The geographic context and black-white disparities in hypertension

by

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Abstract

This dissertation evaluated the contribution of the geographic context to black-white disparities in hypertension. Few studies of area-level factors and hypertension disparities have focused on geographic variation both within and between race groups. Uncovering the mechanisms underlying within-group variation may help elucidate the particular environmental factors that contribute to hypertension disparities and highlight potential targets for interventions. Understanding how the distribution of high blood pressure compares for Blacks versus Whites across different environments helps shed light on the mutability of the disparity and potential ways in which it can be reduced.

The studies in this dissertation investigated (1) regional geographic factors related to hypertension differences among and between Blacks and Whites; (2) the association between metropolitan-level racial residential segregation and hypertension and neighborhood poverty as a mediating pathway; and (3) the link between neighborhood-level racial residential segregation and hypertension and interactions with area- and individual-level factors.

The key finding was that race differences are not invariant. Hypertension prevalence varied significantly within race groups and race differences in hypertension were modified by context. Blacks and Whites born in the South and those living in metropolitan areas located in the South were more likely to be hypertensive than those born or living in other parts of the country. Blacks living in more segregated metropolitan areas had significantly higher odds of hypertension than those in less segregated areas, and the impact of segregation varied by metropolitan area and neighborhood poverty. Race differences in hypertension prevalence ranged from 82% higher for Blacks versus Whites to a low of 13% higher depending on which geographic

groups were compared. Race differences also varied significantly by metropolitan-level segregation and neighborhood poverty; in high segregation, low poverty areas Blacks had over 4 times higher odds of hypertension versus Whites whereas Blacks in low segregation, high poverty areas had just 1.2 times higher odds.

These findings suggest that race differences in hypertension result not from innate differences but from contextual factors. Specifically, eliminating the processes that lead to residential segregation and the resultant inequitable distribution of neighborhood resources could reduce the unequal burden of hypertension in Blacks versus Whites.

Chapter 1:
Introduction

The studies in this dissertation highlight the importance of looking beyond more traditional explanations for disparities like genetic susceptibility or health behaviors to more complex interactions between social, environmental, and biologic factors. Identifying and understanding factors leading to heterogeneity in hypertension within and between race groups allows for examination of the role of the social and physical environment in hypertension disparities within the US. Recognizing variation in the distribution of high blood pressure among Blacks and Whites suggests that increased or decreased susceptibility to hypertension is not inherent to one group versus the other. Uncovering the mechanisms underlying this variation points to the particular social and environmental factors that contribute to hypertension disparities and highlights the aspects that can be targeted for interventions. Along the same lines, understanding how the distribution of high blood pressure compares for Blacks versus Whites across different environments helps shed light on the mutability of the disparity and potential ways in which it can be reduced.

Specific Aims and Hypotheses

The goal of this dissertation is to evaluate geographic variation in hypertension in the US and the impact of racial residential segregation on these differences both among and between Blacks and Whites. To better address the current limitations of the health disparities literature on hypertension this dissertation focuses on three topics: (1) regional and larger area geographic factors related to hypertension differences among and between Blacks and Whites; (2) the association between metropolitan-level racial residential segregation and hypertension and assessment of neighborhood poverty as a mediating pathway; and (3) the link between neighborhood-level racial residential

segregation and hypertension and potential interactions with area- and individual-level factors. More specifically, this dissertation aims to do the following:

Aim 1: To examine regional and larger area geographic factors related to differences in hypertension among and between Blacks and Whites.

Hypothesis 1a: Hypertension prevalence will be higher among Blacks and Whites who were born in the South compared with those born outside the South.

Hypothesis 1b: Hypertension prevalence will be higher among Blacks and Whites residing in metropolitan areas located in the South.

Hypothesis 1c: This geographic variation will be explained by aspects of the neighborhood social and physical environment.

Aim 2: To evaluate metropolitan-level racial residential segregation as a moderator of Black-white differences in hypertension prevalence and the role of racial differences in neighborhood poverty in explaining this moderation.

Hypothesis 2a. Black-white differences in hypertension will be greater in metropolitan areas with more segregation than in areas with less segregation.

Hypothesis 2b. The larger black-white differences in hypertension in more segregated areas will be due to greater Black-white differences in neighborhood poverty in more segregated areas.

Aim 3: To investigate the association between neighborhood-level residential segregation and hypertension prevalence and incidence among Blacks and Whites and the role of neighborhood- and individual-level factors in mediating or moderating this relationship.

Hypothesis 3a: Blacks living in more segregated neighborhoods will have higher hypertension prevalence and incidence than in less segregated neighborhoods.

Hypothesis 3b: The associations between segregation and hypertension will vary by metropolitan area of residence. Segregation will also be more protective at lower levels

of neighborhood poverty and higher levels of neighborhood social cohesion, individual income, and individual education.

Hypertension epidemiology and racial disparities in hypertension

Hypertension is widespread in the US. In 1999-2000, the age-adjusted prevalence of hypertension among participants of the National Health and Nutrition Examination Survey (NHANES) was 28.7% (1). Because hypertension is also a major independent risk factor for coronary heart disease, stroke, congestive heart failure, and kidney disease (2), it is important to better understand its determinants.

The main functions of blood pressure are to facilitate tissue perfusion and to maintain sodium balance (3). Blood pressure readings are measured in millimeters of mercury (mm Hg) and given as two numbers, systolic blood pressure and diastolic blood pressure. Systolic blood pressure (SBP) is a measure of cardiac output, the force with which the heart pushes the blood out into the arteries, and diastolic blood pressure (DBP) is a measure of peripheral resistance, the force with which the arterioles resist this blood flow (4). Normal blood pressure (SBP 90-120 mm Hg; DBP 60-80 mm Hg) is maintained by a balance between cardiac output and peripheral vascular resistance.

Blood pressure is determined by the complex interaction of genetic, environmental and demographic factors that influence cardiac output and peripheral resistance. High blood pressure or hypertension (SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg) occurs when the relationship between output and resistance is altered. Cardiac output mainly depends on blood volume, which is largely influenced by whole body sodium homeostasis, making sodium intake and metabolism important contributing factors (5). Peripheral resistance is primarily determined by a balance between neural and hormonal vasoconstrictors and vasodilators as well as vascular smooth muscle growth and structure (5). Several physiologic mechanisms work to keep blood pressure levels normal including smooth muscle cells of the arterioles; cardiac output from the heart; the renin-angiotensin system's production of the vasodilator bradykinin; vascular endothelial cell production of the vasodilator nitric oxide and the vasoconstrictor endothelin; and secretion of the hormone atrial natriuretic peptide from the atria of the

heart in response to increased blood volume (4). While the exact pathophysiology remains unclear, physiologic dysregulation of any subset of these mechanisms is believed to lead to hypertension.

Traditional risk factors for hypertension

Traditional risk factors for hypertension include older age, low socioeconomic position (SEP), obesity, heavy alcohol use, physical inactivity, high salt intake, and a diet high in red meat and sugar, and low in fruits, vegetables, and whole grains (6). In 1999-2000, 30.1% of all NHANES participants aged 40-59 years were hypertensive, and this percentage increased to 65.4 among adults age 60 and older (1). Hypertension prevalence has also been shown to increase with increasing body mass index (BMI) in men and women aged 20 to 59 years (7). Several studies have shown that people with lower levels of income or educational attainment are more likely to be hypertensive (8, 9). A randomized controlled trial of alcohol consumption and blood pressure showed there was a direct association between alcohol consumption and systolic blood pressure, an association that persisted after adjusting for the weight lost by reducing alcohol intake (10). In a meta-analysis of randomized controlled trials investigating the effects of increased physical activity, researchers found that the randomly assigned aerobic exercise group consistently reduced their systolic blood pressure levels compared with the control group, independent of the intensity of the exercise program (11). Meta-analyses of the efficacy of reducing sodium intake have consistently found it to be associated with a small but significant lowering of systolic blood pressure (12). The Dietary Approaches to Stop Hypertension (DASH) randomized controlled trial showed that a reduction in sodium levels, an increase in fruit and vegetable, whole grain, fish, poultry and nut consumption, and a decrease in red meat and sugary food consumption, effectively reduced blood pressure levels in a sample of male and female non-hypertensives and hypertensives (13). Consumption of specific nutrients including potassium and calcium has been shown to protect against high blood pressure (14, 15, 16, 17).

Epidemiology of racial disparities in hypertension

The burden of hypertension is not shared equally among residents of the US. Blacks are significantly more likely to be hypertensive than the rest of the population. Among 1999-2002 NHANES participants, the age-adjusted hypertension prevalence was 38.6% among non-Hispanic black men compared with 26.6% among non-Hispanic white men (1). Among women, 44.0% of Blacks were hypertensive compared with 29.6% of Whites. Age-specific analyses showed that hypertension prevalence was higher among Blacks than Whites in all age groups.

Several hypotheses have been proposed to explain the Black-white disparity in hypertension. One is that Blacks are intrinsically predisposed to hypertension. Some research has shown that salt sensitivity levels are higher among hypertensive Blacks than hypertensive Whites (18, 19). However, salt sensitivity is similar among normotensive Blacks and Whites (20). There are also other demographic factors that may confound the finding of differential salt sensitivity among hypertensives; in addition to Blacks, salt sensitivity has been shown to be higher among those with higher body weight and among women (20).

A recent study found evidence of substantial variability in hypertension prevalence within persons of African and European descent living in different countries (21, 22). Among those of African descent, hypertension prevalence ranged from 13.5% among those living in Nigeria to 44% for those living in the US. Individuals of European descent living in the US had the lowest hypertension prevalence of the countries investigated (26.8%) while Germans had higher prevalence than US Blacks (55.3%). This suggests that social, environmental, and lifestyle factors may play an important role.

Another hypothesis proposed to explain the black-white hypertension disparity is that the prevalence of key hypertension risk factors is higher in Blacks than Whites. Differences in dietary intake, including sodium, potassium, and fat consumption, have been examined as a possible explanation (18, 19, 23). Although no black-white difference is generally found in salt consumption, Blacks report lower levels of potassium consumption than Whites (23, 24). Black women in particular are also more

likely to consume a diet higher in fat and lower in protein than their white counterparts (23). BMI and physical inactivity are also higher among Blacks than Whites, particularly among women (18, 23).

Differential exposure to psychosocial stressors and certain coping strategies have been linked with racial disparities in hypertension including low SEP, discrimination, and John Henryism (high-effort coping in response to prolonged exposure to stress) (25). There is a consistent inverse relationship between socioeconomic attainment and hypertension among both Blacks and Whites in the literature (9), but given that Blacks are more likely than Whites to be lower SEP, it follows that they may be more likely to be hypertensive. However, several studies have shown that health disparities tend to persist after adjusting for traditional measures of SEP like education and income (26, 27). Findings for studies of discrimination and John Henryism suggest both may be associated with increased hypertension, though results have been mixed (28, 29, 30, 31, 32, 33, 34).

While no single factor is likely to explain racial disparities in hypertension, these explanations are incomplete because they pay insufficient attention to the role of area-level factors. Community resources and local governments determine the quality of neighborhood schools and consequently, opportunities for socioeconomic advancement (35, 36). In addition, the outsourcing of high paying entry-level jobs from city centers where Blacks are often concentrated to the suburbs limits employment opportunities for residents of urban areas (35, 36). The residential environment can also impact the accessibility of behavioral factors associated with hypertension. For example, low neighborhood safety may reduce physical activity levels and a higher density of fast food restaurants may increase obesity (37, 38). Thus, geographic factors may help tie together the cluster of risk factors associated with racial hypertension disparities. A growing number of studies have suggested that the social and physical environment may contribute to racial disparities in hypertension (39, 40, 41), but the mechanisms are not yet fully understood.

Racial disparities in hypertension: the role of geographic context

Large geographic patterning of hypertension and relevance to health disparities

Few studies have been done within the US which point to larger area-level variation (e.g. metropolitan area or region) in hypertension (42, 43, 44, 45), but there is evidence to suggest geographic patterning. Two studies have shown that Blacks and Whites living in the South (as defined by the US Census (46)) have higher hypertension prevalence than those living in the rest of the country (42, 44). In addition, a longitudinal study of young adults found that black men living in Chicago and Minneapolis were significantly less likely to develop elevated blood pressure levels over a 7-year period than black men living in Birmingham, after adjusting for education and established risk factors (43). A similar trend was observed among black women. A study of hypertension awareness, treatment and control found no regional difference in awareness, and trends for better treatment and control among Blacks and Whites living in Stroke Belt compared with other parts of the US (45). The determinants of these geographic differences are not well understood.

Although no studies have investigated associations of region of birth with hypertension prevalence, region of birth has been linked to cardiovascular disease mortality, particularly in African Americans. Two national studies found that, regardless of where the person was living when they died, Blacks born in the South had higher cardiovascular disease mortality rates than those born in other parts of the country (47, 48). A study of black Ohio residents conducted in the 1960s and more recent studies of New York residents found similar results (49, 50, 51). These observed differences have been hypothesized to be due in part to the retention of lifestyle factors like diet and cigarette smoking, as well as the lingering effects of childhood poverty, but the determinants of these differences are not well understood (49, 50, 51).

Neighborhood factors

Blacks are generally more likely to live in poor quality residential environments, which have been linked to increased cardiovascular risk including hypertension (9, 39, 40, 52, 53, 54, 55). Mujahid et al found that better neighborhood social cohesion,

neighborhood safety, walkability, and healthy food availability were associated with decreased hypertension prevalence (40). A study of neighborhood median housing value and incident hypertension in black women found that lower median housing value was associated with increased risk after adjusting for individual-level characteristics (56). An examination of participants of the Chicago Community Adult Health Study found that Blacks were significantly more likely than Whites to be hypertensive after adjustment for individual education and income, but additional adjustment for census-derived neighborhood factors reduced the magnitude of the association between race and hypertension and the resulting difference was no longer statistically significant (39).

Few studies have examined segregation together with neighborhood factors, but there is some evidence suggesting that racial disparities in hypertension depend on the socio-environmental context. Thorpe, Jr. and colleagues conducted a comparative analysis of racial disparities in hypertension in NHANES participants and Exploring Health Disparities in Integrated Communities (EHDIC) Study participants. EHDIC is a study of an integrated community in southwest Baltimore with nearly equal proportions of black and white residents and virtually no race difference in median income. Researchers found that, in both studies, Blacks had a significantly higher odds of hypertension than Whites after adjusting for age, gender, marital status, household income, education level, insurance status, self-rated health, weight status (e.g. obese, overweight, etc), physical inactivity, diagnosis of diabetes, drinking and current smoking status (41). However, the race differences in hypertension prevalence were much larger among NHANES participants (OR: 2.01; 95% CI: 1.63, 2.48) than EHDIC participants (OR: 1.42; 95% CI: 1.09, 1.86). The authors concluded that this reduction was due to increased similarities in social and environmental exposures among Blacks and Whites in the integrated community compared with the general US population.

Racial segregation

One aspect of the geographic context that has received increasing attention in the health sciences literature is racial residential segregation. The segregation of minority race/ethnic groups from the majority has the potential to marginalize

individuals by isolating them from the benefits afforded the rest of society. Those living in segregated areas are more likely to be exposed to violent crime and less likely to have access to grocery stores and healthy food options (57, 58, 59). They may have lower social and political capital, and they may be at greater risk of adverse health outcomes (36, 60, 61). Racial residential segregation is believed to contribute to health disparities by concentrating Blacks into high poverty areas (60, 62). A comparison of neighborhood poverty for poor Blacks and Whites shows that poor Whites are less likely to actually live in high poverty neighborhoods than poor Blacks (63). Neighborhood poverty is in turn associated with adverse health due to factors like reduced economic opportunity and limited exposure to health promoting resources.

Segregation is typically measured in health studies either at the metropolitan level or at the local, or neighborhood, level (61). Metropolitan-level segregation compares the level of segregation in an entire urban area to that of other urban areas and posits that those living in segregated cities will have a different health outcome than those living in more integrated cities. No published studies were found that assessed racial segregation as an explanation for the US black-white disparity in hypertension, but researchers have examined segregation and black-white differences in other outcomes including body mass index, self-rated health, infant mortality, and all-cause mortality (64, 65, 66, 67, 68). These studies have generally found that increased segregation is associated with adverse health outcomes. However, the majority of these studies are ecologic in design which limits their ability to investigate the underlying individual- and neighborhood-level pathways through which segregation may impact health.

Studies of neighborhood segregation are considered extensions of neighborhood effects research and suggest that aspects of the neighborhood context in a segregated neighborhood is different from that of an integrated neighborhood in a way that impacts health (61). Very few studies have assessed the association between neighborhood segregation and hypertension (69), but several other health outcomes have been examined including all-cause mortality, cardiovascular disease mortality, self-

rated health, and homicide. Among Whites, living in a predominantly white neighborhood is typically protective of or unassociated with adverse health outcomes (70, 71). Among Blacks, however, findings are mixed (57, 70, 71, 72). Some studies find that segregation is associated with poor health and increased mortality while others find it to have a protective effect. Those that link segregation to adverse health outcomes attribute it to the increased neighborhood disadvantage and concentrated poverty often found in predominantly black neighborhoods (36, 57). Researchers showing a health protective association point to the ethnic density hypothesis which states that living with a high concentration of one's own race/ethnic group is health promoting because of strong social networks and protection from discrimination (73).

Processes through which the geographic context may affect blood pressure

There are two pathways through which area-level factors may lead to hypertension. One is by creating a chronically stressful living environment. Racial segregation results in the differential distribution of social and economic resources including commercial investment, employment opportunities, wealth, educational opportunities, and political influence (36, 61, 62). Minority groups living in segregated areas may be more likely to live in worse neighborhood conditions and have lower SEP, both factors shown to expose them to more stressors such as low neighborhood safety, financial strain, and low job control (36, 61, 62).

The physiologic response to stress involves a variety of changes to allow for increased physical and mental alertness (74, 75). Upon exposure to a stressor, the sympathetic nervous system elicits a "fight-or-flight" response that includes an increase in heart rate and blood pressure to enhance muscle oxygenation. The hypothalamic-pituitary-adrenal (HPA) axis is a slower response system also activated to handle stress. It stimulates the release of a cascade of hormones designed to help the body cope with stress. The body is well-equipped to handle acute stressors; upon the elimination of the threat the parasympathetic system mediates the return of heart rate and blood pressure to normal and a series of negative feedback loops are activated to shut down the HPA axis response. However, chronic exposure to stress and activation of the stress

response pathways can lead to physiologic dysregulation and several chronic diseases, including hypertension (74, 75, 76).

Interleukin-6 (IL-6) is a pro-inflammatory cytokine believed to play a key role in the stress-induced development of hypertension (75). Stress hormones including cortisol, catecholamines, and angiotensin stimulate the release of IL-6 from the liver and abdominal fat tissue. Cortisol later suppresses IL-6 while the hormones released as part of the sympathetic nervous system continue to increase secretion. The increased release of IL-6 and other pro-inflammatory cytokines due to the dysregulation of stress hormones induces a state of chronic low grade inflammation, which may cause hypertension through a number of mechanisms including endothelial dysfunction and oxidative stress (75).

Stress can also lead to hypertension through the behaviors people adopt to cope. A high fat and carbohydrate diet, heavy alcohol use, cigarette smoking, and physical activity have all been shown to reduce feelings of stress (77). Animal studies suggest that "comfort food eating" or consuming foods high in saturated fat and carbohydrates reduce feelings of anxiety associated with stress by shutting down the signaling cascade elicited by the HPA axis (78, 79). Alcohol use, nicotine consumption, and physical activity all activate the HPA axis but they also stimulate the release of beta-endorphins, endogenous opioids that numb pain and promote feelings of well-being and relaxation (80, 81, 82, 83, 84). Unfortunately, with the exception of physical activity, these behaviors, while effective at reducing feelings of stress, are risk factors for hypertension and more severe cardiovascular diseases.

Another pathway through which area-level factors can lead to hypertension is by limiting access to health promoting resources and increasing access or exposure to health harming resources. The nature of the social environment may help to account for greater opportunities for engaging in negative coping behaviors and fewer opportunities for relying on positive coping alternatives. There is generally limited access to exercise and public recreation resources as well as healthy foods in lower income neighborhoods, both of which have been linked to hypertension risk factors,

specifically reduced physical activity levels and increased obesity (40, 85, 86, 87). Individuals living in low income neighborhoods are also more likely to live in unsafe neighborhoods which could reduce the likelihood that residents will engage in physical activity (38, 40, 85, 88). At the same time, those living in higher poverty, predominantly minority neighborhoods are more likely to live in close proximity to liquor stores and fast food restaurants (37, 58, 89, 90). They are also more likely to be exposed to cigarette- and obesity-related advertising (91, 92, 93), putting them at greater risk of developing hypertension and more serious cardiovascular diseases if these exposures increase their consumption patterns.

Conceptual model: Investigating the multilevel role of place

This dissertation is built on the overall framework that the interaction of social and biologic factors can influence hypertension prevalence and incidence at multiple levels (Figure 1.1). Historic, regional differences may have lasting impacts that shape hypertension among current and former residents. The South was traditionally economically underdeveloped, and this lower SEP at the area level may result in increased hypertension risk, even for those who lived there in childhood and have subsequently moved to other parts of the country. Regional differences in economic development may influence the socioeconomic context of metropolitan areas within the different regions. There may also be regional differences in health behaviors like diet or physical activity.

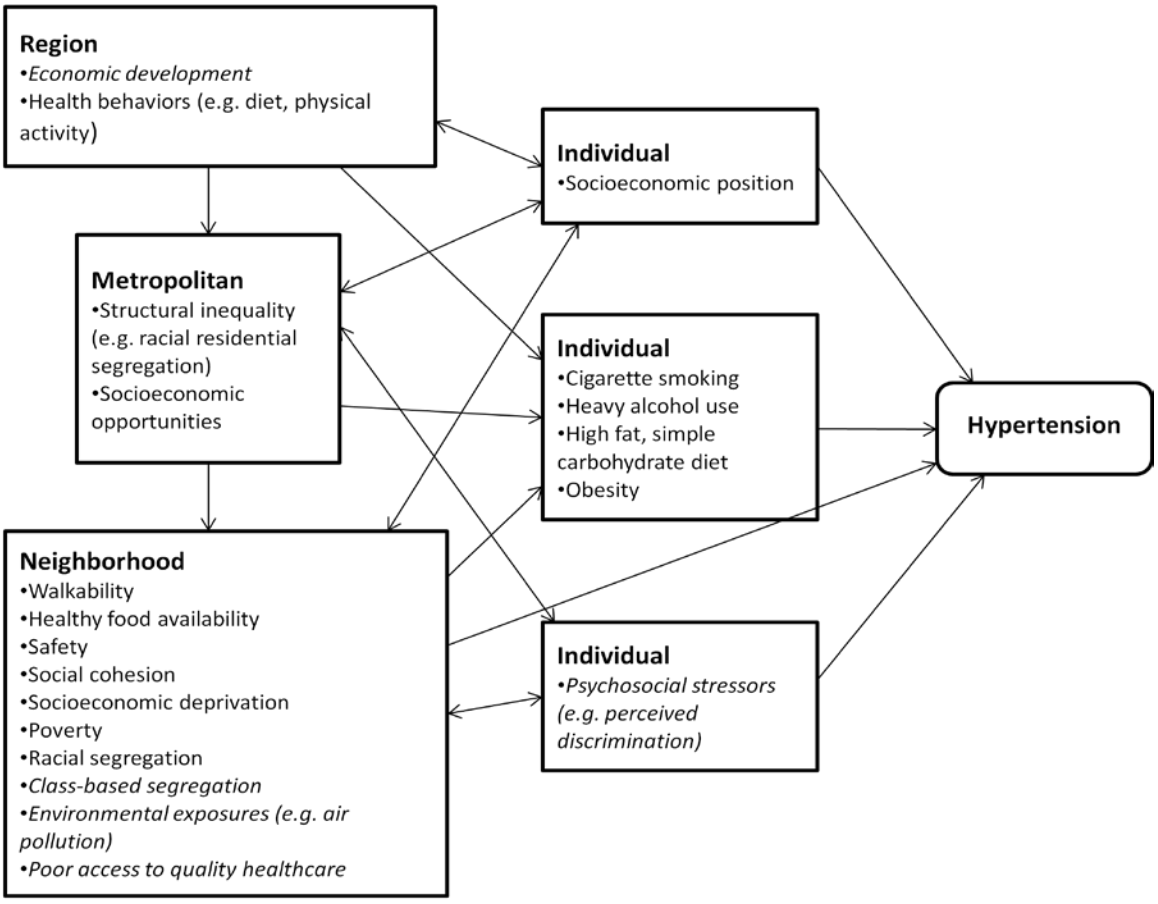
Metropolitan area-level geographic factors may impact hypertension as well. There may be differences in job opportunities and the quality of education received in different metropolitan areas that influence the average socioeconomic attainment of its residents. Differing levels of inequality (in the distribution of social and economic resources) across metropolitan areas may also influence the quality of the neighborhood environment.

The neighborhood environment may also lead to variation in hypertension. Several aspects of the residential context have been linked to racial disparities in CVD risk and hypertension including neighborhood poverty and disadvantage, neighborhood

social cohesion, walkability, healthy food availability, and safety. There may also be differences in exposure to environmental exposures linked to hypertension. In addition, there could be neighborhood-level differences in access to health care. Living in a resource-poor neighborhood may lead to hypertension by increasing levels of stress or by promoting access to health behaviors associated with adverse health while denying access to health promoting behaviors.

Figure 1.1: Conceptual model

Note: Unidirectional arrows indicate a hypothesized causal relationship, and double-headed arrows indicate variables that are associated with each other, but not necessarily causally. Italicized items are those that are believed to impact hypertension but are not assessed in this dissertation.



At the individual level, low socioeconomic position is a strong predictor of hypertension. Individual socioeconomic attainment may be influenced by elements of the area-level environment. For example, the local tax base influences the quality of the education received. Individual SEP may also influence the neighborhoods or metropolitan areas in which an individual is able to live. There are also psychosocial factors like perceived discrimination that may act as stressors and lead to hypertension. Neighborhood-level characteristics could influence perceptions of discrimination (e.g. by impacting level of direct exposure to discrimination). Conversely, perceptions of discrimination may influence the type of neighborhood in which an individual chooses to live; they may prefer to live in segregated neighborhoods in an attempt to avoid discrimination. Individual SEP and psychosocial factors may impact hypertension directly (e.g. by increasing levels of stress) or through health behaviors associated with hypertension such as poor diet.

Each aim in this dissertation was designed to better understand the contribution of area-level factors to variation in hypertension within and/or between race groups. Aim 1 examined variation in hypertension prevalence both among and between race groups by region of birth and place of residence and assessed the role of neighborhood- and individual-level characteristics in explaining these differences. This aim established whether or not hypertension prevalence varied geographically and began to explore what may be driving these differences.

The next two aims evaluated racial residential segregation, a specific area-level characteristic believed to be an important driver of health disparities. Despite the growing recognition of the contribution of area-level characteristics on hypertension and hypertension disparities, the current literature lacks an understanding of the role of segregation. The nonrandom spatial segregation of Blacks from Whites is believed to expose Blacks moreso than Whites to concentrated neighborhood disadvantage. It may also limit individual socioeconomic attainment by denying individuals access to a higher quality education and reducing the earning potential by removing jobs from central cities and moving them to suburban areas. A poor neighborhood environment and low

SEP puts Blacks at greater risk of hypertension and more serious cardiovascular diseases.

Living in a segregated neighborhood has a different outcome for Blacks than for Whites. Among Whites, this segregation is often protective or unassociated with health. For Blacks, however, the impact of segregation seems to vary depending on the level at which it is examined. At the metropolitan level, segregation is often linked to worse cardiovascular-related outcomes, but findings have been mixed at the neighborhood level. Metropolitan-level segregation measures speak to the overall level of structural inequality in the area, but this global measure may not adequately represent the variation in disparities within these places. Thus, there may be interesting differences in the level of segregation and the way this segregation impacts hypertension between versus within these areas.

Chapter 2:
Geographic variation in hypertension prevalence among Blacks and Whites

Introduction

It is well known that hypertension prevalence is higher among African Americans than it is for any other race/ethnic group in the US (27, 94). Despite a growing body of research suggesting that social, environmental, and lifestyle factors may play an important role, the reasons for these disparities remain unclear (21, 22, 39, 95). Understanding potential explanations for this variation within race/ethnic groups could help identify factors leading to hypertension and inform strategies to reduce racial/ethnic disparities.

Studies of race/ethnic differences in hypertension in the United States rarely investigate heterogeneity within groups, although some research has suggested that important geographic heterogeneity may exist. For example, evidence suggests that Blacks and Whites living in the South have higher hypertension prevalence and risk than those living in the rest of the country (42, 44). In addition, a longitudinal study of young adults found that black men living in Chicago, Illinois and Minneapolis, Minnesota were significantly less likely to develop elevated blood pressure levels over a 7-year period than black men living in Birmingham, Alabama after adjusting for education and established risk factors (43). A similar trend was observed among black women. Although no studies have investigated associations of region of birth with hypertension prevalence, being born in the South has been linked to increased cardiovascular disease mortality (48, 49, 51). However, the determinants of these geographic differences are not well understood.

Associations of region of birth and place of residence with the prevalence of hypertension were examined using data from non-Hispanic black and white participants of the Multi-Ethnic Study of Atherosclerosis (MESA). The contribution of the

neighborhood environment, socioeconomic characteristics, and traditional hypertension risk factors to observed geographic differences was also investigated. In addition, black-white differences in hypertension and how these differences varied across geographic subgroups were evaluated.

Methods

Study population

MESA is an observational cohort study designed to examine the determinants of subclinical cardiovascular disease in adults aged 45-84 years (96). Participants free of clinical cardiovascular disease at baseline were recruited from six study sites (New York, New York; Baltimore City and County, Maryland; Forsyth County, North Carolina; Minneapolis, Minnesota; Los Angeles County, California; and Chicago, Illinois) between 2000 and 2002 (96). This study used the cross-sectional data collected at baseline. At each site, random population samples were selected using various lists of area residents. Additional details are provided elsewhere (96). Of the selected persons deemed eligible after screening, 59.8% participated in the study. White participants were recruited from all six study sites; black participants were recruited from all sites except Minneapolis. In order to facilitate comparisons across race groups, these analyses were restricted to the five sites from which both groups were recruited. Institutional Review Board approval was obtained at each site and all participants gave informed consent.

Hypertension definition and measurement

Resting seated blood pressure was measured three times at a single baseline visit by trained and certified clinic staff using a Dinamap PRO 100 automated oscillometric device (Critikon, Tampa, FL), and the average of the last two measurements was used in the analyses (97). Hypertension was defined as having a mean systolic blood pressure greater than or equal to 140 mm Hg, a mean diastolic blood pressure greater than or equal to 90 mm Hg, or a self-reported history of hypertension and report of being on medication for it (98).

Geographic measures

Participants were asked to report the state in which they were born. Place of birth was categorized as southern state, non-southern state, and foreign-born. The South is often defined as those states that seceded from the Union during the Civil War (99). One critique of such a categorization is that it fails to take into consideration the shared history and culture of certain states in the southern part of the country. In an attempt to account for this, the following were included as southern states in these analyses: Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, and West Virginia. Given their physical and cultural distance from the Deep South, Delaware, Maryland, and District of Columbia (states that are considered the South by the US Census (46)) were not included as southern states. Instead, they were added to the non-southern-born category. The foreign-born category included all participants born outside the US. Place of residence was defined as the study site in which the participant lived at baseline: Forsyth County, North Carolina; New York, New York; Baltimore City and County, Maryland; Chicago, Illinois; and Los Angeles, California.

Covariates

Several sets of covariates were examined as potential contributors to observed geographic differences including socioeconomic position; neighborhood factors; and traditional risk factors for hypertension.

Individual socioeconomic position

Parental education was categorized as less than high school (HS), HS, and college or more. Education information was collected on both parents, and the education level of the parent with the higher level of attainment was used in the analyses. Individual education was measured as the highest level completed and categorized as HS or less, some college/technical school, and bachelors degree or more. Annual household income was grouped into quartiles (less than $25,000; $25,000-$39,999; $40,000-$74,999; and $75,000 and greater). Baseline income was available and used for 91.6% of black participants and 97.3% of white participants. When

baseline income was missing, Exam 2 data were used (5.1% of black participants and 1.4% of white participants).

Neighborhood environment

Four survey-based scale measures of the neighborhood environment previously shown to be associated with hypertension in this cohort were examined (40): neighborhood safety (3 items), social cohesion (5 items), walkability (6 items), and healthy food availability (2 items). Each participant's scores were based on the average of the responses given by all other participants living within a mile of the participant, and higher scores represented better environments.

Because some of these neighborhood characteristics were highly correlated, these four scores were entered into a factor analysis with oblique rotation. Two factors were identified that accounted for 81.3 percent of the variation in the data. Factor 1, the physical environment, included neighborhood walkability and healthy food availability. Factor 2, the social environment, consisted of neighborhood safety and social cohesion. Factor-based scores were created for the neighborhood physical and social environments by summing the respective scales within each factor. All factor loadings were comparable (physical environment: 0.87, 0.93; social environment: 0.79, 0.88), so these scales were not weighted by their respective loadings. Number of years living in current neighborhood was included as a control variable to account for varying lengths of exposure to a given neighborhood environment.

Hypertension risk factors

Body mass index (BMI) and health behaviors that are known risk factors for hypertension were assessed as potential mediators of the associations between region of birth/residence, individual SEP and the neighborhood environment and hypertension prevalence. Height and weight measured at baseline were used to calculate BMI. Alcohol use and cigarette smoking were based on self-report and dichotomized as current versus not current. Exercise was measured as metabolic equivalent (MET) hours per day spent in intentional activity and categorized for analyses as high, some, and no activity, with high representing levels above the median (2.0 MET-hours per day).

Statistical analyses

Key covariates were compared across place of birth and place of residence using analysis of variance (ANOVA) and the χ^2 statistic. Because hypertension is not a rare condition, the odds ratio is not a good approximation of the prevalence ratio (100). As recommended in prior work (101, 102), Poisson regression with robust variance estimates was used to estimate prevalence ratios of hypertension comparing the different place of birth (with US birth outside the South as the reference group) and place of residence (with Chicago as the reference group) categories. Although statistically significant interactions between race/ethnicity and place of birth or place of residence were not found, all analyses were conducted separately for white and black participants in order to assess within-race variation in hypertension by area and race-specific predictors. Models were sequentially adjusted for socioeconomic characteristics, neighborhood characteristics, and cardiovascular risk factors.

In addition, analyses pooling Whites and Blacks were conducted in order to contrast different race-geography combinations. Differences by place of birth and place of residence were assessed separately. Two different reference groups were used for these analyses: Whites born in/residing in the area with the highest hypertension prevalence and Whites born in/residing in the area with the lowest hypertension prevalence. This was done to investigate how contrasts between Whites and Blacks differ depending on which geographic subgroups are compared.

Of the 1894 Blacks and 2018 Whites living in the 5 MESA study sites at baseline, 324 Blacks and 249 Whites were missing information on state of birth, parental or personal SEP, or neighborhood characteristics. An additional 8 Blacks and 9 Whites were missing data on BMI or health behaviors, leaving 1562 Blacks and 1760 Whites for analysis. All analyses were conducted using SAS 9.2 (Cary, NC).

Results

Descriptive statistics comparing covariates by place of birth and place of residence for Blacks and Whites are shown in Tables 2.1 and 2.2, respectively. Most participants living in Forsyth at baseline were born in southern states (91.3%), and

between 34.1% and 45.5% of those living in the other sites were born in southern states (not shown). Unadjusted hypertension prevalence among Blacks was highest in those born in the southern states and lowest among the foreign-born. Prevalence of hypertension was highest among those living in Forsyth and lowest in those living in Chicago. Blacks born in southern states generally had the lowest levels of parental education and individual SEP while those born in non-southern states had the highest. New York residents had the lowest levels of parental education and individual education of all the sites, but the best reported neighborhood social environment. Those living in Chicago generally had the highest levels of parental education and individual SEP of all the sites, as well as the best neighborhood physical environment.

Hypertension prevalence among Whites was lower than among Blacks within every place of birth and place of residence category. Just over 67% of Whites living in Forsyth were born in southern states (not shown); a much smaller percentage of Whites living in the other sites were born in southern states (between 2.5% and 10.2%). Whites born in the South had the highest unadjusted hypertension prevalence and foreign-born Whites had the lowest. As with Blacks, Whites living in Forsyth had the highest hypertension prevalence and those living in Chicago had the lowest. Whites born in non-southern states had the highest level of parental education and individual SEP, whereas those born in southern states had the lowest parental and individual educational attainment. Whites living in Forsyth and Baltimore had the lowest parental education levels and individual SEP, while those in Los Angeles had the best. Chicago residents reported the best physical environments and Forsyth residents reported the best social environments.

Table 2.1 Selected socioeconomic, neighborhood, and risk factor characteristics of Blacks by place of birth and place of residence

	Born in southern state (n=801)	Born in non-southern state (n=618)	Foreign-born (n=143)	p-value*	Forsyth (n=401)	New York (n=330)	Baltimore (n=434)	Los Angeles (n=134)	Chicago (n=263)	p-value*
Hypertensive (%)	64.9	51.8	44.1	<0.0001	65.6	56.7	57.4	53	50.6	0.002
Age (years)†	63.4 (9.6)	60.2 (9.9)	57.1 (8.9)	<0.0001	61.6 (9.6)	61.1 (9.9)	61.6 (9.7)	61.6 (10.3)	61.7 (10.3)	0.94
Sex (% male)	44.2	46.9	49	0.42	44.9	41.8	45.4	49.1	52.2	0.23
Parental education less than HS (%)	53.6	30.1	49.7	<0.0001	44.9	48.2	47.5	35	36.6	0.003
Income < $25,000 (%)	33.1	23.8	32.2	0.0005	28.2	27.3	33.2	19.4	44.8	<0.0001
High school education or less (%)	32.3	22.2	35.7	<0.0001	26.2	37.9	31.8	14.8	29.9	<0.0001
Neighborhood physical environment†	6.9 (0.9)	7.3 (0.7)	7.7 (0.6)	<0.0001	6.2 (0.7)	6.6 (0.3)	6.9 (0.4)	7.1 (0.4)	8.0 (0.5)	<0.0001
Neighborhood social environment†	7.1 (0.6)	6.9 (0.5)	6.7 (0.5)	<0.0001	7.5 (0.5)	7.9 (0.3)	7.1 (0.4)	7.0 (0.4)	6.7 (0.4)	<0.0001
BMI (kg/m^2)†	30.0 (5.6)	30.3 (5.8)	29.5 (5.3)	0.36	30.2 (5.4)	30.1 (6.0)	30.5 (5.6)	29.5 (5.8)	29.4 (5.5)	0.07
Current cigarette smoker (%)	16.4	22.2	6.3	<0.0001	15.5	16.1	18	20.5	22.4	0.24
Current alcohol drinker (%)	46.9	56	49	0.003	41.9	50.3	46.5	70.3	53	<0.0001
No intentional exercise (%)	24.1	21.5	25.2	0.44	22.7	18.5	27.7	19.4	29.1	0.008

*p-value for test of overall association between site and each of the covariates

†Values in parentheses are standard deviations

Table 2.2: Selected socioeconomic, neighborhood, and risk factor characteristics of Whites by place of birth and place of residence

	Born in southern state (n=429)	Born in non-southern states (n=1194)	Foreign (n=137)	p-value*	Forsyth (n=501)	New York (n=200)	Baltimore (n=431)	Los Angeles (n=122)	Chicago (n=506)	p-value*
Hypertensive (%)	47.1	35.8	32.9	<0.0001	45.7	31	42.2	36.9	30.8	<0.0001
Age (years)†	62.8 (10.0)	62.6 (9.7)	63.8 (10.5)	0.38	62.6 (9.6)	62.4 (9.7)	64.2 (10.0)	61.2 (10.7)	62.0 (9.7)	<0.0001
Sex (% male)	47.1	49.3	44.5	0.48	49.3	44	50.8	46.1	52.5	0.33
Parental education less than HS (%)	34.7	23.4	25.6	<0.0001	32.3	25	34.8	16.2	15.6	<0.0001
Income < $25,000 (%)	13.8	11.3	15.3	0.21	13.6	7.5	18.6	6.7	14.8	<0.0001
High school education or less (%)	30.5	14.2	17.5	<0.0001	28.3	12	24.6	5.9	18	<0.0001
Neighborhood physical environment†	6.7 (1.0)	7.8 (1.1)	7.9 (1.0)	<0.0001	6.4 (0.7)	8.4 (0.4)	7.1 (0.6)	7.2 (0.3)	8.8 (0.4)	<0.0001
Neighborhood social environment†	7.7 (0.5)	7.3 (0.5)	7.2 (0.5)	<0.0001	7.9 (0.4)	7.1 (0.4)	7.3 (0.5)	7.2 (0.4)	7.2 (0.3)	<0.0001
BMI (kg/m^2)†	27.6 (4.9)	27.4 (5.0)	26.9 (5.0)	0.41	27.7 (4.9)	26.4 (4.7)	28.1 (5.2)	27.6 (5.4)	26.8 (4.8)	<0.0001
Current cigarette smoker (%)	12.4	8	13.1	0.01	11.4	9	8.1	8.9	9.8	0.51
Current alcohol drinker (%)	55.9	77.1	78.8	<0.0001	56.5	81.5	69.6	86.8	68	<0.0001
No intentional exercise (%)	20.1	14	16.8	0.01	17.8	14	19.3	10.5	18.9	0.002

*p-value for test of overall association between site and each of the covariates

†Values in parentheses are standard deviations

Table 2.3 Prevalence ratios of hypertension among Blacks by place of birth and place of residence before and after adjustment for socioeconomic factors, neighborhood characteristics, and hypertension risk factors

	Model 1*	Model 2*	Model 3*	Model 4†	Model 5‡
Born in southern state	1.11 (1.01, 1.23)	1.11 (1.00, 1.23)	1.10 (1.00, 1.22)	1.10 (0.99, 1.22)	1.10 (0.99, 1.22)
Foreign-born	0.90 (0.74, 1.09)	0.90 (0.74, 1.10)	0.88 (0.72, 1.07)	0.88 (0.72, 1.07)	0.87 (0.72, 1.06)
US-born, non-southern state	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Forsyth	1.23 (1.07, 1.42)	1.24 (1.07, 1.42)	1.20 (1.04, 1.38)	1.13 (0.90, 1.42)	1.10 (0.88, 1.38)
New York	1.16 (1.00, 1.34)	1.16 (1.00, 1.34)	1.09 (0.94, 1.26)	1.09 (0.94, 1.26)	1.06 (0.92, 1.23)
Baltimore	1.14 (1.00, 1.31)	1.14 (0.99, 1.31)	1.10 (0.96, 1.27)	1.06 (0.89, 1.26)	1.03 (0.87, 1.23)
Los Angeles	1.05 (0.86, 1.26)	1.05 (0.87, 1.27)	1.00 (0.83, 1.21)	0.97 (0.79, 1.20)	0.97 (0.79, 1.19)
Chicago	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Parental education less than HS		1.07 (0.94, 1.23)	1.04 (0.90, 1.19)	1.03 (0.90, 1.19)	1.02 (0.89, 1.17)
Parental education HS complete		1.09 (0.95, 1.24)	1.08 (0.94, 1.23)	1.07 (0.93, 1.23)	1.05 (0.92, 1.20)
Parental education college complete		1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
HS or less			1.07 (0.96, 1.19)	1.06 (0.95, 1.19)	1.03 (0.93, 1.15)
Some college/technical degree			1.01 (0.91, 1.13)	1.01 (0.91, 1.13)	0.99 (0.89, 1.10)
Bachelors/graduate school			1.00 (ref)	1.00 (ref)	1.00 (ref)
Income <$25,000			1.30 (1.10, 1.54)	1.28 (1.08, 1.52)	1.30 (1.09, 1.54)
Income $25,000 - $39,999			1.33 (1.13, 1.57)	1.32 (1.11, 1.56)	1.32 (1.12, 1.57)
Income $40,000 - $74,999			1.20 (1.02, 1.41)	1.19 (1.01, 1.40)	1.18 (1.01, 1.39)
Income >$74,999			1.00 (ref)	1.00 (ref)	1.00 (ref)
Neighborhood physical environment				0.95 (0.87, 1.04)	0.96 (0.88, 1.04)
Neighborhood social environment				0.97 (0.88, 1.07)	0.96 (0.87, 1.06)

*Adjusted for age and sex in addition to all the variables shown

†Adjusted for age, sex, years in current neighborhood in addition to all the variables shown

‡Adjusted for age, sex, years in current neighborhood, BMI, alcohol use, cigarette smoking, diet, and exercise in addition to all the variables shown

Table 2.3 shows prevalence ratios of hypertension among Blacks by place of birth and place of residence. Blacks born in southern states were 1.11 (95% confidence interval (CI): 1.01, 1.23) times more likely to be hypertensive than those born in non-southern states. This association was not substantially modified after adjustment for individual SEP and neighborhood characteristics (prevalence ratio (PR): 1.10, 95% CI: 0.99, 1.22). Further adjustment for risk factors had little effect. Blacks living in Forsyth (PR: 1.23, 95% CI: 1.07, 1.42), New York (PR: 1.16, 95% CI: 1.00, 1.34), and Baltimore (PR: 1.14, 95% CI: 1.00, 1.31) were all significantly more likely to be hypertensive than those living in Chicago. All associations were slightly attenuated after adjustment for individual SEP but a larger reduction in associations (especially for Forsyth) was observed when neighborhood characteristics were accounted for (Forsyth, PR: 1.13, 95% CI: 0.90, 1.42; New York, PR: 1.09, 95% CI: 0.94, 1.26; Baltimore, PR: 1.06, 95% CI: 0.89, 1.26). These associations were slightly reduced after additional adjustment for hypertension risk factors.

Whites born in southern states were 1.15 (95% CI: 0.98, 1.35) times more likely to be hypertensive than those born in non-southern states (Table 2.4). This association was slightly attenuated with each subsequent adjustment for parental SEP and individual SEP, was not substantially modified after adjustment for neighborhood characteristics, and increased after adjustment for hypertension risk factors (PR: 1.17, 95% CI: 1.00, 1.38). Whites living in Forsyth (PR: 1.32, 95% CI: 1.09, 1.60) and Baltimore (PR: 1.24, 95% CI: 1.05, 1.47) had significantly higher hypertension prevalence compared with those living in Chicago. Los Angeles residents also had higher hypertension prevalence than those in Chicago, though this association was not statistically significant (PR: 1.20, 95% CI: 0.93, 1.56), possibly due to the smaller sample size. These associations were weakened after adjusting for parental and individual SEP but were strengthened after subsequent adjustment for neighborhood characteristics.

Table 2.4: Prevalence ratios of hypertension among Whites by place of birth and place of residence before and after adjustment for socioeconomic factors, neighborhood characteristics, and hypertension risk factors

	Model 1*	Model 2*	Model 3*	Model 4†	Model 5‡
Born in southern state	1.15 (0.98, 1.35)	1.14 (0.97, 1.34)	1.12 (0.95, 1.32)	1.13 (0.96, 1.33)	1.17 (1.00, 1.38)
Foreign-born	0.90 (0.71, 1.14)	0.91 (0.71, 1.15)	0.90 (0.71, 1.14)	0.90 (0.71, 1.15)	0.89 (0.71, 1.13)
Born in non-southern state	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Forsyth	1.32 (1.09, 1.60)	1.30 (1.08, 1.58)	1.23 (1.01, 1.50)	1.32 (0.95, 1.84)	1.29 (0.94, 1.78)
New York	1.01 (0.79, 1.28)	1.00 (0.79, 1.27)	0.99 (0.78, 1.26)	1.01 (0.80, 1.29)	1.03 (0.81, 1.31)
Baltimore	1.24 (1.05, 1.47)	1.22 (1.03, 1.44)	1.17 (0.98, 1.39)	1.26 (0.99, 1.60)	1.21 (0.91, 1.62)
Los Angeles	1.20 (0.93, 1.56)	1.20 (0.93, 1.55)	1.13 (0.88, 1.47)	1.22 (0.90, 1.66)	1.01 (0.85, 1.20)
Chicago	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Parental education less than HS		1.12 (0.95, 1.31)	1.03 (0.87, 1.23)	1.04 (0.87, 1.24)	1.01 (0.85, 1.20)
Parental education HS complete		1.03 (0.88, 1.20)	0.99 (0.84, 1.15)	0.99 (0.85, 1.16)	0.96 (0.82, 1.12)
Parental education college complete		1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
HS or less			1.24 (1.05, 1.47)	1.26 (1.06, 1.49)	1.24 (1.05, 1.47)
Some college/technical degree			1.17 (1.00, 1.36)	1.17 (1.01, 1.36)	1.16 (1.00, 1.35)
Bachelors/Graduate degree			1.00 (ref)	1.00 (ref)	1.00 (ref)
Income <$25,000			0.97 (0.80, 1.18)	0.98 (0.80, 1.21)	0.94 (0.76, 1.16)
Income $25,000 - $39,999			1.02 (0.84, 1.22)	1.03 (0.85, 1.24)	0.98 (0.82, 1.19)
Income $40,000 - $74,999			1.09 (0.94, 1.27)	1.10 (0.94, 1.28)	1.07 (0.92, 1.25)
Income > $74,999			1.01 (0.95, 1.07)	1.00 (ref)	1.00 (ref)
Neighborhood physical environment				1.05 (0.95, 1.16)	1.05 (0.96, 1.15)
Neighborhood social environment				1.05 (0.91, 1.22)	1.05 (0.91, 1.21)

*Adjusted for age and sex in addition to all the variables shown

†Adjusted for age, sex, years in current neighborhood in addition to all the variables shown

‡Adjusted for age, sex, years in current neighborhood, BMI, alcohol use, cigarette smoking, diet, and exercise in addition to all the variables shown

Table 2.5 Prevalence ratios of hypertension by race and study site

	Model 1*	Model 2*	Model 3*	Model 4*
Southern-born Blacks	1.57 (1.41, 1.74)	1.39 (1.23, 1.56)		
Foreign-born Blacks	1.33 (1.09, 1.63)	1.18 (0.95, 1.47)		
Non-southern-born Blacks	1.45 (1.29, 1.62)	1.28 (1.11, 1.48)		
Southern-born Whites	1.13 (0.99, 1.29)	1.00 (ref)		
Foreign-born Whites	0.89 (0.70, 1.13)	0.79 (0.61, 1.02)		
Non-southern-born Whites	1.00 (ref)	0.89 (0.78, 1.01)		
Forsyth Blacks			1.82 (1.43, 2.31)	1.41 (1.25, 1.60)
New York City Blacks			1.61 (1.35, 1.93)	1.26 (1.03, 1.53)
Baltimore Blacks			1.65 (1.35, 2.01)	1.28 (1.11, 1.48)
Chicago Blacks			1.45 (1.21, 1.75)	1.13 (0.92, 1.39)
Los Angeles Blacks			1.49 (1.18, 1.88)	1.16 (0.95, 1.41)
Forsyth Whites			1.29 (1.01, 1.63)	1.00 (ref)
New York City Whites			0.98 (0.77, 1.25)	0.76 (0.58, 1.01)
Baltimore Whites			1.19 (0.97, 1.45)	0.92 (0.78, 1.09)
Los Angeles Whites			1.14 (0.87, 1.51)	0.89 (0.68, 1.16)
Chicago Whites			1.00 (ref)	0.78 (0.61, 0.99)

*Models adjusted for age, sex, place of residence, parental education, individual SEP, and neighborhood characteristics

Table 2.5 shows prevalence ratios for categories based on race and place of birth and race and place of residence in separate models. Two models were reported for place of birth and two models were reported for place of residence: one with Whites with the lowest hypertension prevalence as the reference (models 1 and 3), and another with Whites with the highest hypertension prevalence as the reference category (models 2 and 4). Results show that the magnitude of black-white differences varies substantially depending on which subgroups of Blacks and Whites are compared. Substantial heterogeneity exists when subgroups with different places of birth are compared: the largest race difference was observed when Blacks born in the southern states were compared with non-southern-born Whites (Model 1, PR: 1.57, 95% CI: 1.41, 1.74). In contrast, a much smaller race difference was observed when foreign-born Blacks were compared with southern-born Whites (Model 2, PR 1.18, 95% CI: 0.95, 1.47). The most extreme race difference was observed when Forsyth Blacks are compared with Chicago Whites (PR: 1.82, 95% CI: 1.43, 2.31) whereas the smallest difference was observed when Chicago Blacks are compared with Forsyth Whites (PR: 1.13, 95% CI: 0.92, 1.39).

Discussion

These findings confirm that there is important geographic variation in hypertension prevalence among US Blacks and Whites. Hypertension prevalence was higher among Blacks born in southern states than those born in non-southern states, and was significantly higher among Blacks living in Forsyth, Baltimore and New York than in those living in Chicago. Important heterogeneity was also observed in Whites: Southern-born Whites had marginally higher hypertension prevalence than non-southern-born Whites and Whites living in Forsyth and Baltimore had significantly higher hypertension prevalence than those in Chicago. Adjustment for SEP and neighborhood characteristics reduced many of these associations. Additional adjustment for hypertension risk factors generally had little impact.

An important consequence of this geographic heterogeneity is that differences in hypertension prevalence between Blacks and Whites are not constant but vary

substantially depending on which geographic groups are compared. In a model adjusted for demographics, parental and individual SEP, and neighborhood characteristics, hypertension prevalence was 57% higher among southern-born Blacks compared with non-southern-born Whites. In contrast, hypertension prevalence was only 18% higher among foreign-born Blacks compared with southern-born Whites. The differences were even more striking when comparing race differences in hypertension prevalence across place of residence. Hypertension prevalence was 82% higher among Blacks living in Forsyth compared with Whites living in Chicago. On the other hand, hypertension prevalence was just 13% higher among Blacks living in Chicago compared with Whites living in Forsyth, a difference similar to within-race differences in hypertension prevalence by residence. The presence of large variations in black-white differences suggests that race differences are not immutable and may vary substantially according to the social and environmental context.

Adjustment for hypertension risk factors had little impact on the associations between place of residence and hypertension. Although diet was not included in the analyses due to missing data, the inclusion of a Dietary Approaches to Stop Hypertension (DASH) (13, 103) adherence measure derived from a food frequency questionnaire did not substantially modify results (not shown). The failure of established risk factors to fully explain geographic variability is consistent with prior studies (43, 44). Possible explanations include measurement error in risk factors and an absence of risk factor history, which both limit a thorough examination of the role of these factors as contributors to geographic differences.

Other work has previously reported differences in hypertension incidence and prevalence by region within the US, and the current findings are generally consistent with prior results showing higher levels of hypertension among those living in southern states (42, 43, 44). This study builds on prior research by examining not only place of current residence, but also place of birth. Being born in southern states was associated with increased probability of being hypertensive independent of place of residence. These findings are consistent with mortality studies in Ohio and New York City which

showed that cardiovascular disease mortality rates were higher among Blacks born in the South than those born in other parts of the country regardless of where they later lived (49, 51).

There are several plausible mechanisms through which characteristics of place of birth may influence hypertension. Individuals who leave the South may bring along adverse eating habits. Persons living in the South report consuming lower levels of fiber and higher amounts of sodium and cholesterol compared with persons living in the Northeast, the Midwest, and the West (104). Lower socioeconomic position in childhood may have persistent effects leading to hypertension in adulthood (105, 106, 107), and educational attainment and per capita income have traditionally been lower in the South than other parts of the country (108, 109). In the current study, associations were not substantially modified after adjustment for parental or adult SEP or available behavioral factors, but the available measures were limited. The DASH dietary adherence measure investigated in sensitivity analyses was not associated with hypertension in this sample, and did not contribute to geographic differences. However, diet is notoriously difficult to measure and deserves further exploration as a contributor to geographic differences. In addition, a number of studies have reported an inverse relationship between birth weight and adult blood pressure (110) and the prevalence of low birth weight is higher in the South (111). Unfortunately birth weight data were not available in this sample.

One limitation of this study is that complete information on where the participants lived between when they were born and the time they joined the MESA study was not available. However, a 20-year residential history on 3380 of the black and white participants included in these analyses show that the majority of Blacks and Whites (approximately 80-87% depending on the state) lived in the same state 20 years ago as they did at the start of the study. These data suggest that measures based on current residence reflect long term exposures.

These results reveal substantial geographic heterogeneity in hypertension prevalence within race/ethnic groups and also demonstrate that black-white differences

vary substantially depending on which geographic groups are compared. A better understanding of the presence and causes of geographic differences in hypertension within and across race/ethnic groups may help guide efforts to prevent the disease and reduce the disparity.

Chapter 3:
Metropolitan-level racial residential segregation and black-white disparities in hypertension

Introduction

Hypertension prevalence is significantly higher among blacks than any other race/ethnic group in the US (1), and these disparities often persist in studies after adjustment for individual-level socioeconomic position and traditional risk factors (27, 112, 113). Identifying environments in which black-white hypertension disparities are smaller or nonexistent may help elucidate the role of contextual factors in perpetuating the unequal burden of hypertension as well as provide important clues regarding the causes of this disparity.

A growing number of studies link place to hypertension disparities but the mechanisms are not yet clear (39, 40, 41). No studies have examined the impact of racial residential segregation on black-white disparities in hypertension, but there are studies that indicate that disparities in other cardiovascular disease risk factors including body mass index (BMI) and 10-year predicted heart disease risk may differ based on levels of racial residential segregation (68, 114). Researchers hypothesize that segregation leads to health disparities by leaving Blacks more likely to live in concentrated poverty than Whites (60, 62). Living in concentrated poverty is in turn associated with a wide range of deleterious outcomes that could lead to hypertension disparities, including decreased neighborhood safety and social cohesion, limited access to healthy foods and recreational resources, lower levels of educational attainment, and higher unemployment (39, 40, 115).

In this study, data on adult participants of the 1999-2006 National Health and Nutrition Examination Surveys (NHANES) residing in metropolitan statistical areas (MSAs) were used to evaluate the contribution of racial residential segregation to black-

white hypertension disparities. Whether differences in neighborhood poverty by race contributed to the effect of segregation on racial disparities was also explored. It was hypothesized that higher levels of racial residential segregation would be associated with a stronger race difference in hypertension prevalence by creating greater neighborhood poverty differences between Blacks and Whites.

Methods

Study population

Data used in these analyses came from the black and white participants of the 1999-2006 National Health and Nutrition Examination Surveys (NHANES) aged 25 years and older residing in MSAs. Of the 10,611 eligible black and white study participants, 9262 had complete blood pressure data. In addition, 721 participants were excluded for missing education or income data and another 470 for missing data on BMI, cigarette smoking, diet, or exercise. This left 8071 participants for the analyses.

NHANES is a multi-stage stratified probability sample of US households, with an oversample of Blacks, Hispanics, and older adults which provides estimates for the national non-institutionalized population when sample weights are applied (116). National Center for Health Statistics (NCHS) Research Ethics Review Board approval was obtained for NHANES and informed consent was obtained from all participants.

Hypertension definition and measurement

Resting seated blood pressure was measured up to four times in a single visit by a certified operator using a mercury sphygmomanometer. Approximately 95% of all participants used in this study had at least two blood pressure measurements taken. For these analyses, the average of the last two measurements was used for participants who had three to four measurements taken (113). The second measurement was taken for those who had only two measurements, and the only measurement was used for participants who had just one recorded measurement. Hypertension prevalence was defined as having a mean systolic blood pressure greater than or equal to 140 mm Hg, a

mean diastolic blood pressure greater than or equal to 90 mm Hg, or a self-reported history of hypertension and report of being on medication for it (98).

Racial residential segregation definition and measurement

Massey and Denton conceptualized five geographic dimensions of racial/ethnic residential segregation: evenness, exposure, clustering, centralization, and concentration (117). All are empirically correlated, but each is thought to represent distinct aspects of residential segregation. In this study the isolation index, a measure of the exposure dimension, was used. The isolation index as used here estimates the extent to which Blacks are only exposed to other Blacks, or in other words, are isolated from other race/ethnic groups. This index was chosen because it has provided the strongest evidence of a conceptual link between residential segregation and health (60). Specifically, the residential isolation of Blacks from Whites is hypothesized to concentrate poverty among Blacks and leave them more vulnerable to the adverse health outcomes associated with living in disadvantaged neighborhoods.

The isolation index ranges from 0 to 1, where a score of 0 indicates that Blacks are completely integrated with Whites and 1 means that Blacks are completely isolated from Whites. It is represented mathematically as follows:

$$\text{Isolation index} = {}_xP^*_x = \sum_{i=1}^{n} \left[\frac{x_i}{X} \right] \left[\frac{x_i}{t_i} \right].$$

Where x_i is the number of Blacks in tract i , t_i is the total population in tract i and X is the number of Blacks in the metropolitan area. This proportion is then summed across all n census tracts in the MSA. MSAs were chosen as the larger geographic area as opposed to cities or counties because by design they represent regional housing markets and labor markets which help shape residential segregation and its potential impact on differential disadvantage and adverse health outcomes (61).

Neighborhood poverty definition and measurement

Census tracts were used as proxies for neighborhoods in these analyses. Neighborhood poverty was measured as the percentage of the population living below the US Census-defined poverty threshold and modeled continuously.

Covariates

Individual education was measured as the highest level completed and categorized as less than high school, high school, some college, and college or more. Mean annual family income was broken into the following quartiles:≤ $14,999; $15,000 - $34,999; $35,000 - $64,999; and ≥ $65,000. Gender was analyzed dichotomously as male versus female, and age was measured continuously. Cigarette smoking, based on self-report, was modeled dichotomously as current versus not current. BMI was included continuously (in kg/m^2), using measured height and weight. Intentional exercise was dichotomized as yes versus no based on whether or not participants reported engaging in any amount of moderate or vigorous exercise lasting 10 minutes or longer over the last 30 days.

Diet history was obtained using a 24-hour dietary recall that was administered by a trained interviewer. Poor diet was determined based on levels of consumption of the following nutrients: saturated fat, total fat, protein, cholesterol, fiber, magnesium, calcium, potassium, and sodium. Nutrient targets for everything except sodium were determined based on results of the Dietary Approaches to Stop Hypertension (DASH) trial (103); the target level of sodium consumption was based on the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of Blood Pressure recommendations (118). Dietary accordance with these targets was assessed based on prior work (119). Briefly, for each nutrient, participants were given a score of 1 if they met the target level of consumption. A score of 0.5 was given if they met an intermediate target. A DASH accordance score was created based on the sum of the scores given for each nutrient, and individuals with scores below 4.5 were considered to have a poor diet.

Analyses

Means with standard errors and frequencies were calculated for all continuous and categorical characteristics by race, taking into account the study design and unequal selection probabilities of the study participants. Continuous variables were compared using analysis of variance (ANOVA) and categorical variables using the χ^2 statistic.

Multilevel logistic modeling was used to assess black-white disparities in hypertension and the role of residential segregation and neighborhood poverty in explaining them. A series of three-level random intercept models were utilized in which the 8071 study participants used in these analyses were nested within 1827 census tracts, which were nested within 99 counties. This model was found to be better than a two-level model adjusted for age, sex, race, income, and education (with individuals nested within neighborhoods) based on the likelihood ratio (LR) test ($\chi^2 = 274.8$; $p<0.0001$) . A random intercept was not included to account for clustering at the MSA level because NHANES participants were sampled at the county level, and the majority of MSAs in the study population were only represented by one (64.9%) or two (27%) counties (maximum 3 counties per MSA). There was a median of 12 tracts per county and 8 participants per tract. These geographic identifiers are restricted-use variables and were accessed through the NCHS Research Data Center.

After running a random effects model without any fixed effects (null model), a model was run adjusted for age, gender and race and another further adjusted for income and education. Next, the black isolation was included to determine whether or not this explained some portion of the race difference in hypertension.

A cross-level interaction term was incorporated to allow the race difference in hypertension to vary by level of black isolation. Including a random coefficient for race did not significantly improve the fit of the model, so it was left out of these models (LR test $\chi^2 = 0.35$; $p=0.42$). Models were also run to determine whether the heterogeneity in race differences by levels of segregation were due to race differences in neighborhood poverty. Neighborhood poverty was adjusted for both alone and interacted with race to allow the relationship between poverty and hypertension to vary

by race. Black isolation was only weakly correlated with neighborhood poverty (Pearson r = 0.17), allowing us to assess their independent and interacting effects. To provide a more meaningful interpretation of the results, estimates for isolation and neighborhood poverty correspond to a difference equivalent to one standard deviation (SD) increase (SD = 0.21 and 10.7%, respectively). Neighborhood poverty was also mean-centered (mean = 12.1%) to aid in the interpretation of the interaction terms.

Individual-level sampling weights were incorporated into the multilevel models to account for the study design and unequal selection probabilities of the study participants. These design weights were scaled so that the new weights summed to the level-2 (census tract) cluster sample size (120). Level-2 and level-3 weights (accounting for selection probabilities of the census tracts and counties, respectively) were unavailable and were thus set to 1 in these analyses. All multilevel analyses were conducted using the GLLAMM program (121) in Stata 11 (StataCorp, College Station, TX).

Results

Hypertension prevalence was 40.0% among Blacks compared to 30.8% among Whites (Table 3.1). Blacks were also more likely to have low individual SEP; just under 30% of all Blacks had less than a high school education compared to fewer than 11% of Whites, and 23.6% of Blacks reported annual family incomes below $14,999 versus 9.5% of Whites. Mean BMI was significantly higher for Blacks than Whites; Blacks were also more likely to be current smokers and to have a poorer diet, and less likely to exercise.

On average, Blacks lived in neighborhoods in which 19.8% of the residents were poor whereas Whites resided in neighborhoods in which 8.4% of occupants were classified as poor. In addition, the average black isolation score was higher for Blacks than Whites (0.50 versus 0.36), meaning that black study participants were more likely than white participants to live in areas where Blacks were more spatially isolated from Whites.

Table 3.1 Descriptive statistics for selected covariates by race

	Blacks (n=2382)	Whites (n=5689)	p-value
Hypertensive (%)	40.0	30.8	<0.0001
Age in years (SE)	45.7 (0.3)	49.1 (0.3)	<0.0001
Gender (% male)	45.0	48.9	0.002
% Less than HS	29.9	10.7	<0.0001
% <$19,999	23.6	9.5	<0.0001
BMI in kg/m^2 (SE)	29.9 (0.2)	28.1 (0.2)	<0.0001
No intentional exercise (%)	80.2	73.8	0.0002
Current cigarette smoker (%)	26.8	22.5	0.003
Poor diet (%)	93.8	88.4	<0.0001
% Mean neighborhood poverty (SE)	19.8 (0.7)	8.4 (0.3)	<0.0001
Black isolation score	0.50 (0.02)	0.36 (0.02)	<0.0001

SE = standard error

After adjusting for age and gender, Blacks had a 2.92 (95% Confidence Interval (CI): 2.47, 3.45) times higher odds of hypertension compared with Whites (Table 3.2). This was attenuated some after further adjustment for education and income (Odds Ratio (OR): 2.74; 95% CI: 2.32, 3.25), and then increased slightly after adjusting for the isolation index (OR: 2.81; 95% CI: 2.37, 3.34). However, this association varied significantly by level of segregation (p for interaction 0.006). Blacks living in areas with low black isolation scores (10^{th} percentile; ${}_xP^*_x = 0.06$) had 1.67 (95% CI: 1.08, 2.57) times higher odds of hypertension than Whites, compared to a 3.57 (95% CI: 2.88, 4.42) times higher odds for Blacks versus Whites residing in high isolation areas (90^{th} percentile; ${}_xP^*_x = 0.65$). This significant interaction also indicated that the relationship between black isolation and hypertension was different for Blacks than for Whites. For Blacks, each standard deviation increase in isolation was associated with a 1.18 (95% CI: 1.00, 1.39) times higher odds of hypertension, while the opposite was seen for Whites (OR: 0.90; 95% CI: 0.79, 1.02).

Table 3.2 Odds ratios and 95% confidence intervals from a random intercept three-level logistic model of hypertension

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age		1.10 (1.10, 1.11)	1.10 (1.09, 1.11)	1.10 (1.09, 1.11)	1.10 (1.09, 1.11)	1.10 (1.09, 1.11)
Gender						
Male		0.91 (0.79, 1.06)	0.89 (0.77, 1.04)	0.89 (0.77, 1.04)	0.89 (0.77, 1.04)	0.89 (0.77, 1.03)
Female		1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Race						
Black		2.92 (2.47, 3.45)	2.74 (2.32, 3.25)	2.81 (2.37, 3.34)	1.54 (0.95, 2.50)	1.53 (0.95, 2.47)
White		1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Race difference at 10th percentile segregation					1.67 (1.08, 2.57)*	1.64 (1.07, 2.52)†
Race difference at 90th percentile segregation					3.57 (2.88, 4.42)*	3.37 (2.66, 4.26)†
Education						
Less than HS			1.37 (1.06, 1.77)	1.37 (1.06, 1.77)	1.36 (1.05, 1.76)	1.34 (1.03, 1.74)
HS			1.63 (1.34, 1.98)	1.63 (1.34, 1.97)	1.63 (1.34, 1.97)	1.62 (1.33, 1.97)
Some college			1.29 (1.06, 1.57)	1.29 (1.0, 1.57)	1.28 (1.05, 1.56)	1.28 (1.05, 1.56)
Bachelors or more			1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Income						
Inc1			1.19 (0.94, 1.51)	1.20 (0.94, 1.52)	1.19 (0.94, 1.51)	1.16 (0.92, 1.47)
Inc2			0.99 (0.78, 1.25)	0.99 (0.78, 1.26)	1.00 (0.78, 1.27)	0.98 (0.76, 1.25)
Inc3			0.87 (0.70, 1.10)	0.88 (0.70, 1.11)	0.88 (0.70, 1.10)	0.87 (0.69, 1.09)
Inc4			1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Black isolation score				0.96 (0.86, 1.07)		
Black isolation (Blacks)					1.18 (1.00, 1.39)*	1.17 (0.99, 1.38)†
Black isolation (Whites)					0.90 (0.79, 1.02)*	0.90 (0.79, 1.02)†
Neighborhood poverty						1.07 (0.97, 1.18)

Log likelihood	-8418.30	-6718.83	-6689.82	-6689.83	-6685.82	-6684.81
Random parameter						
Variance for L2 intercept	1.776 (0.204)	1.602 (0.193)	1.594 (0.197)	1.579 (0.179)	1.599 (0.182)	1.597 (0.179)
Variance for L3 intercept	0.045 (0.022)	0.021 (0.029)	0.009 (0.030)	0.016 (0.025)	0.008 (0.031)	0.009 (0.031)

*p for interaction = 0.006

†p for interaction = 0.009

Neighborhood poverty was not significantly associated with hypertension, and adjustment for neighborhood poverty did not explain the race-segregation interaction (i.e. the point estimates for the interactions did not change) (Table 3.2, Model 6). However there was statistical evidence that the association between race and hypertension also varied by level of neighborhood poverty (p for interaction 0.009) in addition to segregation (p for interaction 0.007). Each standard deviation increase in neighborhood poverty was associated with significantly higher odds of hypertension among Whites (not shown; OR: 1.18; 95% CI: 1.03, 1.36) but not among Blacks (not shown; OR: 0.97; 95% CI: 0.88, 1.07).

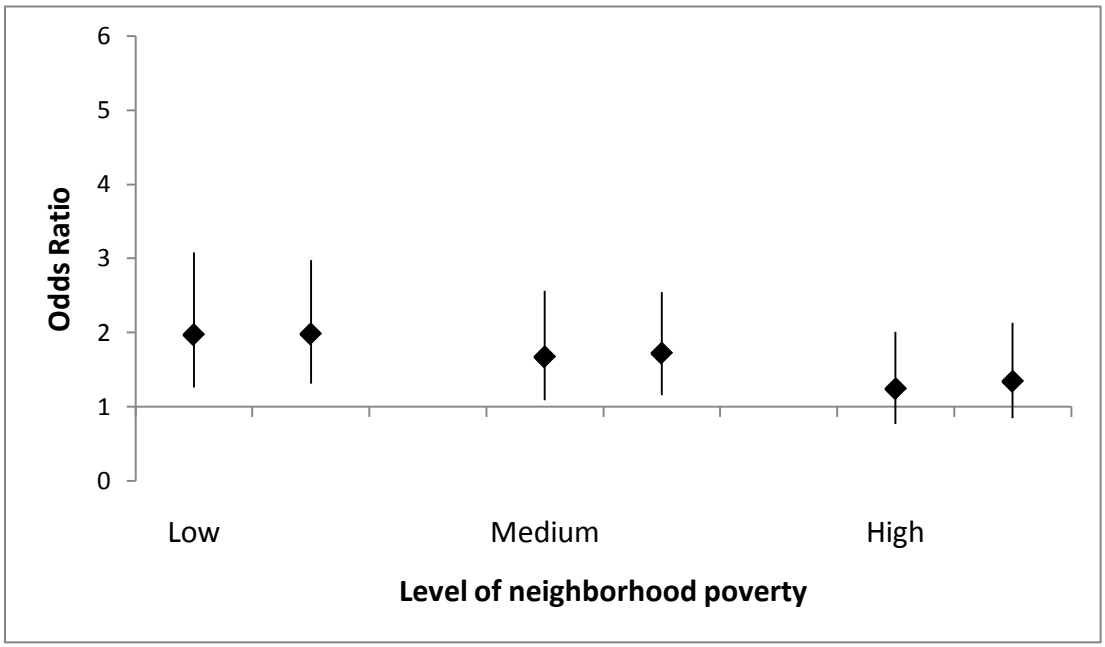
Figure 3.1 shows odds ratios of hypertension for Blacks versus Whites by levels of segregation and neighborhood poverty as estimated from a logistic regression model including both interaction terms. Overall, the odds ratios of hypertension in Blacks versus Whites were higher in areas of high segregation (Figure 3.1a) than in areas of low segregation (Figure 3.1b). In addition odds ratios for Blacks vs. Whites were greater in low poverty than in high poverty areas. For example the odds of hypertension for Blacks versus Whites living in very segregated MSAs ranged from 4.14 (95% CI: 3.18, 5.38) in low poverty neighborhoods (10^{th} percentile; 3%) to 2.61 (95% CI: 1.90, 3.57) in high poverty neighborhoods (90^{th} percentile; 28%). Corresponding ORs for the low segregation MSAs were 1.97 (95% CI: 1.26, 3.08) in low poverty neighborhoods and 1.24 (95% CI: 0.77, 2.01) in high poverty neighborhoods.

Adjustment for BMI and behavioral risks factors attenuated race differences somewhat for those living in high segregation areas but had little impact on race differences in low segregation areas (Figure 3.1). For example, at mean neighborhood poverty, Blacks living in highly segregated areas went from having a 3.50 (95% OR: 2.78, 4.40) times higher odds of hypertension than Whites to a 2.89 (95% CI: 2.30, 3.63) times higher odds after adjusting for risk factors. Meanwhile odds ratios for Blacks versus Whites living in integrated areas shifted slightly, moving from a 1.67 (95% CI: 1.09, 2.56) times higher odds of hypertension versus Whites to a 1.72 (95% CI: 1.16, 2.55) times

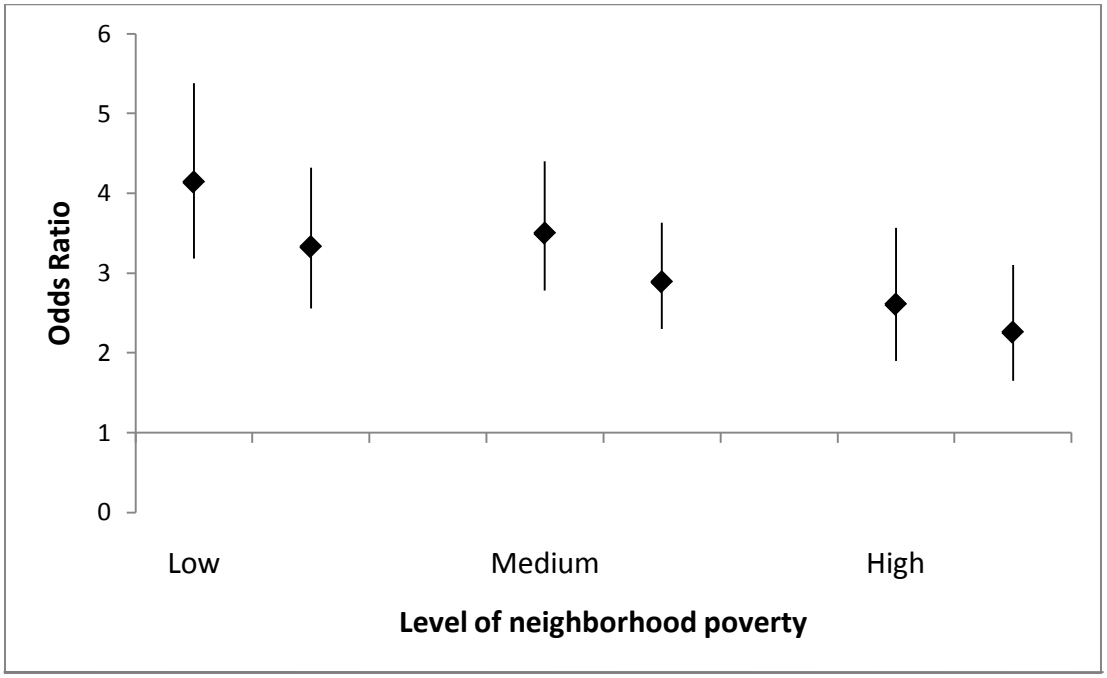
higher odds. Interactions between race and segregation and race and poverty were still statistically significant after risk factor adjustment (p for interactions 0.04 and 0.03, respectively).

Figure 3.1: Odds ratios for association between race and hypertension prevalence by level of neighborhood poverty at (A) low (10th percentile) and (B) high (90th percentile) levels of segregation before and after adjustment for BMI and health behaviors

A



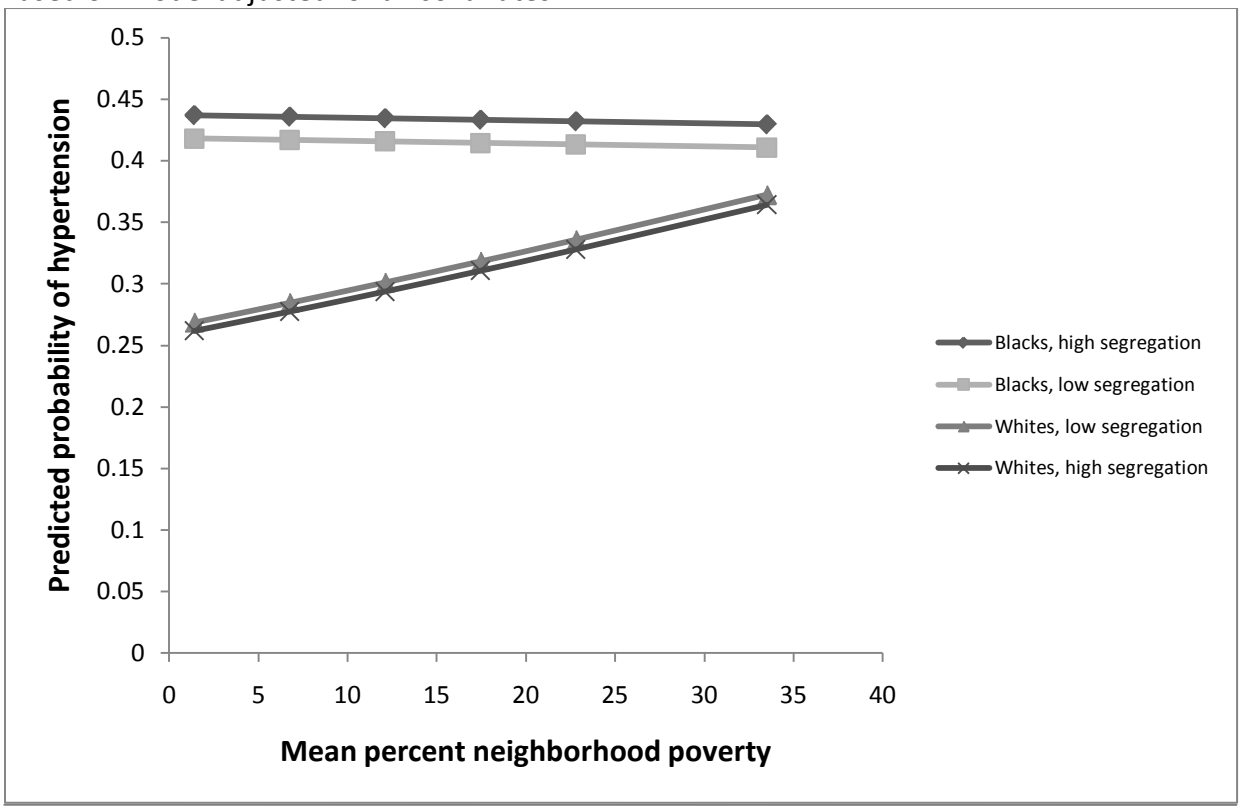
B



An examination of the predicted probabilities by level of neighborhood poverty and segregation for Blacks and Whites (Figure 3.2) shows that the diminished race difference in hypertension is due to increased hypertension prevalence among Whites living in higher poverty neighborhoods. This figure also suggests that while hypertension prevalence among Whites is the same at low and high levels of segregation, among Blacks prevalence is lower for those living in less segregated areas.

Figure 3.2: Predicted probability of hypertension for Blacks and Whites by level of segregation and neighborhood poverty

Based on model adjusted for all covariates



Discussion

This is the first study to investigate how metropolitan-level racial residential segregation is related to black-white disparities in hypertension. The association between race and hypertension varied significantly by level of black isolation; it was smallest among those living in more integrated areas and largest for those living in more segregated areas. This study further adds to the current literature on segregation and health by investigating neighborhood poverty as an underlying pathway through which residential segregation adversely impacts health disparities. Although segregation is hypothesized to impact health disparities through aspects of the neighborhood

environment, this has generally remained untested empirically. Comparison of results before and after adjustment for neighborhood poverty suggested that black-white differences in neighborhood poverty do not explain the larger black-white differences in more segregated areas. However, there was significant heterogeneity in the association between race and hypertension by neighborhood poverty in addition to segregation. When considered together, race differences were greatest in segregated, low poverty areas and weakest in non-segregated, high poverty areas.

Two studies have been conducted on metropolitan-level segregation and other measures of cardiovascular disease risk (68, 114). Using a nationally representative sample, Chang et al found that both BMI and odds of being overweight increased significantly with each standard deviation increase in black isolation for Blacks, but that there was no relationship for Whites (68). A study of BMI and 10-year predicted heart disease risk among low-income, uninsured women found no association between black isolation and BMI but did find that isolation was associated with reduced CHD risk among black women (114). However, that was a very specific population, and those results may not be generalizable to other populations. In addition, the models were only presented adjusted for a wide array of measures of the neighborhood built environment (e.g. number of fitness facilities, fast food restaurants, and full-sized grocery stores) that may mediate the relationships between isolation and adverse health.

Few studies have assessed variation in the relationship between race and hypertension by both level of segregation and neighborhood poverty, but there is some evidence supporting these findings. The Exploring Health Disparities in Integrated Communities (EHDIC) study of a low-income, integrated community in southwest Baltimore found that race differences in hypertension were substantially smaller there than in a national sample (41). After adjusting for race, age, gender, marital status, household income, and education level, the odds ratio for hypertension among Blacks versus Whites was 29% lower among those in the EHDIC sample compared with the

national sample. This is consistent with the current study findings that race differences in hypertension were smaller among those living in more integrated neighborhoods, under more similar living conditions.

The heterogeneity seen in black-white disparities in hypertension by level of neighborhood poverty in addition to segregation may be due to differential exposure to individual-level social stressors. Blacks are typically more likely than Whites to be exposed to social stressors such as financial insecurity or everyday experiences of discrimination (26, 122), and these may be associated with hypertension independent of area-level characteristics. Blacks and Whites living in higher poverty neighborhoods may be more comparable in their exposure to individual- and area-level stressors. In other words, these individuals may be universally worse off than those living in lower poverty neighborhoods due to a combination of negative exposures at the individual and neighborhood levels. On the other hand, there may be more variation in the racially patterned exposure to individual-level social stressors among those living in lower poverty neighborhoods. While access to neighborhood-level resources may be similar among them, there may be more variation in individual-level stressors.

The racial disparities in hypertension seen in this study were further attenuated by adjustment for BMI and behavioral risk factors, but only among those living in more segregated areas. Racial residential segregation leads to the inequitable distribution of social and economic resources, and this can create a stressful living environment. One way to cope with this chronic disadvantage is to engage in certain behaviors, such as cigarette smoking, physical activity, and consumption of a high fat and carbohydrate diet (77). All have been shown to reduce feelings of anxiety and stress, but in some cases at the expense of physical health (78, 79, 80, 81, 82, 84). At the same time, access to coping strategies linked with more positive health outcomes like exercise may be hindered by environmental factors associated with neighborhood poverty like low neighborhood safety or limited availability of recreational resources (86, 87, 88). Thus, it is possible that the contribution of these known individual-level risk factors to black-

white disparities in hypertension depend on the area-level context in which individuals live.

This study is not without limitations. One is that the data are cross-sectional and thus do not allow for the assessment of longitudinal relationships between segregation and hypertension or between the potential pathways through which segregation impacts hypertension. Another limitation is that reasons why race disparities in hypertension varied by level of black isolation or neighborhood poverty remained unclear. Adjustment for BMI and behavioral risk factors somewhat attenuated the race-segregation and race-neighborhood poverty interactions, but they remained statistically significant. Other measures of the neighborhood environment such as supermarket density or recreational resources may better explain the influences of segregation and neighborhood poverty on hypertension. Walkability and healthy food availability have both been found to be associated with lower blood pressure (40, 123). In addition, lower income, predominantly black neighborhoods tend to have more limited access to supermarkets (58, 59).

There may also be differences in unmeasured factors at the individual level that might mediate these relationships. Racial differences in wealth, for example, may be a better representation of disparities in economic status and resources than income or education, and it may be influenced by racial residential segregation (26, 36). Blacks living in highly segregated areas tend to experience smaller growth in housing equity, a major source of wealth, than owners of comparable homes in less segregated areas (124). In addition, racial residential segregation may impact residential returns on individual socioeconomic resources. Segregation based on race means that Blacks are less able to convert improvements in socioeconomic position into better residential quality (26, 125). Racial residential segregation may also be a reflection of the racial climate in the area and may be indicative of other exposures associated with hypertension such as perceived discrimination (34).

A related limitation is that the racial disparities in hypertension were not fully explained. Even after adjustment for all covariates there was still a small disparity between Blacks and Whites living in low segregation, high poverty neighborhoods. As with the persistent race-segregation and race-neighborhood poverty interactions, this lingering disparity may also be due to differences in the aforementioned unmeasured area- or individual-level factors such as healthy food availability, wealth, or perceived discrimination. Future work is needed to better understand the roles of these other factors.

Racial residential segregation is believed to be a fundamental cause of racial health disparities (36). It influences socioeconomic conditions for Blacks both at the individual and neighborhood levels by hindering opportunities for socioeconomic mobility and creating living environments that adversely affect health. This study shows that racial disparities in hypertension depend on the socio-environmental context at both the metropolitan level and the neighborhood level. Effective solutions to health disparities will need to target the processes that create segregation and the differential conditions under which individuals live.

Chapter 4:
Neighborhood-level racial segregation and hypertension among Blacks and Whites

Introduction

Neighborhood-level racial segregation has been linked to several health outcomes including all-cause mortality, cardiovascular disease (CVD) mortality, self-rated health, and homicide risk (57, 70, 71, 72, 126, 127). Studies have shown a protective or null association between neighborhood segregation and health for Whites (57, 70, 71). However, findings for Blacks are mixed; some show a protective association (70, 71), while others show the opposite relationship (57, 127). A New York City study by Fang and colleagues found that Blacks living in more segregated neighborhoods had lower CVD mortality rates (70). Another New York City study reported similar findings for all-cause mortality among Blacks (71). On the other hand, a multi-city study of homicide risk found higher rates of homicide fatalities for Blacks living in segregated neighborhoods (57).

Hypertension is a strong risk factor for cardiovascular disease and the disproportionate burden of this condition among those of lower socioeconomic position (SEP) and Blacks (1, 8) make it an important contributor to CVD disparities. Although several studies have investigated area-level variation in blood pressure (39, 40, 56, 128), very few have examined the possible effects of segregation and the mechanisms through which it may operate to affect blood pressure (69). Segregation of marginalized minority groups like Blacks could affect blood pressure through several processes. Some researchers hypothesize that highly segregated communities in which the minority group is the majority occupant are often more impoverished, leading to a more limited availability of physical activity and food resources and more stressful living conditions (57, 115), neighborhood characteristics that have been shown to be associated with higher levels of hypertension (39, 40, 56). Others suggest that residential segregation

may be protective due to the presence of strong social networks and reduced exposure to direct prejudice (73). In support of this argument, increased social integration and neighborhood social cohesion have been linked to lower levels of hypertension (40, 129).

These plausible competing hypotheses suggest that the nature of the association between segregation and blood pressure may vary by levels of other individual- and area-level characteristics. For example, the negative health effects of segregation may only be apparent in poor neighborhoods, among which segregation may be a stronger predictor of physical and social environments related to blood pressure. Segregation may also have stronger adverse health effects in neighborhoods lacking social cohesion because the adverse material and social consequences of segregation may be greater in neighborhoods lacking social cohesion. Effects could also differ depending on metropolitan area characteristics related to the distribution of resources and the extent to which resource distribution is correlated with segregation. Individual-level characteristics such as low socioeconomic position (SEP) may also modify these effects; for example high income or education may buffer any adverse effects of segregation. No studies have investigated these interactions for CVD-related outcomes, but there is evidence of variation in the effects of neighborhood segregation for other outcomes. An examination of neighborhood segregation and homicide mortality among Blacks and Hispanics found that the association varied by city of residence (57), and an investigation of neighborhood segregation and all-cause mortality among Blacks in Philadelphia found this relationship varied by level of neighborhood social capital (126).

Data from the Multi-Ethnic Study of Atherosclerosis (MESA) were used to examine associations of racial segregation with hypertension prevalence and incidence among Blacks and Whites. Variations in these associations by MESA site of residence, neighborhood poverty, neighborhood social cohesion, individual income, and individual educational attainment were also explored. It was hypothesized that for Blacks, the association between segregation and hypertension would become stronger with

increasing levels of neighborhood poverty and decreasing levels of neighborhood social cohesion, individual income, and individual educational attainment.

Methods

Study population

MESA is an observational cohort study of the determinants of subclinical cardiovascular disease in 6814 adults aged 45-84 years (96). Between 2000 and 2002, participants free of clinical cardiovascular disease at baseline were recruited from six study sites (New York, New York; Baltimore City and County, Maryland; Forsyth County, North Carolina; Minneapolis, Minnesota; Los Angeles County, California; and Chicago, Illinois) (96). At each site, random population samples were selected using various lists of area residents and of the selected persons deemed eligible after screening, 59.8% participated in the study. Additional details are provided elsewhere (96). Three subsequent waves of data collection took place at intervals of approximately 1.5 – 2 years, and retention rates were high (94%, 89%, and 86% for each follow-up wave, respectively). Self-identified non-Hispanic white participants were recruited from all six study sites and self-identified non-Hispanic black participants were recruited from all sites except Minneapolis. All participants gave informed consent, and Institutional Review Board approval was obtained at each site.

Hypertension definition and measurement

Seated resting blood pressure was measured three times at each examination (baseline and three follow-up visits) by trained and certified clinic staff using a Dinamap PRO 100 automated oscillometric device (Critikon, Tampa, FL); the average of the last two measurements was used in the analyses (97). At each examination, hypertension was defined as having a mean systolic blood pressure greater than or equal to 140 mm Hg, a mean diastolic blood pressure greater than or equal to 90 mm Hg, or a self-reported history of being hypertensive and being on medication for it (98).

Neighborhood-level racial segregation

Racial segregation is the extent to which race groups are separated spatially in residential contexts. This is believed to contribute to health disparities because it is not a random distribution of groups across space, but rather one driven by outside forces (e.g. housing discrimination) that result in the differential distribution of resources and opportunities by race/ethnicity (36, 61). Several studies have investigated associations of segregation with outcomes across Metropolitan Statistical Areas (MSAs) (61, 62, 66), but there is also evidence that local neighborhood segregation is related to health outcomes within cities or metropolitan areas (57, 70, 71, 126, 127).

Most studies of segregation and health have used aspatial measures that may not adequately capture local area segregation (130, 131). Whereas aspatial measures focus on the racial composition of a specific neighborhood, spatial measures also account for racial composition of spatially adjacent neighborhoods thus providing a more complete picture of segregation (132). In this study a spatial measure of segregation was used to assess the residential clustering of race groups within the six MSAs represented in MESA (Greensboro-Winston-Salem and Asheville, NC; New York-Newark-Edison, NY-NJ-PA; Baltimore-Towson and Hagerstown, MD; Minneapolis-St. Paul-Bloomington, MN-WI; Los Angeles-Long Beach-Santa Ana, CA; and Chicago-Naperville-Joliet, IL-IN-WI). When members of a certain race group cluster within an urban area, they are less likely to interact with other race groups. This may be detrimental to the health of the isolated group if it is already marginalized in society by concentrating neighborhood disadvantage (e.g. poverty and stressful living conditions) and isolating groups from crucial resources needed to thrive (e.g. better quality schools, access to healthy foods). On the other hand, this isolation may be protective by increasing social capital and limiting exposure to discrimination.

A spatial measure of local segregation (the G_i^* statistic) developed by Getis and Ord (133) was used in these analyses. It is defined as:

$$G_i^* = \frac{\sum w_{ij} x_j - \bar{x} \sum w_{ij}}{S \sqrt{\frac{[N \sum w_{ij}^2 - (\sum w_{ij})^2]}{N-1}}},$$

where *i* is a census tract, w_{ij} is a spatial weight matrix used to weight the impact of the racial composition of tract *j* (neighboring tracts) on the G_i^* statistic for tract *i*, x_j is the proportion of race/ethnic group *x* in tract *j*, $\bar{x}$ is the mean proportion of race group across all *N* tracts in the MSA, and

$$S = \sqrt{\frac{\sum x_j^2}{N} - (\bar{x})^2}.$$

The G_i^* statistic returns a Z-score for each neighborhood (census tract), indicating the extent to which the racial composition in that neighborhood and surrounding neighborhoods deviates from the mean racial composition for the whole MSA. In these analyses an inverse distance weight matrix was used, meaning that the impact of neighboring tracts on the index for tract *i* diminishes with increasing distance. A threshold distance was selected to specify the maximum distance a tract can be from the census tract of interest and still be considered a neighbor. For example, a threshold distance of 1 mile means that all neighboring tracts whose centroids are within 1 mile of the centroid of the tract of interest are included in the calculation of the spatial weight matrix. Because the size and density of the census tracts differed greatly by MESA site, a different threshold distance for the spatial weight matrix was used for each MSA. Specifically, the threshold distance was the one that ensured each tract had an average of 8 neighbors. This was determined to be the ideal number of neighbors to ensure reliable z-scores (134).

An assessment of the MESA census tracts revealed that some were located on the edge of metropolitan area boundaries. Known as "edge effects," the treatment of spaces outside metropolitan boundaries as having no population can distort measures of segregation (131). The majority of the edge tracts were in areas that are not physically surrounded by other settlements (e.g. along coastlines). For those that were, data from neighboring counties were incorporated to reduce the impact of edge effects.

Positive G_i^* Z-scores indicate racial clustering, scores around zero indicate areas where the racial representation is close to the average for the whole area, and negative scores suggest racial under-representation. A statistically significant positive z-score (> 1.96; $p<0.05$) indicates significant clustering and a statistically significant negative z-score (<1.96; $p<0.05$) indicates significant under-representation. Race-specific G_i^* statistics were modeled continuously in these analyses.

Potential moderators

Neighborhood poverty, defined as the percentage of people living in poverty, was modeled continuously. Neighborhood social cohesion was assessed through a score based on five survey questions designed to assess mutual trust and neighborhood solidarity (135). Each participant's score was based on the average of the responses given by all other participants living in the same census tract as the participant (median 12 participants in same tract), with a higher score representing greater social cohesion. Social cohesion was modeled continuously. Income and education were used as indicators of individual socioeconomic position. Income was categorized as < $15,999; $16,000 - $34,999; $35,000 - $74,999; and > $74,999, and education was categorized as less than high school; high school; some college or technical school; and bachelor's degree or higher.

Covariates

Body mass index (BMI) and health behaviors, including alcohol use, cigarette smoking, and intentional exercise, were included as potential mediators of the associations between segregation and hypertension incidence and prevalence. Height and weight measured at baseline were used to calculate BMI. Alcohol use and cigarette smoking were based on self-report and dichotomized as current versus not current. Intentional exercise was assessed using the MESA Typical Week Physical Activity Survey, which was adapted from the Cross-Cultural Activity Participation Study (136). Reported lengths of time engaged in activity were multiplied by activity-specific metabolic equivalent (MET) levels (137). For these analyses, exercise was measured as metabolic

equivalent (MET) hours per day spent in intentional activity and categorized as high, some, and no activity, with high representing levels above the median (2.0 MET-hours per day).

Statistical analyses

Of the 1694 non-Hispanic black and 2433 non-Hispanic white MESA participants enrolled at baseline who agreed to participate in the MESA Neighborhood Study, 173 Blacks and 156 Whites were excluded for missing data on one or more covariates used in the analyses, leaving 1521 Blacks and 2277 Whites. For the analyses of incident hypertension data were used on the 643 Blacks and 1418 Whites with complete baseline covariate data who were not hypertensive at baseline and who participated in at least one follow-up visit.

Race/ethnic-specific descriptive statistics were run for all variables by level of segregation. Chi-square tests were used to detect significant differences in categorical variables and analysis of variance (ANOVA) were used for continuous variables. For the cross-sectional analyses, Poisson regression with robust variance estimates was used to estimate prevalence ratios of hypertension (101, 102). Parametric accelerated failure time models were used to estimate relative hazards of hypertension because events occurred during the interval between follow-up interviews (interval censoring) (138, 139). The Weibull distribution was used as the accelerated failure time function; a comparison of model log-likelihood values showed virtually no difference in fit between the Weibull, exponential, and gamma distributions.

The race-specific associations between neighborhood segregation and hypertension were assessed in stages, adjusting for age, sex, site, income, education, neighborhood poverty, and neighborhood social cohesion as potential confounders. Segregation and neighborhood poverty, segregation and neighborhood social cohesion, and neighborhood poverty and neighborhood cohesion were only weakly to moderately correlated with each other among Blacks (Spearman r = 0.30, -0.07, and -0.43, respectively) and Whites (Spearman r = -0.66, 0.03, -0.20), allowing for the assessment of their independent effects. Current cigarette smoking, current alcohol use, physical

exercise and BMI were also adjusted for as potential mediators. There was no evidence of significant within-neighborhood correlation (age-, gender-, segregation-, and site-adjusted likelihood ratio test for random census tract intercept: $\chi^2 = 0.13$), so this was not accounted for in the models.

In order to test the interaction hypotheses, five separate models were run including interaction terms to assess whether the association between neighborhood racial/ethnic segregation and hypertension varied by study site, neighborhood poverty, neighborhood social cohesion, individual education, and individual income.

Results

Of all the Blacks in the sample, 40.3% were living in areas with significant clustering (z-score > 1.96) (not shown). There were no Whites living in significant clusters, but 1.1% of all Whites were living in areas where they were significantly under-represented (z-score < -1.96). The level of segregation varied widely by study site (Table 4.1). The median segregation z-score among Blacks ranged from 0.91 for those living in Forsyth to 3.44 for those living in Los Angeles. This means that Blacks living in Los Angeles are quite isolated from other race/ethnic groups whereas those in Forsyth are living in more integrated neighborhoods. Among Whites, the neighborhoods they lived in generally had the same composition of Whites as in the larger metropolitan areas in which they resided (i.e. z-scores close to 0). The exception was Minneapolis (median z-score -1.23), where the Whites sampled tended to live in neighborhoods with fewer Whites and a higher than average percentage of Hispanics (not shown).

Table 4.1: Neighborhood segregation by race and study site*

Race	Site					
	Forsyth	New York	Baltimore	Minneapolis	Los Angeles	Chicago
Blacks	0.91 (-0.18 - 2.56)	1.05 (0.26 - 2.17)	1.92 (1.34 - 2.11)		3.44 (2.28 - 5.16)	1.96 (1.47 - 2.01)
Whites	0.53 (0.04 - 0.69)	0.50 (0.08 - 0.93)	-0.11 (-0.96 - 0.43)	-1.23 (-1.43 - -0.25)	0.11 (-0.62 - 0.58)	0.48 (-0.19 - 0.70)

*Values depicted are median segregation score followed by interquartile range

Table 4.2: Race-specific descriptive statistics for selected covariates by level of segregation

	Blacks			Whites		
	Low (n=759)	High (n=762)	p-value	Low (n=1086)	High (n=1191)	p-value
% Hypertensive	54.3	61.2	0.007	35.8	39.0	0.12
Mean age in years*	60.6 (9.5)	62.6 (10.1)	<0.0001	60.9 (10.2)	63.4 (9.9)	<0.0001
% Male	43.2	46.7	0.17	46.1	49.5	0.1
Place of residence			<0.0001			<0.0001
Forsyth	34.8	20.0		14.3	30.8	
New York	27.1	13.5		4.8	11.7	
Baltimore	24.4	32.7		22.4	15.2	
Minneapolis	-	-		40.2	8.6	
Los Angeles	2.8	11.0		4.7	3.9	
Chicago	10.9	22.8		13.6	29.9	
Income			<0.0001			<0.0001
% <$15,999	11.9	18.9		9.2	5.7	
% $16,000 - $34,999	25.3	29.3		22.4	14.1	
% $35,000 - $74,999	41.9	37.0		42.6	32.5	
% >$74,999	21.0	14.8		25.8	47.7	
Education			0.05			<0.0001
% Less than high school	8.7	11.8		5.0	4.5	
% High school	17.5	19.3		19.1	14.4	
% Some college/technical school	34.7	35.4		31.4	24.1	
% Bachelor's degree or higher	39.1	33.5		44.6	57.1	
Mean % neighborhood poverty*	16.3 (13.1)	21.9 (10.8)	<0.0001	14.3 (7.6)	6.2 (3.6)	<0.0001
Mean neighborhood social cohesion*	3.55 (0.34)	3.53 (0.35)	0.22	3.56 (0.25)	3.59 (0.28)	0.002

% Current smoker	17.0	18.9	0.33	13.0	9.4	0.007
% Current drinker	49.7	50.7	0.70	71.1	73.7	0.16
% No intentional exercise	22.1	23.6	0.6	18.0	15.0	0.001
Mean body mass index in kg/m^2*	30.2 (5.8)	30.1 (5.7)	0.66	28.2 (5.3)	27.2 (4.7)	<0.0001

*Values in parentheses are standard deviations

Table 4.2 shows descriptive statistics for Blacks and Whites by level of segregation. Although neighborhood segregation is modeled continuously in the regression analyses, here it is dichotomized at the race-specific medians ($G_i^* = 1.23$ for Blacks; $G_i^* = 0.17$ for Whites) to better facilitate bivariate comparisons. Among Blacks, higher segregation was significantly associated with higher hypertension prevalence. Blacks living in more segregated neighborhoods were more likely to be older, to have lower individual SEP, and to live in higher poverty neighborhoods. They were also more likely to live in Baltimore, Los Angeles, and Chicago and less likely to live in New York and Forsyth. Whites living in more segregated neighborhoods were older, had higher individual SEP, and lived in lower poverty and more cohesive areas. Whites living in segregated neighborhoods were more likely to exercise and have lower BMI. They were also less likely to be current smokers than those living in less segregated neighborhoods.

Hypertension prevalence

Among Blacks, neighborhood segregation was marginally associated with higher hypertension prevalence (Table 4.3). After adjusting for individual demographics and socioeconomic characteristics, hypertension prevalence increased by 3% for every standard deviation increase in segregation (Model 1: Prevalence Ratio (PR): 1.03; 95% Confidence Interval (CI): 0.98, 1.08). This marginally significant association remained unchanged after further adjustment for neighborhood poverty, social cohesion, and hypertension risk factors. There was no association between neighborhood segregation and hypertension prevalence among Whites (Table 4.4; PR: 1.00; 95% CI: 0.94, 1.06), and this remained essentially unchanged after adjustment for neighborhood poverty, social cohesion, and hypertension risk factors.

Table 4.3: Prevalence ratios for hypertension among Blacks by local segregation and selected covariates

	Model 1*	Model 2*	Model 3*	Model 4**
SD of segregation	1.03 (0.98, 1.08)	1.03 (0.98, 1.08)	1.03 (0.98, 1.08)	1.03 (0.98, 1.08)
Education				
Less than HS	1.10 (0.95, 1.26)	1.09 (0.95, 1.26)	1.09 (0.95, 1.26)	1.06 (0.92, 1.22)
HS	1.16 (1.03, 1.31)	1.16 (1.03, 1.31)	1.16 (1.03, 1.31)	1.12 (1.00, 1.26)
Some college / technical school	1.04 (0.94, 1.16)	1.04 (0.94, 1.16)	1.04 (0.94, 1.16)	1.02 (0.91, 1.13)
Bachelors or more	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Income				
< $15,999	1.14 (0.94, 1.37)	1.13 (0.94, 1.37)	1.14 (0.94, 1.38)	1.14 (0.94, 1.38)
$16,000 - $34,999	1.24 (1.04, 1.46)	1.23 (1.04, 1.46)	1.24 (1.04, 1.47)	1.26 (1.06, 1.49)
$35,000 - $74,999	1.23 (1.05, 1.44)	1.23 (1.05, 1.44)	1.24 (1.06, 1.45)	1.23 (1.05, 1.44)
> $74,999	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
SD of neighborhood poverty		1.01 (0.96, 1.05)	1.01 (0.97, 1.06)	1.02 (0.97, 1.07)
SD of neighborhood cohesion			1.03 (0.98, 1.08)	1.02 (0.97, 1.07)

*Models adjusted for age, sex, and site

**Models adjusted for age, sex, site, current cigarette smoking, current alcohol use, intentional exercise and body mass index

SD = standard deviation

Table 4.4: Prevalence ratios for hypertension among Whites by local segregation and selected covariates

	Model 1*	Model 2*	Model 3*	Model 4**
SD of segregation	1.00 (0.94, 1.06)	0.98 (0.91, 1.06)	0.98 (0.91, 1.06)	0.99 (0.92, 1.07)
Education				
Less than HS	1.30 (1.05, 1.61)	1.31 (1.06, 1.62)	1.30 (1.05, 1.61)	1.22 (0.98, 1.53)
HS	1.31 (1.13, 1.51)	1.30 (1.13, 1.51)	1.30 (1.12, 1.51)	1.27 (1.09, 1.48)
Some college / technical school	1.20 (1.04, 1.37)	1.19 (1.04, 1.37)	1.19 (1.04, 1.36)	1.16 (1.02, 1.33)
Bachelors or more	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Income				
< $15,999	1.00 (0.80, 1.24)	1.01 (0.81, 1.25)	1.00 (0.81, 1.24)	0.94 (0.75, 1.17)
$16,000 - $34,999	1.00 (0.84, 1.18)	1.00 (0.84, 1.18)	0.99 (0.84, 1.17)	0.96 (0.81, 1.13)
$35,000 - $74,999	1.05 (0.92, 1.21)	1.05 (0.92, 1.21)	1.05 (0.91, 1.21)	1.01 (0.88, 1.16)
> $74,999	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
SD of neighborhood poverty		0.97 (0.91, 1.04)	0.97 (0.91, 1.04)	0.98 (0.91, 1.04)
SD of neighborhood cohesion			0.98 (0.93, 1.04)	0.98 (0.93, 1.04)

*Models adjusted for age, sex, and site

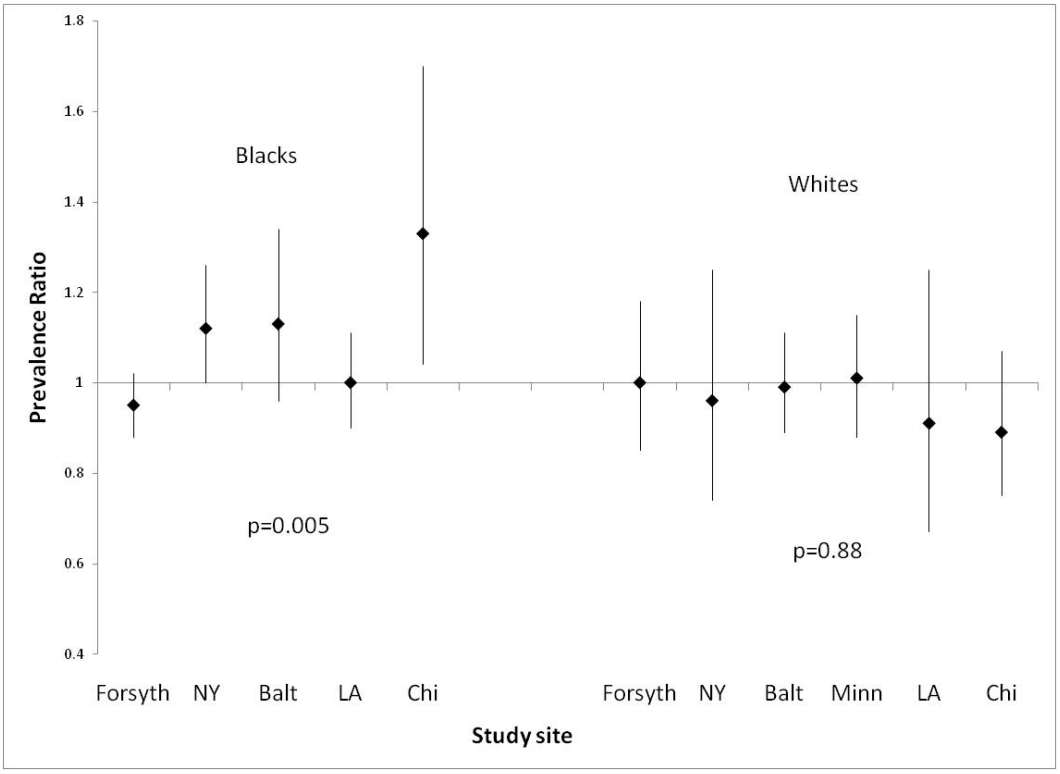
**Models adjusted for age, sex, site, current cigarette smoking, current alcohol use, intentional exercise and body mass index

SD = standard deviation

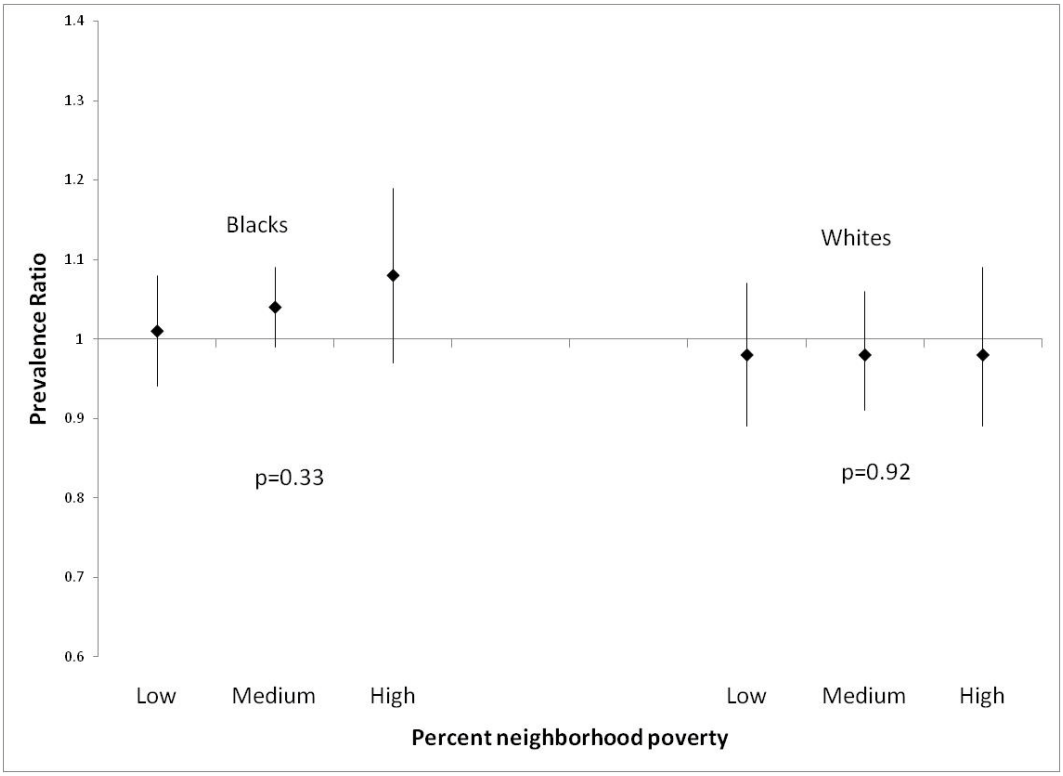
There was statistical evidence among Blacks of variation in the association between neighborhood segregation and hypertension prevalence by study site (p=0.005). The site variation (Figure 4.1a) suggested that neighborhood segregation was associated with increased hypertension prevalence among Blacks living in New York City (demographics-, individual SEP-, and neighborhood covariate-adjusted PR: 1.12; 95% CI: 1.00, 1.26), Baltimore (PR: 1.13; 95% CI: 0.96, 1.34), and Chicago (PR: 1.33; 95% CI: 1.04, 1.70), whereas the association was null in Los Angeles (PR: 1.00; 95% CI: 0.90, 1.11) and somewhat protective in Forsyth County (PR: 0.95; 95% CI: 0.88, 1.02). Though the heterogeneity was not statistically significant (p=0.33), among Blacks the association between segregation and hypertension prevalence became stronger with increasing neighborhood poverty (Figure 4.1b), ranging from a prevalence ratio of 1.01 (95% CI: 0.94, 1.08) in low poverty neighborhoods (10^{th} percentile) to 1.08 (95% CI: 0.97, 1.19) in high poverty neighborhoods (90^{th} percentile). There was no evidence of variation in the association between segregation and hypertension by site or neighborhood poverty among Whites (Figure 4.1), and there was no variation by neighborhood cohesion, income or education for either group (not shown, all p-values ≥ 0.37).

Figure 4.1: Variation in the association between neighborhood segregation and hypertension prevalence by study site (A) and neighborhood poverty (B).
Models are adjusted for segregation, age, sex, site, individual SEP, neighborhood poverty, neighborhood social cohesion, and the interaction between segregation and the area-level characteristic. P-values represent the overall interaction between neighborhood segregation and the area-level characteristic.

A



B



Hypertension incidence

Over the roughly 5-year follow-up period, 243 of the 643 non-hypertensive Blacks and 339 of the 1418 Whites became hypertensive. After adjusting for demographics and individual SEP, results suggested a positive association between neighborhood segregation and hypertension risk for Blacks (Table 4.5; HR: 1.12; 95% CI: 0.96, 1.30), but an inverse association for Whites (Table 4.6; HR: 0.89; 95% CI: 0.78, 1.01). These associations were attenuated after adjustment for neighborhood poverty (Blacks, HR: 1.08; 95% CI: 0.92, 1.27; Whites, HR: 0.93; 95% CI: 0.78, 1.11). The association between neighborhood poverty and hypertension incidence was not statistically significant, but the magnitude of the point estimates suggest that increasing neighborhood poverty was linked to increased hypertension risk (HR=1.11 for Blacks; HR=1.06 for Whites).

Table 4.5: Hazard ratios for hypertension incidence among Blacks by local segregation and selected covariates

	Model 1*		Model 2*		Model 3*		Model 4**	
SD of segregation	1.12	(0.96, 1.30)	1.08	(0.92, 1.27)	1.08	(0.92, 1.27)	1.09	(0.93, 1.28)
Education								
Less than HS	1.97	(1.18, 3.30)	1.90	(1.13, 3.19)	1.90	(1.13, 3.20)	2.08	(1.23, 3.52)
HS	1.33	(0.87, 2.02)	1.31	(0.86, 2.00)	1.32	(0.87, 2.01)	1.15	(0.75, 1.76)
Some college/technical school	1.44	(1.05, 1.98)	1.44	(1.05, 1.97)	1.44	(1.05, 1.97)	1.31	(0.96, 1.80)
Bachelors or more	1.00	(ref)	1.00	(ref)	1.00	(ref)	1.00	(ref)
Income								
<$15,999	0.77	(0.46, 1.29)	0.73	(0.43, 1.23)	0.73	(0.44, 1.24)	0.68	(0.40, 1.17)
$16,000 - $34,999	1.07	(0.72, 1.59)	1.03	(0.69, 1.54)	1.04	(0.70, 1.56)	1.01	(0.67, 1.52)
$35,000 - $74,999	0.92	(0.65, 1.30)	0.91	(0.64, 1.28)	0.92	(0.65, 1.30)	0.9	(0.64, 1.28)
> $74,999	1.00	(ref)	1.00	(ref)	1.00	(ref)	1.00	(ref)
SD of neighborhood poverty			1.11	(0.95, 1.30)	1.13	(0.95, 1.34)	1.10	(0.93, 1.30)
SD of neighborhood cohesion					1.05	(0.91, 1.20)	1.05	(0.92, 1.21)

*Models adjusted for age, sex, and site

**Models adjusted for age, sex, site, current cigarette smoking, current alcohol use, intentional exercise and body mass index
SD = Standard deviation

Table 4.6: Hazard ratios for hypertension incidence among Whites by local segregation and selected covariates

	Model 1*		Model 2*		Model 3*		Model 4**	
SD of segregation	0.89	(0.78, 1.01)	0.93	(0.78, 1.11)	0.93	(0.78, 1.11)	0.96	(0.81, 1.14)
Education								
Less than HS	1.63	(0.96, 2.75)	1.63	(0.96, 2.75)	1.62	(0.96, 2.74)	1.52	(0.89, 2.60)
HS	1.17	(0.84, 1.63)	1.18	(0.85, 1.64)	1.16	(0.83, 1.62)	1.11	(0.79, 1.55)
Some college/technical school	0.94	(0.71, 1.24)	0.94	(0.71, 1.24)	0.93	(0.71, 1.24)	0.94	(0.71, 1.25)
Bachelors or more	1.00	(ref)	1.00	(ref)	1.00	(ref)	1.00	(ref)
Income								
<$15,999	0.86	(0.51, 1.45)	0.84	(0.50, 1.42)	0.83	(0.49, 1.40)	0.75	(0.44, 1.27)
$16,000 - $34,999	1.36	(0.97, 1.91)	1.37	(0.97, 1.92)	1.36	(0.97, 1.91)	1.32	(0.94, 1.86)
$35,000 - $74,999	1.18	(0.89, 1.55)	1.18	(0.89, 1.55)	1.17	(0.89, 1.54)	1.13	(0.86, 1.50)
> $74,999	1.00	(ref)	1.00	(ref)	1.00	(ref)	1.00	(ref)
SD of neighborhood poverty			1.06	(0.92, 1.22)	1.06	(0.92, 1.22)	1.08	(0.94, 1.24)
SD of neighborhood cohesion					0.96	(0.85, 1.07)	0.96	(0.85, 1.07)

*Models adjusted for age, sex, and site

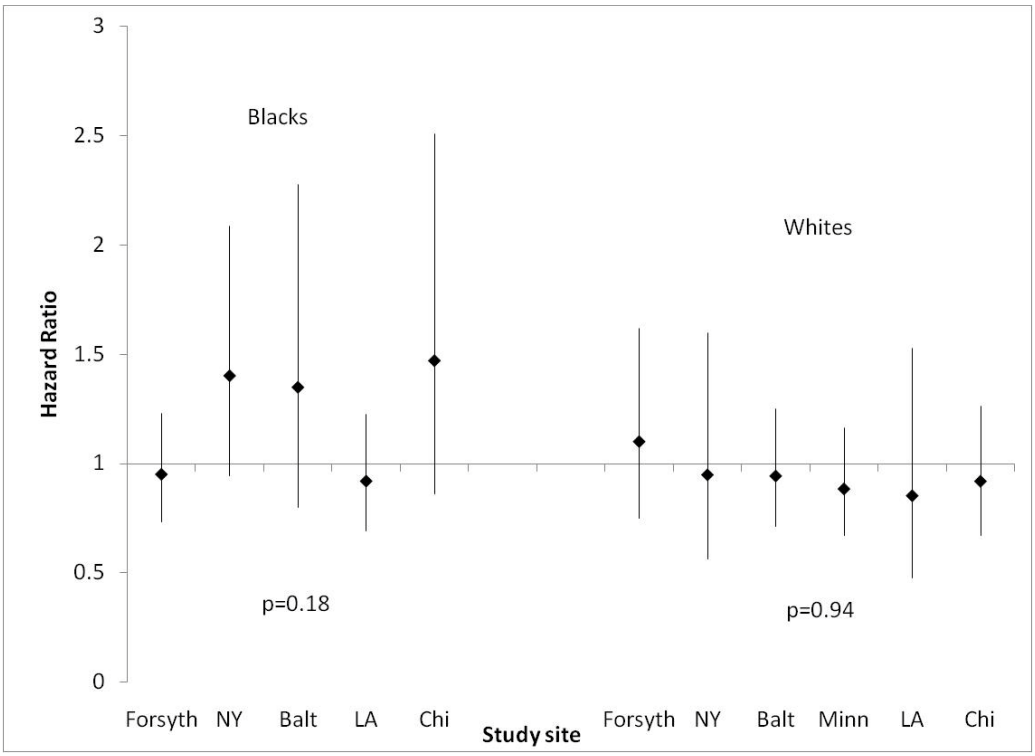
**Models adjusted for age, sex, site, current cigarette smoking, current alcohol use, intentional exercise and body mass index
SD = Standard deviation

There was no statistical evidence of variation in the association between neighborhood segregation and incident hypertension among Blacks (Figure 4.2). However, stratified analyses showed that heterogeneities by site (Figure 4.2a) and poverty (Figure 4.2b) for Blacks had similar patterns as seen in the prevalence analyses: stronger associations in Baltimore, New York City, and Chicago than in the other sites and weakest association in lowest poverty areas. There was no variation in the relationship between segregation and hypertension incidence by site or neighborhood poverty among Whites, but there was by level of neighborhood social cohesion (p=0.05), though not in the expected direction. The association between neighborhood segregation and hypertension incidence became less protective with increasing neighborhood social cohesion (Figure 4.2c), ranging from risk ratios of 0.83 (95% CI: 0.67, 1.02) in the less cohesive neighborhoods (10^{th} percentile) to 1.10 (95% CI: 0.87, 1.39) in the more cohesive areas (90^{th} percentile). A similar pattern was seen for Blacks but the heterogeneity was not statistically significant.

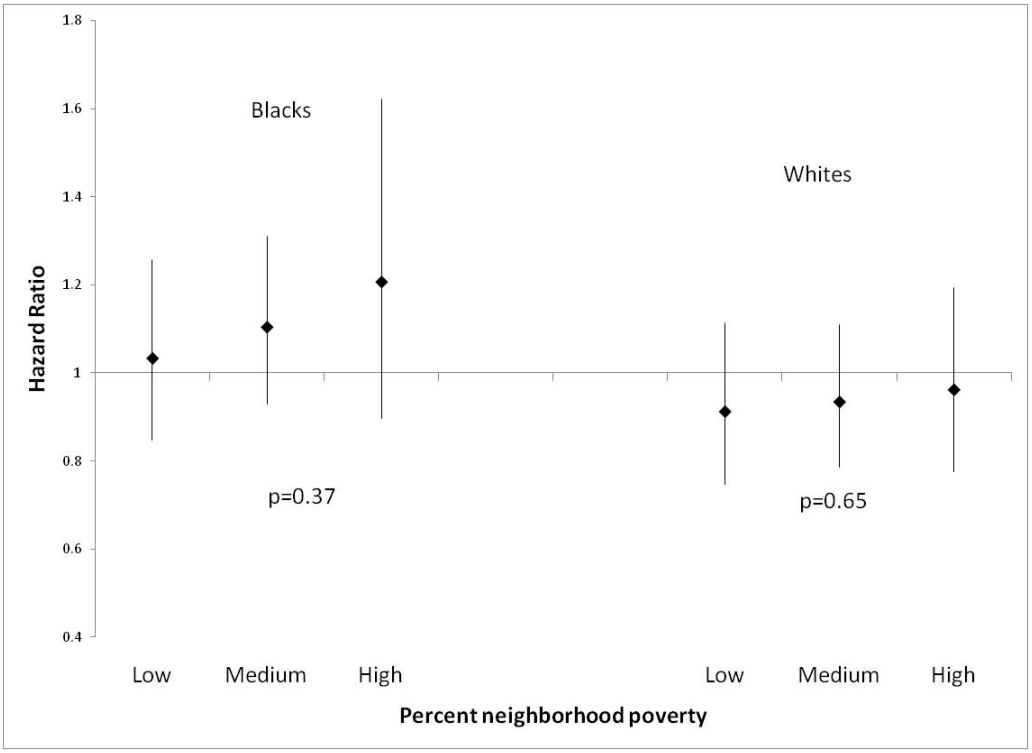
Figure 4.2: Variation in the association between neighborhood segregation and hypertension incidence by study site (A), neighborhood poverty (B), and neighborhood social cohesion (C).

Models are adjusted for segregation, age, sex, site, individual SEP, neighborhood poverty, neighborhood social cohesion, and the interaction between segregation and the area-level characteristic. P-values represent the overall interaction between neighborhood segregation and the area-level characteristic.

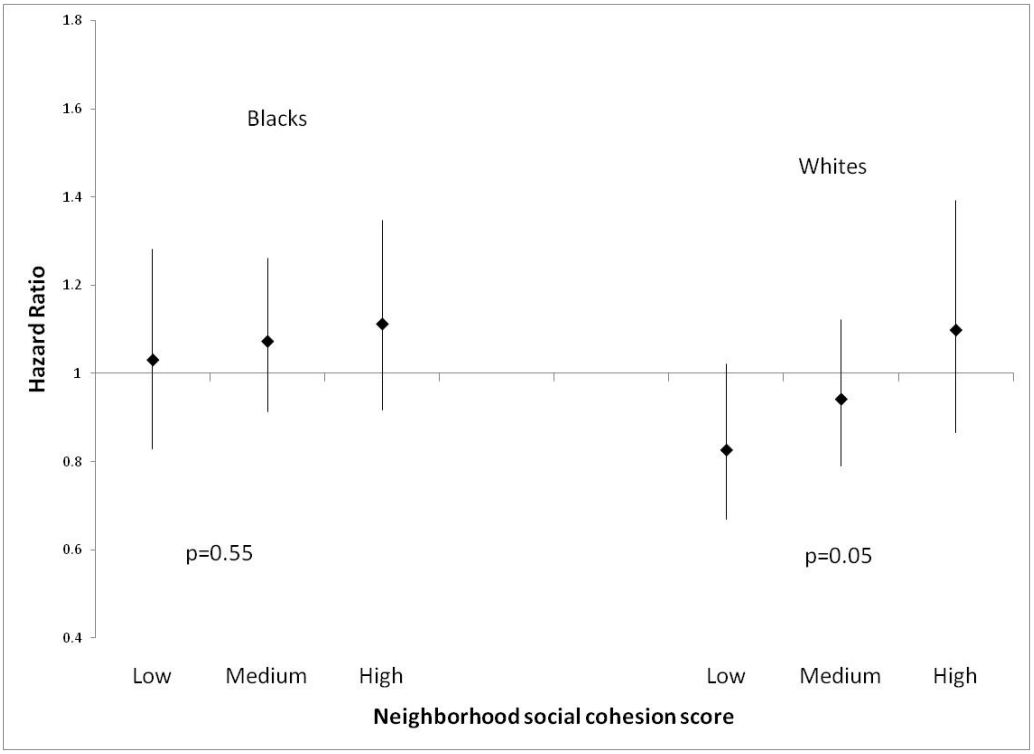
A



B


1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
Hazard Ratio
Blacks
Whites
p=0.37
p=0.65
Low
Medium
High
Low
Medium
High
Percent neighborhood poverty

C



Discussion

This study builds on the current literature by examining how the association between segregation and hypertension is modified by other area- and individual-level factors. No statistically significant associations were found between segregation and hypertension for Blacks or Whites. However, among Blacks, there was significant variation in the association between segregation and hypertension prevalence by site. Similar heterogeneity by site was also observed for hypertension incidence, although it was not statistically significant. Although trends were not statistically significant, there was also some evidence that among Blacks the association between segregation and hypertension prevalence or incidence was only present among those living in higher poverty neighborhoods. Among Whites, there was statistically significant variation in the association between segregation and incident hypertension by level of

neighborhood cohesion such that segregation was only protective for those living in less cohesive neighborhoods.

Unlike the majority of other studies on neighborhood segregation and health, this study was able to examine heterogeneity in the association across several sites. Results showed that the relationship between segregation and hypertension was not the same in every metropolitan area. The sites differed substantially in terms of levels of segregation, but there did not appear to be a relationship between the level of segregation and the strength of association between segregation and hypertension. For example, even though segregation was highest in Los Angeles, it was the site where the relationship between segregation and hypertension was weakest. The variation in the association between segregation and hypertension by site among Blacks suggests there may be contextual differences across these places that explain the impact of segregation. Models were adjusted for neighborhood poverty and social cohesion, but there may be other area-level factors associated with segregation that are leading to these site differences, such as differential distribution of resources (e.g. supermarkets) or the way in which these metropolitan areas are governed (e.g. metropolitan governance versus municipal fragmentation) (58, 59, 60). Future studies will try and determine what specific contextual factors may lead to differences across metropolitan areas.

The finding that the segregation-hypertension association varied by level of neighborhood poverty for Blacks suggests that the distribution of area-level resources is important in determining how segregation impacts health. Low neighborhood poverty is linked to lower hypertension (56), and it may serve as a buffer for the adverse consequences of segregation.

It is not clear why segregation was protective among Whites living in less versus more socially cohesive neighborhoods. Neighborhood social cohesion has been linked to concentrated neighborhood disadvantage (135), and it is possible that the protective relationship between segregation and hypertension only holds for Whites living in more

disadvantaged areas. Heterogeneity in the association between segregation and hypertension was not observed across categories of neighborhood poverty, but it may be that among Whites, neighborhood cohesion is an important marker of area resources or the ability to influence those resources. It is plausible that among Whites living in areas with low cohesion, those living in segregated white areas are less likely to experience material and psychosocial deprivation, and hence are more protected from the development of hypertension. Additional work will need to be done to better understand why greater segregation is protective for Whites living in less cohesive areas.

No evidence was found of interactions with individual-level income or education. Several studies of neighborhood effects on CVD risk indicate that the characteristics of the neighborhood can impact health independent of individual-level SEP (52, 53, 54, 56). Thus, it may be that the neighborhood environment, particularly the social and economic resources available, may be a more important determinant of the impact of segregation on health than an individual's socioeconomic attainment.

There are very few studies of neighborhood-level segregation and cardiovascular disease-related outcomes, and findings are mixed. A study of neighborhood segregation and CVD mortality in New York City found that segregation was associated with decreased coronary heart disease mortality among Whites and older Blacks after adjusting for area-level socio-demographic variables including percent high school education and percent unemployment (70). Segregation was unassociated with coronary heart disease mortality among younger Blacks. In the only study found of local segregation and hypertension, researchers found a significant protective association between local segregation and self-reported high blood pressure among older foreign-born Blacks but no association among US-born or younger foreign-born Blacks living in New York City (69). A national study found that neighborhood proportion Black was associated with increased BMI among men but not women (140). None of these studies assessed variation in the association between segregation and CVD risk by levels of

other neighborhood- and individual-level characteristics, though, which may explain these inconsistent results.

Although very few studies of segregation and health assess heterogeneity by area- or individual-level characteristics, there is some evidence supporting these findings. A multi-site study of neighborhood racial/ethnic composition and homicide among Blacks and Hispanics found substantial variation in the association by city of residence (57). In addition, they found that the extent to which neighborhood disadvantage accounted for the composition-homicide association varied by place of residence. A study of racial composition and all-cause mortality among Blacks living in Philadelphia showed that those living in predominantly black neighborhoods had lower mortality rates than those living in more integrated neighborhoods (126). They also found this protective association varied by level of social capital; among those living in predominantly black neighborhoods, rates were much lower for those living in areas with higher versus lower social capital. No evidence was found of variation by neighborhood cohesion, but it is possible that this characteristic is more important for certain types of conditions or in certain cities. For example, in the sites studied it may be that neighborhood poverty is a more important determinant of the nature of the relationship between segregation and hypertension than social cohesion.

This study is not without limitations. Overall, the evidence of effect modification was weak. It is likely that some of the characteristics examined in this study do not modify the association between segregation and hypertension. The high prevalence of hypertension in the sample, particularly of Blacks, limited the sample size available for incidence analyses and reduced the power to detect meaningful interactions. Another limitation is the representativeness of the data. This sample is not nationally representative nor is it necessarily representative of the MSAs from which study participants were sampled which may limit generalizability.

The association between neighborhood segregation and hypertension may depend on where one lives. While not necessarily protective, neighborhood

characteristics like low neighborhood poverty may mitigate the potentially harmful effects of local segregation among Blacks. For Whites segregation may be protective only for those living in disadvantaged neighborhoods, such as those that are less cohesive. This study provides some evidence that segregation is not universally harmful or helpful, but that it depends on the distribution of social and physical resources that exist in the communities. The relationship between neighborhood segregation and disease is complex, and determining how various elements interact with each other to contribute to adverse health outcomes may help reduce health disparities.

Chapter 5:
Conclusion

Summary of findings

This dissertation assessed the role of the geographic context in racial disparities in hypertension. Specifically, it (1) examined regional and large area geographic factors related to differences in hypertension among and between Blacks and Whites; (2) evaluated metropolitan-level racial residential segregation as a contributor to black-white differences in hypertension prevalence; and (3) investigated the association between neighborhood-level racial segregation and hypertension among Blacks and Whites.

The major finding of this dissertation is that place – be it region of the country, metropolitan statistical area (MSA), or neighborhood – is important for uncovering the mechanisms behind racial disparities in hypertension. Analyses using data from black and white participants of the Multi-Ethnic Study of Atherosclerosis (MESA) showed those born in the South and those living in MSAs located in the South had higher hypertension prevalence than those living in other parts of the country (Chapter 2). Living in the South may influence health through differences in the neighborhood environment, diet, physical activity levels, or socioeconomic opportunities. In addition, the magnitude of the racial disparity in hypertension varied depending on which metropolitan areas were compared. The disparity between Blacks and Whites living in Chicago was quite large, whereas the difference between Blacks living in Chicago and Whites living in Forsyth County, NC was smaller and not statistically significant. This suggests these disparities are not immutable. It also shows that the conditions under which individuals live may play a significant role in contributing to the unequal burden of hypertension in the US.

A deeper investigation of the role of the social environment highlighted the importance of racial residential segregation as a driver of variation in hypertension within race groups and in the disparity between Blacks and Whites (Chapters 3 and 4). The institutional discrimination that segregates Blacks from Whites not only isolates Blacks from Whites. It also concentrates poverty in predominantly black neighborhoods and isolates residents from opportunities for socioeconomic mobility and political capital. It also increases exposure to health-harming characteristics of the neighborhood environment like fast food restaurants, liquor stores, and airborne particulate matter.

Using data from the US Census and the National Health and Nutrition Examination Surveys (NHANES) this dissertation showed that segregation may contribute to hypertension disparities. Living in a segregated MSA was associated with higher hypertension prevalence for Blacks but lower hypertension prevalence for Whites. As seen with the more general assessment of geographic patterning of hypertension, the magnitude of the racial disparity in hypertension varied by place of residence. The disparity was smallest among those living under the most similar conditions, in this case low levels of black isolation and higher levels of neighborhood poverty.

Segregation is often measured at the MSA level, but there is also some important heterogeneity in the spatial distribution of race groups within MSAs. An examination of local neighborhood-level segregation and hypertension using data on black and white MESA participants found no statistically significant association between segregation and hypertension among Blacks or Whites after adjusting for individual SEP and neighborhood characteristics. However, there was evidence this association varied by level of other area-level characteristics. Among Blacks, the association between segregation and hypertension varied by study site; segregation was harmful among those living in Chicago and New York City while somewhat protective among those in Forsyth County. There was also evidence to suggest that segregation was only associated with increased hypertension among Blacks living in high poverty

neighborhoods, though this was not statistically significant. For Whites, segregation was protective against hypertension incidence for those living in less socially cohesive neighborhoods. These findings suggest differential neighborhood conditions, particularly the resources available, may be the more important contributors to the impact of racial segregation on health than the actual demographic composition.

Contributions to the literature

This dissertation addressed several gaps in the current literature on area-level effects on hypertension. Few studies on the contribution of place to health disparities in hypertension have focused on geographic variation within race groups. Uncovering the mechanisms underlying within-group variation may help elucidate the particular social and environmental factors that contribute to hypertension disparities and highlight the aspects that can be targeted for interventions. Region of birth and place of residence were both found to be sources of variation within groups, and these differences were partially explained for Blacks by aspects of the neighborhood physical environment.

Racial residential segregation was also shown to contribute to within-group heterogeneity. Findings from Chapter 3 suggest that living in metropolitan areas where Blacks are isolated from Whites was associated with increased hypertension among Blacks and decreased hypertension among Whites. Chapter 4 results suggest that there is variation in the way neighborhood-level segregation impacts hypertension among Blacks by metropolitan area of residence and level of neighborhood poverty, and among Whites by level of neighborhood cohesion. There may be contextual differences that make neighborhood-level segregation associated with increased hypertension among Blacks in Chicago, New York City, and Baltimore but decreased hypertension in Forsyth. In addition, high neighborhood poverty may make Blacks more vulnerable to the ill effects of living in segregated areas and that segregation may buffer Whites against adverse health in areas with low neighborhood cohesion.

This dissertation also contributed to studies of the influence of area-level factors on black-white disparities in hypertension by assessing the impact of more macro-level

factors like city/MSA of residence or metropolitan-level racial residential segregation. The key finding was that race differences are not invariant. An analysis of MESA data in Chapter 2 showed that hypertension prevalence was 82% higher among Blacks living in Forsyth County, NC compared with Whites living in Chicago, but that there was only a 13% difference when Blacks living in Chicago were compared with Whites living in Forsyth. Chapter 4 results showed that black-white differences ranged from a low odds ratio of 1.24 to a high of 4.14 depending on level of residential segregation and neighborhood poverty. These suggest that these differences result not from innate differences but from environmental characteristics.

Limitations

Cross-sectional study design

The majority of the studies in this dissertation were cross-sectional and cannot be used to show a causal link between place and hypertension. There is no way to be certain whether or not being sick influenced their decision to live in a certain neighborhood or metropolitan area. Hypertension is typically asymptomatic, but it is possible that associated conditions like cardiovascular disease or high BMI influenced an individual's selection into a neighborhood. The residential history data in MESA suggested that area-level variation in hypertension reflected long-term exposures (i.e. the majority of participants did not move much in the 20 years before baseline); data of this nature were not available in NHANES. Future research should use longitudinal data to assess the effects of place of residence and residential segregation on hypertension risk.

Neighborhoods change over time and people move over time. The studies in this dissertation only assess the neighborhood environment at one point in time, and they generally do not account for the characteristics of the different neighborhoods study participants were exposed to over the life course. Few studies have addressed these issues, but there is some evidence to suggest that single-point-in-time measures of neighborhood exposures may underestimate the true impact of the residential environment on CVD risk (141).

Generalizability

One asset of MESA is that it studies CVD risk in adults living in multiple sites across the country. However, the study is not nationally representative and findings may not be generalizable to other parts of the US. In addition, because MESA participants must be free of clinical CVD at baseline, they may not be representative of the general population of adults aged 45-84 years living in the sites from which they were sampled. In addition, those with the most severe hypertension may not be eligible for participation in MESA since they may be more likely to have had a clinical cardiovascular event. NHANES results are more generalizable than MESA; it is designed to be representative of the US civilian, non-institutionalized population.

Measurement of the geographic context

Several aspects of the residential environment have been shown to be associated with hypertension including neighborhood-level SEP, healthy food availability, neighborhood cohesion, walkability, and safety (39, 40, 56). All of these measures were available in MESA but only neighborhood SEP was available in NHANES. In all of the studies of this dissertation, adjustment for the available neighborhood characteristics failed to fully explain associations between more macro-level measures of place and hypertension (i.e. region of birth, place of residence, or metropolitan-level residential segregation). This may indicate that there are unmeasured aspects of the neighborhood environment or individual SEP that would have explained area-level variation in hypertension (e.g. other measures of the childhood socioeconomic environment). There may also be contextual effects at the metropolitan level that influence hypertension independent of neighborhood and individual characteristics. Future work needs to be done to uncover the pathways through which metropolitan level characteristics influence hypertension and CVD risk.

Implications

The findings in this dissertation show that place and context may play an important role in explaining hypertension disparities. Solutions to health disparities

must involve addressing area-level factors that increase the unequal burden of disease in Blacks versus Whites and lower SEP versus higher SEP individuals. In particular, eliminating the inequitable distribution of social and economic resources associated with racial residential segregation may be a crucial step needed to reduce racial disparities in socioeconomic circumstances and cardiovascular disease risk.

Despite decades of research supporting the theory that the processes leading to racial residential segregation have long-term adverse socioeconomic and health-related consequences for Blacks, only one coordinated policy initiative has been introduced to address the issue (61). The Fair Housing Act of 1968 arguably sparked a decline in racial residential segregation between 1970 and 2000 (142, 143, 144), but Blacks still remain the most segregated minority group in the United States (61). Intervention research on housing or neighborhood choice and health show that individuals who are placed in more racially integrated, lower poverty neighborhoods generally have better outcomes (145, 146, 147), but there are few examples of these types of moving opportunities for research to draw from.

Another potential approach to addressing the role of residential segregation in health disparities is by changing the way in which metropolitan areas are run. Areas characterized by regional or metropolitan governance (as opposed to more fragmented forms of governance) have been found to have lower rates of mortality among African Americans and lower levels of racial segregation (148). Thus, policies that promote metropolitan governance may be another way to reduce residential segregation and its negative impact on health disparities.

Other approaches that might help address the ill effects of residential segregation involve intervening on any of the potential pathways through which either segregation is believed to influence health or the impact of segregation on health is shown to vary. The study conducted in Chapter 3 did not find evidence that neighborhood poverty mediated the association between segregation and hypertension, but it is possible that other neighborhood characteristics like the presence of supermarkets or levels of neighborhood safety are mediators. Improving

mediating neighborhood characteristics may an effective solution to the problem of racial segregation and its influence on health.

As suggested in Chapter 4, there may be heterogeneity in the impact of segregation on hypertension by other area-level characteristics. Identifying conditions under which segregation is protective or at the very least not health-harming may aid in the development of effective intervention strategies that do not involve moving individuals to different neighborhoods. This dissertation found some evidence of modification by site and neighborhood poverty (though not statistically significant), but it is possible that there are area-level factors which may impact the relationship between segregation and health. No heterogeneity was found by level of neighborhood cohesion among Blacks, but it is possible that increasing other aspects of social capital may act as a buffer against the negative impacts of segregation on health. It may also be that neighborhood cohesion may produce heterogeneity in the effect of segregation on other health outcomes (126). More research needs to be done to identify conditions that modify the association between segregation and health to better inform area-level interventions.

This dissertation showed that hypertension varied within race groups by region of birth, metropolitan area of residence, and neighborhood. It also identified metropolitan-level racial residential segregation as an important contributor to hypertension disparities between Blacks and Whites. Future research will explore in more detail how segregation influences health and health disparities. Other health outcomes will be examined along with other measures of segregation and potential sources of heterogeneity in the relationship between segregation and health. This dissertation also highlights the need for cross-disciplinary collaborations, particularly with urban planners, sociologists, politicians, and community activists, to better understand the process through which the geographic context leads to disparities and the steps needed to intervene and reduce them.

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