerical value for various occupations, it does not consider other factors that may be important in establishing socioeconomic status. These factors might include two income families and number of children in the household.

Inter-rater reliability procedures provided strong evidence (100% agreement) for assignment to groups when establishing the concordance/discordance dichotomy. However, although the same level of agreement was not true for the MSEI2 difference score continuous measure assignment, raters did not differ slightly when assigning values. One possible explanation for differences in rater's MSEI2 scores for the same subject may have been due to a lack of a one to one correspondence on some of the occupations that subjects stated compared to the options listed on the Duncan MSEI2.

## Clinical Implications

The clinical utility of research on psychosocial risk factors for essential hypertension is diverse. With the increasing number of Behavioral Medicine Sections in hospitals and the advent of multidisciplinary teams for various medical disorders (e.g., sexual dysfunction, chronic pain, oncology), the role of interdisciplinary communication in the treatment of these disorders emphasizes the importance of integative approaches to medicine. Psychological and socio-ecologic factors are of importance in understanding various sympotomatology manifestation. As a result, research in this area can assist in ascertaining mechanisms by which certain symptoms are elicited.

Considering the vast interest in the development of cardiovascular disorders, including essential hypertension, clinical implications for research in this area is considerable. Research in this area can provide a framework for understanding the development and

maintenance of the disorder thereby facilitating treating the disorder adequately. By thoroughly assessing the individual from a biopsychosocial perspective, a more comprehensive treatment plan can be instituted. The Type A Behavior Pattern literature is exemplary of how research on a psychological construct can have practical implications for health professionals. The widespread use of the Jenkins Activity Survey is assessing Type A demonstrates the clinical utility of instruments used to measure constructs that have been demonstrated to be related to health (Booth-Kewley & Friedman, 1987). Considering the disproportionate amount of essential hypertension among Blacks compared to Whites, it would be helpful to have a valid measure of personality characteristics that have been demonstrated to have a relationship to this disorder. Although this study did not provide evidence for the use of the JHAC-12 as a measure of John Henryism as it relates to blood pressure, furthur research on this instrument may justify its clinical utility or may result in the development of other instruments that more adequately measure this psychological construct.

The findings pertaining to intergenerational mobility are important clinically in knowing that individuals who have few resources are probably more at risk than any other group for essential hypertension. Based on the findings of this study, it is not intergenerational mobility per se that is related to blood pressure but the socioeconomic stratum to which the individual belongs during the years when blood pressure tends to increase with age. That is, even though one may have entered an entirely different socioeconomic position than that in which he/she were raised, the accompanying benefits of being middle class may tend to negate the stress of adapting to an unfamiliar social setting. Targeting those individuals who are at greatest risk (i.e. intergenerational poverty) can be a first step in addressing this disorder through public policy, media campaigns, and screening in clinics and doctor's offices. If income and education do in fact protect against the

development of essential hypertension, it would be important to know who is at risk and how the risk factors can be minimized. Educating these individuals would be an important clinical intervention in the management of essential disorder. Education should include not only traditional risk factors (e.g., exercise, diet, weight) but also psychosocial risk factors that are also of importance. The implicatons for treatment are that the type of treatment initiated would depend upon the risk factors that are present.

The failure to adequately establish risk factors for cardiovascular disorders along racial lines makes this research very timely. The shift in focus from inter-racial differences to intra-racial differences is a logical step since many of the traditional inter-racial (i.e., biological) arguments for the etiology of essential hypertension have been called into question. Most of the current research provides converging evidence for individual differences rather than racial groups differences as explanations for this disorder. This shift is not surprising given the statements made by Cooper (1984) that race is not a biological concept but more an economic one. Since most medical research makes racial distinctions based on phenotype and cultural identification rather than genotype, biological distinctions are not accurately established in this way. Race was never meant to be a means of categorizing subspecies but became a convenient way of justifying slavery and more recently institutionalized racism. Racial distinctions (i.e., superiority/inferiority dichotomy) serve an economic function in a capitalistic society, in that those who are classified as inferior are relegated to lowest paying jobs; thereby providing cheap labor to the economy. With this in mind, seeking out racial differences to explain various medical disorders may not be the best approach to explain illness. Given the similarities between Whites and Blacks on many disorders after socioeconomic status and education are controlled for, further evidence is provided for turning to biopsychosocial explanations. Even though Cooper suggests that hypertension may be the one disorder that does in fact fall along racial

lines, previously cited evidence does not completely support this line of thinking. The current research also provides evidence to support the idea that environmental factors (i.e., social mobility) play a role in the development and/or maintenance of at least one cardiovascular disorder, essential hypertension.

Although social mobility has been shown to contribute to the development of other cardiovascular disorders (e.g., angina pectoris, myocardial infarction) in Whites, no studies have been conducted on this phenomenon using a black population. This study provides some evidence that intergenerational mobility interacts with certain personality characteristics (e.g., John Henryism) that are predictive of systolic blood pressure levels after the effects of family history and medication are taken into account. These findings magnify the importance of the effects of medication in this study given that when medication was entered into the statistical analysis, no support was provided for the interaction of John Henryism and social mobility. Future research in this area might do well to eliminate individuals who are currently taking antihypertensives since medication may tend to attenuate the effects of psychosocial risk factors on blood pressure.

Directions for Future Resarch

Utility of John Henryism Active Coping Scale-12. Further research on the validity of the John Henryism construct is needed. It is possible that the current version of the form is inadequate in tapping the characteristic that it is designed to measure given the brevity of the scale. If the workplace is the arena in which the characteristics that are usually associated with John Henryism are elicited, while providing the strongest predictors of John Henryism as James et al. (1983) have proposed, what is the relationship between John Henryism and blood pressure in women who may not be in the Ωworkforce but also are the sole heads of their household. With the large number of female headed households

in the black community, John Henryism may be exhibited for reasons more related to family responsibilities than to job duties. It is unclear if the JHAC12 would generalize to this group of women who might also exhibit characteristics of hard work and determination that is not job related. Similar limitations are apparent with the Jenkings Activity Survey and Structured Interview (SI) as measures of Type A Behavior Pattern (TABP). Booth-Kewely and Friedman (1987) in a meta-analysis of studies investigating TABP found that the JAS and SI factors that were most strongly related to disease were Hard-Driving Competitiveness and Time Urgency respectively, which are most likely to be elicited in a job setting. Consequently, more global measures of these psychological constructs are needed that will be applicable to a broader range of individuals both employed and/or unemployed.

Another consideration is how gender differences might affect the way one perceives his/her identity. If one tends not to define himself/herself in terms of his/her profession, the John Henryism construct may be less applicable. Although women are increasingly establishing careers as well as families, they may still tend to view themselves as wives and mothers first, with their jobs playing a lesser role. Consequently, it might be fruitful to investigate other means of coping that women may tend to use more so than men. One such coping mechanism is unexpressed anger. During the debriefing, many of the women in this study indicated that the reason they thought they had hypertension was because they let things build up inside until they exploded rather than being assertive. Diamond's (1982) review of the literature revealed that anger and hostility appear to play an important role in the development of hypertension, although the mechanism is not clearly specified. More specifically, some researchers have found that anger expression was related to systolic blood pressure, but only in women (Gentry, Chesney, Gary, Hall, and Harburg, 1982). Therefore, sex differences as they apply to psychosocial risk factors should be kept in mind

in future research.

Another aspect of the JHAC12 that needs to be addressed is social desirability. New versions of the form should consider including items unrelated to John Henryism in the scale to minimize a specific response set. Since there was not a large range of John Henryism scores, the discriminatory power of the scale may need to be enhanced. One way of accomplishing this would be to increase the number of test items as James did when he revised the original eight-item, three choice selection to twelve items and five response options.

Consideration of the race of the experimenter may also be a factor in emitting socially desirable responses. This is especially true if both experimenter and subject are black and the questions are being read to the latter by the former. There may be a desire on the part of the subject to please the researcher with "appropriate" responses. Subjects should be required to complete the forms on their own whenever possible. Emphasis should also be placed upon responding based on how the subject really perceive themselves rather than how they would like to perceive themselves. Changing the directions in the scale to stress this latter point might facilitate minimizing socially desirable response sets. This might have been an issue with subjects who may not accurately perceive themselves either because of an ingruence between how they would like to see themselves or how they would like the experimenter to see them.

Prospective Studies. The arguments presented for the use of prospective studies as they relate to Type A Behavior Pattern and Cardiovascular heart disease also, apply to John Henryism and essential hypertension. Prospective studies are more effective in establishing relationships between personality characterics and the subsequent development of illness and disease and should be used ideally when possible. However, issues of time, money, and random assignment often dictate that cross-sectional research be conducted as an

alternative means of doing research in this area.

Type A Behavior Pattern (TABP) in Blacks. Lastly, as mentioned above , Type A Behavior Pattern research has focused primarily on white males in attempts to establish linkages between personality characteristics and risk for cardiovascular disease. Despite the tremendous amount of research that has been done in this area, it remains unclear if any of these findings are applicable to Blacks in general, and women in particular. More research is needed in this area on the segment of the population.

John Henryism. In addition to TABP in Blacks, the John Henryism concept shows some promise based on previous research done by James and his colleagues. The current study did partially support James' findings on John Henryism in interaction with another psychosocial variable (i.e., social mobility) were related to blood pressure, although the associaton was not in the direction that would be expected based on James' hypotheses. However, the significant interaction of personality characteristics and social-environmental factors with blood pressure, is in agreement with James et al. (1983, 1987) findings. This study provides evidence that more research is needed to ascertain what other biologic, psychologic, and sociologic variables are operating to increase or decrease blood pressure as well as what factors tend to attenuate (i.e., coping resources) the effects of these variables on blood pressure.

Self-efficacy. James makes a distinction between John Henryism and self-efficacy, stating that the former is characterized by active coping that oftentimes results in deleterious physical ramifications, unlike the latter. Furthermore, unlike self-efficacy, active coping was derived from the psychophysiological literature which has provided empirical evidence that active coping with a stressor in the laboratory can result in sustained blood pressure

elevations. However, it appears that John Henryism and self-efficacy have at least one aspect in common in terms of coping. Bandura (1977) postulates that expectations of personal efficacy determine whether coping behavior will be intitiated, how much effort will be expended, and how long it will be sustained in the face of obstacles and aversive experiences. This ties in with James' hypothesis that individuals who exhibit high levels of John Henryism attempt to actively cope with their environment. Furthermore, to the degree that these individuals feel that they have some control over their environment, is the degree to which he/she will sustain their active coping. However, James would postulate that if these same individuals had low levels of resources (i.e., income, education) that resulted in greater frustration in achieving their goals, they would be at greater risk for developing essential hypertension (a negative outcome) because of their coping efforts rather than a positive outcome, as one would expect with self-efficacy theory. Consequently, even though self-efficacy and John Henryism have similar components (i.e. environmental mastery) the outcomes of their presence or absence is different.

Mobility. Since this study provided some evidence for the influence of social mobility on blood pressure and prior research has implicated a relationship mobility in general (i.e, cultural, occupational, religious, geographical) on blood pressure (Marmot et al., 1975; Kaplan et al., 1971; Lehr et al., 1973, Rhoads et al. 1976), future studies in the area of mobility among Blacks might do well to include geographic as well as occupational and social mobility. It is possible that moving from rural areas in the South to urban areas in the North, which is typical of many Blacks in the inner city, may be related to blood pressure. Research in the area of social mobility specifically should focus on components of social mobility that relate to blood pressure. Lifestlye factors and cognitive appraisal of stressful situations are specific facets of social mobility that might further our understanding

of the effects of this phenomenon on physical health.

Considering the complexity of determining the etiology of illness and disease, biopsychosocial models are becoming more prevalent in attempting to explain incidence and prevalence rates among certain individuals. The decreased emphasis on inter-racial differences and the more appropriate emphasis on intra-racial differences has taken research in this area a step closer to understanding the role of biologic, psychologic, and socio-ecologic factors in physical disorders. Attempts to integrate knowledge in various disciplines to more adequately explain illness and disease is difficult but necessary if a comprehensive understanding of the interaction between psyche and soma, in the context of environmental considerations, is to emerge. Medical research on underlying hypotheses of essential hypertension are necessary but not sufficient in providing an explanation. Furthermore, results of biological research that attempts to establish inter-racial differences should consider alternative psychosocial explanations (i.e, sodium retention hypothesis) when intepreting the data, if indicated. Conversely, psychosocial researchers should consider biologic factors (e.g., genetic predisposition) when attempting to explain physical disorders with psychosocial data. Although much work is still needed in refining biopsychosocial approaches, great strides have been taken compared to a strictly biologic approach which has prevailed in the past and continues to be prominent in many health care facilities.

Implications for the biopsychosocial model are signficant in the areas of research, assessment, and treatment. Much of the research on essential hypertension has resulted in changes in assessment and treatment of this disorder. Studies on biofeedback and relaxation (McCaffrey & Blanchard, 1985; Blanchard, McCoy, Musso, Gerardi, Pallmeyer, Gerardi, Cotch, Siracusa, & Andrasik, 1986) have provided evidence for the efficacy of non-pharmacologic treatment of this disorder. Thus, valid and reliable

assessment instruments need to be constructed to aid in targeting individuals for specific types of psychological intervention. As long as empirical evidence can be provided to demonstrate the efficacy of non-pharmacologic as well as pharmacologic treatment of essential hypertension it strengthens the rationale for using the biopsychosocial model in research on health and illness.

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# APPENDIX A

CONSENT BY SUBJECT FOR PARTICIPATION IN RESEARCH PROTOCOL
THE UNIVERSITY OF CHICAGO
DIVISION OF THE BIOLOGICAL SCIENCES

Protocol Number: Patient Name:

Title of Protocol: Psychosocial Risk Factors of Essential Hypertension Among Black Women

Doctor directing Research: Sara Knight, Ph.D. Phone: 702-1526
Researcher: Stephanie Livingston, M.A. Phone: 702-6189

You are being asked to participate in a research study. The doctors at The University of Chicago Medical Center and the Division of the Biological Sciences study the nature of disease and attempt to develop improved methods of diagnosis and treatment. In order to decide whether or not you want to be a part of this research study, you should understand enough about its risks and benefits to make an informed judgement. This process is known as informed consent. This consent form gives detailed information about the research study which will be discussed with you. Once you undersatnd the study, you will be asked to sign this form if you wish to participate.

I. NATURE AND DURATION OF PROCEDURE

The researcher, Stephanie Livingston, or an assistant, will be conducting the interviews for the purpose of investigating biological, psychological, and sociological risk factors in essential hypertension among black women. As part of the study, you will be asked to complete one demographic questionnaire and two psychological inventories. These three forms should take approximately twenty minutes to complete. You will have an opportunity to ask questions about the study. All of the information you give will be kept strictly confidential, and used for scientific purposes only. Your name will not be used i any reports or publications.

II. POTENTIAL RISKS AND BENEFITS

You will not be subjected to any harmful procedures in this study. Consequently, there is minimal or no risk to your health or well being because of participation. Although this study is not designed to diredtly help any current medical condition that you may have, information that you may receive regarding the results could prove to be indrectly beneficial to persons with essential hypertension. You are free to discontinue your participation at any time.

AGREEMENT TO CONSENT

The research project and the treatment procedures associated with it have been fully explained to me. All experimental procedures have been identified and no guarantee has been given about the possible results. I have had the opportunity to ask questions concerning any and all aspects of the project and any procedures involved. I am aware that I may withdraw my consent at any time and such withdrawal will not restrict my access to

health care services normally available a The University of Chicago Medical Center. Confidentiality of records concerning my involvement in this project will be maintained in an appropriate manner. When required by law, the records of this research may be reviewed on an anonymous basis by applicable government agencies.

I understand that in the event of physical injury resulting from this research, The University of Chicago Medical Center will provide me with free emergency care, if such care is necessary. I also understand that if I wish, but the Center assumes no responsibility to pay for such care or provide me with financial compensation.

I, the undersigned, hereby consent to participate as a subject in the above described research project c onducted at the The University of Chicago Medical Center. I have received a copy of this consent form for my records. I understand that if I have any questions concerning this research or my rights in connection with the research, I can contact the doctor named above or the Clinical Investigation Committee, at 312/702-1472.

After reading the entire consent form, if you have no further questions about giving consent, please sign where indicated.

Doctor: Signature of Subject

Witness: Date:

Time: AM/PM

# APPENDIX B

# DEMOGRAPHIC QUESTIONNAIRE

ID#

## DEMOGRAPHIC SHEET
## FOR OFFICE USE ONLY

| | |
|---|---|
| Marital Status S M D W | Blood Pressure |
| Age | Cholesterol |
| Sex M F | Smoking Yes No |
| Race B W H O | Exercise Yes No |
| Height | Family History Yes No |
| Weight | Body Mass Index |

## MEDICAL HISTORY

1. Do you currently or have you ever had any of the following illnesses? Indicate at what age.

| | AGE | | AGE |
|---|---|---|---|
| heart attack | | stroke | |
| kidney failure | | glaucoma | |
| high blood pressure | | heart disease | |

a. Do you currently take medications for any of these illnesses?
Yes No

b. If you do take medications, please list the prescriptions that you take for each illness.

| | |
|---|---|
| heart attack | stroke |
| kidney failure | glaucoma |
| high blood pressure | heart disease |

2. Do you take these medications as prescribed by your doctor? Yes No

3. Do/did either of your parents ever have any of the following illnesses?

| Mother | AGE | Father | AGE |
|---|---|---|---|
| heart attack | | heart attack | |
| kidney failure | | kidney failure | |
| high blood pressure | | high blood pressure | |
| stroke | | stroke | |
| glaucoma | | glaucoma | |
| heart disease | | heart disease | |

4. Do you currently smoke cigarettes? Yes No

   a. If yes, how many hours per week? Number: _____

5. Do you exercise regularly?

   a. If yes, how many hours per week? Number: _____

SOCIAL HISTORY

CURRENT SOCIAL HISTORY

6. What kind of work (do you/did you) normally do? That is, what (is/was) your job called?

   OCCUPATION:

   a. What (do/did) you actually do in that job?

      DUTIES:

   b. What kind of place (do/did) you work for?

      INDUSTRY:

7. What is the highest grade you completed in school?

| | | | |
|---|---|---|---|
| a. grammar school | 1 2 3 4 5 6 7 8 | | |
| high school | 9 10 11 12 | diploma/GED | Yes No |
| college | 13 14 15 16 | degree? | Yes No |
| grad school | 17 18 19 20 | degree? | Yes No<br>M.A. Ph.D |

8. On a scale of 1 to 10 (1=minimum stress, 10=maximum stress), how much stress would you say you have experienced in the last 12 months?

1 2 3 4 5 6 7 8 9 10

9. How many children do you currently have living with you? NUMBER:_____

PAST SOCIAL HISTORY

10. What kind of work did your (father/head of household) usually do for a living when you were about 16 years old?

OCCUPATION:

a. What did he/she actually do in that job?

DUTIES:

b. What kind of place did he/she work for?

INDUSTRY:

11. What is the highest grade that your father/head of household completed when you were age 16?

| | | | |
|---|---|---|---|
| grammar school | 1 2 3 4 5 6 7 8 | | |
| high school | 9 10 11 12 | diploma/GED | Yes No |
| college | 13 14 15 16 | degree? | Yes No |
| grad school | 17 18 19 20 | degree? | Yes No<br>M.A. Ph.D. |

12. Which of these categories comes closest to the type of place where you were living when you were 16 years old?

    a. In an open country but not on a farm

    b. On a farm

    c. In a small city or town (under 50,000)

    d. In a medium-sized (50,000-250,000)

    e. In a suburb near a large city

    f. In a large city (over 250,000)

    g. Don't Know

13. How many brothers and sisters did you have living with you when you were growing up?

| | NUMBER | | NUMBER |
|---|---|---|---|
| brothers: | | sisters: | |

# APPENDIX C

## The John Henryism Active Coping Scale (JHAC-12)

The questions below concern how you see yourself, today, as a person living and doing things in the real world. Read each question carefully, and then write the number of the response which best describes how you feel on the line next to the question. Each person is different, so there are no "Right" or "Wrong" answers. We would simply like an honest appraisal of how you generally see yourself.

*FOR EACH OF THE FOLLOWING STATEMETS, PLEASE SELECT ONE OF THESE RESPONSES*

Completely True = 5
Somewhat True = 4
Somewhat False = 2
Completely False = 1
Don't Know = 3

____1. I've always felt that I could make of my life pretty much what I wanted to make of it.

____2. Once I make up my mind to do something, I stay with it until the job is completely done.

____3. I like doing things that other people thought could not be done.

____4. When things don't go the way I want them to, that just makes me work even harder.

____5. Sometimes I feel that if anything is going to be done right, I have to do it myself.

____6. It's not always easy, but I manage to find a way to do the things I really need to get done.

____7. Very seldom have I been disappointed by the results of my hard work.

____8. I feel that I am the kind f individual who stands up for what he believes in regardless of the consequences.

____9. In the past, even when things got really tough, I never lost sight of my goals.

___10. It's important for me to be able to do things the way I want to do them rather than the way other people want me to do them.

___11. I don't let my personal feelings get in the way of doing a job.

___12. Hard work has really helped me to get ahead in life.

# APPENDIX D

| Census occupational category | | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|---|
| 001 | Accountants | 70.17 | 64.58 | 73.33 |
| 002 | Architects | 78.16 | 80.11 | 93.15 |
| | Computer Specialists | | | |
| 003 | Computer programmers | 63.57 | 66.05 | 65.35 |
| 004 | Computer systems analysts | 75.39 | 74.10 | 80.02 |
| 005 | Computer specialists, n.e.c. | 73.01 | 71.19 | 77.15 |
| | Engineers | | | |
| 006 | Aeronautical and astronautical engineers | 83.21 | 83.53 | 89.57 |
| 010 | Chemical engineers | 85.39 | 87.14 | 91.97 |
| 011 | Civil engineers | 75.33 | 76.79 | 79.87 |
| 012 | Electrical and electronic engineers | 78.32 | 79.04 | 83.59 |
| 013 | Industrial engineers | 71.62 | 72.02 | 75.51 |
| 014 | Mechanical engineers | 76.21 | 76.84 | 81.10 |
| 015 | Metallurgical and materials engineers | 81.92 | 80.34 | 87.94 |
| 020 | Mining engineers | 74.22 | 75.49 | 78.56 |
| 021 | Petroleum engineers | 90.55 | 82.32 | 86.12 |
| 022 | Sales engineers | 77.08 | 78.16 | 82.08 |
| 023 | Engineers, n.e.c. | 75.27 | 76.36 | 79.85 |
| 024 | Farm management advisors | 77.74 | 80.25 | 82.33 |
| 025 | Foresters and conservationists | 47.48 | 49.57 | 45.84 |
| 026 | Home management advisors | 47.10 | 65.83 | 45.28 |
| | Lawyers and judges | | | |
| 030 | Judges | 79.87 | 79.68 | 85.42 |
| 031 | Lawyers | 86.96 | 88.42 | 93.81 |
| | Librarians, archivists, and curators | | | |
| 032 | Librarians | 65.23 | 65.46 | 66.83 |
| 033 | Archivists and curators | 57.14 | 61.22 | 57.30 |
| | Mathematical specialists | | | |
| 034 | Actuaries | 84.37 | 80.37 | 90.55 |
| 035 | Mathematicians | 84.33 | 84.39 | 90.55 |
| 036 | Statisticians | 71.45 | 65.12 | 74.91 |
| | Life and physical scientists | | | |
| 042 | Agricultural scientists | 62.73 | 63.90 | 64.36 |
| 043 | Atmospheric and space scientists | 77.70 | 74.58 | 82.91 |
| 044 | Biological scientists | 76.95 | 76.73 | 81.27 |
| 045 | Chemists | 75.56 | 77.76 | 79.89 |
| 051 | Geologists | 85.59 | 87.35 | 92.12 |
| 052 | Marine scientists | 80.72 | 82.48 | 86.30 |
| 053 | Physicists and astronomers | 85.16 | 87.00 | 91.64 |
| 054 | Life and physical scientists, n.e.c. | 81.72 | 80.05 | 87.37 |

| Census occupational category | | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|---|
| | Teachers, college and university | | | |
| 102 | Agriculture teachers | 82.58 | 85.71 | 88.26 |
| 103 | Atmospheric, earth, marine, and space teachers | 81.82 | 85.04 | 87.23 |
| 104 | Biology teachers | 81.67 | 83.80 | 87.05 |
| 105 | Chemistry teachers | 81.96 | 85.03 | 87.42 |
| 110 | Physics teachers | 80.41 | 84.22 | 85.56 |
| 111 | Engineering teachers | 83.30 | 84.88 | 89.25 |
| 112 | Mathematics teachers | 79.43 | 82.46 | 84.24 |
| 113 | Health specialties teachers | 86.63 | 80.74 | 93.36 |
| 114 | Psychology teachers | 84.20 | 85.53 | 90.22 |
| 115 | Business and commerce teachers | 83.03 | 82.91 | 88.81 |
| 116 | Economics teachers | 84.10 | 87.11 | 90.09 |
| 120 | History teachers | 80.04 | 83.61 | 84.97 |
| 121 | Sociology teachers | 79.15 | 82.28 | 83.87 |
| 122 | Social science teachers, n.e.c. | 82.13 | 85.04 | 87.64 |
| 123 | Art, drama, and music teachers | 78.42 | 79.74 | 83.03 |
| 124 | Coaches and physical education teachers | 78.57 | 81.43 | 83.21 |
| 125 | Education teachers | 85.34 | 86.20 | 91.74 |
| 126 | English teachers | 79.15 | 80.81 | 83.85 |
| 130 | Foreign language teachers | 77.74 | 78.97 | 82.12 |
| 131 | Home economics teachers | 74.84 | 73.13 | 79.16 |
| 132 | Law teachers | 88.65 | 90.45 | 95.89 |
| 133 | Theology teachers | 76.14 | 80.48 | 80.08 |
| 134 | Trade, industrial, and technical teachers | 66.90 | 68.84 | 69.21 |
| 135 | Miscellaneous teachers, college and university | 79.02 | 81.93 | 83.73 |
| 140 | Teachers, college and university, subject not specified | 75.22 | 77.13 | 79.08 |
| | Teachers, except college and university | | | |
| 141 | Adult education teachers | 59.33 | 59.56 | 60.25 |
| 142 | Elementary school teachers | 68.99 | 70.88 | 71.15 |
| 143 | Prekindergarten and kindergarten teachers | 50.32 | 58.51 | 48.75 |
| 144 | Secondary school teachers | 73.02 | 75.14 | 76.20 |
| 145 | Teachers, except college and university, n.e.c. | 48.92 | 51.64 | 47.46 |
| | Engineering and science technicians | | | |
| 150 | Agriculture and biological technicians, except health | 37.65 | 39.10 | 33.92 |
| 151 | Chemical technicians | 45.71 | 48.48 | 43.66 |

| Census occupational category | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|
| 202 Bank officers and financial managers | 66.48 | 62.13 | 69.21 |
| 203 Buyers and shippers, farm products | 33.63 | 34.31 | 29.65 |
| 205 Buyers, wholesale and retail trade | 48.70 | 45.80 | 47.84 |
| 210 Credit men | 56.60 | 50.89 | 56.95 |
| 211 Funeral directors | 58.33 | 60.47 | 59.06 |
| 212 Health administrators | 68.80 | 61.90 | 71.97 |
| 213 Construction inspectors, public administration | 38.91 | 39.63 | 36.10 |
| 215 Inspectors, except construction, public administration | 44.89 | 44.92 | 43.24 |
| 216 Managers and superintendents, building | 40.74 | 38.43 | 38.02 |
| 220 Office managers, n.e.c. | 56.34 | 48.48 | 56.99 |
| 221 Officers, pilots, and pursers; ship | 36.65 | 35.28 | 33.73 |
| 222 Officials and administrators; public administration, n.e.c. | 59.78 | 57.09 | 61.25 |
| 223 Officials of lodges, societies, and unions | 53.17 | 52.47 | 53.45 |
| 224 Postmasters and mail superintendents | 45.23 | 39.84 | 43.89 |
| 225 Purchasing agents and buyers, n.e.c. | 56.73 | 54.52 | 57.55 |
| 226 Railroad conductors | 36.42 | 33.75 | 33.66 |
| 230 Restaurant, cafeteria, and bar managers | 36.80 | 32.51 | 33.41 |
| 231 Sales managers and department heads, retail trade | 45.36 | 41.58 | 43.75 |
| 233 Sales managers, except retail trade | 69.25 | 68.09 | 72.39 |
| 235 School administrators, college | 80.14 | 79.49 | 85.59 |
| 240 School administrators, elementary and secondary | 84.98 | 83.39 | 91.35 |
| 245 Managers and administrators, n.e.c. | 50.39 | 49.13 | 50.66 |
| 246 Managers and administrators, except farm-allocated | 41.07 | 40.07 | 38.55 |
| 260 Advertising agents and salesmen | 60.93 | 58.71 | 62.38 |
| 261 Auctioneers | 35.94 | 34.40 | 32.79 |
| 262 Demonstrators | 41.68 | 25.69 | 39.13 |
| 264 Hucksters and peddlers | 32.79 | 25.64 | 28.26 |
| 265 Insurance agents, brokers, and underwriters | 54.57 | 53.43 | 54.78 |
| 256 Newsboys | 16.43 | 19.40 | 8.49 |
| 270 Real estate agents and brokers | 55.28 | 50.92 | 55.72 |

| Census occupational category | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|
| 350 Tabulating machine operators | 30.27 | 28.05 | 24.92 |
| 355 Office machine operators, n.e.c. | 26.29 | 23.73 | 20.26 |
| 360 Payroll and timekeeping clerks | 33.41 | 27.38 | 29.00 |
| 361 Postal clerks | 29.92 | 30.25 | 24.83 |
| 362 Proofreaders | 43.71 | 35.25 | 41.49 |
| 363 Real estate appraisers | 64.33 | 64.93 | 66.52 |
| 364 Receptionists | 37.33 | 29.00 | 33.26 |
| Secretaries | | | |
| 370 Secretaries, legal | 47.79 | 34.73 | 46.03 |
| 371 Secretaries, medical | 44.21 | 35.57 | 41.76 |
| 372 Secretaries, n.e.c. | 45.99 | 31.75 | 44.25 |
| 374 Shipping and receiving clerks | 19.49 | 21.93 | 12.20 |
| 375 Statistical clerks | 37.24 | 31.28 | 33.65 |
| 376 Stenographers | 50.66 | 30.02 | 50.22 |
| 381 Stock clerks and storekeepers | 22.93 | 25.26 | 16.29 |
| 382 Teacher aides, exc. school monitors | 40.85 | 31.56 | 37.20 |
| 383 Telegraph messengers | 20.46 | 24.40 | 13.19 |
| 384 Telegraph operators | 29.02 | 28.48 | 23.89 |
| 385 Telephone operators | 33.98 | 21.89 | 29.82 |
| 390 Ticket, station, and express agents | 38.04 | 39.28 | 34.61 |
| 391 Typists | 28.69 | 25.23 | 23.09 |
| 392 Weighers | 19.15 | 20.33 | 11.90 |
| 394 Miscellaneous clerical workers | 40.22 | 32.93 | 37.22 |
| 395 Not specified clerical workers | 34.13 | 27.74 | 29.78 |
| 396 Clerical and kindred workers-allocated | 29.65 | 27.19 | 28.34 |
| 401 Automobile accessories installers | 19.81 | 22.03 | 12.67 |
| 402 Bakers | 18.48 | 19.22 | 11.16 |
| 403 Blacksmiths | 19.35 | 20.72 | 12.30 |
| 404 Boilermakers | 26.07 | 25.55 | 20.78 |
| 405 Bookbinders | 24.65 | 19.88 | 19.77 |
| 410 Brickmasons and stonemasons | 22.15 | 22.52 | 15.86 |
| 411 Brickmasons and stonemasons, apprentices | 21.51 | 23.63 | 14.82 |
| 412 Bulldozer operators | 18.29 | 19.54 | 11.05 |
| 413 Cabinetmakers | 19.04 | 20.89 | 11.83 |
| 415 Carpenters | 21.43 | 22.58 | 14.87 |
| 416 Carpenter apprentices | 17.74 | 21.31 | 9.96 |
| 420 Carpet installers | 21.65 | 23.07 | 15.18 |
| 421 Cement and concrete finishers | 20.08 | 21.10 | 13.25 |
| 422 Compositors and typesetters | 28.11 | 28.02 | 23.01 |
| 423 Printing trades apprentices, exc. pressmen | 18.34 | 22.06 | 10.71 |

| Census occupational category | | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|---|
| 486 | Railroad and car shop | 17.87 | 19.60 | 10.47 |
| 491 | Mechanic, exc. auto, apprentices | 24.43 | 24.83 | 18.30 |
| 492 | Miscellaneous mechanics and repairmen | 24.96 | 26.40 | 19.03 |
| 495 | Not specified mechanics and repairmen | 25.71 | 26.87 | 20.01 |
| 501 | Millers; grain, flour, and feed | 16.77 | 18.58 | 9.13 |
| 502 | Millwrights | 29.17 | 27.58 | 24.72 |
| 503 | Molders, metal | 17.71 | 18.66 | 10.31 |
| 504 | Molder apprentices | 20.82 | 23.90 | 13.74 |
| 505 | Motion picture projectionists | 29.95 | 32.75 | 24.92 |
| 506 | Opticians, and lens grinders and polishers | 30.28 | 29.21 | 25.43 |
| 510 | Painters, construction and maintenance | 18.58 | 20.71 | 11.28 |
| 511 | Painter apprentices | 15.38 | 18.91 | 7.13 |
| 512 | Paperhangers | 22.65 | 23.40 | 16.29 |
| 514 | Pattern and model makers, exc. paper | 33.74 | 31.49 | 30.24 |
| 515 | Photoengravers and lithographers | 37.66 | 34.33 | 34.94 |
| 516 | Piano and organ tuners and repairmen | 30.11 | 33.12 | 24.96 |
| 520 | Plasterers | 20.92 | 21.55 | 14.37 |
| 521 | Plasterer apprentices | 17.92 | 20.28 | 10.60 |
| 522 | Plumbers and pipe fitters | 28.09 | 27.24 | 23.27 |
| 523 | Plumber and pipe fitter apprentices | 21.44 | 24.42 | 14.56 |
| 525 | Power station operators | 34.65 | 33.59 | 31.19 |
| 530 | Pressmen and plate printers, printing | 26.16 | 25.70 | 20.77 |
| 531 | Pressman apprentices | 19.97 | 22.80 | 12.80 |
| 533 | Rollers and finishers, metal | 25.16 | 23.87 | 19.70 |
| 534 | Roofers and slaters | 17.97 | 19.73 | 10.59 |
| 535 | Sheetmetal workers and tinsmiths | 26.54 | 26.12 | 21.30 |
| 536 | Sheetmetal apprentices | 19.66 | 23.12 | 12.30 |
| 540 | Shipfitters | 22.30 | 23.21 | 15.93 |
| 542 | Shoe repairmen | 15.50 | 17.29 | 7.49 |
| 543 | Sign painters and letterers | 23.72 | 25.80 | 17.48 |
| 545 | Stationary engineers | 28.08 | 28.34 | 23.06 |
| 546 | Stone cutters and stone carvers | 17.50 | 19.63 | 9.92 |
| 550 | Structural metal craftsmen | 29.06 | 27.99 | 24.48 |
| 551 | Tailors | 18.52 | 19.13 | 11.26 |
| 552 | Telephone installers and repairmen | 32.46 | 32.59 | 28.33 |

| Census occupational category | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|
| 634 Meat wrappers, retail trade | 16.55 | 15.10 | 8.55 |
| 635 Metal platers | 18.18 | 19.94 | 10.77 |
| 636 Milliners | 21.96 | 17.68 | 15.86 |
| 640 Mine operatives, n.e.c. | 18.16 | 20.24 | 10.73 |
| 641 Mixing operatives | 17.19 | 19.32 | 9.55 |
| 642 Oilers and greasers, exc. auto | 18.69 | 20.00 | 11.50 |
| 643 Packers and wrappers, except meat and produce | 15.42 | 16.07 | 7.28 |
| 644 Painters, manufactured articles | 16.33 | 17.99 | 8.56 |
| 645 Photographic process workers | 31.90 | 28.46 | 27.24 |
| Precision machine operatives | | | |
| 650 Drill press operatives | 17.10 | 18.25 | 9.46 |
| 651 Grinding machine operatives | 20.82 | 21.21 | 14.20 |
| 652 Lathe and milling machine operatives | 21.28 | 22.16 | 14.67 |
| 653 Precision machine operatives, n.e.c. | 22.24 | 22.17 | 15.93 |
| 656 Punch and stamping press operatives | 17.07 | 17.54 | 9.46 |
| 660 Riveters and fasteners | 14.16 | 15.93 | 5.80 |
| 661 Sailors and deckhands | 19.91 | 21.71 | 12.92 |
| 662 Sawyers | 13.80 | 16.39 | 5.36 |
| 663 Sewers and stitchers | 14.78 | 14.48 | 6.58 |
| 664 Shoemaking machine operatives | 11.83 | 14.29 | 2.94 |
| 665 Solderers | 16.63 | 15.90 | 8.86 |
| 666 Stationary firemen | 20.32 | 20.99 | 13.52 |
| Textile operatives | | | |
| 670 Carding, lapping, and combing operatives | 11.44 | 14.12 | 2.48 |
| 671 Knitters, loopers, and toppers | 14.92 | 15.42 | 6.76 |
| 672 Spinners, twisters, and winders | 11.80 | 13.98 | 2.91 |
| 673 Weavers | 11.62 | 14.32 | 2.69 |
| 674 Textile operatives, n.e.c. | 12.67 | 14.76 | 3.98 |
| 680 Welders and flame-cutters | 19.76 | 20.66 | 12.83 |
| 681 Winding operatives, n.e.c. | 18.32 | 17.17 | 11.05 |
| 690 Machine operatives, miscellaneous specified | 18.43 | 18.86 | 11.12 |
| 692 Machine operatives, not specified | 18.00 | 18.63 | 10.57 |
| 694 Miscellaneous operatives | 18.34 | 19.30 | 10.91 |
| 695 Not specified operatives | 18.93 | 19.43 | 11.70 |
| 696 Operatives, except transport-allocated | 18.01 | 17.97 | 10.56 |
| 701 Boatmen and canalmen | 22.02 | 23.09 | 15.59 |
| 703 Bus drivers | 21.16 | 21.47 | 14.40 |

| Census occupational category | | MSEI2 | TSEI2 | MSEI3 |
|---|---|---|---|---|
| | Cleaning service workers | | | |
| 846 | Farm laborers and farm foremen-allocated | 14.41 | 17.41 | 6.01 |
| 901 | Chambermaids and maids, except private household | 15.74 | 15.24 | 7.60 |
| 902 | Cleaners and charwomen | 14.52 | 15.97 | 6.16 |
| 903 | Janitors and sextons | 15.80 | 18.51 | 7.72 |
| | Food service workers | | | |
| 910 | Bartenders | 22.71 | 23.96 | 16.06 |
| 911 | Busboys | 15.23 | 19.04 | 6.89 |
| 912 | Cooks, except private household | 18.06 | 17.52 | 10.43 |
| 913 | Dishwashers | 16.35 | 19.03 | 8.21 |
| 914 | Food counter and fountain workers | 18.52 | 20.43 | 10.83 |
| 915 | Waiters | 22.38 | 18.88 | 15.42 |
| 916 | Food service workers, n.e.c., except private household | 17.55 | 17.81 | 9.68 |
| | Health service workers | | | |
| 921 | Dental assistants | 34.22 | 27.15 | 29.79 |
| 922 | Health aides, exc. nursing | 27.11 | 25.65 | 21.16 |
| 923 | Health trainees | 33.95 | 45.49 | 29.24 |
| 924 | Lay midwives | 28.54 | 23.58 | 22.78 |
| 925 | Nursing aides, orderlies, and attendants | 21.09 | 19.52 | 13.86 |
| 926 | Practical nurses | 24.20 | 24.94 | 17.67 |
| | Personal service workers | | | |
| 931 | Airline stewardesses | 35.17 | 51.51 | 31.51 |
| 932 | Attendants, recreation and amusement | 24.89 | 28.84 | 18.51 |
| 933 | Attendants, personal service, n.e.c. | 23.37 | 27.09 | 16.66 |
| 934 | Baggage porters and bellhops | 19.25 | 23.22 | 11.71 |
| 935 | Barbers | 18.19 | 20.45 | 10.75 |
| 940 | Boarding and lodging house | 31.16 | 26.69 | 26.11 |
| 941 | Bootblacks | 11.56 | 14.91 | 2.59 |

# APPENDIX E

## DEBRIEFING

Subjects were given an opportunity to ask question following the interview and completion of the questionnaires. They were also given the name and number of the researcher for future reference. Subjects were then told that they would be notified in writing of the results of the study or would have an opportunity to come to a group feedback session.

# APPENDIX F

Table 11

Variable Names and Labels

*John Henryism Active Coping Scale*

| Name | Label |
|---|---|
| John Henryism | JH |
| John Henryism Median Split | JHMS |
| 1=low JH | |
| 2=high JH | |

*Duncan Socioeconomic Index--Revised (MSEI2)*

| Name | Label |
|---|---|
| MSEI2 Difference Scores | MSEIDIF |
| Concordance/Discordance | CONDIS |
| 1=concordant | |
| 2=discordant | |

Table 12

Demographic Variables

| Value | Variable |
|---|---|
| Age of the subject | AGE |
| Subject's education level in years | PTED |
| Subject's head of household education level in years | HHED |
| City size<br>1=rural<br>2=farm<br>3=small town (<50,000)<br>4=medium town (50,000-250,000)<br>5=suburb<br>6=large city (>250,000) | CITY |
| Number of children | CHILDREN |
| Number of siblings at age 16 | SIBLINGS |
| Marital status<br>1=single<br>2=married<br>3=divorced/separated<br>4=widowed | MARITAL |
| Stress level in the past year<br>1-10<br>1=minimum amount<br>10=maximum amount | STRESS |

Table 13

Medical Factors

| Value | Variable |
|---|---|
| Family history of hypertension<br><br>1=positive<br>2=negative<br>3=don't know | FHX |
| Number of cigarettes smoked per day | SMOKING |
| Medication usage<br><br>1=with<br>2=without | RX |
| Systolic blood pressure | SBP |
| Diastolic blood pressure | DBP |
| Diagnosis<br><br>1=hypertensive<br>2=normotensive | DX |
| Age of onset of hypertension | AGEONSET |
| Exercise | EX |
| Cholesterol | CHOL |
| Body mass index | BMI |

Table 14

2 (John Henryism) X 2 (Social Mobility) ANOVA Summary Table for SBP

| | DF | F | p |
|---|---|---|---|
| Main Effects (Overall) | 2 | 2.67 | .07 |
| JHMS | 1 | .19 | .66 |
| CONDIS | 1 | 5.17 | .02 * |
| 2-way Interactions | | | |
| JHMS X CONDIS | 1 | .02 | .87 |

TOTAL MEAN=131.28

| | Cell Means | | Cell Means |
|---|---|---|---|
| JHMS | 132.37 | | 130.31 |
| CONDIS | 136.98 | | 126.20 |

| | | CONDIS | |
|---|---|---|---|
| | | **1** | **2** |
| **JHMS** | **1** | 132.37 | 130.31 |
| | **2** | 136.98 | 126.20 |

Note: JHMS=John Henryism Median Split
1=low John Henryism; 2=high John Henryism
CONDIS=Concordant/Discordant Social Mobility
1=concordant; 2=discordant
*p <.05 **p <.01 ***p <.001

Table 15

2 (John Henryism) X 2 (Social Mobility) ANOVA Summary Table for DBP

| | DF | F | p |
|---|---|---|---|
| Main Effects (Overall) | 2 | 1.82 | .16 |
| JHMS | 1 | .25 | .61 |
| CONDIS | 1 | 3.39 | . 06 |
| 2-way Interactions | | | |
| JHMS X CONDIS | | .03 | .85 |

TOTAL MEAN=87.45

| | Cell Mean | | Cell Mean |
|---|---|---|---|
| JHMS | 36.71 | | 88.11 |
| CONDIS | 90.10 | | 85.09 |

| | | CONDIS | |
|---|---|---|---|
| | | **1** | **2** |
| **JHMS** | **1** | 89.09 | 84.62 |
| | **2** | 91.00 | 85.52 |

Note: JHMS=John Henryism Median Split
1=low John Henryism; 2=high John Henryism
CONDIS=Condcordant/Discordant Social Mobility
1=concordant; 2=discordant

Table16

2 (John Henryism) X 2 (Social Mobility) ANCOVA Cell Means for SBP

Covariates=FHX, AGE, RX, $S_s$ ED, BMI, SMOKING, HHED

TOTAL MEAN=130.64

| | | Cell Mean | Cell Mean |
|---|---|---|---|
| | | 1 | 2 |
| JHMS | | 131.96 | 129.46 |
| CONDIS | | 136.17 | 125.72 |
| | | **CONDIS** | |
| | | **1** | **2** |
| **JHMS** | **1** | 138.52 | 125.92 |
| | **2** | 134.00 | 125.55 |

Note: JHMS=John Henryism Median Split
1=low John Henryism; 2=high John Henryism
CONDIS=Concordant/Discordant Social Mobility
1=condordant; 2=discordant
Covariates: FHX=family history of hypertension; RX=medication, $S_s$ Educational level; HHED=head of household's education

Table 17

2 (John Henryism) X 2 (Social Mobility) ANCOVA Cell Means for DBP

Covariates=FHX, AGE, RX, $S_s$ ED, BMI, SMOKING, HHED

TOTAL MEAN=87.07

| | | Cell Mean | Cell Mean |
|---|---|---|---|
| JHMS | | 1 | 2 |
| | | 86.56 | 87.52 |
| CONDIS | | 89.48 | 84.93 |
| | | **CONDIS** | |
| | | **1** | **2** |
| **JHMS** | **1** | 89.09 | 84.24 |
| | **2** | 89.84 | 85.52 |

Note: JHMS=John Henryism Median Split
1=low John Henryism; 2=high John Henryism
CONDIS=Concordant/Discordant Social Mobility
1=concordant; 2=discordant
Covariates: FHX=family history of hypertension; RX=medication; $S_s$ Ed=subject's educational level; BMI=body mass index; HHED=head of household's educational level

Table 18

Multivariate ANCOVA

| Covariate | t | p |
|---|---|---|
| | **SBP** | |
| Family History | -.69 | .49 |
| Medication | -.76 | .45 |
| Body Mass Index | -.80 | .43 |
| Smoking | 1.31 | .19 |
| Subject's Education | -1.87 | .06 |
| Age | -3.69 | .00*** |
| | **DBP** | |
| Family History | -.80 | .42 |
| Medication | -.85 | .39 |
| Body Mass Index | -.39 | .70 |
| Smoking | 1.92 | .06 |
| Subject's Education | -2.50 | .01** |
| Age | 2.58 | .01** |

*p <.05 ** p <.01 ***p <.001

Table 19

Intercorrelations among Selected Risk Factors and Blood Pressure

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. John Henryism | | | | | -.01 | -.10 | .13 | -.28** | .12 | .01 |
| 2. Social Mobility | | | | -.01 | | .05 | -.26 | -.21** | -.11 | .03 |
| 3. Stress | | | | -.10 | -.10 | | -.04 | .09 | .13 | -.18* |
| 4. Age of Onset | | | | .13 | .13 | -.04 | | -.01 | .23 | -.28 |
| 5. # of Children | | | | -.28** | -.28** | .09 | -.01 | | .08 | -.00 |
| 6. # of Siblings | | | | .12 | .12 | .13 | .23 | .08 | | .10 |
| 7. Exercise | | | | .01 | .03 | -.18 | -.28 | -.00 | .10 | |

Note: Age of Onset=Age of onset of hypertension
* p <.05 **p <.01 ***p<.001

APPROVAL SHEET

The dissertation submitted by Stephanie L. Livingston has been read and approved by the following committee:

Dr. Steven Brown, Co-Director
Professor, Counseling & Educational Psychology, Loyola University of Chicago

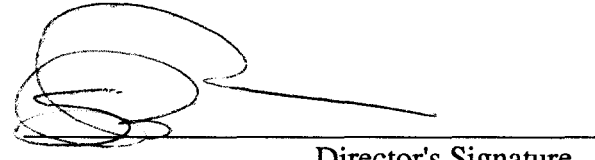
Dr. Kevin Hartigan, Co-Director
*Assistant Professor, Counseling & Educational Psychology, Loyola University of Chicago

Dr. Sara Knight
Assistant Professor, Department of Psychiatry, The University of Chicago

The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

2/14/90
Date


Director's Signature

*At the time of the dissertation proposal approval, Dr. Hartigan was assistant professor at Loyola. He is currently at the Evanston Psychological Group.