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Mediators of Heightened Pressor Responses to Phenylephrine

A Dissertation submitted in partial satisfaction of the requirements for the degree

Doctor of Philosophy

in

Clinical Psychology

by

KaMala S. Thomas

Committee in charge:

University of California, San Diego

Joel E. Dimsdale, Chair
Sonia Ancoli-Israel
Michael Ziegler

San Diego State University

Karen Coleman
Linda Gallo
Vanessa Malcarne

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The Dissertation of KaMala S. Thomas is approved, and it is acceptable in quality and form for publication on microfilm:

Chair

University of California, San Diego

San Diego State University

2007

DEDICATION

To my mother, Gloria Thomas, whose prayers, support, and sacrifices made it possible for me to accomplish this goal.

To my sister, LaTonya Thomas-Lavender, and her children, Caleb Thomas-Lavender, and Kalen Lavender. Thank you for believing in me.

To my grandmother, aunts, uncles, and cousins who supported me through this process. To my uncles, Michael Jones and Hersy Jones, Jr, whose example taught me the importance of excellence and integrity in the face of challenges.

EPIGRAPH

If the misery of the poor be caused not by the laws of nature, but by our institutions, great is our sin. ~Charles Darwin

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VITA

Education

June, 2007	Doctor of Philosophy, San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology Specialty track: Behavioral Medicine
June, 2007	APA-Approved Clinical Psychology Internship University of California, Los Angeles Neuropsychiatric Institute
June, 2006	Master of Public Health, San Diego State University, School of Public Health, Health Promotion concentration.
May, 2004	Master of Science, Clinical Psychology, San Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology
June, 2001	Master of Arts, General Experimental Psychology, California State University, San Bernardino
June, 1998	Bachelor of Arts, Psychology, California State University, San Bernardino, Psychology Honors Graduate

Research Grants and Fellowships

2002-2006	American Psychological Association Minority Fellowship Program Mental Health Research Award. National Institutes of Mental Health Predoctoral Fellowship (F31). $20,772
2002	EXPORT Grant. San Diego EXPORT Center, National Center of Minority Health and Health Disparities, National Institutes of Health. $9,000
2001	University of California, San Diego Predoctoral Fellowship. National Institutes of Health. $16,000

2001	Eugene Cota Robles Fellowship. University of California, San Diego. $16,000
2000	California Predoctoral Scholarship. California State University, Chancellor's Office. $5,500
1999	Associated Students Incorporated Research Grant. California State University, San Bernardino. $500
1998	Graduate Equity Fellowship. California State University, Chancellor's Office. $2,000
1998	Associated Students Incorporated Travel Grant. California State University, San Bernardino. $400
1997	Associated Students Incorporated Research Grant. California State University, San Bernardino. $250

Honors and Awards

2004	American Psychological Association Outstanding Student Poster Recognition
2004	Society of Behavioral Medicine Meritorious Student Paper Recognition
1998	Psychology Honors Graduate, California State University, San Bernardino
1998	Psi Chi, the National Honor Society in Psychology
1997	McNair Scholars Fellow

Professional Affiliations

Society of Behavioral Medicine, Student Member

American Psychological Association, Student Member

Professional Activities

2004 – 2006	Graduate Student Liaison, Office of Graduate Studies and Research, University of California, San Diego
2001 – 2006	Diversity Advisory Committee, Office of Graduate Studies and Research, University of California, San Diego
1997 – 1998	Mentor, Learners Enroute Toward Success, Pomona, California.

Clinical Experience

2006 – 2007:	Health and Behavior Psychology Intern, UCLA Neuropsychiatric Institute, Los Angeles, CA Supervisors: David Wellisch, Ph.D. and Gail Wyatt, Ph.D.
2004 – 2005:	Clinical trainee, University of California San Diego Veteran's Affairs Health System Sleep Disorders Clinic, San Diego, CA Supervisor: Sonia Ancoli-Israel, Ph.D.
2003 – 2004:	Clinical trainee, University of California San Diego Mission Valley Veterans Affairs Behavioral Medicine Clinic, San Diego, CA Supervisor: Ariel Lang, Ph.D.
2002 – 2003:	Student Therapist, San Diego State University Psychology Clinic, San Diego, California. Supervisors: Rick Shulte, Ph.D. and Linda Gallo, Ph.D.

Research Experience

2002 – 2006:	Research Assistant, Stress Physiology Lab, UCSD Department of Psychiatry Faculty Mentor: Joel Dimsdale, M.D.
2002 – 2003:	Assistant to the Associate Editor, Psychology of Women Quarterly Supervisor/Associate Editor: Hope Landrine, Ph.D.

2001 – 2002:	Research Assistant, Behavioral Health Institute, SDSU/UCSD Joint Doctoral Program in Clinical Psychology Faculty Mentors: Elizabeth Klonoff, Ph.D and Hope Landrine, Ph.D.
1999 – 2000:	California Predoctoral Program Summer Research Internship, San Diego State University, San Diego, California. Faculty Mentor: Vanessa Malcarne, Ph.D.
1998 – 1999:	Research Assistant, Orange County Social Service Agency, Santa Ana, California. Supervisor: Diane Halpern, Ph.D.
1997 – 1998:	McNair Scholars Program Summer Research Internship, California State University, San Bernardino. Faculty Mentor: Michael Lewin, Ph.D.

Teaching Experience

1999 – 2000	Teaching Assistant, Experimental Psychology Lab, California State University, San Bernardino.
1999	Teaching Assistant, Lecturer, Psychology 115: Personal and Social Adjustment, California State University, San Bernardino.

Manuscripts

Lim, W., Thomas, K. S., Bardwell, W. A., Dimsdale, J. E. (in press). Which measures of obesity are related to depression and in whom? *Psychosomatics.*

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Abstracts and Academic Presentations

Thomas, K. S., Nelesen, R. A., Bardwell, W. A., Dimsdale, J. E. (March, 2005). *Relationship between depression and pressor sensitivity.* Poster presented at the annual meeting of the American Psychosomatic Society.

Thomas, K. S., Nelesen, R. A., Bardwell, W. A., Dimsdale, J. E. (July, 2004). *Job demands,decisional control, and vascular reactivity in African Americans and Caucasian Americans.* Outstanding student poster presented at the annual meeting of the American Psychological Association.

Thomas, K. S., Nelesen, R. A., Dimsdale, J. E. (March, 2004). *Effects of ethnicity and discrimination on pressor sensitivity.* Poster presented at the annual meeting of American Psychosomatic Society.

Thomas, K. S., Bardwell, W. A., Dimsdale, J. E. (March, 2004). *Relationships between ethnicidentity, sleep architecture, and fatigue.* Meritorious Student Paper presented at the annual meeting of the Society of Behavioral Medicine.

Thomas, K. S., Malcarne, V., Dimsdale, J. (March, 2003). *Ethnic identity differences between black and white students and non-students.* Poster presented at the annual meeting of the American Psychosomatic Society.

Thomas, K. S., Fernandez, S., Klonoff, E., & Landrine, H. (November, 2002). *Ethnic differences in cigarette smoking among college students: Examining the role of racism.* Poster presented at the 2002 National Conference on Tobacco or Health.

Landrine, H., Klonoff, E., Kashima, K., Parekh, B., Fernandez, S., Hickman, N., Thomas, K., Brouillard, C., Zolessi, M., Jensen, J., & Weslowski, Z. (2002, Invited). *Preliminary investigation of the marketing and availability of the new tobacco-related products.* Paper presented at National Conference on Tobacco or Health, San Francisco, CA, November 19, 2002.

Landrine, H., Fernandez, S., Thomas, K., Klonoff, E. A., & Parekh, B. (2001). *Experimental analysis of minors' access to tobacco.* Paper presented at the Tobacco-Related Disease Research Program AIM Conference, Los Angeles, CA, December 4-6, 2001.

Thomas, K. S. & Malcarne, V. (January, 2000). *Perceived discrimination as a predictor of depressive and anxious symptoms: Direct and moderating relationships.* Poster presented at the annual APA Multicultural Conference.

Thomas, K. S. & Lewin, M. R. (November, 1999). *Predictors of fear and worry in a sample of African-American and Caucasian-American college students.* Poster presented at the annual meeting of the Association for the Advancement of Behavior Therapy.

Thomas, K. S. & Lewin, M. R. (April, 1999). *Cultural conditioning and the differential report of anxiety symptoms.* Poster presented at the annual meeting of the Western Psychological Association.

Thomas, K. S. & Lewin, M. R. (April, 1998). *Differences in the report of anxiety across*

cultures. Poster presented at the annual meeting of the Western Psychological Association.

Thomas, K. S. & Lewin, M. R. (November, 1997). *The assessment of fear, anxiety, and worry in a sample of African-Americans, Asian-Americans, Hispanics, and Caucasians*. Poster presented at the annual meeting of the Association for the Advancement of Behavior Therapy.

ABSTRACT OF THE DISSERTATION

Mediators of Heightened Pressor Responses to Phenylephrine

by

KaMala S. Thomas

Doctor of Philosophy in Clinical Psychology

University of California, San Diego, 2007

San Diego State University, 2007

Dr. Joel Dimsdale, Chair

This study examined whether individual and neighborhood socioeconomic status (SES) relate to vascular reactivity to phenylephrine (PE) in 105 African-Americans and 106 Caucasian-Americans. Aim 1 examined whether SES mediates the relationship between ethnicity and pressor responsiveness to PE. Aim 2 examined how neighborhood SES and individual SES relate to pressor responsiveness to PE using multilevel modeling. Aim 3 tested the hypothesis that SES moderates the relationship between perceived discrimination and pressor responsiveness to PE in African-Americans. Neighborhood SES (% below the poverty line, per capita income) was assessed using census block data gathered from the Census Bureau.

Individual SES (education and occupation) was measured using the Hollingshead Index of Social Position. The Scale of Ethnic Experience was used to assess perceived discrimination. Pressor responsiveness was calculated as the systolic and diastolic blood pressure (BP) response to a 100-microgram phenylephrine (PE) bolus administered to participants intravenously. The results revealed that education and occupation each partially mediated ethnic differences in diastolic pressor responsiveness to PE (Aim 1). Low education and occupation were each associated with larger BP increases when given PE. Multilevel analyses revealed that higher education was associated with smaller BP responses to PE only when percentage of neighborhood poverty was 5% or less. As neighborhood poverty increased, the beneficial effect of education on pressor responses was no longer significant. Contrary to what was expected, there was no interaction between SES and perceived discrimination on pressor responses in African-Americans. These results suggest that SES plays an important role in explaining ethnic differences in vascular reactivity to PE.

INTRODUCTION

Epidemiology of Hypertension

Hypertension (HTN) is among the most common health problems affecting Americans today and is associated with an increased risk of stroke and cardiovascular morbidity and mortality (Henderson, Bretsky, Henderson, & Stram, 2001). Epidemiological data indicate that African-Americans have higher rates of HTN than any other ethnic group in this country (Bassett, Fitzhugh, Crespo, King, & McLaughlin, 2002). Prevalence rates for HTN among Hispanic Americans, Caucasian-Americans, and Native Americans are roughly equivalent, ranging from 24%-27%. However, approximately 34% of African-American men and women have HTN, and it may account for over 40% of all deaths in this group (Burt et al., 1995). Further, there is an increased risk of fatal complications from untreated HTN in African-Americans. Compared to Caucasian-Americans, African-Americans have 1.8 times the risk of fatal stroke (Ferdinand, 2006), 1.5 times the risk fatal heart disease, and 4.2 times the rates of end-stage kidney disease (NKUDIC, 2005).

Several factors have been identified as possible mechanisms underlying ethnic disparities in hypertension rates. These include physiological and genetic factors (Campese, Amar, Anjali, Medhat, & Wurgaft, 1997; Chen et al., 2001; Kotchen et al., 2002; Snieder, Harshfield, & Treiber, 2003; Thiel et al., 2003; Treiber et al., 2003; Vita, 2003; Zhu et al., 2003), lifestyle factors (Bassett et al., 2002; Moreau et al., 2001; Vollmer et al., 2001), and chronic stress (Lovallo & Gerin, 2003) .

Chronic Stress

Recent findings reveal that chronic or uncontrollable stress may play a significant role in the development of HTN. According to the reactivity hypothesis, structural vascular changes in response to chronic stress can lead to greater vascular resistance, increasing the risk of HTN and CVD (Lovallo & Gerin, 2003). This hypothesis is supported by epidemiological studies, which demonstrate that there is a higher incidence of CVD morbidity and mortality in individuals who report high levels of occupational stress (Alfredsson et al., 2002; Gallo et al., 2004; Greenlund et al., 1995; Kamarck et al., 2004; Kristensen, 1996; Theorell & Karasek, 1996).

African-Americans may be particularly vulnerable to the effects of stress on blood pressure (BP). Chronic stressors may interact with genetic and lifestyle factors to increase the risk for CVD in this group. Racial discrimination is a unique stressor that may negatively affect cardiovascular health (Broman, 1996; Brondolo, Rieppi, Kelly, & Gerin, 2003; Cooper, 2001; Krieger & Sidney, 1996; Wyatt et al., 2003) and research suggests that racial discrimination is a common experience for African-Americans (Broman, 1996; Krieger & Sidney, 1996; Nelson, 2003; Schulz et al., 2000; Schulz et al., 2000). Recent reports from the Institute of Medicine reveal that African-Americans and other ethnic minorities receive lower quality healthcare than Caucasian-Americans (Nelson, 2003). Further, African-Americans have higher unemployment rates and a longer mean duration of unemployment than Caucasian-Americans (Darity, 2003). These chronic stressors may interact with biological, psychosocial, and behavioral risk factors to increase SNS activity in African-Americans (Anderson, Myers, Pickering, & Jackson, 1989). Over time,

repeated episodes of SNS activation may cause structural vascular changes, leading to the development of HTN.

Stress and Sympathetic Nervous System Activity

Studying neuroendocrine changes that occur during stress may increase our understanding of how chronic stress is associated with the development of HTN and heart disease. When an individual perceives an event as stressful, hormones are released. These hormones act in a number of ways to facilitate responses to stressors. During stress, sympathetic nervous system (SNS) activity increases with a corresponding catecholamine release (Krantz & Falconer, 1997). These changes, along with other neuroendocrine changes increase the availability of glucose, redistribute blood to skeletal muscle and away from the skin and other areas, and support whatever coping responses are applied. The catecholamines, epinephrine (E) and norepinephrine (NE), as well as corticosteroids (CO) and their metabolites have been the most widely studied neuroendocrine factors in stress research and play a major role in the initiation and regulation of stress responses.

SNS agonists act on receptors in various muscles to cause either constriction or dilation in a particular area of the vasculature, depending on the function of the receptors in that vascular bed. For instance, vasoconstriction occurs when α adrenergic receptors in blood vessels affecting the skin, skeletal muscles, heart, and visceral organs are stimulated. On the other hand, vasodilation occurs when beta adrenergic receptors in the heart and skeletal muscles are stimulated (Krantz & Falconer, 1997).

Research consistently demonstrates that African-Americans have greater BP reactivity than Caucasian-Americans (Barnes et al., 2000; Guyll, Matthews, & Bromberger, 2001; Stein, Lang, Singh, He, & Wood, 2000; Wilson, Kliewer, Plybon, & Sica, 2000). Since vasoconstriction is mediated by stimulation of α adrenergic receptors, studies have examined α receptor sensitivity to stress in an effort to understand its role in the development of HTN in African-Americans.

Alpha Receptors and Sympathetic Nervous System Activity

Alpha adrenergic receptors mediate actions in the sympathetic nervous system through the binding of the catecholamines, epinephrine and norepinephrine. These receptors mediate catecholamine induced changes such as vasoconstriction and cardiac contractility. Alpha adrenergic receptors are subdivided into α_1 and α_2 receptors and exist in varying concentrations in a variety of organs and systems such as the heart, lungs, and kidneys (Mills & Dimsdale, 1988); Robertson, Parfyonova, Menshikov, & Hollister, 1988).

Alpha-2 receptors are primarily located in the central nervous system presynaptically where they mediate feedback inhibition of NE, or postsynaptically where their activation causes an inhibition of adenylate cyclase activity and as a result, a decrease in cellular cAMP levels. The sensitivity of these receptors can be measured through tests of platelet rich plasma. Platelet α_2 receptors are not good models of SNS responses to acute stress because these receptors do not change significantly in response to acute stressors (Mills & Dimsdale, 1993). Thus, little work has been conducted examining α_2 receptors in behavioral medicine research.

Alpha-1 adrenergic receptors are postsynaptic and located in the periphery. Typically, α_1 receptor sensitivity is measured using pharmacological infusions. Alpha-1 receptors are better model of SNS responses to acute stressors than α_2 receptors.

Alpha1 Agonist Infusions

Calculations of BP responsiveness to pharmacological challenges can be used to measure α_1 receptor sensitivity. In many pharmacological studies of α receptor sensitivity, phenylephrine (PE) is given to participants. PE is an α_1 agonist that stimulates the same pressor receptors as norepinephrine. This drug mimics the short-term effects of stress on BP by increasing vasoconstriction (Hamilton & Reid, 1983; Ziegler et al., 1995).

There are a number of ways to measure responses to PE. One way is to measure the PE dose required to increase mean arterial pressure by 25 mmHg (known as PD25). The PD25 index is inversely related to vascular α receptor responsiveness. Thus, individuals whose BP exceeds 25mmHg with a lower dose of pheylephrine are considered to have greater α receptor sensitivity.

Another way of assessing responses to PE is to measure pressor responsiveness to a fixed dose of PE during baroreflex testing. Baroreflex testing is a procedure that involves stimulation of the carotid or aortic baroreceptors by administering vasoactive drugs that either augment or decrease BP and heart rate. Changes in arterial BP elicited by these drugs are sensed by baroreceptors. In

response, baroreceptors generate signals to the brain that lead to compensatory adjustments in sympathetic and parasympathetic nerve traffic. During baroreflex testing, a 100 μg PE bolus is administered to individuals and BP responses are assessed immediately. Studies consistently demonstrate that PE infusions lead to increased BP (Dimsdale, Graham, Ziegler, Zusman, & Berry, 1987; Sherwood, Steffen, Blumenthal, Kuhn, & Hinderliter, 2002; Stein et al., 2000). However, this effect is much greater in hypertensives and individuals with impaired baroreflexes.

Ethnic Differences in Pressor Responsiveness

Studies have shown that African-Americans have increased α adrenergic receptor sensitivity to pharmacological challenges (Crisostomo et al., 1998; Dimsdale et al., 1987; Merritt, Sollers, Evans, Zonderman, & Thayer, 2003; Sherwood & Hinderliter, 1993; Sherwood et al., 2002; Stein et al., 2000; Thomas, Nelesen, Ziegler, Bardwell, & Dimsdale, 2004). This heightened pressor responsiveness may lead to greater cardiovascular reactivity in African-Americans in response to stress, placing them at greater risk for developing HTN.

In one of the earliest studies of ethnic differences in pressor responses to an α_1 agonist, Dimsdale and colleagues (Dimsdale et al., 1987) found that African-Americans had greater increases in BP than Caucasian-Americans when given NE infusions. Sherwood and Hinderliter (Sherwood & Hinderliter, 1993) also examined pressor sensitivity in 13 African-American and 13 Caucasian-American males matched for age, BMI, and BP status. Participants were given an α adrenergic receptor agonist (PE) to determine how much of it would be required to increase

mean arterial BP by 25 mmHg (PD_{25}). African-Americans had higher α receptor sensitivity than Caucasian-Americans, suggesting that exaggerated vascular reactivity to sympathetic nervous system activation in African-Americans is due to heightened α adrenergic receptor sensitivity.

Stein and colleagues (Stein et al., 2000) also found ethnic differences in adrenergic responsiveness in a study examining vascular reactivity in 10 African-Americans and 10 Caucasian-Americans. After controlling for gender, diet, sodium, caffeine, and alcohol intake, African-Americans had greater vascular resistance than Caucasian-Americans in response to a cold pressor test and required less PE than Caucasian-Americans to decrease forearm blood flow by 25%. Participants' response to PE was not affected by family history of HTN and was not related to resting BP in African-Americans or Caucasian-Americans, suggesting that heritability and tonic BP levels do not completely explain heightened α receptor sensitivity in African-Americans.

In another study of BP responses to PE, Sherwood and colleagues (Sherwood et al., 2002) examined the relationship between nighttime BP dipping and sympathetic nervous system activity in a biracial sample of 172 adult men and women. These researchers found that compared to Caucasian-Americans, African-Americans had a smaller drop in BP and norepinephrine from day to nighttime and had greater increases in BP in responses to PE. They also found that nondippers were more likely to have heightened BP responses to PE.

Very little research has been conducted examining whether the increased pressor sensitivity to PE that has been observed in African-Americans is associated

with psychosocial factors (Hughes, Sherwood, Blumenthal, Suarez, & Hinderliter, 2003; Thomas et al., 2004). However, the results of studies in this area suggest that psychosocial factors may be associated with increased pressor sensitivity to pharmacological challenges. For instance, Thomas et al. (Thomas et al., 2004) examined the association between job strain and vasoconstriction in response to PE in African-Americans and Caucasian-Americans. Similar to other studies, these researchers found that African-Americans had greater BP responses to PE than Caucasian-Americans. They also found an interaction between ethnicity and job strain such that African-Americans who reported low control over decisions at work had the greatest increase in BP when given PE. This suggests that psychosocial factors may be particularly important in understanding α receptor sensitivity in African-Americans.

SES and Census Data

Census data has been used in epidemiological studies to examine the relationship between SES and CVD. These studies reveal that there is a higher incidence of CVD in lower SES individuals compared to higher SES individuals (Singh & Siahpush, 2002). Further, studies of census data show that African-Americans living in lower SES neighborhoods have more risk factors for CVD than those in higher SES neighborhoods (Cubbin, Hadden, & Winkleby, 2001). Thus, using census data to characterize neighborhood SES may shed light on the higher rates of CVD among African-Americans. Along with having lower SES, African-Americans tend to live in communities that are more structurally disadvantaged than

those occupied by Caucasian-Americans (Robert & Reither, 2004). Living in disadvantaged communities may be associated with poor health due to poor neighborhood safety and lack of resources within the community to support healthy lifestyles (i.e., physical activity and healthy diets).

African-Americans are more likely to live in lower SES neighborhoods than Caucasian-Americans with the same income and education level (Diez-Roux et al., 2001; Jargowsky, 1997). Thus, using both individual and aggregate measures of SES in research may provide a more complete picture of the role of economic disadvantage in health disparities. In a study examining associations between ethnicity, community disadvantage, and BMI, Robert and Reither (Robert & Reither, 2004) found that individual and neighborhood SES were each independently related to BMI in African-Americans. Similarly, in a study examining whether neighborhood SES relates to physical health, Boardman (Boardman, 2004) found that even after controlling for individual SES and other demographic factors, low neighborhood SES was associated with poor health.

Krieger and colleagues (Krieger, Chen, Waterman, Rehkopf, & Subramanian, 2005) examined associations between area-based socioeconomic data and ethnic health inequalities using public health surveillance data in Massachusetts and Rhode Island. These researchers found that the incidence of cardiovascular mortality was highest in census tracts with a higher percentage of individuals living below the poverty line. Further, adjusting for census tract level poverty substantially reduced the risk of CV mortality in African-Americans and Hispanics compared with Whites. This suggests that ethnic disparities in CV mortality may

partially be associated with neighborhood SES.

Neighborhood disadvantage may be more closely related to poor CV health in African-Americans than individual SES due to the stress of living in unsafe neighborhoods and struggling to meet living expenses with limited resources to do so. It has also been suggested that aggregate measures of SES (census data) are more effective than individual SES in controlling for SES confounding of relationships between ethnicity and health outcomes (Soobader, LeClere, Hadden, & Maury, 2001). However, few studies have examined relationships between SES characterized by census tract data and SNS activity in African-Americans**.** Thus, this area should be explored.

SES, Ethnicity, and Alpha Receptor Sensitivity

There is substantial evidence that low SES is associated with poor CV health. Epidemiological data demonstrate that there are greater incidences of HTN and stroke among low SES individuals (Alter, Iron, Austin, & Naylor, 2004; Gillum, Mussolino, & Madans, 1997; Keil et al., 1993; Mensah, Mokdad, Ford, Greenlund, & Croft, 2005). Further, studies have found greater vascular reactivity among individuals in lower SES brackets (Steptoe, Willemsen, Kunz-Ebrecht, & Owen, 2003).

African-Americans tend to have lower SES and live in poorer neighborhoods than Caucasian-Americans. Compared to African-Americans in higher SES brackets, those in lower SES groups tend to have higher rates of hypertension and CVD mortality (Cubbin et al., 2001; Matthews et al., 2002) Thus, it seems likely

that socioeconomic factors may partially account for the greater α receptor sensitivity in African-Americans as compared to Caucasian-Americans. However, few studies have examined the association between SES and α adrenergic responsiveness in African-Americans. Among those studies that have been conducted, there have been inconsistent findings, with some researchers reporting that SES is associated with exaggerated hemodynamic responses to laboratory stress (Kapuku, Treiber, & Davis, 2002; Wilson et al., 2000) and others not finding such as association (Jackson, Treiber, Turner, Davis, & Strong, 1999; Musante et al., 2000).

Wilson and colleagues (Wilson et al., 2000) examined the effects of individual and neighborhood SES on BP reactivity to a video game challenge in African-American adolescents who resided in Memphis, Tennessee and Richmond, Virginia. Neighborhood SES was assessed using census tract data by calculating the percentage of household incomes below the poverty line in each census tract. These data were then used to create two groups, high versus low SES neighborhoods. Family SES was also assessed using self-reported parental income and education. The results of this study revealed that adolescents who lived in poorer neighborhoods had lower BP reactivity if their parents were more educated or had a higher annual income (Wilson et al., 2000).

In a study examining BP responses to a video game and cold pressor test, Kapuku and colleagues (Kapuku et al., 2002) found that living in a lower SES neighborhood was related to increased diastolic BP reactivity in African-American males. Similarly, Cook and colleagues (Cook et al., 2001) found that low SES and

family history of HTN were both related to exaggerated diastolic blood pressure (DBP) and total peripheral resistance (TPR) responses to behavioral challenges in African-American and Caucasian-American youth (Cook et al., 2001). However, one limitation of this study was that data from African-Americans and Caucasian-Americans were combined and the association between SES and hemodynamic responsiveness was not examined in each ethnic group separately. Thus, it is unclear whether the relationship was the same in each ethnic group.

To understand the association between SES and BP reactivity in African-Americans, the John Henryism hypothesis has been proposed. According to the 'John Henryism' hypothesis, the stress of struggling to achieve a successful lifestyle with fewer resources and opportunities to do so can lead to chronic medical problems, including HTN (Dressler, Bendon, & Negger, 1998; James, Keenan, Strogatz, Browning, & Garrett, 1992). Merritt and colleagues (2004) examined relationships between SES, John Henryism, and cardiovascular reactivity to an anger recall task in African-American adult males. These researchers observed that the combination of high John Henryism and low educational attainment was associated with higher DBP during anger recall and recovery.

Contrary to findings that low SES African-Americans have greater BP reactivity than those in higher SES brackets, some studies have reported heightened reactivity among African-Americans in higher SES groups (Barnes et al., 2000; Jackson et al., 1999; Musante et al., 2000). Musante et al. (Musante et al., 2000) examined the association between SES and hemodynamic responses to laboratory stress in 483 African-American and Caucasian-American adolescents in Augusta,

Georgia. The two laboratory stressors included a car driving simulation and a 10 minute social stressor interview in which participants discussed a recently experienced stressful situation. SES was measured using census tract data as well as Hollingshead's Index of Social Status. There was no relation between SES and TPR responses to either laboratory stressor. However, there was a main effect of SES on DBP reactivity during the social stressor interview. Contrary to studies finding greater reactivity among those with lower SES, Musante et al. (2000) found that individuals with higher family SES had greater DBP reactivity. In a similar study, Jackson and colleagues (Jackson et al., 1999) found an interaction between race and neighborhood SES such that there was heightened BP reactivity among low SES Caucasian-Americans and high SES African-Americans who underwent several acute laboratory challenges. Thus, the effect of SES on BP may not be the same in African-Americans and Caucasian-Americans.

Limitations and Future Directions

It is possible that methodological differences among studies concerning the manner in which SES is measured may account for inconsistent findings in studies of SES and BP reactivity. For instance, some researchers have measured SES using census data (Cook et al., 2001; Musante et al., 2000; Wilson et al., 2000). With this method, individuals are classified as high, medium, or low SES based on the median income and education in their census tract. Although this method seems to be a more objective measure of SES than self-report data, it also has limitations. Within some neighborhoods, there may be a great deal of variability in the income and

education of individual households. Consequently, households that share the same census tract may have a significantly different economic makeup. This limits the precision with which this measure assesses SES and can limit one's ability to determine the true effects of SES on α receptor sensitivity.

Other studies have assessed SES using self-report instruments (Kapuku et al., 2002; Merritt, Bennett, Williams, Sollers, & Thayer, 2004; Musante et al., 2000). One self-report instrument that has been widely used is Hollingshead's measure of social status. This is a self-report measure that classifies individuals based on education and occupation. This scale was developed in the 1950's. It has been well validated and is widely used in studies examining associations between SES and health. However, it has been criticized as being outdated. Further, it can be difficult to assess SES in individuals who do not work outside of the home (i.e., homemakers), individuals who work part-time, and students. There is also evidence that African-Americans and Caucasian-Americans with similar income and education may not be equivalent on Hollingshead (Anderson & Armstead, 1995). Thus, ethnic differences in SES identified through the use of this scale may not reflect actual differences that exist in the population.

More research should be conducted to understand the nature of the relationship between SES and α adrenergic responsiveness. To date, no published studies have examined the relationship between SES and α receptor sensitivity to a pharmacological challenge. Thus, studies should be conducted to extend the literature in this area.

Purpose of the Current Study

The purpose of the current study was to examine whether neighborhood SES and individual SES (education and occupation) are associated with vascular reactivity to PE using data from two previous protocols. Data from baroreflex testing conducted on all participants was used to assess pressor responsiveness to PE. Individual SES was assessed using Hollingshead data and neighborhood SES was assessed using census block data gathered from zipcode and address information collected from all participants. Mediational analyses were conducted to test the hypothesis that SES mediates the relationship between ethnicity and pressor responsiveness (Aim 1).

A major aim of the current study was to determine whether neighborhood SES (census block data) and individual SES (education and occupation) interact to predict pressor responses to PE. Specifically, multilevel modeling was used to examine whether the effect of individual SES on vascular reactivity is nested within neighborhood SES. It was expected that there would be a stronger relationship between neighborhood SES and pressor responsiveness than individual SES and pressor responsiveness (Aim 2). It was also expected that individual education would buffer the effects of neighborhood SES on pressor responsiveness (Aim 2). This was a unique aspect of this study. To date, no studies have examined the effects of SES on pressor responses to PE. Further, no published study has utilized multilevel modeling to assess the interaction between neighborhood and individual SES factors. The proposed study will do so.

Census data have been widely used in public health studies to characterize

SES, using median income and education in each census tract. Recently, behavioral medicine researchers have taken advantage of this methodology as a means of objectively measuring SES. Behavioral medicine studies typically have access to smaller samples than studies in public health. One possible limitation of this is that there may be restricted variability within the sample. Although this is a possible limitation, studies using smaller sample sizes have been able to characterize high and low SES using census data and have found that it correlates with physiological measures. For instance, Wilson et al (2000) used census tract data to characterize neighborhood SES in 86 participants. This study revealed greater BP reactivity in African-Americans in low SES neighborhoods compared to those in high SES neighborhoods.

Perceived discrimination data, was collected from approximately one half of the participants. This data was used to examine whether SES moderates the relationship between perceived discrimination and pressor responses to PE in a subset of the sample. It was expected that African Americans in lower SES neighborhoods would have more pressor responsiveness to PE than those in higher SES neighborhoods (Aim 3). It was also expected that SES would moderate the relationship between perceived discrimination and pressor responsiveness to PE in African Americans (Aim 3).

METHODS

Participants

Participants for the current study included 211 individuals who participated in two protocols of research on stress, ethnicity, and BP. There were 105 African-Americans (55 men) and 106 Caucasian-Americans (62 men). The sample consisted of 117 males and 94 females. Participants were recruited from the San Diego community via advertisement and referrals. Participants were between the ages of 25 and 52 (Mean=37.6, SEM=0.50), with an ideal body weight between 90% and 130% (Metropolitan life tables, 41) and resting blood pressure (BP) lower than 180/110 mm Hg at screening. Screening BP was defined as the average of 3 seated BP's. Women were excluded if postmenopausal, diagnosed with premenstrual syndrome, taking oral contraceptives, or pregnant. Individuals with major medical conditions other than hypertension or with a psychiatric disorder were excluded from the study. Participants were excluded if they had a medical illness other than hypertension. Screening BP was taken using Dinamap model 1846-SZ with appropriate size cuffs. BP readings were taken in the right arm and defined as the average of 3 seated BP's. Participants were recumbent and their hands were kept at heart level. They were instructed to refrain from moving their hand during the recording period. Participants whose systolic BP was above 140 mmHg and/or whose diastolic BP was above 90 mmHg were considered hypertensive. Concerning BP status, 50 of the participants were hypertensive (32 African-Americans, 18 Caucasian-Americans) and 160 were normotensive.

Measures

Scale of Ethnic Experience (SEE; (Malcarne, Chavira, Fernandez, & Liu, 2006)). This 32-item, 5-point Likert-type self-report questionnaire measures the experience of ethnicity across several dimensions. We conducted analyses using the "Perceived Discrimination" subscale. Perceived discrimination assesses whether an individual believes that members of his or her ethnic group have been discriminated against in society. Sample items include, "In my life, I have experienced prejudice because of my ethnicity," and "My ethnic group does not have the same opportunities as other ethnic groups." High scores on this subscale indicate greater endorsement of the construct being assessed. Internal consistency coefficients for the perceived discrimination subscale were .86 and .81 in a validation sample of African-Americans and Caucasian-Americans respectively (Malcarne et al., 2006). The SEE was developed to be administered to individuals of any ethnic background. It was validated on a college sample from diverse ethnic groups and has been found to have sound psychometric properties, with internal consistency coefficients ranging from .82 to .89 for the subscales, and concurrent validity demonstrated through correlations with existing group-specific instruments in predicted directions.

Two-Factor Index of Social Position (Hollingshead, 1958). Individual SES was measured using the Hollingshead two-factor index of social position. This two-factor scale measures an individual's social status. The two factors that determine social position include occupation and education. Each factor has a 1 to 7 range,

with lower scores representing a higher social status. Scores on the education and occupation factor can be combined to obtain an index of social class. Social Class is scaled in five categories, with lower scores representing a higher social class. Validation studies support the utility of this scale as a reliable and valid measure of social status

Neighborhood Demographic Characteristics

Census data were used to characterize neighborhood SES. Address and zip code data were collected from all participants in each of our protocols. This information was entered into a database and used to determine the census block that participants reside in. Census block, which is a subdivision of a census tract, enables researchers to determine neighborhood SES with greater precision than using zip code data alone. A census block is the smallest geographic unit created by the Census Bureau. These areas are bounded on all sides by visible features such as streets, roads, streams, and railroad tracks as well as by invisible features such as city, town and county limits, and property lines. Since households that share the same zipcode may not reside in the same census block, census block data is more sensitive to subtle changes in SES between streets in the same zip code. Information regarding the demographic characteristics of each participant's census blocks was downloaded from the Census Bureau's website and matched with participants' identification number using American Fact Finder Census 2000 datasets (2000, 2001; Census, 2001).

Three variables were used to assess demographic characteristics in each census block in the current study. These include per capita income, percentage of individuals living below the poverty line, and percentage of African-Americans residing in the census block. To aid in interpretation of the results, percentage of individuals living below the poverty line and percentage of African-Americans residing in the census block were multiplied by 10 and entered as whole numbers in the database.

Pressor Sensitivity

Data from baroreflex testing was used to assess pressor sensitivity sensitivity. During baroreflex testing, a Finapres BP cuff (Ohmeda, 2300) was used to measure BP signals which were relayed to an A/D converter (Data Translation, DT2801), sampling at 1 kHz per channel (Global laboratory software, Data Translation) and stored in an IBM PC compatible computer in 3-minute epochs. The Finapres BP cuff was placed on the third or fourth digit of the hand opposite the venous injection site. Hand position and cuff location were adjusted so that the Finapres readings were within 5 mmHg of casual BP determinations.

The participants were tested for their response to PE in the UCSD General Clinical Research Center in the afternoon. After resting supine for at least twenty minutes, baseline data were collected over the last three minutes of the twenty minute resting period. Immediately following baseline, a 100 μg PE bolus was administered intravenously. Pressor sensitivity was assessed by recording the changes in BP in response to PE and was calculated as peak level BP in response to

PE dosage minus baseline BP.

Statistical Analysis

The following statistical analyses were employed to test the hypotheses in each Aim.

Aim 1

The first aim in this dissertation study was to test the hypothesis that SES mediates the relationship between ethnicity and pressor responses to PE. This hypothesis was tested using the test for assessing indirect effects recommended by Preacher and Hayes (Preacher & Hayes, 2004). This test of indirect effects assesses mediation directly by testing whether the product of paths *a* and *b* (the indirect effect) are significantly different from zero.

According to Preacher and Hayes (2004), the following two conditions must be met in order to demonstrate that mediation exists. First, it must be demonstrated that the independent variable significantly relates to the outcome variable (Figure 1, Panel A, path *c*). Second, it must be demonstrated that the indirect effect (Figure 1, Panel B, path *c'*), which includes the product *ab*, is statistically significant in the direction predicted by the mediation hypothesis. Preacher and Hayes (Preacher & Hayes, 2004, 2005) created a macro for testing mediation while controlling for potential covariates. This macro was downloaded into SPSS and used to examine whether SES mediates the relationship between ethnicity and pressor responses to PE in SPSS. In these analyses, ethnicity was entered as the independent variable and the outcome variables included systolic and diastolic BP responses to PE.

Individual SES variables (education, occupation) and neighborhood SES variables (% living below poverty, per capita income) were tested as proposed mediators. Each mediator was tested in a separate analysis. Covariates included baseline BP and BMI.

Aim 2

Multilevel analyses were used to examine whether neighborhood demographic characteristics moderated the association between individual education level and BP responsiveness to PE. These analyses were conducted using Hierarchical Linear and Nonlinear Modeling (HLM 6.0, Student Version; Raudenbush, Bryk, Congdon, 2005) statistical software according to the methods described by Raudenbush and Bryk (Raudenbush & Bryk, 2002). The procedures for conducting the multilevel analyses were as follows.

Level-1 Predictor Variables

Level-1 predictor variables included ethnicity and individual education level. These variables were entered on level-1 of the analysis. An important step in conducting multilevel analyses is determining whether predictor variables should be centered around the mean of the predictor variable in their level-2 group (group centered), centered around the mean of the predictor variable in the entire sample (grand-mean centered), or uncentered. Determining whether to group or grand mean center a predictor variable depends on whether one thinks the relationship between the level-1 variable and the outcome is the same in different level-2 groups.

A predictor variable should be group mean centered if it is believed that the relationship between predictor variable and the outcome will be different across level-2 groups. A predictor variable should be grand mean centered if it is believed that the relationship is the same in different level-2 groups.

Although centering does not affect the outcome of analyses, it does affect the interpretation of the intercept in level-1 models. If a variable is group mean centered, then change scores should be interpreted as deviations from the level-2 group mean. If it is grand mean centered, then change scores should be interpreted as deviations from the grand mean.

Ethnicity was dummy coded as 0 (Caucasian-American) or 1 (African-American). It is recommended (Raudenbush & Bryk, 2002) that dummy coded variables with a restricted range remain uncentered because their mean is not meaningful. Therefore, this variable was entered into the analysis as an uncentered variable.

Education was scaled into 6 categories and coded in the following manner: graduate or professional training = 6; bachelor's degree = 5; partial college = 4; high school = 3; partial high school = 2; junior high school = 1. Since it was hypothesized that the effect of education on pressor responses to PE is not the same in each neighborhood SES group, education was entered as a group mean centered variable so that the effect of education could be examined separately within each neighborhood SES group. Therefore, the intercept represents the mean BP response to PE at each level 2 group's average education level. Further, the slope of education on BP response to PE represents the changes in pressor response to PE as

participants' education level deviates from the average education level in their neighborhood SES group.

Baseline BP and body mass index (BMI) were entered as covariates in the study. These variables were both grand-mean centered. Therefore, the intercepts for these variables represent the predicted BP response to PE when an individual's baseline BP and BMI are equal to the mean for the entire sample.

Level-2 Predictor Variables

Neighborhood variables were entered as level-2 predictor variables in analyses. These included percentage of individuals living below the poverty line, per capita income in block, and percentage of African-Americans living in block. In order to assess the effect of level-2 predictors on associations between level-1 predictors and outcome variables, level-1 data must be aggregated to each level-2 group. To do this, neighborhood per capita income was transformed into 20 groups ranging from the lowest 5% per capita income to the highest 5% per capita income. This created 20 level-2 groups representing different per capita incomes in the sample. Each level-2 group had approximately 10 participants. A separate level-2 dataset was then created in which level-1 data were aggregated to their corresponding level-2 group's identification number.

Level-2 predictor variables can be entered as either grand-mean centered or uncentered varables. Neighborhood poverty level and percentage of African-Americans living in the block were both entered as uncentered variables and per capita income was entered as a grand-mean centered variable.

Outcome Variables

Outcome variables included systolic and diastolic blood pressure responses to PE. These variables were calculated as peak level BP in response to PE minus baseline BP.

Aim 3

Aim 3 examined relationships between SES, perceived discrimination, and pressor responses to PE using a subset of the data. The first hypothesis is that African-Americans report more perceived discrimination than Caucasian-Americans. A t-test was used to test this hypothesis, with ethnicity entered as the independent variable and perceived discrimination scores entered as outcome variable. A correlation analyses was used to test the hypothesis that African-Americans in lower SES groups will report more discrimination than African-Americans in higher SES groups (Hypothesis 2). In this analysis, the bivariate correlation between discrimination and SES was examined in the African-American sample.

The final hypothesis in Aim 3 is that discrimination will moderate the relationship between SES and pressor response to PE in African-Americans. To test this hypothesis, hierarchical regression analyses were conducted examining the interaction between discrimination and each of the SES variables on pressor response variables. Covariates were entered on Step 1 of the regression analyses. Perceived discrimination and SES were entered on Step 2, with individual and neighborhood SES variables entered in a separate regression analysis. The

interaction term (perceived discrimination*SES) was entered on Step 3 of the analysis. Outcome variables included systolic and diastolic pressor response to PE.

RESULTS

Sample Characteristics

Ethnic differences in sample characteristics are listed in Table 1. African-Americans had higher baseline systolic BP ($t_{1, 198}$ = -2.85, $p < .01$), a higher BMI ($t_{1, 202}$ = -4.75, $p < .01$), and were more likely to be hypertensive ($t_{1, 202}$ = -2.26, $p < .05$) than Caucasian-Americans. African-Americans also had a higher systolic ($t_{1, 146}$ = -2.65, $p < .01$) and diastolic ($t_{1, 146}$ = -3.03, $p < .01$) pressor responses to PE. Concerning SES, African-American had less education ($t_{1, 204}$ = 3.70, $p < .01$) and less skilled occupations ($t_{1, 204}$ = 3.31, $p < .01$) than Caucasian-Americans. They were also more likely to live in neighborhoods with more poverty ($t_{1, 192}$ = -2.40, $p < .05$) and a lower per capita income ($t_{1, 191}$ = 5.90, $p < .01$) than Caucasian-Americans.

Differences in sample characteristics between the two protocols are listed in Table 2. Individuals in the 1995 protocol were more likely to be diagnosed with hypertension($t_{1, 208}$ = 2.60, $p < .01$) and had a higher systolic ($t_{1, 150}$ = 2.23, $p < .05$) and diastolic ($t_{1, 150}$ = 3.59, $p < .01$) pressor response than individuals in the 2000 protocol. Further, those in the 1995 protocol were slightly older ($t_{1, 207}$ = 2.14, $p < .05$), and were more likely to be African-American ($t_{1, 209}$ = 2.49, $p < .05$) than individuals in the 2000 protocol. They also were more likely to be less educated ($t_{1, 205}$ = -4.90, $p < .01$), and live in neighborhoods with more poverty ($t_{1, 196}$ = 3.23, $p < .01$) and lower per capita income ($t_{1, 195}$ = -2.76, $p < .01$) than those in the 2000 protocol.

Aim 1

Education as a Mediator of Ethnicity and Systolic Pressor Responses to PE. The full model accounted for 10.9% of the variance in systolic pressor responses to PE ($F_{4, 132} = 4.06$, $p = .004$). BMI (B=-.51, $p = .02$) and baseline SBP (B= .19, $p = .01$) both significantly predicted systolic pressor responses to PE. The first condition for demonstrating mediation involved testing whether ethnicity predicted systolic pressor responses to PE. Analysis revealed that ethnicity was significantly associated with systolic BP responses to PE (B=5.44, $p = .018$). The *ab* path testing whether education mediates the relationship between ethnicity and systolic BP responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met and education did not mediate ethnic differences in systolic pressor responses to PE.

Education as a Mediator of Ethnicity and Diastolic Pressor Responses to PE. The full model accounted for 11.8% of the variance in diastolic pressor responses to PE ($F_{4, 132} = 4.42$, $p = .002$). BMI significantly predicted diastolic pressor responses to PE (B=-.306, $p = .02$). There was not a significant relationship between baseline DBP and diastolic pressor responses to PE. The first condition for demonstrating mediation involved testing whether ethnicity predicted diastolic pressor responses to PE. Analysis revealed that ethnicity was significantly associated with diastolic BP responses to PE (B=4.36, $p = .003$). Path *a* was statistically significant, demonstrating that African-Americans had lower educational attainment than Caucasian-Americans (B=-.564, $p = .003$). Path *b*

testing whether education predicts diastolic pressor responses to PE was marginally significant (B=-1.17, p = .07). The *ab* path testing whether education mediates the relationship between ethnicity and systolic BP responses to PE was statistically significant (B=-.665, p<.05), demonstrating that education did mediate ethnic differences in diastolic pressor responses to PE. After the effect of education was taken into account, ethnicity remained a statistically significant predictor of diastolic pressor responses to PE (B=3.69, p<.05). Thus, education only partially mediated the relationship between ethnicity and diastolic pressor responses to PE (see Figure 2).

Occupation as a Mediator of Ethnicity and Systolic Pressor Responses to PE. The full model accounted for 13.4% of the variance in systolic pressor responses to PE ($F_{4, 131}$ = 5.07, p =.008). BMI (B=-.49, p = .02) and baseline SBP (B= .20, p = .005) both significantly predicted systolic pressor responses to PE. The *ab* path testing whether occupation mediates the relationship between ethnicity and systolic BP responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met.

Occupation as a Mediator of Ethnicity and Diastolic Pressor Responses to PE. The full model accounted for 16.2% of the variance in diastolic pressor responses to PE ($F_{4, 131}$ = 6.32, p <.001). BMI significantly predicted diastolic pressor responses to PE (B=-.271, p = .03). There was not a significant relationship between baseline DBP diastolic pressor responses to PE. Ethnicity was significantly

associated with diastolic BP responses to PE (B=4.94, $p < .001$). Path *a* was statistically significant, demonstrating that African-Americans had lower skilled occupations than Caucasian-Americans (B=-.781, $p = .013$). Path *b* testing whether occupation predicts diastolic pressor responses to PE was statistically significant (B=-.788, $p = .03$). The *ab* path testing whether occupation mediates the relationship between ethnicity and diastolic BP responses to PE was statistically significant (B=-.615, $p<.05$). After the effect of occupation was taken into account, ethnicity remained a statistically significant predictor of diastolic pressor responses to PE (B=4.33, $p<.05$). Thus, occupation only partially mediated the relationship between ethnicity and diastolic pressor responses to PE (see Figure 3).

Per Capita Income as a Mediator of Ethnicity and Systolic Pressor Responses to PE. The full model accounted for 9.9% of the variance in systolic pressor responses to PE ($F_{4, 121} = 3.31$, $p = .013$). The *ab* path testing whether per capita income mediates the relationship between ethnicity and systolic BP responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met.

Per Capita Income as a Mediator of Ethnicity and Diastolic Pressor Responses to PE. The full model accounted for 9.7% of the variance in systolic pressor responses to PE ($F_{4, 121} = 3.25$, $p = .014$). The *ab* path testing whether per capita income mediates the relationship between ethnicity and diastolic BP

responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met.

Poverty Level as a Mediator of Ethnicity and Systolic Pressor Responses to PE. The full model accounted for 10% of the variance in systolic pressor responses to PE ($F_{4, 121} = 3.37$, $p = .012$). The *ab* path testing whether poverty level mediates the relationship between ethnicity and systolic BP responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met.

Poverty Level as a Mediator of Ethnicity and Diastolic Pressor Responses to PE. The full model accounted for 9.6% of the variance in systolic pressor responses to PE ($F_{4, 121} = 3.25$, $p = .014$). The *ab* path testing whether % of individuals living below the poverty level mediates the relationship between ethnicity and diastolic BP responses to PE was not statistically significant. Therefore, the second condition for demonstrating mediation was not met.

Aim 2

Education Nested Within Neighborhood SES

Association Between Level-1Predictors and Systolic Pressor Response. Table 3 demonstrates the results of the multilevel analysis relating education and neighborhood SES to systolic pressor responses. The level-1 analysis examined whether ethnicity and education were related to pressor response to PE after

controlling for baseline BP and BMI. The chi square tests assessing whether relationship between level-1 variables and systolic pressor response varied between level-2 groups were not significant. Therefore, all level-1 error terms were set as fixed effects. The intercept for the level-1 model was significant (B=34.60, $t_{16, 106}$ = -3.97, p = .001). Less education was associated with a greater increase in SBP when given PE (B=-13.34, $t_{16, 106}$ = -2.38, p = .019). Ethnicity was unrelated to systolic pressor response to PE after education and the covariates were taken into account.

Multilevel Predictors and Systolic Pressor Response. There was not a significant main effect of neighborhood variables on systolic pressor responses to PE. However, there was a significant interaction between percentage of individuals living below the poverty line and education on systolic pressor response. Analysis of the multilevel model revealed that the effect of educational attainment on systolic pressor response was dependent on neighborhood poverty level (B=.554, $t_{16, 106}$ = 2.23, p = .028). Simple slope analyses were calculated to determine the nature of this interaction. Figure 4 depicts the relationship between education and systolic pressor response to PE when 3%, 15%, and 25% of individuals in the neighborhood live below the poverty level. These analyses revealed that less education was associated with a greater systolic pressor response to PE when less than 5% of individuals in the neighborhood lived below the poverty line (B= -12.23, $t_{16, 106}$ = -2.03, p = .044. The effect of education on systolic pressor response to PE was not significant in individuals who lived in neighborhoods where more than 5% of the

residents lived below the poverty line. None of the other neighborhood variables related to systolic pressor response.

Association Between Level-1Predictors and Diastolic Pressor Response. Table 4 demonstrates the results of the multilevel analysis relating education and neighborhood SES to diastolic pressor responses. The chi square test assessing whether relationship between level-1 variables and systolic pressor response varied between level-2 groups was significant for baseline BP (χ^2=22.45, p = .013) and BMI (χ^2=25.53, p = .005), demonstrating that the relationship between these variables and diastolic pressor response was not the same across level-2 groups. Therefore, these variables were set as random effects. All other level-1 error terms were set as fixed effects. The intercept for the level-1 model was significant (B=23.43, $t_{16, 106}$ = 4.40, p < .001), indicating that there is significant variability among participants on diastolic pressor response to PE when all predictor variables are set at 0. Less education was associated with a greater increase in DBP when given PE (B=-7.66, $t_{16, 106}$ = -2.21, p = .029). Ethnicity was unrelated to systolic pressor response to PE after education and the covariates were taken into account.

Multilevel Predictors and Diastolic Pressor Response. There was not a significant main effect of the neighborhood variables on diastolic pressor response. However, there was a significant interaction between percentage of individuals living below the poverty line and education on systolic pressor response. Analysis of the multilevel model revealed the effect of educational attainment on diastolic

pressor response was nested within neighborhood poverty level (B=.35, $t_{16, 106}$ = 2.32, p = .022). Simple slope analyses revealed that less education was associated with a greater diastolic pressor response to PE when less than 5% of individuals in the neighborhood lived below the poverty line (B= -6.96, $t_{16, 106}$ = -2.08, p = .039). The effect of education on diastolic pressor response to PE was not significant in individuals who lived in neighborhoods where more than 5% of the residents lived below the poverty line. Figure 5 depicts the relationship between education and diastolic pressor response to PE when 3%, 15%, and 25% of individuals in the neighborhood live below the poverty level. None of the other neighborhood variables interacted with education to predict diastolic pressor response.

Occupation Nested Within Neighborhood SES

Association Between Level-1 Predictors and Systolic Pressor Response. The level-1 analysis examined whether ethnicity and occupation were related to diastolic pressor response to PE after controlling for baseline BP and BMI. The chi square tests assessing whether relationship between level-1 variables and systolic pressor response varied between level-2 groups were not significant. Therefore, all level-1 error terms were set as fixed effects. The intercept for the level-1 model was significant (B=30.49, $t_{16, 106}$ = 3.55, p = .003), indicating that there is significant variability among participants on diastolic pressor response to PE when all predictor variables are set at 0. However, none of the level-1 variables significantly predicted systolic pressor response to PE.

Multilevel Predictors and Systolic Pressor Response. There was not a significant main effect of the neighborhood variables on systolic pressor response. There was also no interaction between the level-1 and neighborhood variables on systolic pressor response.

Association Between Level-1 Predictors and Diastolic Pressor Response. The chi square tests assessing whether the relationship between level-1 variables and diastolic pressor response varied between level-2 groups was significant for occupation (χ^2=18.60, p = .01), demonstrating that the relationship between occupation and diastolic pressor response was not the same across level-2 groups. Therefore, occupation was set as a random effect. All other level-1 error terms were set as fixed effects because their relationship to diastolic pressor response did not vary accross level-2 groups. The intercept for the level-1 model was significant (B=20.56, $t_{16,\ 105}$ = 4.77, p < .001), indicating that there is significant variability among participants on diastolic pressor response to PE when all predictor variables are set at 0. Education and ethnicity were unrelated to diastolic pressor response to PE controlling for the covariates.

Multilevel Predictors and Diastolic Pressor Response. There was not a significant main effect of the neighborhood variables on diastolic pressor response. Further, neither education nor ethnicity significantly interacted with neighborhood variables to predict diastolic pressor response.

Aim 3

Analyses were conducted on a subset of the data to examine associations between SES, ethnicity, and perceived discrimination. These data were collected during the 2000 protocol. The results of these analyses were as follows.

SES, Ethnicity, and Perceived Discrimination. African-Americans reported more perceived discrimination than Caucasian-Americans, $t_{138} = 13.17$, $p<.01$. None of the neighborhood SES variables were associated with perceived discrimination scores in the African-American sample.

SES X Perceived Discrimination in African-American Sample

SES as a Moderator of Perceived Discrimination and Systolic Pressor Response. Hierarchical regression analyses assessed whether SES moderated the relationship between perceived discrimination and systolic pressor responses to PE in African-Americans. Separate regression equations were conducted testing whether each of the SES variables (education, occupation, percent below poverty, per capita income, percent below poverty) interacted with perceived discrimination to predict systolic pressor responses to PE. In these analyses, covariates were entered on Step 1, perceived discrimination and SES variables were entered on Step 2, and the interaction between SES and perceived discrimination was entered on Step 3. None of the covariates or predictor variables significantly predicted systolic pressor responses to PE. Further, none of the SES variables interacted with perceived discrimination to predict systolic pressor responses to PE.

SES as a Moderator of Perceived Discrimination and Diastolic Pressor Response. Hierarchical regression analyses assessed whether SES moderated the relationship between perceived discrimination and diastolic pressor responses to PE in African-Americans. Separate regression equations were conducted testing whether each of the SES variables (education, occupation, percent below poverty, per capita income, percent below poverty) interacted with perceived discrimination to predict diastolic pressor responses to PE. In these analyses, covariates were entered on Step 1, perceived discrimination and SES variables were entered on Step 2, and the interaction between SES and perceived discrimination was entered on Step 3. None of the covariates or predictor variables significantly predicted diastolic pressor responses to PE. Further, none of the SES variables interacted with perceived discrimination to predict diastolic pressor responses to PE.

DISCUSSION

Aim 1

The current study examined the relationships between SES, ethnicity, and vascular reactivity to PE. Aim 1 tested the hypothesis that SES mediates the relationship between ethnicity and pressor responses. There was evidence to support this hypothesis. Both education and occupation partially mediated the relationship between ethnicity and diastolic pressor responses to PE. Individuals with low education had greater diastolic pressor responses to PE. This suggests that individual SES factors may partially explain ethnic differences in diastolic blood pressure responsiveness. This is the first study that has examined the association between SES and pressor response to PE. Most of the work in this area has examined reactivity to a behavioral stressor. These studies have also found greater increases in BP among low SES individuals (Barnes et al., 2000; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Everson et al., 2001; Lynch, Everson, Kaplan, Salonen, & Salonen, 1998; Steptoe, Kunz-Ebrecht, Rumley, & Lowe, 2003; Steptoe et al., 2003; Suchday, Krantz, & Gottdiener, 2005; Wilson et al., 2000). Research suggests that increased BP reactivity is a risk factor for CVD (Armario et al., 2003; Treiber et al., 2003). Thus, the current results provide preliminary evidence that one mechanism through which SES is associated with CVD may be heightened pressor response to α adrenergic stimulation.

Interestingly, the individual SES variables did not mediate the relationship between ethnicity and systolic pressor responses to PE. PE is an α agonist that

stimulates pressor receptors, leading to vasoconstriction and in turn, increasing BP. The current results demonstrate that individual SES partially mediates ethnic differences in diastolic, but not systolic pressor responses to PE. It has been posited that different types of stressors may operate on the body via different physiological pathways, uniquely impacting systolic and diastolic pressure. For instance, tasks that require active coping efforts may produce an increase in heart rate and systolic BP, while tasks that require quiet attentiveness and vigilance may lead to decreased cardiac output, as well as increased total peripheral resistance and diastolic blood pressure (Krantz & Falconer, 1997). It is possible that individuals in low SES brackets utilize more passive coping strategies and increased vigilance to cope with the stress associated with stress of struggling to make ends meet with limited resources. Within the limits of this study, it is impossible to determine whether this is the case. Thus, future research should examine associations between SES, coping strategies, and pressor responses.

Contrary to what was expected, none of the neighborhood SES variables mediated the relationship between ethnicity and systolic or diastolic pressor responses. In fact, there was no relationship between neighborhood SES variables and pressor responses to PE. This suggests that individual SES factors (i.e., education and occupation) may play a greater role in BP reponsiveness than the socioeconomic characteristics of one's neighborhood. This does not support the results of research conducted by Kapuku and colleagues (Kapuku et al., 2002) in which living in low SES neighborhoods was associated with greater DBP reactivity among African-American males. This may be due to differences in sample

characteristics between the studies. The current study was conducted using data collected from African American and Caucasian American men and women. The relationship between neighborhood SES and pressor responses to PE were examined in the total sample and not in African American males alone. It is possible that the relationship between neighborhood SES and pressor responses to PE is different in African American males than in the total sample. However, this was not explored due to sample size limitations.

Aim 2

Aim 2 examined the interaction between individual SES factors and neighborhood SES on pressor responses to PE. The first hypothesis in this Aim was that neighborhood SES will have a stronger relationship to pressor responses to PE than individual SES. There was no support for this hypothesis. Multilevel analyses revealed that there was no main effect of neighborhood SES on systolic or diastolic pressor responses. Instead, the effect of education on pressor response to PE was dependent on neighborhood poverty level. Specifically, less education was associated with heightened pressor responsiveness to PE only for participants who resided in neighborhoods with less than 5% of individuals living below the poverty level. Interestingly, when poverty level in the neighborhood increased, education was no longer associated with pressor responsiveness. This was the case for both systolic and diastolic pressor responses to PE. These findings support the results of a review conducted by Krieger and Fee (Krieger & Fee, 1994) in which it was found that education has little effect on health in individuals with household incomes that

are below the poverty level. This suggests that although education may be associated with positive cardiovascular health, it cannot buffer the negative effects of neighborhood poverty on pressor responsiveness.

Initially, it was expected that educational attainment would buffer the negative effects of low neighborhood SES on pressor responses to PE. Based on research finding that African-American adolescents who lived in poorer neighborhoods had lower BP reactivity if their parents were more educated or had a higher annual income (Wilson et al., 2000), we expected that education would be associated with smaller pressor responses to PE in individuals who reside in low SES neighborhoods (Hypothesis 2). There was no support for this hypothesis. In fact, the results reveal that the opposite was true in our sample. Even after controlling for ethnicity, BMI, and baseline BP, education was only associated with pressor response to PE in individuals who reside in neighborhoods in which less than 5% of individuals live below the poverty line.

These findings are noteworthy for several reasons. The current findings suggest that the health benefits associated with educational attainment alone may not be potent enough to counteract the adverse effects of living in neighborhoods with more poverty. This seems to highlight a broader public health issue. It points to the possibility that along with focusing on the individual, efforts to eliminate disparities in cardiovascular disease should also target those aspects of the neighborhood that are associated with poor cardiovascular health.

Some characteristics of the neighborhood that may have adverse effects on cardiovascular health include higher crime rates (Sundquist et al., 2006), lack of

access to healthy foods (Echeverria, Diez-Roux, & Link, 2004; Krummel et al., 2001), limited recreation and environments that discourage physical activity (Echeverria et al., 2004), as well as lack of access to health care and health information (Blair, Lloyd-Williams, & Mair, 2002). It was beyond the scope of this dissertation study to determine whether each of these factors contributed a significant proportion of variability in cardiovascular functioning. However, future research should "un-package" neighborhood poverty to determine which of the above aspects should be targeted in public health interventions and public policies designed to eliminate health disparities.

Within the current study, ethnicity was not a significant predictor of pressor response to PE when the SES variables were taken into account. This suggests that ethnic differences in pressor response to PE may partially result from SES differences between African-Americans and Caucasian-Americans. Thus, regardless of ethnicity, as neighborhood poverty increases, the effect of education on pressor response decreases. These findings support the results of a study by Krieger and colleagues (Krieger et al., 2005) who found that the incidence of cardiovascular mortality was highest in census tracts with a higher percentage of individuals living below the poverty line and that adjusting for census tract level poverty substantially reduced the risk of CV mortality in African-Americans and Hispanics compared with Whites.

Since African-Americans tend to live in lower SES neighborhoods than their Caucasian-American counterparts with the same income and education level (Cubbin et al., 2001; Matthews et al., 2002), the current findings may have

significant implications for this group. They suggest that along with individual factors, neighborhood disadvantage may partially explain the higher rates of cardiovascular disease in this group.

In multilevel analyses in which neighborhood factors were taken into account, occupation did not explain a significant amount of variance in pressor responses to PE. Although occupation mediated ethnic differences in diastolic pressor responses to PE, it no longer related to pressor responses to PE when neighborhood SES was taken into account. It is possible that collinearity between occupation and neighborhood poverty inhibited the ability to detect relationships between occupation and pressor response to PE in the multilevel model.

Aim 3

The final aim of this study was to examine relationships between SES, perceived discrimination and pressor responses to PE in the African-American sample. These analyses were conducted on a subset of the data. There was support for the hypothesis that African-Americans would report more perceived discrimination than Caucasian-Americans (Hypothesis 1). However, contrary to what was expected, SES was unrelated to perceived discrimination scores in African-Americans (Hypothesis 2). Low power due to a modest sample size of African-Americans with perceived discrimination data may explain these findings.

It was also expected that SES would moderate the effect of perceived discrimination on pressor responses to PE in African-Americans. The results of the study revealed that this was not the case. There was no interaction between

perceived discrimination and any of the SES variables on systolic or diastolic pressor responses to PE. This may be due to limited power in the African-American sample. Analyses were conducted on only 37 African-Americans who had complete data on perceived discrimination, SES variables, and pressor responses variables. Thus, limited power may have compromised our ability to detect the relationship between these variables. Future studies should examine relationships between these variables using larger sample sizes.

Limitations and Conclusions

There are several methodological issues that should be taken into account when interpreting the results of the current analyses. First, this study was conducted using a modest sample size. Because of the power needed to reliably detect interactions between level 1 and level 2 variables in multilevel analyses, it is recommended that larger sample sizes are used. In the current study, multilievel analyses were conducted on only 137 individuals who had complete data on all variables entered into the analyses. Thus, reduced power may have limited our ability to detect relationships among variables. Future studies should be conducted using larger sample sizes.

Another limitation of the current study was that data were combined from two different protocols to conduct this study. Descriptive statistics conducted prior to performing analyses revealed SES differences between the samples; the 1995 sample had lower SES than the 2000 sample. Combining the samples may have introduced additional error variance into the study, resulting in less power to detect

effects. However, in spite of limited power, analyses revealed a significant interaction between neighborhood poverty and education on pressor responses to PE. The fact that this effect emerged with the above power issues suggests that it is a salient effect and worthy of further examination in future studies.

Although interesting relationships emerged between SES and heightened pressor response to PE, it is difficult to determine what aspects of SES are associated with heightened pressor responses. The 'John Henryism' hypothesis states that the stress of struggling to cope with psychosocial demands with limited resources and opportunities can lead to elevated BP (Dressler, Bendon, & Negger, 1998; James, Keenan, Strogatz, Browning, & Garrett, 1992). This may explain why education partially mediated ethnic differences in diastolic pressor responses. Merritt and colleagues (Merritt et al., 2004) also found that African-Americans who had low educational attainment and adapted an active coping style (John Henryism) had greater cardiovascular reactivity than those who adapted a passive coping style.

Lifestyle factors associated with achieving SES in our society may also explain our findings. Skills and social benefits associated with increasing educational levels, such as greater access to health information, a greater sense of control, and socialization to adopt health-promoting behaviors (Yen & Moss, 1999) may have positive cardiovascular effects. Thus, it is possible that African-Americans in higher educational brackets may adapt a healthier lifestyle, through increased physical activity and engaging in healthier eating habits. This may serve as a protective factor against the negative effects of stress on cardiovascular

functioning. Future studies should examine whether lifestyle factors mediate relationships between SES and cardiovascular functioning in African-Americans.

Summary

The current dissertation study revealed interesting relationships between SES, ethnicity, and pressor response to PE. Although this study was exploratory and should be replicated on larger samples, the current findings suggest that SES may play an important role in the heightened pressor responsiveness in African-Americans. Specifically, education and occupation partially mediated ethnic differences in diastolic pressor responsiveness to PE. When these SES variables were taken into account, the association between ethnicity and diastolic pressor responses to PE was weakened. There was also evidence that the association between education and pressor responses to PE is nested within neighborhood poverty level. In both African-Americans and Caucasian-Americans, education was only associated with pressor responsiveness to PE in individuals who lived in higher SES communities. As neighborhood poverty level increased, the association between education and pressor responses was no longer significant.

This is the first study that has examined the association between neighborhood SES and vascular reactivity in a multilevel model, and the current results suggest that the relationship between SES and pressor responsiveness is complex with individual and neighborhood SES factors interacting to predict pressor responses to PE. Since neighborhood poverty seems to play an important role in cardiovascular health (Krieger et al., 2005), research should continue

examining its association with pressor responsiveness as well as other cardiovascular indicators.

The current results highlight the importance of community interventions designed to promote healthy lifestyles among individuals who live in low SES neighborhoods. These results reveal that individuals with low SES have greater pressor responsiveness to PE, a risk factor for hypertension. Further, this study suggests that education alone may not be a protective factor against CVD for those who live in low SES neighborhoods. Interventions targeting low SES communities may reduce the risk of CVD in these individuals. More research is needed to determine which factors associated with low SES would be most relevant to incorporate into such an intervention. However, this type of research would be extremely important in reducing risk of CVD in low SES individuals and may be particularly beneficial for African Americans, a group that is already especially vulnerable to CVD.

Table 1: Ethnic Differences in Sample Characteristics

Variable	**African-Americans (N=105)**	**Caucasian-Americans (N=106)**
Education[2]		
BA and Above	24 (23.1%)	46 (44.7%)
Partial College	36 (34.6%)	29 (28.2%)
High School Graduate	34 (32.7%)	27 (26.2%)
Partial High School	10 (9.6%)	1 (1.0%)
% of Individuals in Block Identified as Black[2]	16.35% (SEM=1.6%)	6.0% (SEM=.84%)
% of Individuals in Block Living Below Poverty Level[1]	19.9% (SEM=1.3%)	15.8% (SEM=1.1%)
Hypertensive[1]	32 (30.5%)	18 (17.1%)
	M±SEM	**M±SEM**
Age	38.3 ± 0.7	36.8 ± 0.7
BMI[2]	28.6 ± 0.6	25.1 ± 0.4
Baseline SBP (mmHg)[1]	130.4 ± 1.6	124.3 ± 1.5
Baseline DBP	77.6 ± 1.1	75.0 ± 1.0
SBP Pressor Change (mmHg)[2]	32.2 ± 1.6	26.6 ± 1.3
DBP Pressor Change (mmHg)[2]	20.3 ± 0.9	16.3 ± 0.8
Per Capita Income in Block[2]	$16,520 ± $855	$23,718.58 ± $863

[1] *p<.05*
[2] *p<.01*

Table 2: Differences Between the Protocols in Sample Characteristics

Variable	**1995 Protocol (N=62)**	**2000 Protocol (N=149)**
Ethnicity		
African-American	39 (62.9%)	66 (44.3%)
Caucasian-American	23 (37.1%)	83 (55.7%)
Education[2]		
BA and Above	6 (10%)	64 (43.5%)
Partial College	23 (38.3%)	42 (28.6%)
High School Graduate	25 (41.7%)	36 (24.5%)
Partial High School	6 (10%)	5 (3.4%)
% of Individuals in Block Identified as Black[2]	16.4% (SEM=2.2%)	8.7% (SEM=.87%)
% of Individuals in Block Living Below Poverty Level[1]	22.2% (SEM=1.9%)	16.0% (SEM=.91%)
Hypertensive[1]	22 (35.5%)	28 (18.9%)
	M±SEM	**M±SEM**
Age	39.2 ± 0.7	36.9 ± 0.6
BMI[2]	26.6 ± 0.5	26.9 ± 0.5
Baseline SBP (mmHg)[1]	130.0 ± 2.4	126.4 ± 1.2
Baseline DBP	78.6 ± 1.6	75.4 ± .8
SBP Pressor Change (mmHg)[2]	33.1 ± 2.5	28.1 ± 0.9
DBP Pressor Change (mmHg)[2]	21.7 ± 1.5	16.9 ± 0.6
Per Capita Income in Block[2]	$17,398 ± $1019	$23,718 ± $790

[1] *p<.05*
[2] *p<.01*

Table 3: Effects of Education and Neighborhood SES on Systolic Pressor Response*

Fixed Effects	Coefficient	Std Error	T-ratio	df	p-value
		Intercept 1, B0			
Intercept 2, G00	34.60	8.71	3.9	16	0.001
% Black, G01	-0.54	0.42	-1.3	16	0.21
Per Capita Income, G02	-0.00053	0.00049	-1.06	16	0.31
% Below Poverty, G03	-0.12	0.44	-0.28	16	0.79
		Baseline SBP, B1			
Intercept 2, G10	-0.44	0.42	-1.03	106	0.31
% Black, G11	0.021	0.020	1.02	106	0.31
Per Capita Income, G12	0.00003	0.00002	1.12	106	0.27
% Below Poverty, G13	0.020	0.018	1.13	106	0.26
		Ethnicity, B2			
Intercept 2, G20	1.50	11.32	0.13	106	0.89
% Black, G21	0.45	0.57	0.79	106	0.43
Per Capita Income, G22	0.00048	0.00076	0.56	106	0.57
% Below Poverty, G23	-.066	0.53	-0.13	106	0.90
		BMI, B3			
Intercept 2, G30	-.234	1.12	-2.09	106	0.04
% Black, G31	0.001	0.06	0.016	106	0.43
Per Capita Income, G32	0.00007	0.00008	0.91	106	0.57
% Below Poverty, G33	0.097	0.054	1.81	106	0.90
		Educational Attainment, B4			
Intercept 2, G40	-13.34	5.61	-2.38	106	0.019
% Black, G41	0.32	0.33	0.96	106	0.34
Per Capita Income, G42	0.00063	0.003	1.82	106	0.07
% Below Poverty, G43	0.554	0.249	2.22	106	0.02

**Table 3 demonstrates the interaction between level-1 predictors (B1, B2, B3, B4) and level-2 predictors (G01, G02, G03) on systolic pressor responses to PE. As shown in the final block of the table, educational attainment (B4) interacts with % below poverty to predict systolic pressor responses to PE (G43). Overall, education is associated with less of an increase in SBP in response to PE (G40). However, as % below poverty increases, the beneficial effect of education on systolic pressor responses diminishes.*

Table 4: Effects of Education and Neighborhood SES on Diastolic Pressor Response

Fixed Effects	Coefficient	Std Error	T-ratio	df	p-value
		Intercept 1, B0			
Intercept 2, G00	23.43	5.28	4.4	16	0.001
% Black, G01	-0.42	0.24	-1.7	16	0.11
Per Capita Income, G02	-0.00047	0.00029	-1.6	16	0.12
% Below Poverty, G03	-0.16	0.27	-0.61	16	0.55
		Baseline SBP, B1			
Intercept 2, G10	0.12	0.35	.34	16	0.73
% Black, G11	0.028	0.021	1.34	16	0.20
Per Capita Income, G12	0.000004	0.00002	1.17	16	0.87
% Below Poverty, G13	0.016	0.015	-1.06	16	0.30
		Ethnicity, B2			
Intercept 2, G20	0.91	7.02	0.13	16	0.89
% Black, G21	0.17	0.37	0.47	16	0.65
Per Capita Income, G22	0.00023	0.00047	0.48	16	0.64
% Below Poverty, G23	0.085	0.32	0.26	16	0.79
		BMI, B3			
Intercept 2, G30	-2.07	0.67	-3.08	106	0.003
% Black, G31	0.001	0.06	0.016	106	0.95
Per Capita Income, G32	0.00008	0.00005	1.66	106	0.09
% Below Poverty, G33	0.092	0.032	2.89	106	0.005
		Educational Attainment, B4			
Intercept 2, G40	-7.66	3.47	-2.21	106	0.02
% Black, G41	0.04	0.18	0.24	106	0.81
Per Capita Income, G42	0.0003	0.0002	1.80	106	0.07
% Below Poverty, G43	0.35	0.15	2.32	106	0.02

**Table2 demonstrates the interaction between level-1 predictors (B1, B2,B3, B4) and level-2 predictors (G01, G02, G03) on diastolic pressor responses to PE. As shown in the final block of the table, educational attainment (B4) interacts with % below poverty to predict diastolic pressor responses to PE (G43). Overall, education is associated with less of an increase in DBP in response to PE (G40). However, as % below poverty increases, the beneficial effect of education on diastolic pressor responses diminishes.*

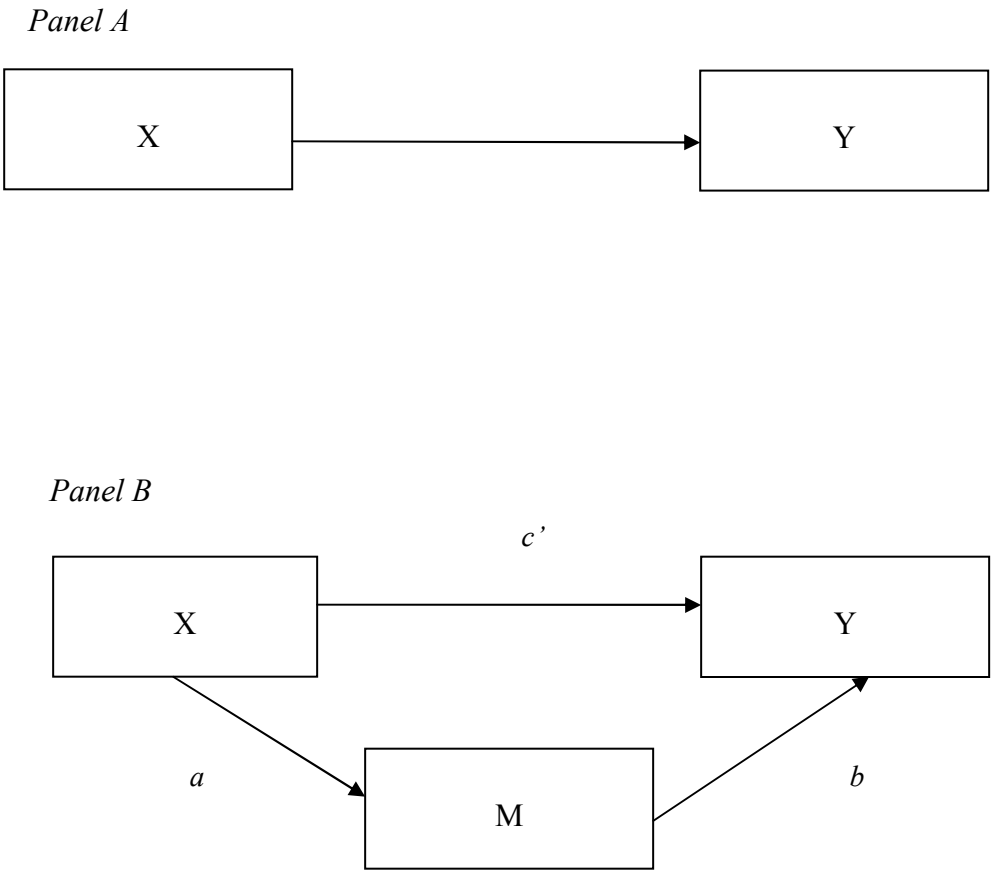
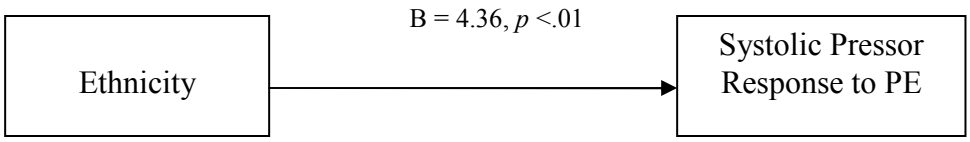


Figure 1: Illustration of a mediation analysis using the method recommended by Preacher and Hayes (2004). Panel A illustrates the direct effect of X on Y. Panel B illustrates the mediation design. X indirectly affects Y through M.

Panel A



Panel B

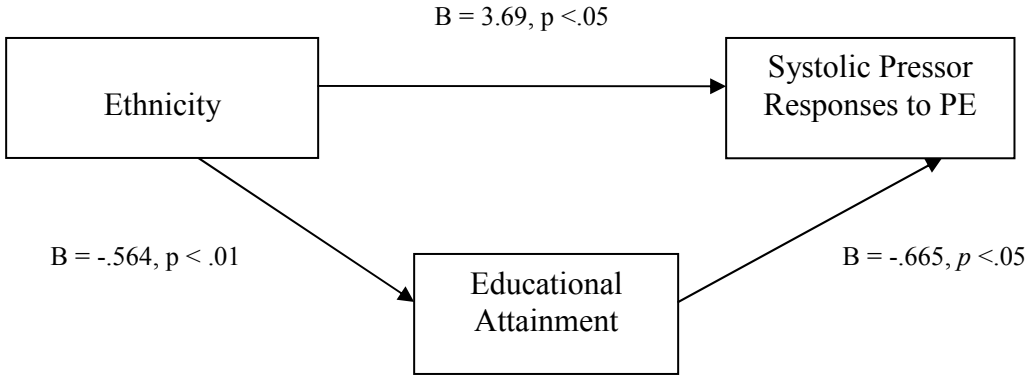
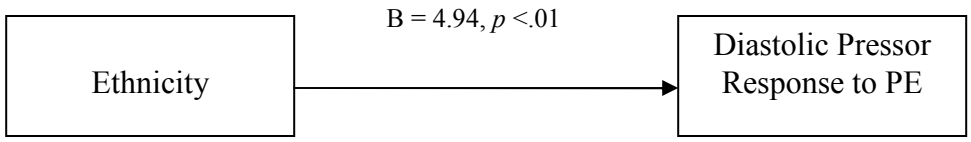


Figure 2: Path model relating ethnicity and educational attainment to systolic pressor responses to PE. Panel A illustrates the direct relationship between ethnicity and systolic pressor responses to PE. Panel B depicts the indirect relationship between ethnicity and systolic pressor responses to PE through educational attaiment. After ontrolling for covariates (baseline BP and BMI), education mediated the relationship between ethnicity and systolic pressor responses to PE (p<.05).

Panel A



Panel B

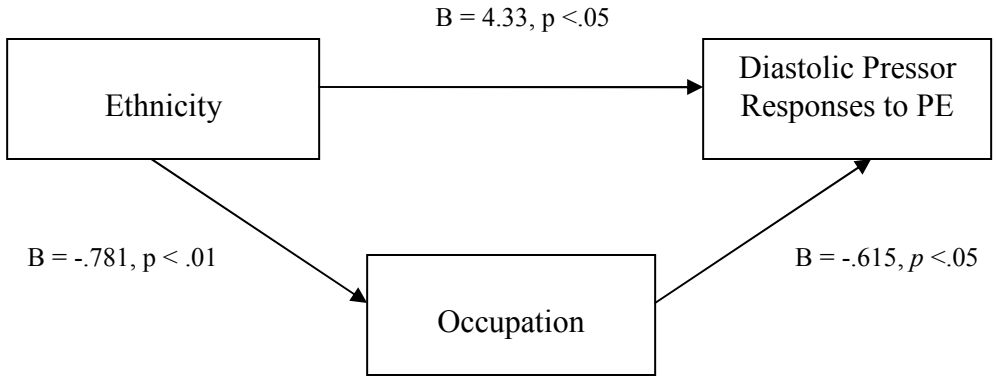


Figure 3: Path model relating ethnicity and occupation to diastolic pressor responses to PE. Panel A illustrates the direct relationship between ethnicity and diastolic pressor responses to PE. Panel B depicts the indirect relationship between ethnicity and diastolic pressor responses to PE through occupation. After controlling for covariates (baseline BP and BMI),occupation mediated the relationship between ethnicity and diastolic pressor responses to PE (p<.05).

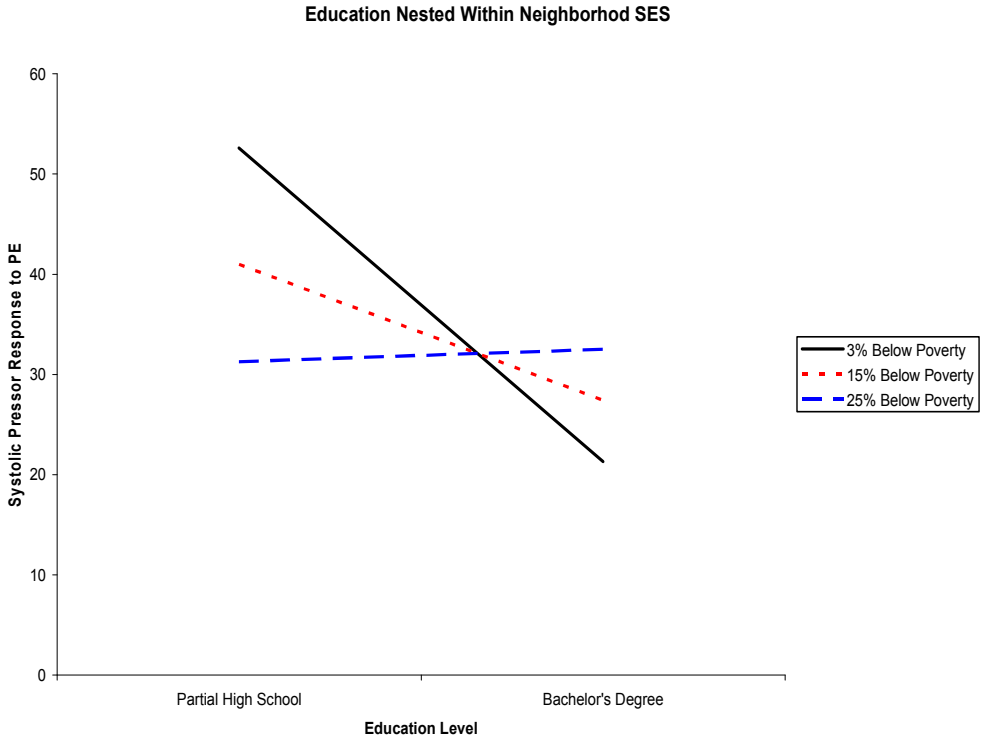


Figure 4: Interaction between education and neighborhood poverty level on systolic pressor responses to PE. Higher education was associated with lower systolic pressor responses to PE in neighborhoods with less than 5% of individuals living below the poverty level ($p<.05$). As neighborhood poverty increased, the protective effect of education on systolic pressor responses was no longer apparent.

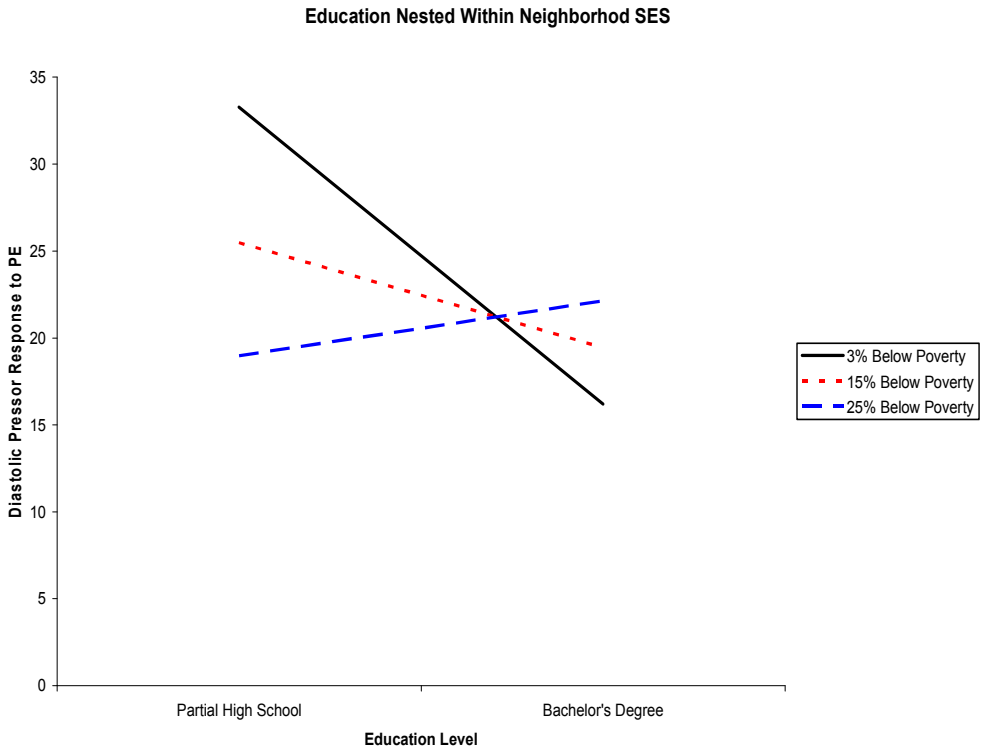


Figure 5: Interaction between education and neighborhood poverty level on diastolic pressor responses to PE. Higher education was associated with lower diastolic pressor responses to PE in neighborhoods with less than 5% of individuals living below the poverty level ($p<.05$). As neighborhood poverty increased, the protective effect of education on diastolic pressor responses was no longer apparent.

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