Interaction of social factors and environmental pollutants in black-white health disparities: The case of lead and hypertension

by

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For Maia Eriko,

Thank you for letting me work.

Now it’s time to play!

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CHAPTER ONE

INTRODUCTION AND OVERVIEW

Black-white disparities in hypertension

Hypertension is one of the most common forms of cardiovascular disease in the United States (US), with recent age-adjusted prevalence at about 29% for both men and women (Lloyd-Jones et al., 2009). Hypertension increases the risk of stroke, kidney failure, and other forms of cardiovascular disease, the latter of which is the number one cause of mortality in this country (Heron et al., 2009). However, these prevalence estimates mask the reality that hypertension is not distributed equally across social groups, and black-white disparities in hypertension are particularly large and persistent. The recent age-standardized hypertension prevalence is at roughly 43% for black adults, while the it is roughly 33% for white adults (Lloyd-Jones et al., 2009). The disparity is particularly large between black and white women, as they experience the highest and lowest prevalence rates, respectively, not only in the US, but the world (Ong, Cheung, Man, Lau, & Lam, 2007; Wolf-Maier et al., 2003).

The causes of hypertension are not completely understood, although there are well-known individual-level risk factors such as smoking and obesity. However, these

risk factors fail to explain the black-white *disparities* in hypertension. For example, although black women have higher rates of obesity and poverty compared to white women, these factors explain only a small portion of the hypertension disparity (Flegal, Carroll, Ogden, & Curtin, 2010; Geronimus, Bound, Keene, & Hicken, 2007; Current Population Survey, US Census Bureau, 2009). Furthermore, it is not likely that smoking accounts for the hypertension disparities given that black women have lower rates of smoking than white women. (Centers for Disease Control and Prevention, 2008)

Genetic differences between black and white Americans have also been proposed to explain the hypertension disparities (Ferdinand & Ferdinand, 2008; A. L. Taylor et al., 2005). However, racial categories have been shown to be socially constructed and fluid over time rather than biologically-based taxonomic groups (Kittles & Weiss, 2003; Williams, 1997). The social nature of race becomes clearer when comparing phenotypically "black" populations in different sociopolitical environments. If race were based solely on genetic ancestry, then one would expect diverse black populations to have similar rates of hypertension, and that all of these groups would have higher rates than white Americans. Yet, West African and foreign-born black Americans have rates of hypertension that are similar to those of White Americans. US-born black adults stand apart as having higher rates than all of these other groups (R. S. Cooper & Rotimi, 1997; R. S. Cooper et al., 1997; Hicks, Fairchild, Cook, & Ayanian, 2003; Mensah, Mokdad, Ford, Greenlund, & Croft, 2005; Read & Emerson, 2005). The sequencing of the human genome and advances in molecular biology techniques have provided the opportunity to test these genetic hypotheses more directly, but analyses have failed to produce any "uniformly detectable effects on the predisposition to hypertension" that differ along

racial lines (Kardia et al., 2003; Kittles & Weiss, 2003; Province et al., 2003, p. 145; Thiel et al., 2003).

The role of social and physical environments in hypertension disparities

If not individual-level health behaviors, poverty, or genetic predisposition, what could explain the excess risk of hypertension among black Americans? The unequal social and physical environments experienced by black and white Americans contribute substantially to racial disparities in health, including hypertension (R. S. Cooper, 1993; S. A. James, Hartnett, & Kalsbeek, 1983; Lillie-Blanton & Laveist, 1996; Morello-Frosch & Lopez, 2006; Williams & Collins, 2001; Yen & Syme, 1999). For example, in a study conducted in Chicago, black adults experience nearly double the odds of hypertension compared to white adults. However, this disparity is completely attenuated when accounting for residential environment, which can be thought of as a composite of many aspects of the social and physical environments (Morenoff et al., 2007).

Moreover, recent research suggests that there may be interactions between the social and physical environments such that the social environment increases the harmful effects of environmental pollutants on health (Clougherty et al., 2007; Glass et al., 2009; Peters et al., 2007). For example, traffic-related pollution has been shown to be associated with a 50% increase in the odds of asthma in children, but only for those whose parents report high levels of stress (Shankardass et al., 2009). While the literature on racial disparities in health has focused separately on the social and physical environments, researchers have called for the integration of these fields to examine the notion that social factors increase susceptibility to the harmful effects of environmental

pollutants (Clougherty & Kubzansky, 2009; Evans & Pilyoung, 2010; Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006). I examine the role of such an interaction with regard to black-white disparities in hypertension. To accomplish this, I take advantage of an unexplained racial pattern in the effect of lead on blood pressure.

Susceptibility to the effect of lead and black-white disparities in hypertension

Lead has been causally related to blood pressure[1] increase and risk of hypertension in a dose-dependent manner (Cheng et al., 2001; Navas-Acien, Guallar, Silbergeld, & Rothenberg, 2007; Vaziri, 2008). Following the legislation in the 1980s governing lead use, lead exposure has decreased substantially in the general US population (Muntner, Menke, DeSalvo, Rabito, & Batuman, 2005; Pirkle et al., 1998). For example, in the NHANES II (1976-1980), unadjusted mean blood lead levels for black and white women were 13.2 $^{\mu g}/_{dl}$ and 12.1 $^{\mu g}/_{dl}$, respectively (Sorel et al., 1991). In that sample, blood lead was associated with blood pressure for *both* black and white women (Harlan, 1988).

More than a decade later, in the NHANES III (1988-1994), unadjusted mean blood lead levels had dropped to 2.3 $^{\mu g}/_{dl}$ and 2.1 $^{\mu g}/_{dl}$ for black and white women, respectively (Den Hond, Nawrot, & Staessen, 2002). These more recent blood lead levels are markedly lower for both black *and* white women. Yet, a curious finding is that the blood lead–blood pressure association was not present for white women but remained large and statistically-significant for black women (Den Hond et al., 2002;

[1] Although hypertension is the disease state measured and discussed when describing health, it represents an arbitrary threshold risk of cardiovascular, cerebrovascular and metabolic morbidity and mortality; this risk occurs along a continuum of blood pressure (Britton, Gaziano, & Djousse, 2009; Franklin et al., 2001; Vasan et al., 2001). Therefore, blood pressure, as a continuous variable, is an important marker of cardiovascular health related to hypertension.

Muntner et al., 2005; Vupputuri et al., 2003). What might explain this finding? One possibility is that at low blood lead levels, lead has no influence on blood pressure except in the presence of a susceptibility factor. If this argument is true, then the reason why lead is related to blood pressure among black but not white adults is due to the presence of some other factor. I hypothesize that this "other factor" is the poor social environment experienced by black adults. Specifically, I hypothesize that factors from the poorer social environments experienced by black compared to white adults increase their susceptibility to the harmful effects of lead on blood pressure. This results in the positive effect of blood lead on blood pressure among black but not white adults.

Although no one has yet examined the interaction of lead and social factors with regard to black-white health *disparities*, there is some literature on the interaction with regard to health *in general*. For example, the effect of lead on cognitive functioning has been shown to be stronger for those who live in residential areas characterized by high levels of psychosocial hazards (e.g., number of vacant houses, number of liquor stores, etc) or those who report higher levels of perceived stress compared to those who live in residential areas characterized by low levels of psychosocial hazards or who report low levels of perceived stress, respectively (Glass et al., 2009; Peters et al., 2008). Furthermore, the effect of lead on blood pressure has been shown to be present only for men who report high levels of perceived stress and not for those who report low levels of stress (Peters et al., 2007).

Both lead and stress act on the same biological stress systems and the same cardiovascular tissues (Harrison & Gongora, 2009; Pajovic, Radojcic, & Kanazir, 2008; Vaziri, 2008; Virgolini, Chen, Weston, Bauter, & Cory-Slechta, 2005). It may be that

lead and stress interact biologically. This is suggested by the finding that adults who have higher allostatic load scores, a biomarker composite of chronic stress, have a stronger association between blood lead and hypertension compared to those with lower allostatic load scores (Zota, Shenassa, & Morello-Frosch, 2010).

Organization of this document

I describe my research in the following five chapters. Chapter Two contains a discussion on the way in which poor social environments are sources of stressors and constraints with few resources that result in psychosocial stress. This discussion includes a description of my conceptual model, which emphasizes that disparities in health arise not only from the main effects of exposure to stress or environmental pollutants but that the interaction between the two affects health (Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006; Schulz et al., 2005). I introduce the proxy measurements of social stressors and psychosocial stress measured in National Health and Nutrition Examination Survey (NHANES), the dataset I use: educational attainment, family poverty-to-income ratio (PIR or poverty status), and depressive symptoms (Lillie-Blanton & Laveist, 1996; Mirowsky & Ross, 2003). The discussion of each of these factors includes the theoretical and empirical rationale for their use. Finally, I outline my research questions.

Chapter Three contains a description of my research questions and methods. Overall, I hypothesize that stress, broadly defined, increases the harmful effects of low lead levels on blood pressure. Specifically, I hypothesize that the lead–blood pressure association in black but not white adults is only present at low levels of education, high

levels of poverty, and high levels of depressive symptoms. I hypothesize that this interaction between these markers and lead is significant beyond their individual main effects. To test these hypotheses, statistical interactions within standard linear and logistic regression models are used with data from the most recent waves of the NHANES (2001-2006), a nationally-representative, population-based survey of health in the US.

Chapters Four and Five contain the results from all analyses. Analyses are separated based on the two different biomarkers of lead I use: (1) *blood* lead and blood pressure; and (2) *bone* lead and hypertension, the justification for which, I outline in Chapters Two and Three (Cheng et al., 2001; Navas-Acien et al., 2007). In Chapter Four, I present results for the effect of education, poverty, and depressive symptoms on the association between blood lead and blood pressure. In Chapter Five, I present the results for the effect of education and poverty on the association between bone lead and hypertension.

Finally, in Chapter Six, I synthesize my results with the literature to explain how the social and physical environments interact to produce and maintain black-white disparities in hypertension. I also discuss implications for policies, interventions, and research.

CHAPTER TWO

BACKGROUND AND SIGNIFICANCE

Black-white disparities in hypertension are significant and have been well-documented for decades (Boyle, 1970; Geronimus et al., 2007; Mensah et al., 2005). The disparities in the social and physical environments appear to be fundamental determinants of the disparities in hypertension (Gee & Payne-Sturges, 2004; Link & Phelan, 1996; Morello-Frosch & Lopez, 2006). In addition to their independent contributions to hypertension disparities, the social and physical environments may also *interact* to further contribute to the disparities.

In this chapter, I describe my conceptual model, which links race to racial disparities in hypertension through the interaction of the social and physical environments. The literature suggests that disparities in hypertension are the result of the greater social stress experienced by black compared to white adults (Williams, 1999; Williams, Neighbors, & Jackson, 2003; Wyatt et al., 2003). The literature also suggests that social stress increases the harmful effects of physical hazards on health (Clougherty et al., 2007; Glass et al., 2009; Peters et al., 2007). I combine these two lines of research to examine the notion that the greater social stress experienced by black compared to adults results in more harmful effects of physical substances on health. To test this

idea, I take advantage of an unexplained racial pattern in the harmful effects of lead on blood pressure. Specifically, lead is related to blood pressure in black but not white adutls.

My research is based on the notion that race is a socially-constructed group of categories that is fluid over time and place (Kittles & Weiss, 2003; Williams, 1997). Although racial groups are socially constructed, the meanings attached to each group determine their social, economic, political, and health realities. In a race-conscious and racially-hierarchical society such as the US, racial discrimination segregates black and white Americans into unequal social and physical environments. These environments are the conduit through which disparities in health are produced and maintained (Blank, Dabady, & Citro, 2004; Williams, 1997; Williams & Collins, 2001).

Black-white disparities in hypertension

Hypertension, defined as either a systolic blood pressure over 140mmHg or a diastolic blood pressure over 90 mmHg, is one of the most common forms of cardiovascular disease in the United States. Age-adjusted prevalence in 2005-2006 for both men and women is roughly 29% (Lloyd-Jones et al., 2009). Hypertension increases the risk of stroke, kidney failure, and other forms of cardiovascular disease, the latter of which is the number one cause of mortality in this country (Heron et al., 2009). It is estimated that in 2010, hypertension will cost $76.6 billion in direct health care and indirect productivity costs (Lloyd-Jones et al., 2009). This includes $9 billion in lost productivity due to morbidity and $12.7 billion in lost productivity due to mortality (Lloyd-Jones et al., 2009).

However, these estimates mask the reality that hypertension risk is not distributed equally across social groups; black adults have had higher rates of hypertension compared to white adults for decades (Heymsfield et al., 1977; Lennard & Glock, 1957; Mensah et al., 2005). Recent age-standardized hypertension prevalence is at roughly 43% for black adults while it is roughly 33% for white adults (Lloyd-Jones et al., 2009).

The disparities in hypertension are reflected in the larger burden in health and economic costs of hypertension carried by black compared to white Americans. For example, Figure 2.1 outlines the disparities in mortality rates due to hypertension in 2006. Mortality rates are roughly 15 deaths per 100,000 people for white men and women; the mortality rate for black women is *40* per 100,000 and is over *50* per 100,000 for black men (Lloyd-Jones, Adams et al. 2009). The disparities in hypertension account for the highest number in the disparities in years of lost life compared to *any* other health condition (Wong, Shapiro et al. 2002). Economically speaking, if black Americans had the hypertension rates of white Americans, about $400 million would be saved in out-of-pocket health care expenses, about $2 billion would be saved in private insurance costs, and $375 million would be saved from Medicare and Medicaid – *per year* (Waidmann, 2009).

Approaches to the study of black-white disparities in hypertension

The causes of the disparities in hypertension are not completely understood. Current approaches can be divided into two general categories: (1) disparities in individual-level hypertension risk factors and (2) disparities in the social and physical environments. The first set of approaches is characterized by research into disparities in

genetic composition, health behaviors, and/or socioeconomic status. Because certain factors may be important risk factors for hypertension in the general population, researchers posit that disparities in their prevalences may explain disparities in hypertension.

Some have argued that genetic differences explain the hypertension disparities. However, the social rather than biological nature of race becomes clearer when comparing phenotypically "black" populations in different sociopolitical environments. For example, Nigerian and Cameroon women have hypertension rates of 13.1% and 16.3%, respectively, which is lower than the rate of 23.9% for white American women (R. S. Cooper et al., 1997; R. S. Cooper et al., 2005). On the other hand, black American women have a substantially higher rate of 44.8% (R. S. Cooper et al., 2005). In fact, black American women have some of the highest hypertension rates in the world (R. S. Cooper et al., 2005; Wolf-Maier et al., 2003).

Even within the US, there is substantial variation in hypertension based on nativity, length of time in the US, and geographic location (Borrell, Crawford, Barrington, & Maglo, 2008; Hicks et al., 2003). For example, black adults residing in the northern US have hypertension rates of roughly 35% while black adults residing in the southern US have rates of roughly 42% (Hicks et al., 2003). In Africa, there is substantial variation in hypertension based on urban/rural residence (Kaufman, Owoaje, James, Rotimi, & Cooper, 1996). For example, hypertension rates in the 1990s for Tanzanian men and women in an urban setting was roughly 38% while rates for those in a rural setting were roughly 27% (Edwards et al., 2000). Taken together, these results

indicate that there is substantial variation in hypertension in populations of African descent and that the variation may have social determinants.

Although the literature on genetically distinct racial groups has faded into the background in recent years, the underlying notion that there are biological differences has not. Interest in these differences has renewed with the sequencing of the human genome and advances in molecular biology techniques that have provided the opportunity to test genetic hypotheses more directly (Frank, 2007; Lee, 2009). However, researchers report that there are more genetic differences between two members of the same racial group than between two members of two different racial groups (Garte, 2002; Mountain et al., 2002). Furthermore, analyses have thus far failed to produce any genes important for the development of hypertension that differ along racial lines (Kardia et al., 2003; Poston et al., 2001; Province et al., 2003; Thiel et al., 2003). In fact, socially-ascribed racial categories, particularly when interacted with SES, explain variation in blood pressure whereas genetically-determined ancestry categories do not (Gravlee, Non, & Mulligan, 2009). Thus, the research to date does not support the notion of race as a way "to provide a useful categorization of genetic information about the response to drugs, diagnosis, or causes of disease." (R. S. Cooper, Kaufman, & Ward, 2003, p. 1168).

Some argue that a higher prevalence of hypertension risk factors in black compared to white adults explains a large part of the disparities in hypertension. However, black adults generally have lower rates of obesity, smoking and alcohol use compared to white adults (Centers for Disease Control and Prevention, 2008; Geronimus, Neidert, & Bound, 1993; Jan, Marilyn, & Sonja, 2001; Serdula, Brewer, Gillespie, Denny, & Mokdad, 2004). Even when black adults have higher levels of these risk

factors, they do not explain the disparities in hypertension. For example, black women have higher rates of obesity compared to white women, but risk for obesity accounts for only a small portion of the black-white hypertension disparity in women (Flegal et al., 2010; Geronimus et al., 2007).

Finally, some regard race as merely a proxy for SES. Because socioeconomic status (SES) is negatively associated with hypertension and because black adults have lower levels of SES, it has been suggested that SES explains the disparities in hypertension (discussed in Farmer & Ferraro, 2005). However, evidence does not support this either as the disparity remains substantial after accounting for education, income, or poverty (Flegal et al., 2010; Geronimus et al., 2007; Current Population Survey, US Census Bureau, 2009).

If the hypertension disparities are not explained by genetics, health behaviors, and SES, what factors are important? A growing literature indicates that social and physical environments are important determinants of health – and the disparities in these environments are important determinants of disparities in health, including hypertension (Boardman, 2004; Dressler, 1996; Morello-Frosch & Lopez, 2006; Mujahid et al., 2008; Williams & Jackson, 2005). The social environment is the source of social stressors and psychosocial stress. Furthermore, evidence suggests that psychosocial stress may increase the harmful effects of physical hazards (Clougherty & Kubzansky, 2009; Glass et al., 2009; Peters et al., 2007). Therefore, disparities in social stressors and psychosocial stress may contribute to disparities in hypertension by increasing the harmful effects of physical hazards.

My conceptual model of black-white hypertension disparities

My conceptual model, shown in Figure 2.2, outlines the mechanisms by which race results in racial disparities in hypertension through the interaction of the social and physical environments. While similar models of health disparities exist (Adler & Ostrove, 1999; Gee & Payne-Sturges, 2004; Schulz et al., 2005), there are several unique qualities about my model. First, it combines an explicit social meaning of race with a biologically-plausible mechanism linking race to racial disparities in hypertension (Lee, 2009). Second, it incorporates a theoretically-based meaning of stress as it related to US racial categories and racial disparities in hypertension (Geronimus & Thompson, 2004; Williams, 1999). Third, it includes interactions between the social and physical environments (Clougherty & Kubzansky, 2009; Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006).

I take an *ecosocial approach* to the fundamental cause theory of social disparities in health. An ecosocial approach integrates social and biological processes, moving beyond the description of social patterns of disease to the incorporation of biological mechanisms that link these social factors to disease (Krieger, 1994, 2001). An ecosocial perspective places the biological body, along with health behaviors and other proximal health risk factors, within the context of community, larger social norms, and political and economic constraints (Yen & Syme, 1999). The *fundamental causes* theory underscores the importance of distal, structural-level factors over proximal, individual-level factors in the production and maintenance of social disparities in health, including hypertension (Link & Phelan, 1995; Link & Phelan, 1996). It is these fundamental factors that determine access to resources and exposure to constraints through which

disease is determined (Link & Phelan, 1995; Link & Phelan, 1996). While I incorporate *biological mechanisms* in my research, by integrating the fundamental causes theory, I maintain the focus of the *social causes* of disparities in hypertension.

Racial discrimination results in the segregation of black and white Americans to unequal social and physical environments (Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006; Williams & Collins, 2001). It is the inequalities in these environments that fundamentally determine the inequalities throughout my model (Link & Phelan, 1996). The poorer social and physical environments experienced by black compared to white Americans result in greater activation of the biological stress processes. This greater activation is thought to ultimately result in hypertension disparities (Adler & Rehkopf, 2008; Geronimus et al., 2007; Geronimus, Hicken, Keene, & Bound, 2006).

I divide the discussion of the social and physical environments, as they are generally addressed in separate literatures, before bringing them together to discuss how they may interact to result in hypertension disparities. First, I discuss the physical environment, with an emphasis on lead. Then, I discuss the social environment, which is the source of social stressors, resources, constraints, and psychosocial stress. I focus my discussion on the three markers related to the social environment that I use. Educational attainment and poverty status are proxies for resources and constraints. Depressive symptoms are a proxy for psychosocial stress.

A note on blood hypertension and blood pressure

Although hypertension is the disease state measured and discussed when describing health, it represents an arbitrary threshold risk of cardiovascular, cerebrovascular and metabolic morbidity and mortality. Data show that blood pressure

has a "continuous, graded, strong, independent, etiological significant relationship" (Stamler et al, 1993, p. 598) to disease (Britton et al., 2009; Stamler et al., 1993; Vasan et al., 2001). Therefore, blood pressure, as a continuous variable, is an important marker of cardiovascular health related to hypertension. I discuss both hypertension and blood pressure.

The physical environment, race, and hypertension: The case of lead

The physical environment "includes the built environment, such as age and quality of housing stock, transportation systems, and age and location of industrial activities" (Schulz et al., 2005, p. 1819). Included in this definition are the by-products from these systems and activities, such as air and noise pollution and hazardous waste. These aspects of the physical environment are important determinants of health, including hypertension (Schulz et al., 2005). I focus on one environmental pollutant, lead, and its effect on blood pressure.

Lead is a naturally occurring metal found in ore deposits – but the current lead levels in soil and water are due to human activity (US Agency for Toxic Substances and Disease Registry, 2007). Automobile exhaust from leaded gasoline was the largest source of lead until 1989 (US Agency for Toxic Substances and Disease Registry, 2007). Recent US policies, such as the prohibition of lead-containing gasoline and paint, have decreased the amount of environmental lead, yet these historical sources, coupled with current sources, such as drinking water and battery waste, still result in human exposure (Patrick, 2006; US Agency for Toxic Substances and Disease Registry, 2007). Figure 2.3

shows the dramatic decline in blood lead levels for black and white men and women over the period when major lead-containing products were removed from the market.

Even before birth, fetuses may accumulate lead from their mothers, shown by the association between blood lead from the umbilical cord and the mother (Rothenberg, Karchmer et al., 1996). Pregnancy is a time of bone remodeling to meet fetal calcium needs; lead is released from maternal bone with calcium (Rothenberg, Karchmer et al., 1996). During childhood, lead is ingested primarily through housing-related sources (e.g., water from pipes with lead solder, lead paint chips) (US Agency for Toxic Substances and Disease Registry, 2007).

Nearly the entire bodily lead burden (~94%) is found in bone, yielding a lead half-life of up to 25-30 years and making bone lead a marker of cumulative, long-term lead exposure. Although lead sequestered in bone is not readily available for transfer to other body systems where it can have its harmful effects, lead is released into the blood stream during periods of bone remodeling (e.g., aging, poor nutrition, pregnancy, lactation, or menopause), thus providing a potential continuous internal source of lead long after any environmental exposure has passed.

The remaining bodily lead is found in soft tissue and blood, with less than two percent in blood. Because nearly all blood lead is bound to red blood cells (RBCs), the approximately one month half-life of blood lead is due to the RBC life-span. This means that blood lead is a marker of recent lead exposure coming from either external environmental or internal bone reserves during periods of bone remodeling. In sum, bone lead is a marker of long-term, cumulative lead exposure, while blood lead is a marker of short-term lead exposure.

Black-white disparities in lead exposure

Black Americans have historically been at greater risk of lead exposure than white Americans. Black adults have higher levels of bone lead than adults from white or other race/ethnic groups (Lin, Kim et al. 2004; Martin, Glass et al. 2006). Because bone lead is a marker of long-term, cumulative lead exposure, this suggests a long history of greater lead exposure for black Americans compared to other social groups.

Environmental lead exposure has decreased substantially for *both* black and white adults since the removal of leaded gasoline and other lead-containing products from the market. In addition to the decrease in absolute lead levels, there has also been a large decrease in the black-white *disparity* in lead levels, shown in Figure 2.4. During 1988-1994, the age-adjusted geometric mean blood lead levels for black and white adults were $3.29^{\mu g}/_{dL}$ and $2.65^{\mu g}/_{dl}$, respectively (Muntner et al., 2005). During 1999-2002, the levels for black and white adults were $1.85^{\mu g}/_{dL}$ and $1.58^{\mu g}/_{dL}$, respectively.

The pattern is similar when considering the percentage of black and white adults with elevated ($\geq 5^{\mu g}/_{dL}$) or high ($\geq 10^{\mu g}/_{dL}$) blood lead levels, shown in Figure 2.5. For example, during 1988-1994, the age-standardized percentages of black and white adults with elevated blood lead were 30.5% and 18.6%, respectively. During 1999-2002, these levels dropped to 8.1% and 4.3%, respectively (Muntner et al., 2005). The *disparity* dropped from 11.9 to 3.8 percentage points. Although there are still disparities in mean blood lead and in rates of elevated and high blood lead, they are small. Yet, as will be discussed in the next section, there are disparities in the *effect* of lead.

Lead and black-white disparities in blood pressure and hypertension

Lead is causally related to blood pressure and hypertension.(Navas-Acien et al., 2007) In general, blood lead, a marker of recent lead exposure, is positively associated with blood pressure while bone lead, a marker of cumulative lead exposure, is positively associated with hypertension risk (Cheng et al., 2001; Glenn, Stewart, Links, Todd, & Schwartz, 2003; Hu et al., 1996; Korrick et al., 2002; Martin et al., 2006). For example, a doubling of blood lead is associated with a one mmHg increase in systolic blood pressure and a $25^{\mu g}/_{g}$ increase in patella bone lead is associated with twice the odds of hypertension (Korrick, Hunter, Rotnitzky, Hu, & Speizer, 1999; Nawrot, Thijs, Den Hond, Roels, & Staessen, 2002; J. Schwartz, 1995).

The associations between lead and blood pressure and hypertension appear to be dose-dependent (Ahamed & Siddiqui, 2007; Cheng et al., 2001; Hu et al., 1996). This dose-dependent nature coupled with the dramatic decline in lead levels over the past decades likely explains a new pattern in the blood lead-blood pressure association. In recent decades, white adults no longer exhibit the positive association between lead and blood pressure (Den Hond et al., 2002; Muntner et al., 2005; Vupputuri et al., 2003). Nevertheless, black men continue to exhibit a roughly three mmHg increase in systolic blood pressure for every doubling of blood lead (Den Hond et al., 2002). Black women exhibit an even higher four mmHg increase in systolic blood pressure for every doubling of blood lead (Den Hond et al., 2002). Although black adults have higher blood lead levels compared to white adults, their blood lead levels are low, as shown in Figures 2.4 and 2.5. The small disparities in blood lead levels likely to not explain the disparities in the blood lead-blood pressure association.

Race, the social environment, and hypertension

The social environment "includes the groups to which we belong, the neighborhoods in which we live, the organization of our workplaces, and the policies we create to order our lives" (Yen & Syme, 1999, p. 288). These social factors and processes are the fundamental mechanisms that link the social environment to health (Link & Phelan, 1995). The social environment is the source of constraints, resources, social stressors, and psychosocial stress.

Disparities in social environments are a fundamental cause of disparities in health, including hypertension (Geronimus et al., 2007; Link & Phelan, 1996; Williams & Collins, 2001). For example, in Chicago, black residents have an 80% greater odds of hypertension compared to white residents, even after adjusting for individual SES (Morenoff et al., 2007). This disparity is completely attenuated when accounting for residential area, a composite of many aspects of the social environment.

Black-white disparities in constraints and resources: Educational attainment and poverty status

Resources and constraints are factors that shape exposure to the risk of poor health (Link & Phelan, 1995; Yen & Syme, 1999). I use two markers of these resources and constraints – educational attainment and poverty status (Lillie-Blanton & Laveist, 1996). Educational attainment is an important determinant of other social and economic resources, as it determines occupational and earnings potential (Card, 1999). Poverty status is a major constraint, as it limits the ability to purchase goods necessary for a healthy life such as healthy food, proper clothing, and safe shelter (Citro & Michael,

1995). The balance of resources and constraints may serve as stressors in themselves, but they may also transform daily activities into stressors. For example, providing healthy meals for one's family may not, in itself, be a stressful activity. However, it may be transformed into a stressor when met with inadequate financial resources and a lack of easily accessible grocery stores.

Research shows that black Americans are often met with an imbalance in constraints and resources that make the negotiation of daily activities stressful. For example, access to healthy food in communities composed predominantly of black residents is more difficult compared to other communities. Detroit, an oft-examined case-study in racial segregation and inequality, has only nine supermarket-type chain grocery stores within its predominantly-black-residence city limits, while the surrounding 15-mile largely-white-residence suburban radius contains 160 such stores (Zenk, Schulz et al. 2005).

There are also inequalities in access to adequate health care resources in areas composed largely of black residents. For example, pharmacies in New York City vary in their supplies of important pain medications by racial composition of the residential area. In areas composed of more than 80% white residents, 72% of the area pharmacies stock adequate pain medication. On the other hand, in areas composed of more than 40% black residents, only 30% of the area pharmacies were adequately stocked (Morrison, Wallenstein, Natale, Senzel, & Huang, 2000).

On the other hand, residential areas composed largely of black residents have more convenience and liquor stores, pawn brokers, short-term/high-interest lenders, and fast food establishments (Block, Scribner, & DeSalvo, 2004; Heynen, Perkins, & Roy,

2006; LaVeist & Wallace, 2000; Morello-Frosch & Lopez, 2006). Furthermore, insurance agencies and mortgage lenders engage in practices that limit the availability of affordable resources to those living in areas with predominantly black residents (Charles & Hurst, 2002; Galster & Booza, 2008; Ondrich, Ross, & Yinger, 2003; Squires, Velez, & Taeuber, 1991). Taken together, these studies indicate that residential areas composed primarily of black residents are characterized by fewer resources important for health.

SES has been extensively examined with regard to race. On average, black adults have lower levels of education compared to white adults. For example, roughly eight percent of white adults over the age of 25 are without a high school education. However, black adults have double that rate; between 16%-18% are without a high school education (US Census Bureau, 2008). Poverty rates are also substantially higher for black adults. Figure 2.6 shows that the poverty rates for black adults are more than 2.5 times the rates for white adults. Importantly, because black women are more likely to serve as household heads compared to white women, their low income translates into particularly low household incomes for black families (Williams & Collins, 2001).

Compounding the disparities in the levels of SES is fact that there are disparities *within levels* of SES. In other words, SES markers hold different meanings for black and white Americans (David, Selina, Jacinta, & Chiquita, ; Kaufman, Cooper, & McGee, 1997). Black adults have lower occupational status and lower wages for the same educational attainment (Anderson & Shapiro, 1996; Williams, 1999; Wilson, Tienda, & Wu, 1995). Others show that middle-income black adults are often constrained to similar residential environments as low-income black adults (Pattillo, 1999, 2005). In the grocery store example in Detroit, even when comparing only poor residents, poor black

residents must travel over one additional mile than their poor white counterparts to reach a supermarket, not an insignificant distance when travelling on foot or by public transportation (Zenk, Schulz et al. 2005).

Black-white disparities in psychosocial stress: Depressive symptoms

The disparities in the social environment result in disparities in psychosocial stress both because black adults are more likely to experience generally stressful circumstances and because their greater experience of constraints and fewer resources transform daily activities into stressors. This greater stress then results in greater levels of psychological distress and depressive symptoms. In general, black men and women report higher levels of distress and depressive symptoms compared to white men and women (Eaton & Kessler, 1981; Kessler & Neighbors, 1986; Nuru-Jeter, Williams, & LaVeist, 2008; Ulbrich, Warheit, & Zimmerman, 1989). Psychosocial stress and social constraints, marked by SES, account for a large portion of these disparities (Eaton & Kessler, 1981; J. Taylor & Turner, 2002; R. J. Turner & Avison, 2003).

Social stressors result in psychosocial stress, which challenges the body biologically to adapt (McEwen, 1998). I use a proxy measure of psychosocial stress, depressive symptoms, that mark the physiological and psychological response to the psychosocial stress that results when demands exceed adaptive resources (Mirowsky & Ross, 2003; J. R. Turner, 2009). In this sense, depressive symptoms are used as a marker of physiological and psychological allostatic load (McEwen, 2003).

Because SES holds a different meaning for black and white adults, race and SES interact to result in more complex patterns of depressive symptoms (Kessler &

Neighbors, 1986; Nuru-Jeter et al., 2008). For example, when an automobile plant in Michigan closed, less educated black men and women who had lost their jobs reported particularly high levels of psychological distress compared to their white counterparts or more educated black men and women (Broman, Hamilton, Hoffman, & Mavaddat, 1995). It may be that this group of less educated men and women understood the long-term economic ramifications of the loss of low-skill jobs. Black adults, particularly black men, with low educational attainment are overlooked during the hiring process (Holzer, Offner, & Sorensen, 2005b).

The social environment and black-white disparities in hypertension

Educational attainment and poverty status are related to hypertension (Adler & Ostrove, 1999; Mensah et al., 2005; Pickering, 1999). Furthermore, evidence suggests that low SES is particularly harmful to the health of black adults. For example, black men who are both poor and report high effort coping with stressors have particularly high rates of hypertension compared to both nonpoor black men and poor black men who do not report high effort coping (S. A. James et al., 1983). This suggests that attempting to cope with stressors with the severe constraints of poverty are particularly harmful to the cardiovascular system. Similarly, evidence suggests that buying into the dominant cultural lifestyle is also particularly harmful to black adults of low SES (Dressler, 1990).

Distress and depressive symptoms are related to hypertension (Levenstein, Smith et al. 2001; Raikkonen, Matthews et al. 2001; Hamer, Molloy et al. 2008). However, evidence indicates that this association is not constant across black and white race. For example, black adults show a stronger association between depressive symptoms and

later hypertension development compared to white adults (Davidson, Jonas, Dixon, & Markovitz, 2000; Jonas, Franks, & Ingram, 1997). This suggests that reports of depressive symptoms have different meanings across race; it may be that, in addition to reporting higher levels of depressive symptoms, each level of depressive symptoms represents higher levels of psychosocial stress for black compared to white adults.

In sum, the evidence indicates that the social environment is an important determinant of black-white hypertension disparities. The social environment is the source of constraints, resources, and stressors, which result in psychosocial stress. In addition to its more direct effects on hypertension, evidence suggests that stress may also increase the harmful effects of physical hazards (Clougherty et al., 2007; Glass et al., 2009; Peters et al., 2007). In the next section, I bring the evidence on the social and physical environments together to examine their interaction.

The interaction of the social and physical environments

Generally addressed in separate disciplinary literatures, there is a need to examine interactions between the social and physical environments, as these are often spatially correlated and may present cumulative or synergistic effects on health (Clougherty & Kubzansky, 2009; Evans & Pilyoung, 2010; Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006; Schulz, Williams, Israel, & Lempert, 2002; Yen & Syme, 1999). In particular, social stressors and psychosocial stress appear to increase the harmful effects of physical hazards.

This idea has been explored to a limited extent in the literature. For example, traffic-related pollution is associated with a 63% increase in the odds of asthma in

children, but only for those with a high exposure to violence, which can be considered a social source of stress (Clougherty et al., 2007). This heightened association between traffic-related pollution and incident asthma is also reported for children whose parents report high levels of stress (Chen, Schreier, Strunk, & Brauer, 2008; Shankardass et al., 2009). Traffic-related pollution is also associated with coronary artery calcification (a type of heart disease), but only in those who live in neighborhoods with high unemployment (Dragano et al., 2009). These studies suggest that an important aspect of the physical environment is its interaction with the social environment.

Psychosocial stress and the harmful effects of lead

Recent evidence suggests that psychosocial stress may also increase the harmful effects of lead on health. For example, there is an negative association between bone lead and cognitive outcomes but only for those who live in neighborhoods with high levels of psychosocial hazards (Glass et al., 2009). This negative association between bone lead and cognitive outcomes is also shown only in those who report high levels of perceived stress (Peters et al., 2008).

Lead is associated with blood pressure and hypertension; however, evidence suggests that stress modifies this association as well. For example, the association between blood lead and hypertension is stronger in adults with higher allostatic load scores, a measure of the wear and tear on the body's systems due to psychosocial stress (McEwen, 1998; Zota et al., 2010). Furthermore, *bone* (as opposed to blood) lead is not generally associated with blood pressure, as discussed earlier. Yet there is an association between bone lead and blood pressure, but only for men who report high levels of

perceived stress (Peters, Kubzansky et al. 2007). A similar pattern is reported for bone lead and heart rate variability, a risk factor for cardiovascular disease (unpublished results described in Clougherty & Kubzansky, 2009, p. 1351). No one has applied this relation to the black-white disparity in the association between lead and hypertension.

A biologically-plausible model of the social environment, lead, and blood pressure

Although I do not test biological mechanisms directly, my research is based on the biological evidence linking lead, stress, and blood pressure. Lead and psychosocial stress share several biological mechanisms important for blood pressure changes, including actions on the biological stress response and cardiovascular systems (Ahamed & Siddiqui, 2007; Harrison & Gongora, 2009; Payton et al., 1993; Virgolini, Bauter, Weston, & Cory-Slechta, 2006). These shared pathways are thought to result in synergistic effects (Vyskocil, Fiala et al. 1991; Virgolini, Chen et al. 2005; Cory-Slechta, Virgolini et al. 2008; Vaziri 2008). However, the exact mechanisms of this synergy is not known. One possible mechanism is that lead may continually activate the biological stress response systems. This continual activation would increase wear and tear – or allostatic load – on the stress and related cardiovascular systems. This allostatic load state is characterized by a lack of appropriate control of the cardiovascular system, resulting in hypertension (McEwen & Seeman, 1999; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997).

For example, in a healthy stress system, cortisol increases in response to a stressor and then decreases with the removal of the stressor. Children with low blood lead levels

show this pattern of cortisol increase and decrease in response to a laboratory stressor. However, children with high blood lead levels do not exhibit a cortisol decrease after removal of the stressor (Gump et al., 2008). Similarly, animals with both high levels of lead and stress showed particularly high levels of corticosterone (the animal equivalent of cortisol) compared to animals with either high lead or high stress (Virgolini et al., 2006). These studies suggest that both psychosocial stress and lead wear away the stress response systems making further proper response to stress difficult.

The intersection of race and gender

While my research focuses on race, I recognize that gender intersects with race in ways that are important to health (Mullings, 2002; Schulz & Mullings, 2006). More specifically, gender interacts with racial discrimination to result in gendered racial stereotypes, ultimately placing black men and women in qualitatively different physical and social environments from each other and from white men and women (Browne & Misra, 2003; Darrell, Jeffery, & John, 1998; Mullings, 2002). This suggests that one gender group – black men or black women – does *not* necessarily experience *more* stressful social environments than the other, but that they experience *different* stressful social environments.

Gender modifies the mechanisms that link race to racial disparities in hypertension at several points in my conceptual model, with two highlighted. Overall, race and gender interact to result in disparities in hypertension. Black women have the highest rates compared to black men or white men and women (Geronimus et al., 2007). Race and gender also interact with regard to SES. For example, black men have the

highest rates of unemployment. In 2009, the unemployment rate for black men was 16.3% while the rate for black women was 11.5% and the rates for white men and women were 8.8% and 6.8%, respectively (US Burear of Labor Statistics, 2010). In fact, race and gender interact to result in group differences in numerous factors I examine.

Research questions

There are only a handful of studies that examine the interaction of the social and physical environments in the production and maintenance of social health disparities. There are no studies that examine this interaction with regard to black-white disparities in hypertension. My research addresses this gap in the literature. To test the notion that social stress increases the harmful effects of lead, I take advantage of the pattern of black-white disparities in the association between lead and blood pressure, documented in the literature. Specifically, I hypothesize that the association between lead and blood pressure among black but not white adults is due to greater imbalance of constraints and resources, marked by the proxies of educational attainment and poverty status, and higher levels of psychosocial stress, marked by the proxy of depressive symptoms.

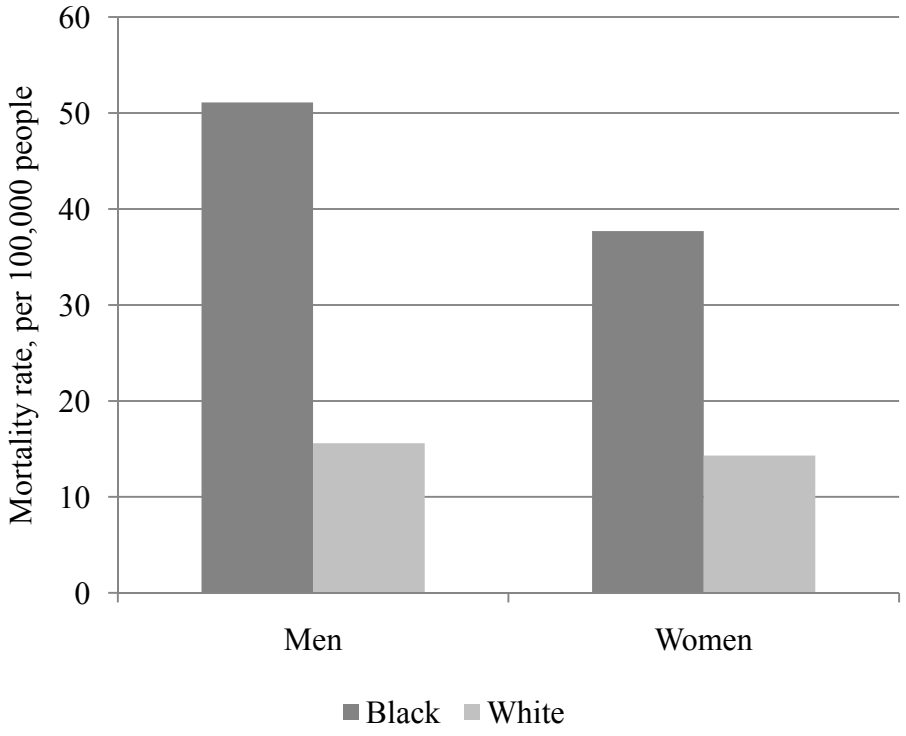
There are five research questions addressed, with the first three documented in the literature, but not in my dataset.

1. What is the current state of black-white hypertension disparities?
2. What is the current state of black-white lead level disparities?
3. Does lead predict hypertension for both black and white adults?
4. Do educational attainment and poverty status, as proxies of the constraints and resources, explain the association between lead and hypertension among black but not white adults?

5. Do depressive symptoms, as a proxy of psychosocial stress, explain the association between lead and hypertension among black but not white adults?

In the next chapter, I discuss my analytic model, drawn from my conceptual model, and my detailed research questions and hypotheses. I also provide a detailed explanation of the methods involved in testing each of my hypotheses.

Figure 2. 1 Mortality rates due to hypertension, 2006

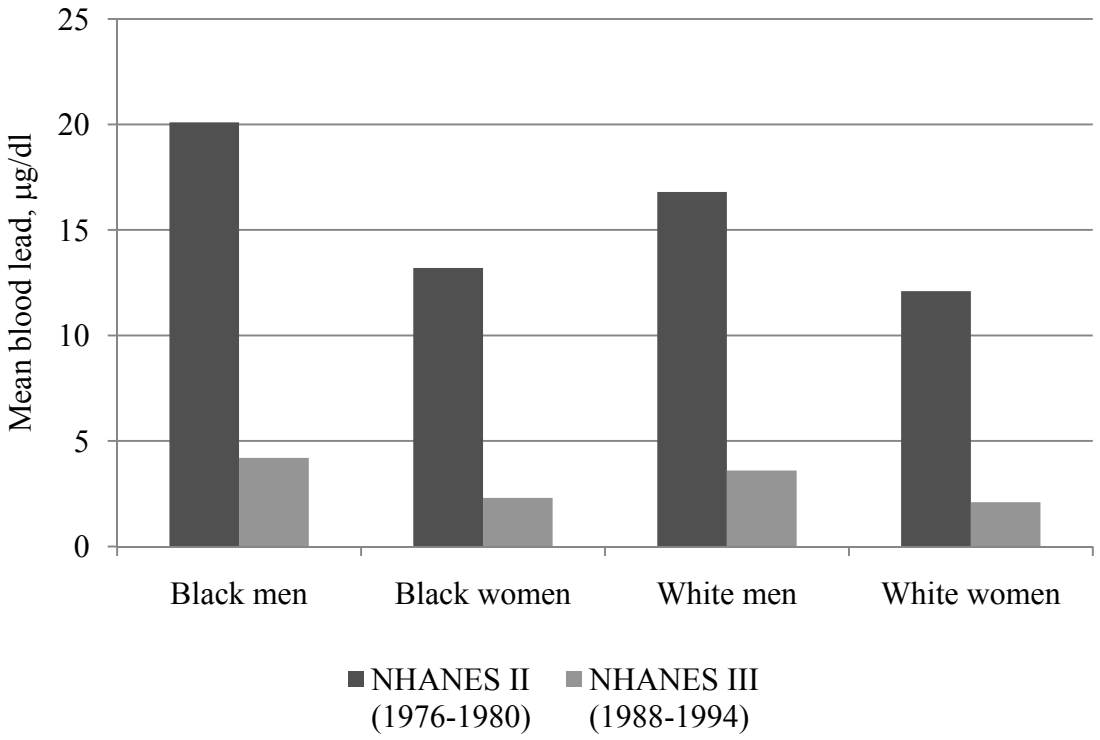


Source: Adapted from data in (Lloyd-Jones et al., 2009), from the National Center for Health Statistics.

Figure 2. 2 Conceptual model linking race to racial disparities in hypertension through psychosocial stress

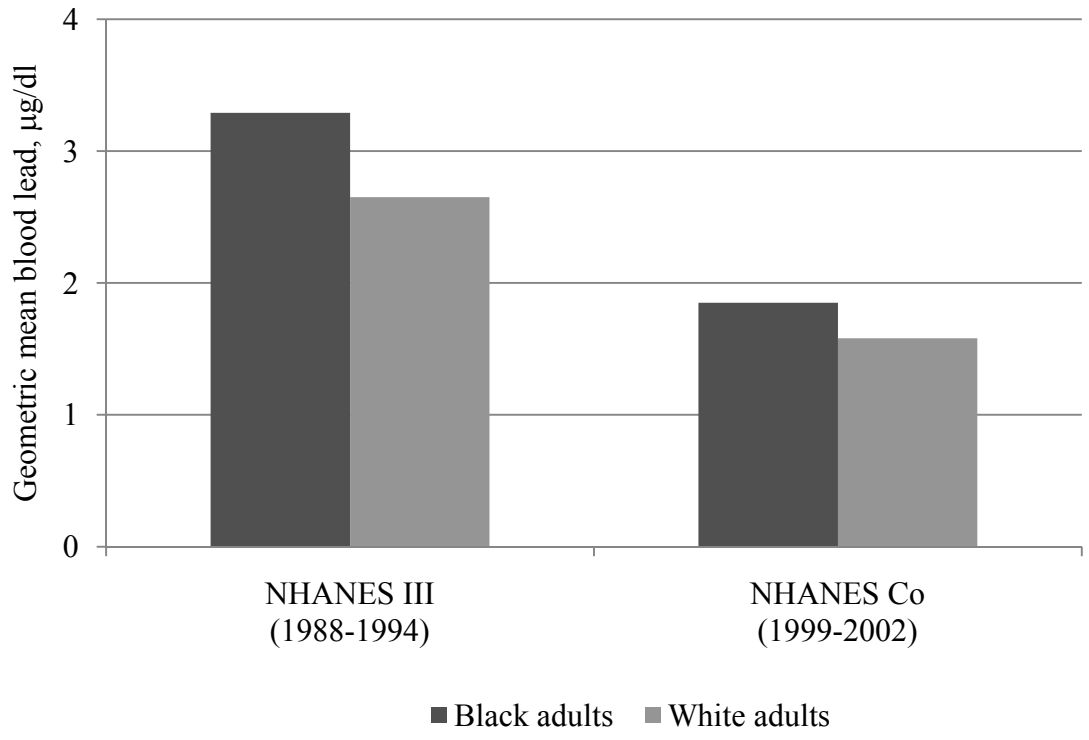
Notes: Factors in **BOLD SMALL CAPITAL LETTERS** denote variables tested in the analyses.

Figure 2. 3 Unadjusted mean blood lead levels, NHANES II and III



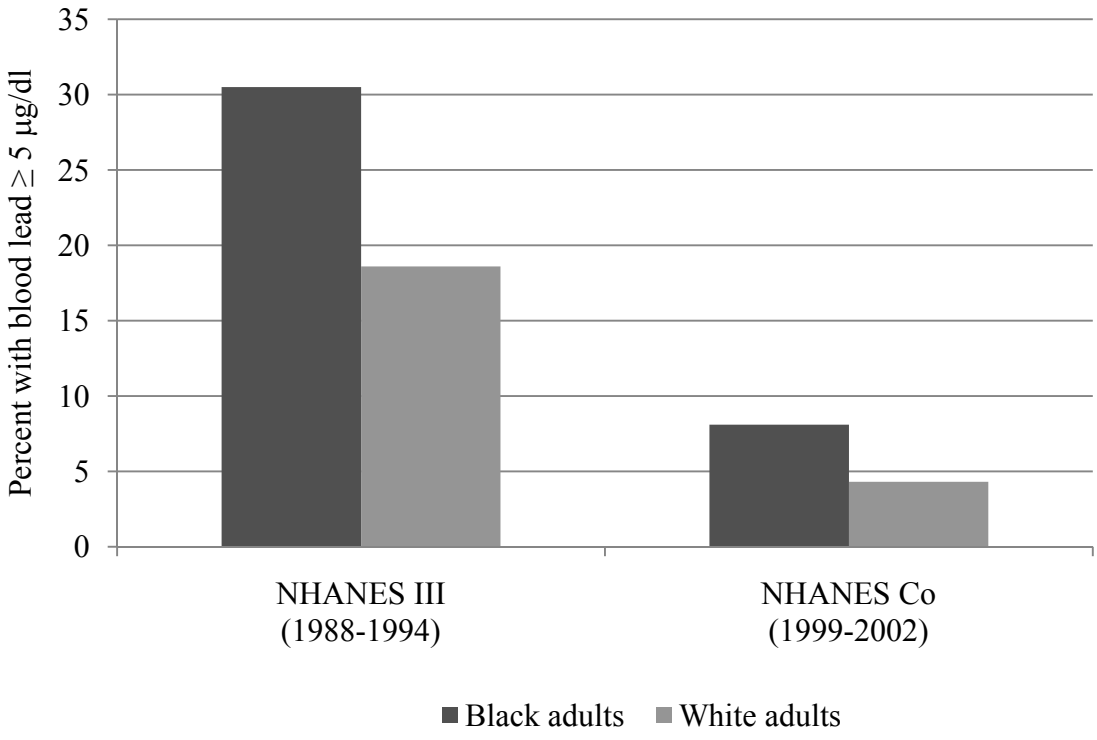
Source. Adapted from data in (Sorel et al., 1991, NHANES II adults 18-74) and (Den Hond et al., 2002, NHANES III adults 18 and older)

Figure 2. 4 Age-adjusted geometric mean blood lead levels, NHANES III and continuous NHANES



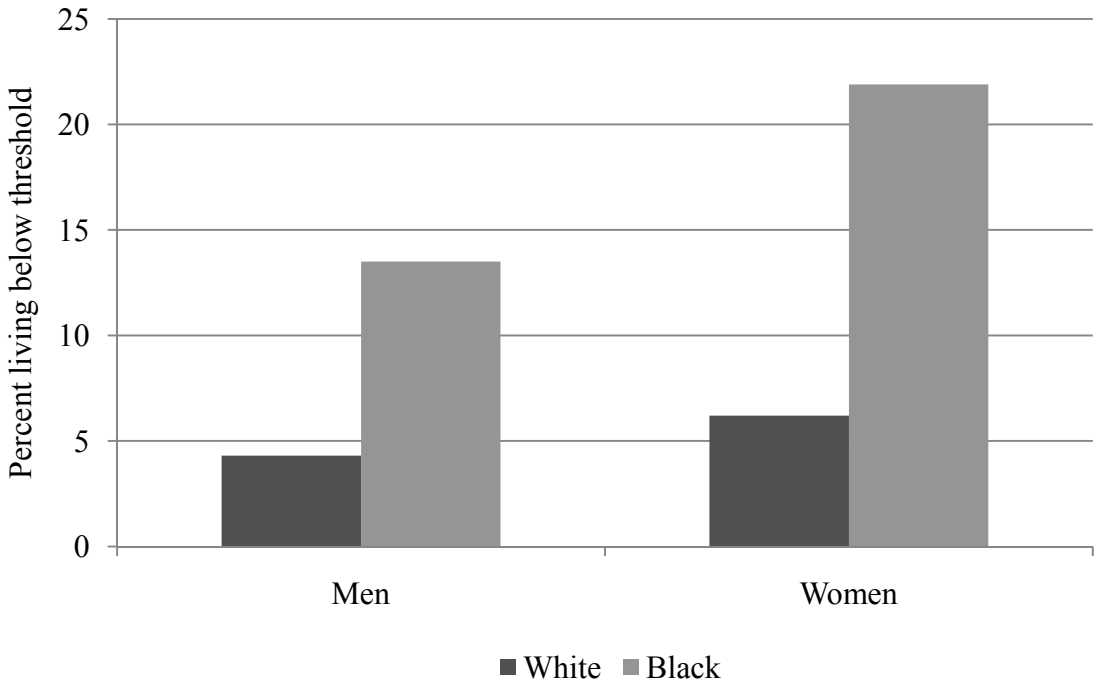
Source: Adapted from data in (Muntner et al., 2005)

Figure 2. 5 Age-adjusted percentage of elevated blood lead levels, NHANES III and continuous NHANES



Source: Adapted from data in (Muntner et al., 2005)

Figure 2. 6 Poverty rates, 2008 Census



Source: Data from (US Census Bureau, 2009)

CHAPTER THREE

METHODOLOGY

This chapter contains the methodology used to test the research questions broadly outlined in Chapter Two. I begin with an introduction to my analytic model, which is drawn from the conceptual model (Chapter Two, Figure 2.2) and narrows the discussion to the specific variables and pathways I examine. Specific research questions and hypotheses are then outlined. Finally, a description of my dataset and analytic approach are provided.

The analytic model: The interaction of stress and lead

The analytic model, shown in Figure 3.1 outlines the factors and pathways linking race to racial disparities in blood pressure and hypertension that I test. The solid black rectangles represent the focal factors in my analyses: race, SES, lead, depressive symptoms, blood pressure, and hypertension. The dashed black rectangles represent confounding factors: nutritional status, renal function, and health behaviors. I control for these factors to better estimate the effects of the focal variables. The gray solid box represents the biological stress process. While I cannot test this directly, I keep it in my analytic model because it is a key mechanism linking the social environment to blood

pressure and hypertension.

I examine multiple markers of blood pressure and lead and multiple proxies for social stressors and psychosocial stress. First, as the outcome, I examine both systolic and diastolic blood pressures as continuous variables and hypertension as a dichotomous variable. Although hypertension is the disease state measured and discussed when describing health, it represents an arbitrary threshold risk of cardiovascular, cerebrovascular and metabolic morbidity and mortality; this risk occurs along a continuum of blood pressure (Britton et al., 2009; Franklin et al., 2001; Vasan et al., 2001). Therefore, blood pressure, as a continuous variable, is an important marker of cardiovascular health, particularly when assessing health at the population level. Because anti-hypertensive medication reduces blood pressure, but not the underlying social determinants of hypertension, the inclusion of participants taking these medications may increase the error of my models. Therefore, I control for medication use in models with systolic or diastolic blood pressure.

I also examine two biomarkers of lead – bone lead, a marker of long-term, cumulative exposure, and blood lead, a marker of recent exposure (which may be from the external environment or from leaching from bone) (Hu, 1998). In studies that collect bone lead, data is gathered from two types of bone. The bone types differ on their structure – one is more solid and undergoes relatively less remodeling (e.g., tibia), and the other is more spongy and undergoes relatively more remodeling (e.g., patella). Bone lead is generally not collected in large, population-based studies because of the resources required. However, an algorithm to estimate bone lead from blood lead was recently developed and is used in this study (S. K. Park et al., 2009).

When using blood lead, several serum biomarkers are included as controls: hematocrit, iron, calcium, vitamin D, and phosphorous. Hematocrit is included in the analyses because 99% of blood lead is bound to red blood cells, and thus "inactive" for damage to tissue. The blood lead that is free in serum (the non-cell portion of blood) is available for damage to tissue. Hematocrit is also associated with cardiovascular conditions, including hypertension (Cirillo, Laurenzi, Trevisan, & Stamler, 1992). Iron is an essential nutrient needed in the formation of red blood cells. However, it has also been shown to affect the absorption and maintenance of lead in tissue, meaning that it may be related to the effect of lead (Kwong, Friello, & Semba, 2004). Calcium is one of the most important minerals in the body and is involved in proper muscle, nerve, and heart function. It is an important component of certain cell-signaling systems. Calcium and lead are both divalent cations, with research indicating that lead substitutes for calcium at calcium receptors (Vaziri, 2008). Research also suggests that calcium is related to blood lead level and may modify the effect of lead on blood pressure (Ettinger et al., 2009; Proctor, Rotnitzky, Sparrow, Weiss, & Hu, 1996). Calcium is governed in large part by the vitamin D system. Research also indicates that vitamin D has a negative association with hypertension (Pilz, Tomaschitz, Ritz, & Pieber, 2009). Phosphorous is interrelated with calcium and affects calcium balance, which is important for the effect of lead on tissue. GFR is included in all analyses because kidney function is intimately related to hypertension, there are racial disparities in kidney disease, and some effects of lead operate through kidney dysfunction (Muntner, He, Vupputuri, Coresh, & Batuman, 2003; Nzerue, Demissochew, & Tucker, 2002; Wedeen, 1988).

In general, bone lead is related to hypertension and blood lead is related to blood pressure (Cheng et al., 2001; Glenn et al., 2003; Hu, 1998; Hu et al., 1996; Korrick et al., 2002; Martin et al., 2006). Because bone lead marks lead exposure over decades, it may better mark long-term dysregulation of the cardiovascular system that results in hypertension. On the other hand, blood lead as a marker of short-term lead exposure, marks temporary perturbations in the cardiovascular system. In a cross-sectional study, such as the NHANES, blood lead may better correlate with blood pressure perturbations as opposed to hypertension, as blood pressure may reflect both temporary and relatively permanent changes in the cardiovascular system. Hypertension, on the other hand, may mark the end of a long-term, permanent decline of the cardiovascular system. Therefore, I restrict analyses to blood lead with blood pressure and bone lead with hypertension.

I use educational attainment and poverty status as proxies for the constraints and resources and depressive symptoms as a proxy for psychosocial stress (Aneshensel, Rutter, & Lachenbruch, 1991; Lillie-Blanton & Laveist, 1996; McEwen, 2003; Mirowsky & Ross, 2003). Evidence further indicates that SES and depressive symptoms have different meanings across black and white race (Kaufman et al., 1997; Kessler & Neighbors, 1986; Nuru-Jeter et al., 2008). In other words, although race is correlated with SES and depressive symptoms, an additive model of race with SES and depressive symptoms inadequately capture the black-white disparities *within* level of SES and depressive symptoms. Therefore, race is interacted with education, poverty status, and depressive symptoms to reflect the qualitative differences by race. This race-specific measure is then interacted with lead in order to examine the effect of lead on blood pressure and hypertension for these race-education, race-poverty, and race-depressive

symptoms groups. For example, I examine the effect of lead on blood pressure for poor and nonpoor black adults and poor and nonpoor white adults. This result in a three-way interaction.

From my model, both lead and psychosocial stress result in activation of the biological stress systems (Cohen et al., 2006; Cole, 2008; O'Connor et al., 2009; Virgolini et al., 2006). Chronic activation of the biological stress systems may result in psychological distress or allostatic load, marked as depressive symptoms (McEwen, 2003, 2005). In other words, theoretically, depressive symptoms can results from both psychosocial stress and lead, which would be difficult to disentangle using regression techniques. However, lead is not associated with depressive symptoms in the dataset I use (Golub, Winters, & van Wijngaarden, 2009).

From my model, depressive symptoms are a result of dysregulation of the biological stress response systems. However, note that they are not in the direct mechanistic pathway leading to hypertension. Although there is a consistent association between depressive symptoms and cardiovascular disease, evidence suggests that this is not a causal association in either direction (Yan et al., 2003). Both depressive symptoms and cardiovascular disease, including hypertension, may be the allostatic load result of a common dysregulation of the stress response systems (Grippo & Johnson, 2009).

Although depressive symptoms result from dysregulation of the biological stress response systems, this does not necessarily mean that they are a direct measure of psychosocial stress. Dysregulation of the stress response systems are associated with and may result from numerous other factors. Therefore, it may be more accurate to view

depressive symptoms as a marker of a broad range of factors that increase susceptibility to disease – including susceptibility to the harmful effects of lead.

Depressive symptoms may indirectly result in blood pressure increases and hypertension through changes in hypertension risk factors such as BMI, smoking, and alcohol use (Dallman et al., 2003; Paperwalla, Levin, Weiner, & Saravay, 2004; Plante, 2005; Sullivan, Fiellin, & O'Connor, 2005). BMI is a rough marker of obesity, which is a risk factor for hypertension (Mikhail, Golub, & Tuck, 1999). Obesity may be negatively related to bone loss, which is in turn is associated with bone lead release into the bloodstream (Lan-Juan et al., 2008; Nash, Magder, Sherwin, Rubin, & Silbergeld, 2004; Tsaih et al., 2001). Both smoking and alcohol use are also associated with blood lead burden as well as hypertension (Bowman, Gaziano, Buring, & Sesso, 2007; Halperin, Gaziano, & Sesso, 2008; Hense, Filipiak, & Keil, 1994; Mannino, Homa, Matte, & Hernandez-Avila, 2005). Because I use depressive symptoms as a marker of the overactivation of the biological stress systems (i.e., psychological allostatic load), I control for these hypertension risk factors to estimate the net effects of depressive symptoms on blood pressure and hypertension.

All analyses are stratified by gender because there are social and biological differences by gender/sex that are related to blood lead burden, hypertension risk, and depressive symptoms (Geronimus et al., 2007; Muntner et al., 2005; Nolen-Hoeksema, 2001). Furthermore, evidence indicates that there are gender differences not only in the *levels* of the variables I use (e.g., differences in alcohol use), but in the pathways linking variables (e.g., association between stress and alcohol use). In other words, there are gender differences the *meaning* within the variables. For example, there are documented

gender differences in reports of depressive symptoms that do not reflect differences in the effect of stress, but rather differences in coping strategies (Rosenfield, 1999). Specifically, women tend to report more depressive symptoms, but men report certain behavioral coping mechanisms such as alcohol use in relation to stress (M. L. Cooper, Russell, Skinner, Frone, & Mudar, 1992). Because of qualitative differences at multiple points in the model that cannot be addressed by simply controlling for gender within the statistical models, I stratify all analyses by gender.

Finally, age is included in all analyses as a confounder for several reasons: there are black-white differences in the age distribution, age is associated with blood lead, blood pressure, and hypertension, and there are black-white disparities in age at which these associations develop (Geronimus et al., 2007; Muntner et al., 2005).

Research questions and hypotheses

My central research question is: "Does the poor social environment experienced by black adults further increase the harmful effects of lead on hypertension?" Equation 3.1 outlines the general model I use (control variables are omitted for simplicity):

$$Y = \beta_0 + \beta_1 race + \beta_2 lead + \beta_3 social + \beta_4 race*social + \beta_5 race*lead + \beta_6 social*lead + \beta_7 race*social*lead \quad \text{(Equation 3.1)}$$

where: Y = blood pressure or hypertension status variables:
systolic blood pressure (mmHg)
diastolic blood pressure (mmHg)
hypertension status (absent=0, present=1)
race = race variable:
black race (white=0, black=1)

lead = lead variables:
- blood lead ($^{\mu g}/_{dl}$)
- estimated tibia bone lead ($^{\mu g}/_{g}$)
- estimated patella bone lead ($^{\mu g}/_{g}$)

social = social markers:
- educational attainment (≥high school=0, <high school=1)
- poverty status (nonpoor=0, poor=1)
- depressive symptoms (low=0, high=1)

I have two hypothesis sets – one addressing blood lead and blood pressure and one addressing bone lead and hypertension. (I describe these variables in following sections).

Hypothesis set 1: Blood lead and blood pressure

Hypotheses 1a: There are black-white disparities in the association between blood lead and blood pressure.

- Hypothesis 1a.1: Black but not white adults exhibit a positive association between blood lead and systolic blood pressure.
- Hypothesis 1a.2: Black but not white adults exhibit a positive association between blood lead and diastolic blood pressure.

Hypothesis 1b: Black-white disparities in blood lead levels do *not* explain the black-white disparities in the association between blood lead and blood pressure.

- Hypothesis 1b.1: This positive association between blood lead and systolic blood pressure is not explained by black-white disparities in blood lead level.
- Hypothesis 1b.2: This positive association between blood lead and diastolic blood pressure is not explained by black-white disparities in blood lead level.

Hypothesis 1c: Black-white disparities in the social environment explain the black-white disparities in the association between blood lead and blood pressure.

Hypothesis 1c.1: The positive association between blood lead and systolic blood pressure is explained by racial disparities in educational attainment.

Hypothesis 1c.2: The positive association between blood lead and systolic blood pressure is explained by racial disparities in poverty status.

Hypothesis 1c.3: The positive association between blood lead and systolic blood pressure is explained by racial disparities in depressive symptoms.

Hypothesis 1c.4: The positive association between blood lead and diastolic blood pressure is explained by racial disparities in educational attainment.

Hypothesis 1c.5: The positive association between blood lead and diastolic blood pressure is explained by racial disparities in poverty status.

Hypothesis 1c.6: The positive association between blood lead and diastolic blood pressure is explained by racial disparities in depressive symptoms.

Hypothesis set 2: Estimated bone lead and hypertension

Hypothesis 2a: There are black-white disparities in the association between estimated bone lead and hypertension.

Hypothesis 2a.1: Black but not white adults exhibit a positive association between estimated tibia lead and hypertension.

Hypothesis 2a.2: Black but not white adults exhibit a positive association between estimated patella lead and hypertension.

Hypothesis 2b[2]: Black-white disparities in the social environment explain the black-white disparities in the association between estimated bone lead and hypertension.

Hypothesis 2b.1: The positive association between estimated tibia lead and hypertension is explained by racial disparities in educational attainment.

Hypothesis 2b.2: The positive association between estimated patella lead and hypertension is explained by racial disparities in educational attainment.

Hypothesis 2b.3: The positive association between estimated tibia lead and hypertension is explained by racial disparities in poverty status.

Hypothesis 2b.4: The positive association between estimated patella lead and hypertension is explained by racial disparities in poverty status.

The dataset

The data analyzed are from the continuous National Health and Nutrition Examination Survey (NHANES). The NHANES is a nationally-representative repeated cross-sectional survey of non-institutionalized children and adults (not on active military duty) administered by the National Center for Health Statistics (NCHS) of the Centers for Disease Control (CDC). A detailed interview, health examination, and laboratory analysis comprise each survey. The interview includes questions on such factors as sociodemographic information, social support, health behaviors, dietary intake, diseases, and medical conditions. The health examination includes items such as anthropometric measurements, a dental exam, and a medical exam. The laboratory analysis includes

[2] There is no "Hypothesis 2b" to correspond to "Hypothesis 1b" because the Oaxaca-Blinder method that is used was not available for nonlinear models in STATA 11.0 at the time of data analysis. However, it is now available and will be employed in analyses for manuscript preparation.

measures of nutrition, cardiovascular and renal health, and toxicants. There have been three large waves of the NHANES, starting in the 1960s. In 1999, NCHS began to administer this survey in a continuous manner such that there is data collection every year. Currently, each two-year wave includes approximately 10,000 adults and children, with oversampling of certain populations such as low-income adults, teens, older adults, and Mexican and African Americans.

For the examination of black-white disparities in lead and hypertension/blood pressure, I include only adults 20 years of age and older who identify as non-Hispanic black or white and who participated in the clinic portion of the three most recent waves, 2001-2002, 2003-2004, 2005-2006, yielding a starting sample size of 10,532. I exclude children and teens because the literature indicates that lead and bone exhibit different kinetics in adults and children (US Agency for Toxic Substances and Disease Registry, 2007). I include adults 20 years and older because research has shown that black-white disparities in hypertension are evident as early as 20 years of age (Geronimus et al., 2007). Furthermore, the NHANES surveys frequently use different survey instruments for participants 19 years and younger, thus precluding the definition of "adult" as 18 years and older.

I exclude women who are pregnant or lactating, as this is a period of heightened bone remodeling, with increased risk of lead release from bone into the bloodstream (Rothenberg et al., 1994). This excludes 621women, reducing the sample size to 9,911. Although menopause is also a time of heightened bone remodeling, I do not exclude these women that would exclude nearly all women above the age of 55 years. Rather, I run models with and without controls for menopausal status. If these models are similar,

I report only on the more parsimonious model.

I exclude men and women missing blood lead and other key variables for a final sample size of 6,390. For analyses using bone lead, the final sample size is restricted to the first two waves due to the availability of key variables, for a final sample size of 4,233. Analyses using depressive symptoms are restricted to the last wave due to the availability of those variables, for a final sample size of 2,167. In the section "Missing variables", I discuss my plan to address this large number of missing variables.

It is likely that blood lead levels are very low in this 2001-2006 dataset, which may impact the ability to detect any effect of lead on blood pressure. However, my research is aimed at testing the notion that social factors amplify the effects of lead. It may be that there are no detectable effects of lead in this dataset. In which case, the NHANES III can be used. However, if there are black-white disparities in the effect of blood lead in the NHANES 2001-2006, it will serve as further support that some social factor related to race amplifies the effect of lead – even at very low levels.

Variables

The variables included in the analytic models are divided into dependent, explanatory, and confounding. Additionally, there are several dataset weight and design variables that are necessary to account for the complex survey design of the NHANES.

Dependent variables

Systolic and diastolic blood pressures

Blood pressure was measured using a sphygmomanometer by a trained physician during the physician examination which took place at the mobile examination center (MEC). Up to four sitting blood pressure measurements were taken on every person eight years of age and older. The protocol used was that recommended by the American Heart Association for the measurement of human blood pressure using a sphygmomanometer.

I calculate average systolic and diastolic blood pressures, mmHg, in STATA based on the NHANES guidelines provided in the blood pressure documentation. If only one blood pressure reading was obtained, that reading is the average. If there is more than one blood pressure reading, the first reading is always excluded from the average. If only two blood pressure readings were obtained, the second blood pressure reading is the average. If there is one diastolic reading of zero and one or more with a number greater than zero, then the diastolic reading of zero is not used to calculate the diastolic average. If two out the three diastolic reading are zero, the one diastolic reading that is not zero is used to calculate the diastolic average.

Hypertension

I calculate hypertension as a dichotomous variable (absent=0/present=1) in STATA from the information gathered from the MEC blood pressure measurement and the home interview on anti-hypertensive medication use and recent health care visits. Hypertension is defined as an MEC average systolic blood pressure reading of 140mmHg

or more, *or* diastolic blood pressure reading of 90mmHg or more, *or* an affirmative response to the question, “Are you now taking prescribed medication [for high blood pressure/hypertension]?”, *or* an affirmative response to the question, “Were you told on two or more different [clinic] visits that you had hypertension, also called high blood pressure?” (Joint National Committee, "Hypertension prevalence and the status of awareness, treatment, and control in the United States. Final report of the Subcommittee on Definition and Prevalence of the 1984 Joint National Committee," 1985; Lloyd-Jones et al., 2009).

Explanatory variables

Race

Racial and ethnic category was selected by the respondent from a list of responses. Respondents were allowed to select more than one category. The list of categories is as follows:

Racial group categories

- White
- Black/African American
- Indian (American)
- Alaska Native
- Native Hawaiian
- Guamanian
- Samoan
- Other Pacific Islander
- Asian Indian
- Chinese
- Filipino
- Japanese
    - Korean
    - Vietnamese
    - Other Asian

Hispanic group categories

- Puerto Rican
- Dominican (Republic)
- Mexican/Mexicano
- Mexican American
- Chicano
- Cuban
- Cuban American
- Central or South American
- Other Latin American (Specify)
- Other Hispanic (Specify)
- Refused
- Don’t know

- Some Other Race (Specify)
- Refused
- Don't Know

If the respondent selected more than one racial group, s/he was asked to select a single category that best describes him/her. NHANES staff recoded these responses to create a single race/ethnicity variable with the following categories five: non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic, and other race/multi-racial. The analyses presented here include only those who identify as non-Hispanic white or non-Hispanic black.

For models without interaction terms, I create a dummy variable in STATA, coded as 0=white and 1=black. For analyses where race is included in the interaction term, I use weighted effects coding in the creation of the dichotomous variable, with the foundation coding of -1=white and +1=black. Then I add to the category for black adults to correspond to their lesser proportion in my sample compared to white adults. This is done to account for the sample size differences.

Blood lead

Whole blood lead was measured in all participants aged one year and older using inductively coupled plasma mass spectrometry. The mass spectrometer instrument has lower-bound detection limits. If blood lead levels are below this limit, the reading is not reliable. To distinguish nondetectable laboratory test results from a very low measured lead test result, a special value was imputed by NHANES staff. This value is equal to the lower-bound detection limit for that wave divided by the square root of two. The lower-bound detection limits and corresponding imputed values for each wave are as follows:

0.28µg/dL and 0.20µg/dL (2001-2002); 0.28µg/dL and 0.20µg/dL (2003-2004); and 0.25µg/dL and 0.18µg/dL (2005-2006). In 2005-2006, there were two lower-bound detection limits, 0.25µg/dL and 0.30µg/dL with no guidance in the NHANES documentation as to which limit is to be used, therefore, the former value was selected, as had been done in the literature (Navas-Acien et al., 2009). Participants with blood lead levels below the limit of detection were dropped from analyses including blood lead. The number of observations dropped is 181: 125 (2001-2002), 24 (2003-2004), and 32 (2005-5006). Finally, blood lead was natural-log-transformed due to its skewed distribution which resulted in heteroskedasticity of the error terms.

Estimated tibia and patella bone lead

Bone lead is predicted from blood lead and other variables. The algorithm was developed using a sample of older white men from the Normative Aging Study (NAS), a longitudinal study started in 1961 by the US Veterans Administration. Twenty-eight potential predictors shown to be important in the determination of bodily lead burden through previous studies were selected for inclusion. Then, using the least absolute shrinkage and selection operator (LASSO) method, the researchers identified a set of 14 variables that best predicted tibia and patella lead from blood lead in the NAS sample:

- Age
- Blood lead
- Body mass index
- Education
  - HS
  - college
- Hematocrit
- Serum calcium
- Serum creatinine
- Serum phosphorus

- Serum uric acid
- Smoking amount
- Smoking status
  - former
  - current
- Total cholesterol
- Total high-density lipoprotein
- White collar occupation

I calculate the estimated tibia and patella lead levels in STATA from the reported regression coefficients obtained from the LASSO procedure on the NAS sample dataset, based on Equations 3.2 and 3.3, respectively:

Tibia lead	=	-12.603			(Equation 3.2)
	+	1.605	*	blood lead	
	+	0.604	*	age	
	+	-4.244	*	high school diploma dummy	
	+	-7.362	*	college degree dummy	
	+	-2.994	*	white collar occupation dummy	
	+	0.027	*	pack-years smoking	
	+	1.808	*	former smoker dummy	
	+	2.044	*	current smoker dummy	
	+	-3.668	*	serum phosphorus	
	+	1.048	*	serum uric acid	
	+	-0.403	*	serum calcium	
	+	-0.223	*	serum creatinine	
	+	0.019	*	total cholesterol	
	+	0.060	*	total high-density lipoprotein	
	+	-0.257	*	hematocrit	
	+	0.156	*	body mass index	

Patella lead	=	-42.927			(Equation 3.3)
	+	1.851	*	blood lead	
	+	0.911	*	age	
	+	-4.125	*	high school diploma dummy	
	+	-8.295	*	college degree dummy	
	+	-4.374	*	white collar occupation dummy	

+	-0.073	*	pack-years smoking
+	1.805	*	former smoker dummy
+	3.648	*	current smoker dummy
+	-4.178	*	serum phosphorus
+	1.108	*	serum uric acid
+	0.226	*	serum creatinine
+	0.124	*	total high-density lipoprotein
+	-0.106	*	hematocrit
+	0.362	*	body mass index

The dummy variables required for these equations are created from the categorical variables provided in NHANES. The educational attainment, smoking status, and BMI categorical variables are discussed below, as they are used as covariates in my analytic models. The white collar occupation dummy variable is coded as "1" if the respondent reported that their longest held job (including their current job) was of the following categories:

- Executive, administrators, and managers
- Management related occupations
- Engineers, architects and scientists
- Health diagnosing, assessing and treating occupations
- Teachers
- Writers, artists, entertainers, and athletes
- Other professional specialty occupations
- Technicians and related support occupations
- Supervisors and proprietors, sales occupations
- Sales representatives, finance, business, & commodities ex. retail
- Sales workers, retail and personal services
- Secretaries, stenographers, and typists
- Information clerks
- Records processing occupations
- Material recording, scheduling, and distributing clerks
- Miscellaneous administrative support occupations
- Private household occupations
- Health services occupations
- Farm operators, managers, and supervisors

Four additional serum biomarkers are also used in the algorithm: creatinine, high-density lipoprotein (HDL), total cholesterol, and uric acid. These biochemistry tests were conducted on blood collected during the MEC physician's examination. Creatinine and uric acid measurements were performed on blood from participants 12 years and older. HDL and total cholesterol measurements were performed on blood from participants three years and older. Phosphorous and hematocrit are used as control variables as well and are discussed later.

Creatinine is a waste product produced in the muscles from creatine (part of the muscle contraction process). Nearly all creatinine is excreted by the kidneys, so serum creatinine is used as a marker of how the kidneys are functioning. Higher levels of serum creatinine suggest that the kidneys may not be functioning properly. Uric acid is the waste product from the breakdown of a group of molecules called purines, which are found in DNA. Because uric acid is produced through the digestion of food and cellular turnover, it is a marker of metabolic disorders. Because it is primarily excreted by the kidneys, it is also a marker of kidney function. HDLs are a class of lipoproteins, molecules made of fat and protein that carry cholesterol, an essential molecule, in the body. HDLs contain primarily protein with a small amount of cholesterol which they carry from tissues to the liver for disposal. High levels of HDL are associated with lower risk for cardiovascular disease because of their cholesterol-removing function. Total cholesterol is a measure of both HDL and LDL, or low-density lipoprotein. LDL carries higher amounts of cholesterol and deposits it in places where it can be harmful, such as in vascular walls. A high total cholesterol, if elevated due to high LDL, is associated with a risk for cardiovascular disease.

Although there are several components of the algorithms that are also used as covariates in my analyses, this will not result in multicollinearity between estimated bone lead and the covariates. However, it may be that when adjusting for these factors, the effect of bone lead may disappear. I discuss this further in Chapter Six. Following Park et al (2009), I include seven of the components of the algorithm as covariates in my regression models (age, BMI, smoking status, pack-years smoked, white-collar occupation, and hematocrit).

Educational attainment

Respondents 20 years and older were asked to report the highest level of education that they had completed. The response categories are:

- Less than 9^{th} grade
- 9^{th}-11^{th} grade (which includes 12^{th} grade and no diploma)
- High school graduate/GED
- Some college or an associate's degree
- College degree or higher

I create a variable with three categories: 1=less than high school, 2=high school, 3=more than high school, for use as a control variable. I create a dichotomous variable that is: 0=less than high school; 1=high school or more, for use in interaction terms. This dichotomous variable is used in the three-way interactions among race, educational attainment, and lead.

Family poverty-to-income ratio

The US Census Bureau uses a set of earnings thresholds that vary by family size to determine who is poor. The family poverty-to-income ratio (PIR) is the ratio of the

family's gross income to the family's poverty threshold – which is generally calculated with an algorithm to determine the family's needs. PIR was calculated by the NHANES staff and is top-coded at 5.0. When creating the dichotomous variable used in these analyses, a PIR below 1.85 is defined as poor, as this is the poverty guideline used by certain means-tested social programs (US Department of Agriculture, 2008). The coding for this variable is: 0=PIR<1.85 (poor), 0=PIR≥1.85 (nonpoor).

Depressive symptoms

Depressive symptoms are measured using the Patient Health Questionnaire (PHQ), a nine-question version of the Primary Care Evaluation of Mental Disorders (Prime-MD) diagnostic instrument. Nine questions that assess depressive symptoms plus an additional question on overall impairment by these symptoms comprise the NHANES measure. Respondents 18 years and older scored the following questions on a scale of :

- 0 not at all
- 1 several days
- 2 more than half the days
- 3 nearly everyday

The tenth question is on a scale of:

- 0 not at all difficult
- 1 somewhat difficult
- 2 very difficult
- 3 extremely difficult

The questions are:

- Over the last two weeks, how often have you been bothered by the following problems:
  - little interest or pleasure in doing things
  - feeling down, depressed, or hopeless

        - trouble falling or staying asleep, or sleeping too much
        - feeling tired or having little energy
        - poor appetite or overeating
        - feeling bad about yourself – or that you are a failure or have let yourself or your family down
        - trouble concentrating on things, such as reading the newspaper or watching TV
        - moving or speaking so slowly that other people have noticed, or the opposite – being so fidgety or restless that you have been moving around a lot more than usual
        - thoughts that you would be better off dead or of hurting yourself in some way
    - How difficult have these problems made it for you to do your work, take care of things at home, or get along with people?

The scale reliability coefficient, α, is: 0.84.

I create a dichotomous variable based on the gender-specific distribution of the mean of the depressive symptoms scores. The variable is coded as follows: 0=lower three quartiles of the distribution; 1=highest quartile of the distribution. This dichotomous variable is used in the three-way interactions among race, depressive symptoms, and lead.

Because depressive symptoms were only collected during the last wave (2005-2006), analyses including these variables are restricted to this wave. The bone lead algorithm contains occupation, which is only collected in the first two waves (2001-2004). This means that there are no models with depressive symptoms and bone lead. Although the use of depressive symptoms as a marker of psychosocial stress brings with it challenges, including the fact that it is only available in the third wave of my dataset, it is the only marker related to the effects of psychosocial stress available in the NHANES.

Confounding variables

Gender/sex

Sex was determined by the interviewer during the home interview portion of the NHANES. It is coded as a dummy variable with 0=men and 1=women.

Age

Age is the age of the respondent at the time of the screening interview and is top-coded at 85 years.

Smoking.

Smoking is measured in two ways – smoking status and amount smoked. I create a smoking status categorical variable of "never," "former," and "current" using the responses from a combination of questions asked of adults 20 years and older during the home interview. First, respondents were asked if they had smoked 100 cigarettes in their lifetime. If they answered "no," they are coded as "never smokers." If they answered "yes," they were asked the age at which they started smoking regularly. If they responded that they never smoked regularly, they are also coded as "never smokers." Those who answered "yes" to the first question were also asked if they now smoked. If they answered "no" to that question, they are coded as "former smokers." If they answered "everyday" or "some days," they are coded as "current smokers."

The second measure of smoking is a rough estimate of the cumulative smoking amount in pack-years. The continuous variable is calculated as follows:

1. If respondents are coded as a "never smoker" above, then they are coded as "0" pack-years.

2. If respondents are coded as "former smokers" above, the following equation is used to calculate the number of pack-years they had smoked:

   ((age last smoked regularly – age started smoking regularly) * #cigarettes smoked per day when last smoked regularly) / 20

3. If respondents are coded as "current smokers" above, the following equation was used to calculate the number of pack-years they have smoked:

   (#cigarettes smoked per day now * #years smoked this amount) / 20

Body mass index (BMI)

BMI was calculated by NHANES staff on all participants two years and older using the standard formula from height and weight measured during the MEC physician's examination:

$$\text{BMI} = \frac{\text{weight (kilograms)}}{\text{height}^2\ (\text{meters}^2)} \qquad \text{(Equation 3.4)}$$

Alcohol use

Alcohol use was assessed during the in-home interview using a battery of questions and responses were reported for adults aged 20 years and older. The average number of alcoholic beverages consumed per day over the past 12 months was calculated by NHANES staff. If respondents said that they had not had more than 13 drinks in their lifetime, their average weekly alcohol consumption was coded as "0."

Anti-hypertensive medication use

Participants were asked during the home interview, "Are you now taking medication [for high blood pressure]?" I created a dummy variable from this question that equals one if the respondent reported taking medication.

Hematocrit

Hematocrit was measured from whole blood taken during the MEC physician's examination of all participants aged one year and older.

Vitamin D

Vitamin D was measured from serum taken as part of the MEC physician's examination for participants one year and older.

Calcium

Calcium was measured from serum taken as part of the MEC physician's examination in participants 12 years and older.

Iron

Iron was measured from serum taken as part of the MEC physcian's examination in participants 12 years and older.

Phosphorous

Phosphorous was measured from serum taken as part of the MEC physician's examination in participants 12 years and older.

Glomerular filtration rate (GFR)

GFR is the best measure of kidney function, according to the National Kidney Foundation. There are two major formulas used to calculate GFR from serum creatinine and other health and demographic variables. The formula preferred by renal experts is Modification of Diet and Renal Disease (MDRD) Study equation which is:

$$\text{GFR } (^{ml}/_{min}/1.73\ m^2) = 186 * \text{serum creatinine}^{-1.154} * \text{age}^{-0.203} * 0.742 \text{ (if female)} * 1.212 \text{ (if black)} \quad \text{(Equation 3.5)}$$

Because this equation controls for black race, I opted for the second major formula, called the Cockcrost-Gault equation, as is as follows:

$$\text{GFR } (^{ml}/_{min}/) = (140 - \text{age} * \text{weight (in kilograms)} * 0.85 \text{ (if female)}) / (72 * \text{serum creatinine}) \quad \text{(Equation 3.6)}$$

Models are run with and without GFR. If there are no differences, the more parsimonious models are presented.

Sampling and complex design weights and variance units

The NHANES sample represents the total civilian noninstitutionalized population, two months and older residing in the 50 states. A four-stage sample design was used: (1) primary sampling units (PSUs) which are mostly single counties; (2) area segments within PSUs; (3) households within area segments; and (4) persons within households.

There are several weight and design variables that account for complex survey design and must be included for the calculation of unbiased estimates. Sampling weights are required to make population-level inferences about the NHANES sample and reflect the unequal selection probabilities, non-response adjustments, and adjustments to independent population controls. Each wave has its own MEC and interview sampling weight. Because MEC data is used in all analyses, the MEC sampling weights are used. I calculate sampling weights required when combining waves based on the NHANES staff guidelines. The sampling weight for each wave was divided by the total number of waves to be included in the final dataset and then these quotients from the different waves are summed to create the new multi-wave sampling weight.

The design weights adjust the estimates to account for the complex stratification and clustering design of NHANES and are required for proper calculation of standard errors. The NHANES staff includes both stratum and PSU variables used by STATA.

Missing data

As mentioned earlier, there is considerable loss of observations due to missing data. I considered several approaches to dealing with this situation: (1) listwise deletion, (2) dummy variable adjustment, (3) multiple imputation of missing variables, and (4)

omission of variables with many missing data. Listwise deletion is a common way to handle missing data and it is my starting point. As an example, I discuss the analyses that include blood lead and blood pressure, as these analyses are based on all three waves of the NHANES.

There are 9,911 black and white adults 20 years of age and older who participated in the clinic portion of the NHANES 2001-2006. When I include only those who have blood pressure and blood lead variables – the dependent and the focal independent variables, my sample size is reduced by 1,066 to result in a sample size of 8,845. When I include only those who have data on the remaining 11 variables used in the analyses in Hypothesis Set 1, my sample size is reduced by an additional 2,455 to result in a final sample size of 6,390.

Although I have not introduced my analytic approach yet, I discuss my most basic regression model with a two-way interaction, simply to serve as an example of the result of listwise deletion of missing data. Table 3. 2 contains the linear regression results of the following model:

$$\text{Blood pressure} = \beta_0 + \beta_1 \text{race} + \beta_2 \text{lead} + \beta_3 \text{race*lead} \qquad \text{(Equation 3.7)}$$

I have run this model on two samples (stratified by gender): (1) the sample of 8,845 adults and (2) the sample of 6,390 that are not missing any data. The results in Table 3.1 show that there is little difference in these two samples. There is less than half of one mmHg difference in the black-white disparity in the lead-systolic blood pressure association.

I decided not to use the dummy variable adjustment because it has been shown to yield more biased estimates compared to listwise deletion (Allison, 2002). Regarding multiple imputation, I have investigated this approach and the new version of STATA (11.0) has the ability to perform multiple imputation techniques. The major reason that I do not impute the missing data is that, for the time is required (for me as a novice to this technique) it may not produce better results than listwise deletion for this dissertation. However, I plan to investigate to examine this directly by comparing the models with listwise deletion and those with imputed data for manuscript preparation.

A last approach to handling missing data is to examine the covariates with large numbers of missing data. For example, the family poverty-to-income ratio (PIR) variable contains an additional 487 observations from the base of 8,845, resulting in a sample size of 8,358. However, it is a focal variable in the final set of analyses. The alcohol use variable contains an additional 1,318 observations missing, resulting in a sample size of 7,040. I keep alcohol use in my final models to follow the literature on lead and hypertension. However, it is a candidate for either omission or imputation during manuscript preparation. I am confident that altering my final models by excluding or imputing the alcohol use variable does not change the pattern of results or my interpretation. Table 3. 3 shows the results of the linear regression based on Equation 3.7 with and without alcohol use in the model. These results show that these models are similar with regard to black-white comparisons in the association between blood lead and blood pressure. Therefore, for this first documentation of these associations, I opt to use listwise deletion to handle missing variables, with the plan to compare these results with imputed data for manuscript preparation.

Analytic approach

All analyses were conducted in STATA 11.0 which can account for complex survey design. All analyses account for complex survey design (STATA command: svy). I also use robust standard errors, as the regression diagnostic tests revealed slight homoskedasticity of error terms. Most analyses are based on the three most recent waves of the NHANES available at the time of the dissertation prospectus defense, 2001-2006. Due to the availability of certain key variables, some analyses are conducted using either one or two waves. Specifically, any analyses that include estimated bone lead are restricted to the first two waves of the overall dataset (2001-2004) because a key variable in the prediction algorithm, occupation, is only available in these waves. Any analyses that include depressive symptoms are restricted to the last wave of the overall dataset (2005-2006), as this was the only wave in which these variables were collected. I divide my analyses in to two sets. Set 1 contains the analyses for the associations among estimated bone lead, education, poverty status, and hypertension. Set 2 contains the analyses for the associations among blood lead, education, poverty status, depressive symptoms, and blood pressure.

Regarding how best to address the role of gender in these analyses, there are two analytic strategies to consider. One strategy is to interact gender with race. However, I test my central research questions using interactions. Adding an interaction with gender would result in a four-way interaction and add substantial interpretive complexity. An alternative strategy is to stratify by gender. I choose this strategy because my research is fundamentally about race as a powerful social determinant of health through the sorting and stratification of racial groups in the US. Therefore, as a first test of my conceptual

model, I focus on my fundamental ideas on race and health, leaving comparisons between black men and women for future work.

All analyses are weighted to account for complex survey design, as outlined in the NHANES documentation (National Center for Health Statistics, 2006). When using interactions, continuous variables are centered and the categorical race variable is weighted to account for unequal sample sizes. When calculating margins for tables and graphs, all covariates are held at their means.

Descriptive statistics

I estimate means with standard deviations (STATA commands: means; estat sd) for all continuous variables and category percentages with 95% confidence intervals (STATA command: prop) for all categorical variables, stratified by race and gender. I test for racial differences and disparities in means and percentages (STATA command: lincom). To examine bivariate associations between my dependent and focal independent variables, I calculate correlation matrices for all covariates, stratified by race and gender. I use Pearson's correlation coefficient (STATA command: pwcorr) for all continuous variables. For all categorical variables, I create dummy variables for each level and use Spearman's correlation coefficient (STATA command: spearman). I test for significance in correlation pairs using a Bonferroni-corrected test at the $p<0.01$ level.

Multivariate "descriptive" statistics

In addition to simple descriptive statistics, I estimate the margins of several variables to document any black-white disparities. Although disparities have been

documented in these variables in the literature, they have not been established in the NHANES. Specifically, following the literature on blood and bone lead, I estimate black-white disparities in predicted mean blood pressures, blood lead levels, and bone lead levels, and predicted probabilities in hypertension prevalence, controlling for certain variables (Den Hond et al., 2002; S. K. Park et al., 2009; Vupputuri et al., 2003).

For predicted means, I first use linear regression to estimate the mean values, β_1 in Equations 3.8 and 3.9 (STATA command: regress). Then I calculate predicted means for black and white adults post-estimation, holding all covariates at their means (STATA command: margins). Because blood lead is ln-transformed, the geometric mean is estimated taking the black-white difference in ln-transformed lead and exponentiating the result. I test for significant differences between black and white predicted means and probabilities.

For hypertension prevalence, I use logistic regression (STATA command: logistic) to estimate the odds ratio of hypertension, β_1 in Equation 3.8. Predicted probabilities of hypertension for black and white men and women are calculated post-estimation (STATA command: predict), using the Wald test for significance from zero.

$$Y = \beta_0 + \beta_1 \text{race} + \beta_2 \text{covariates} \qquad \text{(Equation 3.8)}$$

where:

Y	=	systolic blood pressure (mmHg) or diastolic blood pressure (mmHg) or hypertension status (absent=0, present=1)
race	=	black race (white=0, black=1)
covariates	=	age, age^2, alcohol use, BMI, educational attainment, GFR, poverty status, smoking status, smoking amount

$$lead = \beta_0 + \beta_1 race + \beta_2 covariates \quad \text{(Equation 3.9)}$$

where: lead = ln-transformed blood lead ($^{\mu g}/_{dl}$) or estimated tibia lead ($^{\mu g}/_{g}$) or estimated patella lead ($^{\mu g}/_{g}$)
race = black race (white=0, black=1)
covariates = age, age^2, alcohol use, BMI, calcium, educational attainment, GFR, hematocrit, iron, phosphorous, poverty status, smoking status, smoking amount, vitamin D

Analytic set 1: Blood lead and blood pressure

In this first set of analyses, I examine whether SES or depressive symptoms increases the harmful effects of blood lead on blood pressure. I accomplish this in two steps. First, I examine if there are black-white disparities in the effect of blood lead on blood pressure. Second, I examine whether SES or depressive symptoms increase this harmful effect for black but not white adults.

1a: Black-white disparities in the association between blood lead and blood pressures

Systolic and diastolic blood pressure is linearly regressed on ln-transformed blood lead, based on the following equation:

$$BP = \beta_0 + \beta_1 race + \beta_2 lead + \beta_3 race*lnlead + \beta_4 covariates \quad \text{(Equation 3.10)}$$

where: BP = systolic blood pressure (mmHg) or diastolic blood pressure (mmHg)
race = black race (white=0, black=1)
lnlead = ln-transformed blood lead ($^{\mu g}/_{dl}$)
covariates = age, age^2, alcohol use, BMI, calcium,

educational attainment, GFR, hematocrit, iron, phosphorous, poverty status, smoking status, smoking amount, vitamin D

The effect of lead on blood pressure for white adults is β_2 in Equation 3.10. The effect of lead for black adults is the sum of β_2 and β_3. Wald tests are run to examine any statistically significant disparities between black and white adults.

1b: Disparities in lead level as the reason for disparities for lead effect

To examine black-white disparities in *level* of lead as the reason for the black-white disparities in the *effect* of lead on blood pressure, Oaxaca-Blinder decompositions are modeled (STATA command: oaxaca) (Blinder, 1973; O'Donnell, 2008; Oaxaca, 1973). This procedure divides the black-white disparity in blood pressure into a part that is explained by the characteristics of black and white adults and a part that that not explained by these characteristics. For example, if the mean systolic blood pressure for black and white men is 125mmHg and 118mmHg, respectively, there is a 7mmHg disparity. The decomposition method divides this disparity into a part that is explained by black-white differences in lead and any other covariates, called "endowments." What is unexplained by these differences is thought to be due to differences in the effects of lead and other covariates.

1c: Education, poverty status, depressive symptoms, and black-white disparities in the blood lead-blood pressure association

I first test the hypothesis that SES, as a marker of constraints and resources in the social environment, explains the positive effect of blood lead on blood pressure among

black but not white adults. I then test the hypothesis that depressive symptoms, as a marker of the allostatic load resulting from psychosocial stress, explain the positive effect of blood lead. Both hypotheses are based on the following equation:

$$BP = \beta_0 + \beta_1 race + \beta_2 lnlead + \beta_3 social + \beta_4 race*lnlead + \beta_5 race*social + \beta_6 social*lnlead + \beta_7 race*social*lnlead + \beta_8 covariates \quad \text{(Equation 3.11)}$$

where:
- BP = systolic blood pressure (mmHg) or diastolic blood pressure (mmHg)
- race = black race (white=0, black=1)
- lead = estimated tibia lead ($^{\mu g}/_{g}$) or estimated patella lead ($^{\mu g}/_{g}$)
- social = educational attainment (≥high school=0, <high school=1) or poverty status (nonpoor=0, poor=1) or depressive symptoms (low=0, high=1)
- covariates = age, age^2, alcohol use, BMI, calcium, educational attainment, GFR, hematocrit, iron, phosphorous, poverty status, smoking status, smoking amount, vitamin D

I calculate the coefficients for each race-by-social marker group using the coefficient combinations outlined in Table 3. 1:

Table 3. 1 Regression coefficient combinations for the calculation of the net effect of lead for each race-by-social marker group

		social marker	
		low education, high poverty, or high depressive symptoms	high education, low poverty, or low depressive symptoms
race	black	$\beta_2 + \beta_4 + \beta_6 + \beta_7$	$\beta_2 + \beta_4$
	white	$\beta_2 + \beta_6$	β_2

I test for significance that the linear combination of each of these coefficient combinations is greater than zero.

Analytic set 2: Bone lead and hypertension

In this second set of analyses, I examine whether SES increases the harmful effect of bone lead on hypertension in black and white adults. As in with the previous set of analyses, this requires two steps. First, I estimate the black-white disparity in the association between bone lead and hypertension. In other words, I test if black but not white adults exhibit the association between bone lead and hypertension. Then I examine whether SES increases this harmful effect. Analyses for this set use NHANES 2001-2004, as these are the years in which occupation, an important component of estimated bone lead, is available.

2a: Black-white disparities in the bone lead-hypertension association

Black-white disparities in the association between estimated tibia and patella lead and hypertension are estimated, based on the following equation:

$$HTN = \beta_0 + \beta_1 race + \beta_2 lead + \beta_3 race*lead + \beta_4 covariates \qquad \text{(Equation 3.12)}$$

where:
- HTN = hypertension status (absent=0, present=1)
- race = black race (white=0, black=1)
- lead = estimated tibia lead ($^{\mu g}/_{g}$) or estimated patella lead ($^{\mu g}/_{g}$)
- covariates = age, age^2, alcohol use, BMI, GFR, hematocrit, smoking status, smoking amount

Rather than stratifying by race, as is generally done in the lead literature, I estimate the race-specific effect of lead through the interaction of race with lead. The effect of lead on blood pressure for white adults is β_2 in Equation 3.12; the effect of lead for black adults is the sum of β_2 and β_3. I run tests for significance to examine any statistically significant disparities between black and white adults (STATA command: lincom).

2b: Education, poverty status, and the black-white disparities in the bone lead-hypertension association

I test the hypothesis that education and poverty status, as markers of constraints and resources in the social environment, explains the positive effect of estimated bone lead on hypertension among black but not white adults, based on the following equation:

$$\begin{aligned} HTN = {} & \beta_0 + \beta_1 race + \beta_2 lead + \beta_3 social \\ & + \beta_4 race*lead + \beta_5 race*social + \beta_6 social*lead \\ & + \beta_7 race*social*lead \\ & + \beta_8 covariates \end{aligned} \qquad \text{(Equation 3.13)}$$

where:

HTN	= hypertension status (absent=0, present=1)
race	= black race (white=0, black=1)
lead	= estimated tibia lead ($^{\mu g}/_{g}$) or estimated patella lead ($^{\mu g}/_{g}$)
social	= educational attainment ($\geq$high school=0, <high school=1) or poverty status (nonpoor=0, poor=1)
covariates	= age, age^2, alcohol use, BMI, GFR, hematocrit, smoking status, smoking amount

I use three-way interactions to test these hypotheses because I examine the interaction between lead and education and poverty status – but education and poverty status interact

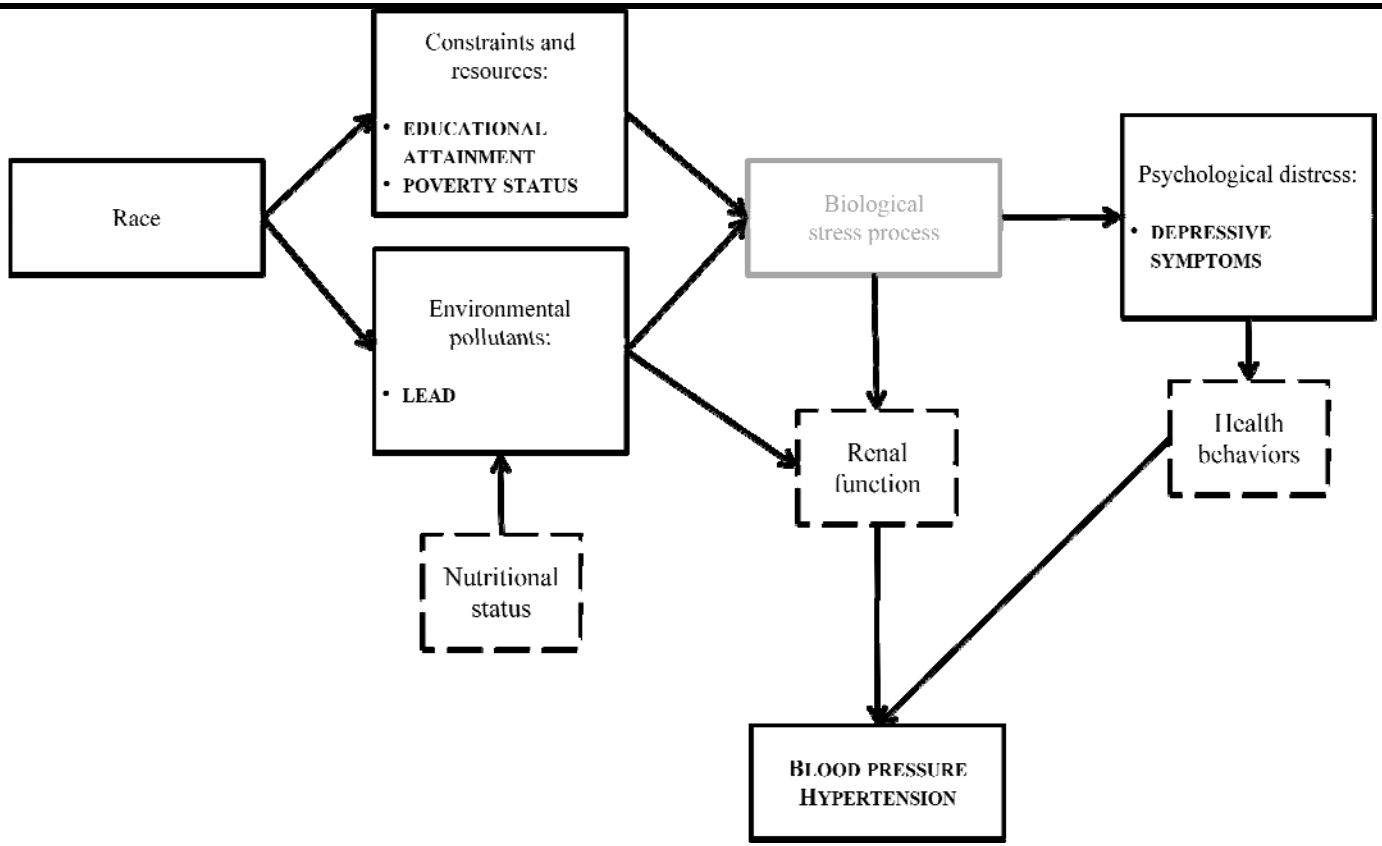
with race. Based on Equation 3.13, I calculate four coefficients for the effect of estimated bone lead as shown in Table 3. 1.

Interaction graphs

I graph the interaction results for all linear and logistic regression results by estimating predicted margins in STATA and then graphing the results in Microsoft Excel 2007. First, in STATA, I estimate predicted probability of hypertension at the following percentiles of the bone lead distribution: 1^{st}, 25^{th}, 50^{th}, 75^{th}, and 99^{th} (STATA command: margins). I then graph the curves, with bone lead on the x-axis and predicted probability of hypertension on the y-axis. I repeat these steps for the graphing of predicted mean systolic and diastolic blood pressures.

In the next two chapters, I present the results from these analyses. First, I present the results from blood pressure and blood lead and second, I present hypertension and estimated bone lead.

Figure 3. 1 Analytic model outlining the associations between race and hypertension that are tested



Solid black boxes represent focal variables; dashed black boxes represent confounding variables; gray box represents variables that cannot be tested.

Table 3. 2 Comparison of respondents with and without missing data on control variables in the lead-SBP association, NHANES 2001-2006

	Men		Women	
	(1)	(2)	(1)	(2)
	N=4552	N=3220	N=4293	N=3170
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	3.5***	4.0***	5.7***	6.0***
	(0.8)	(1.0)	(0.7)	(0.7)
Blood lead	0.3	0.5	-0.7	-1.1
	(0.6)	(0.7)	(0.7)	(0.8)
Interactions				
Race*lead	2.5*	2.1^{+}	3.5***	3.7***
	(1.0)	(1.1)	(0.8)	(1.1)
Intercept	114.4***	114.1***	97.5***	97.8***
	(1.8)	(2.2)	(1.9)	(2.2)

Notes:
All results are weighted to account for complex survey design. All models are adjusted for age and age^2. Models are black and white adults 20 years of age and older, who are not pregnant or lactating, who participated in the NHANES clinic:

(1) who have data on systolic blood pressure and blood lead;
(2) who have data on systolic blood pressure and blood lead AND on the remaining 11 variables used in full analytic models.

Abbreviations are: SE, standard error

[a] Omitted category in parentheses.

$^{+}$p<0.10; *p<0.01; **p<0.05;***p<0.001

Table 3. 3 Comparison of respondents with and without missing data on alcohol use in the lead-SBP association, NHANES 2001-2006

	Men		Women	
	(1)	(2)	(1)	(2)
	N=4316	N=3561	N=4042	N=3479
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	3.6***	4.0***	5.8***	6.2***
	(0.8)	(0.8)	(0.7)	(0.7)
Blood lead	0.4	0.4	-0.8	-1.0
	(0.7)	(0.7)	(0.7)	(0.8)
Interactions				
Race*lead	2.6*	2.3*	3.3***	3.7***
	(1.0)	(1.0)	(0.9)	(1.1)
Intercept	114.7***	114.3***	97.5***	98.3***
	(1.9)	(2.0)	(1.9)	(2.0)

Notes:
All results are weighted to account for complex survey design. All models are adjusted for age and age^2. Models are black and white adults 20 years of age and older, who are not pregnant or lactating, who participated in the NHANES clinic, who have data on systolic blood pressure and blood lead, who:

(1) do not have the alcohol use variable;
(2) do have the alcohol use variable.

Abbreviations are: SE, standard error

[a] Omitted category in parentheses.

$^+$p<0.10; *p<0.01; **p<0.05;***p<0.001

CHAPTER FOUR

RESULTS, PART 1: BLOOD LEAD AND BLOOD PRESSURE

In this chapter, I present the results from my analyses on black-white disparities in blood lead and blood pressure and the role of education, poverty status, and depressive symptoms. There are two lines of results that I present in sequence – first blood pressure and then blood lead. For each, I discuss the descriptive characteristics of my sample, the bivariate associations, and additive multivariate associations. Then I bring these two lines together and present the results on black-white disparities in the association between blood lead and blood pressure (the two-way interactions). Finally, I present the results on the amplifying effects of educational attainment, poverty status, and depressive symptoms, as an explanation for the disparities in this association (the three-way interactions).

Black-white disparities in blood pressure

Table 4.1 contains the descriptive characteristics of the three most recent waves of the National Health and Nutrition Examination Survey (NHANES 2001-2006). Results

that include depressive symptoms are restricted to the most recent wave (2005-2006). This table contains the mean and standard deviations for all continuous variables and percentages for all categorical variables. Results are stratified by race and gender.

Black adults have higher systolic and diastolic blood pressures compared to white adults. Systolic blood pressures are 127.5mmHg and 123.6mmHg for black and white men, respectively. This is a disparity of 3.9mmHg ($p<0.001$). Systolic blood pressures are 125.2mmHg and 122.2mmHg for black and white women – a disparity of 3.0mmHg ($p<0.001$). Diastolic blood pressures are 74.5mmHg and 73.0mmHg for black and white men, which is a disparity of 1.5mmHg ($p<0.05$). Diastolic blood pressures are 72.3mmHg and 70.7mmHg for black and white women – a disparity of 1.6mmHg ($p<0.01$).

Black adults generally have lower levels of hypertension risk factors such as smoking, obesity, and alcohol use compared to white adults. For example, black adults are more likely to have never smoked. In fact, 70% of black women report never smoking, compared to only 57% of white women ($p<0.001$). Although they are less likely to quit once they have started smoking (i.e., be "former smokers"), overall, black adults have smoked about half the number of pack-years as white adults (5.3 and 10.5 pack-years[3] for black and white men, respectively, $p<0.001$; 3.0 and 6.3 pack-years for black and white women, respectively, $p<0.001$). An exception to the pattern of lower levels of risk factors is that black women have higher rates of obesity, as marked by body

[3] A pack-year is a measure of the amount of cigarette smoking over time, determined by multiplying the number of packs a person has smoked per day by the number of years a person has smoked this amount. One pack-year is equivalent to one pack of cigarettes smoked per day for one year. One pack-year is also equivalent to smoking one-half pack per day for six months.

mass index (BMI). Fifty-two percent of black women are obese, while only 26% of white women are in this category ($p<0.001$).

Black adults experience substantially lower educational attainment and higher poverty status than white adults. Black men and women have poverty rates of roughly 39% and 48%, respectively. White men and women have about half these rates at roughly 18% and 23%, respectively ($p<0.001$ for both men and women). However, there are no black-white differences in either mean depressive symptoms or in the likelihood of having high depressive symptoms. About 20% of black and white men report relatively high depressive symptoms. Roughly 32% and 26% of black and white women are in this category (p=NS).

Tables 4.2a and 4.2b contain the correlation matrices of the blood pressure-related factors for men and women, respectively. Pearson's correlation coefficients are reported for all continuous variables. For categorical variables, dummy variables were created for each level and Spearman's correlation coefficients are reported. Bonferroni-adjusted tests for significance are reported at the $p<0.01$ level.

In general, systolic blood pressure is correlated with hypertension risk factors such as age, smoking, obesity, and leanness in expected ways. For example, systolic blood pressure is negatively correlated with leanness ($r=-0.15$, $p<0.01$ for men; $r=0.20$, $p<0.01$ for women) and positively correlated with obesity ($r=0.11$, $p<0.01$ for men; $r=0.15$, $p<0.01$ for women). On the other hand, systolic blood pressure is correlated with smoking in some unexpected directions. While it is positively correlated with pack-years smoked ($r=0.15$, $p<0.01$ for men; $r=0.08$, $p<0.01$ for women), it is negatively correlated with current smoker status ($r=-0.08$, $p<0.01$ for men; $r=-0.16$, $p<0.01$ for women).

Of the measures that I include in the three-way interactions with race and lead (education, poverty status, and depressive symptoms), systolic blood pressure is correlation only with education. It is weakly correlated with lack of a high school education for men and women ($r=0.08$, $p<0.01$ and $r=0.11$, $p<0.01$, respectively), and it is weakly and negatively correlation with post-secondary education, but for women only ($r=-0.11$, $p<0.01$).

In general, diastolic blood pressure is not correlated with the other measures, and when it is correlation, it is only weakly so. Diastolic blood pressure is correlated *negatively* with age ($r=-0.10$, $p<0.01$ for men; $r=-0.08$, $p<0.01$ for women). It is negatively correlated with leanness ($r=-0.13$, $p<0.01$ for men; $r=-0.10$, $p<0.10$ for women) and positively correlated with obesity ($r=0.11$, $p<0.01$ for men; $r=0.01$, $p<0.01$ for women).

In sum, the results show black-white disparities in systolic and diastolic blood pressure. Furthermore, black adults generally have lower levels of hypertension risk factor and these factors are correlated with systolic (but not diastolic) blood pressure. On the other hand, black adults have lower SES measures, but of these measures, only educational attainment is correlated with blood pressure – and only systolic blood pressure. Do these risk factors and SES explain the blood pressure disparities? Tables 4.3a and 4.3b contain the linear regression coefficients for systolic and diastolic blood pressures, respectively, regressed on these factors. Models are stratified by gender. These tables contain only the focal coefficients for race and the intercept. The tests for significance are for differenced from zero. The first model adjusts for age and anti-

hypertensive medication use only. The second model adjusts for hypertension-risk factors and the third model adjusts SES.

For men, there is a large black-white disparity of 5.2mmHg in age-adjusted systolic blood pressure (Table 4.3a, Model 1, $p<0.001$) that is not altered by the addition of hypertension risk factors or SES (Model 2, b=5.4mmHg; Model 3, b=5.0mmHg, respectively). For women, the disparities in blood pressure are similar to those seen in men, however, the addition of hypertension risk factors explain a small portion of these disparities. For example, the 6.1mmHg disparity in systolic blood pressure (Model 1, $p<0.001$) is reduced to 5.0mmHg with the addition of BMI, smoking, and alcohol use; note however that the disparity remains substantial and statistically significant (Model 2). The reduction in disparity is primarily due to the addition of BMI. The addition of SES does not change the disparity (Model 3, b=4.8mmHg).

The pattern of results is similar for diastolic blood pressure. For both men and women, there is a 1.6mmHg disparity (Table 4.3b, Models 1, $p<0.01$). For men, the addition of the risk factors or SES does not change the disparity (Model 2, b=1.6mmHg; Model3, b=1.4mmHg). This is also the case for women (Model 2, b=1.2mmHg, Model 2, b=1.2mmHg).

In sum, there is a black-white disparity in both systolic and diastolic blood pressure that is generally not explained hypertension risk factors or SES. One exception is that, for women, BMI explains a small portion of the disparity in systolic blood pressure.

Black-white disparities in blood lead

Table 4.4 contains the lead-related descriptive characteristics for the NHANES 2001-2006 sample. All results presented are means with their standard deviations, with the exception of blood lead, which is reported as the geometric mean, the mean of the ln-transformed lead values. There are no racial differences in blood lead among men. Among women, racial differences in lead are statistically significant, but these differences are small (1.4 $^{\mu g}/_{dl}$ and 1.2 $^{mg}/_{dl}$ for black and white women, respectively, $p<0.01$). Overall, there are small black-white differences in most of the other biomarkers. There are some large differences – in iron and vitamin D, for example. Black men and women have vitamin D levels around $15^{mg}/_{dl}$, while white men and women have levels of roughly $25^{mg}/_{dl}$ while ($p<0.001$ for black-white difference for both men and women).

Tables 4.5a and 4.5b contain the correlation matrices for lead-related factors for men and women, respectively. Blood lead is correlated with systolic but not diastolic blood lead for both men ($r=0.21$, $p<0.01$) and women ($r=0.30$, $p<0.01$). Blood lead is moderately correlated with numerous other health and demographic factors, particularly for women. It is moderately correlated with age for men ($r=0.43$, $p<0.01$) and women ($r=0.48$, $p<0.01$). It is also correlated with risk factors of lead exposure such as smoking and alcohol use. For example, blood lead is negatively correlated with never smoker status for men ($r=-0.32$, $p<0.01$), but positively correlated for women ($r=0.19$, $p<0.01$).

Blood lead is moderately correlated with both educational attainment and poverty in men, but only weakly correlated with these markers in women. For example, it is negatively correlated with post-secondary education ($r=-0.18$, $p<0.01$) and positively

correlated with poverty status ($r=0.14$, $p<0.01$) for men. It is not correlated with high depressive symptoms for either men or women ($r=0.05$, p=NS for men; $r=0.04$, p=NS for women).

In sum, there are small black-white disparities in crude mean blood lead and these correlations are more clearly present among women than men. Black adults have lower levels of behavior risk factors associated with lead exposure such as smoking and alcohol use compared to white adults. However, they experience substantially lower levels of SES, which are correlated with blood lead to varying degrees. Furthermore, there are several factors, such as age, that are related to both race and blood lead. Does adjusting for these factors like age alter the black-white disparities in blood lead?

Table 4.6 contains the results for ln-transformed blood lead linearly regressed on these factors for men and women separately. I present only the focal coefficients of race and the intercept. These coefficients have been exponentiated to be interpretable as untransformed lead values. The first model adjusts for age and serum biomarkers only. The second model adds the lead-related (and hypertension-related) risk factors. The third model adds SES.

There is a small disparity in blood lead of $1.2^{\mu g}/_{dl}$ (Model 1, $p<0.001$) for men. The disparity decreases slightly to $1.1^{\mu g}/_{dl}$ with the addition of risk factors (Model 2, $p<0.001$) and does not change further with the addition of SES (Model 3, $p<0.001$). For women, the disparity is also small at $1.3^{\mu g}/_{dl}$ (Model 1). This disparity does not change with adjustment for risk factor and SES (Models 2 and 3). In sum, there are small black-white disparities in blood lead.

Black-white disparities in the association between blood lead and systolic blood pressure

Although there are small black-white disparities in blood lead levels, the levels for both black and white adults are low. What does this mean for the disparity in the *effect* of blood lead that had been documented in the literature? In order to address this question, I interact race with blood lead in my regression models to get race-specific effects of blood lead on blood pressure. The results in Tables 4.7a and 4.7b contain the results for systolic and diastolic blood pressure regressions, respectively. Results are stratified by gender. The tables contain the focal coefficients of race, blood lead, the interaction, and the intercept. The change in blood pressure due to lead is estimated by the following model:

$$\text{Blood pressure} = \beta_0 + \beta_1 \text{race} + \beta_2 \text{lead} + \beta_3 \text{race*lead} \qquad \text{(Equation 4.1)}$$

White adults are the reference category for race. Hence, β_0 represents the conditional mean blood pressure for white adults, and β_2 represents the change in blood pressure due to lead for white adults. The sum of β_2 and β_3 is the change in blood pressure due to lead for black adults.

Table 4.7a shows that white men do not have an association between blood lead and systolic blood pressure in any model (the "blood lead" term in Table 4.7a) On the other hand, black men have a stronger association as indicated by the significant interaction term (Model 1, $b=2.5$, $p<0.05$). For black men, there is a net 3.1mmHg increase in systolic blood pressure for each doubling of blood lead ($\beta_2 + \beta_3$ in Equation

4.1; Table 4.7a, Model 1, b=0.6 + 2.5). Neither the overall blood lead-systolic blood pressure association nor the black-white disparity in this association changes with the adjustment for hypertension risk factors or SES.

Figure 4.1 depicts the black-white disparity in the blood lead-systolic blood pressure association for men and women. Along the x-axis is the level of ln-transformed blood lead at certain points in its gender-specific distribution: 1st, 25th, 50th, 75th, and 99th. Along the y-axis is the predicted mean systolic blood pressure. The gray line represents white men and women and the black line represents black men and women. These slopes correspond to the results under Model 3 in Table 4.7a. This figure shows that black men and women experience an increase in systolic blood pressure with blood lead while white men and women do not.

For diastolic blood pressure, there is no interaction between lead and race (see Table 4.7b). For men, when risk factors and SES are included (Table 4.7b, Model 3), there is an interaction that is marginally statistically significant (b=1.4; p=0.104). However, because there are no models that reached statistical significance at $p<0.10$, I do no examine diastolic blood pressure in any further analyses.

It may be that the slightly higher *level* of blood lead accounts for the stronger *effect* of blood lead among black adults. To examine this possibility, I divide the black-white difference in mean systolic blood pressure into the different parts explained by blood lead. One part is the difference in systolic blood pressure due to differences in the *level* of blood lead. The other part is the difference in systolic blood pressure due to the differences in the *effect* of blood lead. Table 4.8 contains the results of the Oaxaca-Blinder decomposition of means, the method I use to divide the difference in systolic

blood pressure. Although only women show a statistically-significant disparity in blood lead, I present decomposition results for both men and women for completeness.

Under the heading "Systolic blood pressure" is the unadjusted mean systolic blood pressure for black and white adults. For example, the mean systolic blood pressures are 125.2mmHg and 123.2mmHg for black and white women, respectively. Next is the black-white difference in mean systolic blood pressure. For example, the black-white difference in systolic blood pressure is 125.2-123.2=3.0mmHg. It is *this* difference that is then divided into parts explained by the level of lead and effect of lead.

The row marked "Level (endowments)" shows how much systolic blood pressure would change if black adults had the same levels of the variables (age, lead, hematocrit, calcium, vitamin D, iron, phosphorous and anti-hypertensive medication use) as white adults. These results show that there would be *no change* for black adults if they had the same levels of these variables as white adults (0.0mmHg p=NS for men; -0.2mmHg p=NS for women).

The row marked "Effect (coefficients)" shows how much systolic blood pressure would change if black adults had the same effects of those variables as white adults. The results show that systolic blood pressure for black men and women would decrease by 4.1mmHg ($p<0.001$) and 5.5mmHg ($p<0.001$) if they had the same effects of these variables as white men and women, respectively.

The remaining two rows show how much the systolic blood pressure of black adults would change if *blood lead specifically* were changed. For black men, there would be no change in systolic blood pressure if they had either the same level or same effect of the blood lead as white men. For black women, there would be a tiny decrease in systolic

blood pressure of 0.3mmHg if they had the same *level* of blood lead as white women ($p<0.05$). On the other hand, black women would experience a small decrease 1.2mmHg ($p<0.01$) if they had the same *effect* of lead as white women.

In sum, the results from this section indicate that black but not white adults exhibit a positive association between blood lead and systolic blood pressure that is not due to hypertension risk factors, SES. This effect of lead is also not due simply to the fact that black adults have higher levels of blood lead.

Education, poverty status, and depressive symptoms and black-white disparities in the lead-blood pressure association

The results thus far show that black-white disparities in the *level* of SES do not explain the black-white disparities in the association between blood lead and blood pressure. However, it may be that there are black-white disparities in the *meaning* of SES that explain the association disparities. For example, for those with the same educational attainment, black men have lower wages and lower wealth compared to white men (Williams, 1999). Evidence suggests that there is also a difference in the meaning of depressive symptoms (Davidson et al., 2000; Jonas et al., 1997). To account for this disparity in meaning, I interact race with educational attainment, poverty status, and depressive symptoms (in separate models). This results in a three-way interaction with lead to further address the question of whether the social environment explains the stronger effect of blood lead on systolic blood pressure among black adults. This is shown by the following equation:

$$SBP = \beta_0 + \beta_1 race + \beta_2 lead + \beta_3 social + \beta_4 race*lead + \beta_5 social*lead + \beta_6 race*social + \beta_7 race*social*lead \quad \text{(Equation 4.2)}$$

where: SBP = systolic blood pressure
social = educational attainment (≥high school=0, <high school=1) or
poverty status (nonpoor=0, poor=1) or
depressive symptoms (low=0, high=1)

Tables 4.9a through 4.9c show the results of the linear regression models with the three-way interaction of race-by-lead-by-social marker, stratified by gender, using: educational attainment (Table 4.9a), poverty status (Table 4.9b), and depressive symptoms (Table 4.9c). Each of these three measures is dichotomized to "poor" and "nonpoor" categories. The "poor" categories are: low educational attainment, poverty status, or high depressive symptoms. These tables contain the coefficients only for the focal variables of race, educational attainment, poverty status, depressive symptoms, blood lead, the interactions, and the intercept. Model 1 adjusts for age, serum biomarkers, and anti-hypertensive medication use. Model 2 further adjusts for the hypertension risk factors of smoking, BMI, and alcohol use.

Figures 4.2 and 4.3 illustrate these interactions for men and women, respectively. Each figure contains three graphs – one for each of educational attainment, poverty status, and depressive symptoms. Along the x-axes is ln-transformed blood lead at the following percentiles of the gender-specific distribution: 1^{st}, 25^{th}, 50^{th}, 75^{th}, and 99^{th}. Along the y-axes is the predicted mean systolic blood pressure. The gray lines represent white men and women while the black lines represent black men and women. The solid lines represent the "nonpoor" social marker while the dashed lines represent the "poor" social marker. These slopes are adjusted for age, serum biomarkers, anti-hypertensive

medication use, BMI, smoking, and alcohol use and correspond to Model 2 in Tables 4.9a and 4.9b. Slopes for depressive symptoms correspond to the only model in Table 4.9c and do not account for BMI, smoking, and alcohol use, due to sample size restrictions.

In Figure 4.2, the black dashed line, representing black men with a poor social marker, is steeper for black men without a high school education (Figure 4.2a) and black men in the highest quartile of the depressive symptoms distribution (Figure 4.2c). There are no differences in the slopes for black men who are poor and nonpoor with regard to poverty status (Figure 4.2b).

The results are similar for women, shown in Figure 4.3. Black women with low educational attainment (Figure 4.3a, black dashed line) have a slightly steeper slope compared to black women with a higher educational attainment (black solid line). The slope for black women in the highest quartile of depressive symptoms is substantially steeper (Figure 4.3c, black dashed line) compared to black women in the lower quartiles (black solid line). There are no differences in the slopes for black women who are poor and nonpoor with regard to poverty status (Figure 4.3b). Although there are some exceptions, such as poverty status, the general pattern of results suggests that black adults with poor social markers show a stronger effect of lead on systolic blood pressure.

Summary

Blood lead levels are low for both black and white adults and the disparity is small. Nevertheless, black but not white adults show a marked association between blood lead and systolic blood pressure. Both black and white adults show similar

associations between blood lead and diastolic blood pressure. Black men show a stronger association compared to white men, but this difference is marginally statically significant.

Hypotheses are partially supported, particularly for men. When examining the explanatory role of education, poverty, and depressive symptoms, the results are mixed. For black men, low education and high depressive symptoms resulted in a stronger blood lead-systolic blood pressure association compared to high education and low depressive symptoms, respectively. For black women, only high depressive symptoms yielded this pattern. For both black men and women, both poor and nonpoor adults showed similar blood lead-systolic blood pressure associations.

Table 4. 1 Blood pressure-related health and demographic characteristics, NHANES 2001-2006

	Men			Women		
	Black (N=842)	White (N=2378)	p[a]	Black (N=860)	White (N=2310)	p
Systolic blood pressure, mmHg, mean (SD)	127.5 (18.9)	123.6 (9.9)	***	125.2 (21.8)	122.2 (13.0)	***
Diastolic blood pressure, mmHg, mean (SD)	74.5 (14.8)	73.0 (7.6)	*	72.3 (12.8)	70.7 (7.2)	**
Hypertension-related factors						
Age, years, mean (SD)	41.6 (16.4)	46.3 (10.4)	***	43.3 (15.8)	48.9 (10.7)	***
Anti-htn meds use, %	20	18		29	21	***
Smoking, pack-years, mean (SD)	5.3 (13.5)	10.5 (12.8)	***	3.0 (9.1)	6.3 (8.8)	***
Smoking status, %						
never	55	46	***	70	57	***
former	15	29	***	12	22	***
current	30	25	*	18	21	
BMI, %						
<25 $^{kg}/_{m^2}$	31	28		21	42	***
25-29.9 $^{kg}/_{m^2}$	36	42	**	27	26	
≥30 $^{kg}/_{m^2}$	33	31		52	31	***
Alcohol, drinks/week, mean (SD)	1.4 (2.2)	1.8 (1.5)	***	0.5 (1.2)	0.9 (1.0)	***
Socioeconomic status						
Education, %						
<High school	28	10	***	24	11	***
High school dipl	57	57		59	60	
>High school	15	33	***	17	29	***
Family PIR <1.85, %	39	18	***	48	23	***
Depressive symptoms						
Depressive symptoms[b], mean (SD)	0.2 (0.4)	0.2 (0.2)		0.4 (0.5)	0.3 (0.3)	
High depressive symptoms, %	18	19		32	26	

Notes: All results are weighted to account for complex survey design. Abbreviations are: SD, standard deviation; BMI, body mass index; dpl, diploma; htn, hypertension; PIR, poverty-to-income ratio.

[a] Test for black-white difference different from zero: $^{+}p<0.10$; *p<0.05; **p<0.01; ***p<0.001

[b] Depressive symptoms are available only in the 2005-2006 wave, reducing the sample sizes to: black men, 315; white men, 782; black women, 322; white women, 748

Table 4. 2a Blood pressure-related factors correlation matrix for men (N=3220), NHANES 2001-2006

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Systolic BP	1.00								
(2)	Diastolic BP	0.33*	1.00							
(3)	Age	0.37*	-0.10*	1.00						
(4)	AH meds use	0.23*	0.00	0.41*	1.00					
(5)	Pack-yrs smoked	0.15*	-0.08*	0.37*	0.15*	1.00				
(6)	Never smoker	-0.06	0.05	-0.18*	-0.08*	-0.90*	1.00			
(7)	Former smoker	0.14*	-0.05	0.40*	0.19*	0.60*	-0.59*	1.00		
(8)	Current smoker	-0.08*	-0.01	-0.21*	-0.11*	0.40*	-0.52*	-0.37*	1.00	
(9)	BMI $<25^{kg}/_{m}{}^{2}$	-0.15*	-0.13*	-0.12*	-0.16*	0.01	-0.04	-0.12*	0.17*	1.00
(10)	BMI 25-29.9$^{kg}/_{m}{}^{2}$	0.03	0.01	0.10*	-0.01	0.01	0.01	0.07*	-0.09*	-0.53*
(11)	BMI $\geq 30^{kg}/_{m}{}^{2}$	0.11*	0.11*	0.01	0.17*	-0.02	0.04	0.04	-0.08*	-0.42*
(12)	Alcohol use	0.07*	0.01	0.13*	-0.02	0.14*	-0.21*	0.09*	0.14*	0.04
(13)	>High school	0.09*	-0.06	0.10*	0.05	0.15*	-0.15*	0.01	0.16*	0.10*
(14)	=High school	-0.02	0.03	-0.12*	-0.01	0.04	-0.04	-0.03	0.08*	-0.06
(15)	<High school	-0.06	0.02	0.04	-0.03	-0.18*	0.18*	0.03	-0.24*	-0.02
(16)	Poverty status	0.02	0.00	-0.05	0.00	0.08*	-0.09*	-0.08*	0.19*	0.13*
(17)	High dep symp[a]	-0.05	0.08	-0.07	-0.04	0.06	0.11*	-0.12*	0.01	0.13
		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	
(10)	BMI 25-29.9$^{kg}/_{m}{}^{2}$	1.00								
(11)	BMI $\geq 30^{kg}/_{m}{}^{2}$	-0.54*	1.00							
(12)	Alcohol use	0.07	-0.12*	1.00						
(13)	>High school	-0.07	-0.03	-0.07*	1.00					
(14)	=High school	0.01	0.05	-0.01	-0.53*	1.00				
(15)	<High school	0.05	-0.03	0.07*	-0.29*	-0.66*	1.00			
(16)	Poverty status	-0.09*	-0.03	-0.10*	0.30*	-0.01	-0.25*	1.00		
(17)	High dep symp	0.00	-0.04	0.03	0.01	0.08	0.04	-0.11*	1.00	

Notes: Pearson's r reported for continuous variables; Spearman's ρ reported for dichotomous variables. Abbreviations: BP, blood pressure; AH meds, anti-hypertensive medication use; pk-yrs, pack-years; BMI, body mass index; dep symp, depressive symptoms. Bonferroni-adjusted test for difference from zero, *p<0.01 [a] High depressive symptoms are only available in the 2005-2006 wave.

Table 4.2b Blood pressure-related factors correlation matrix for women (N=3170), NHANES 2001-2006

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Systolic BP	1.00								
(2)	Diastolic BP	0.28*	1.00							
(3)	Age	0.57*	-0.08*	1.00						
(4)	AH meds use	0.39*	0.05	0.45*	1.00					
(5)	Pack-yrs smoked	0.08*	-0.04	0.19*	0.01	1.00				
(6)	Never smoker	0.04	0.01	0.00	0.02	-0.95*	1.00			
(7)	Former smoker	0.11*	0.01	0.22*	0.09*	0.62*	-0.63*	1.00		
(8)	Current smoker	-0.16*	-0.02	-0.22*	-0.12*	0.54*	-0.59*	-0.25*	1.00	
(9)	BMI $<25^{kg}/_{m^2}$	-0.20*	-0.10*	-0.11*	-0.19*	0.01	-0.03	-0.02	0.07	1.00
(10)	BMI 25-29.9$^{kg}/_{m^2}$	0.06	0.00	0.12*	0.02	0.00	0.01	0.03	-0.04	-0.46*
(11)	BMI $\geq 30^{kg}/_{m^2}$	0.15*	0.10*	-0.01	0.17*	-0.01	0.03	0.00	-0.03	-0.57*
(12)	Alcohol use	0.02	0.05	0.05*	-0.16*	0.12*	-0.23*	0.14*	0.14*	0.20*
(13)	>High school	0.11*	-0.04	0.14*	0.12*	0.06	-0.05	-0.03	0.10*	-0.08*
(14)	=High school	0.01	0.01	-0.02	-0.01	0.06	-0.06	0.01	0.06	-0.08*
(15)	<High school	-0.11*	0.02	-0.09*	-0.10*	-0.12*	0.11*	0.01	-0.16*	0.16*
(16)	Poverty status	0.03	-0.07	-0.02	0.04	0.04	-0.04	-0.11*	0.17*	-0.07*
(17)	High dep symp[a]	0.03	0.04	-0.02	0.04	0.08	-0.10	-0.03	0.16*	-0.07
		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	
(10)	BMI 25-29.9$^{kg}/_{m^2}$	1.00								
(11)	BMI $\geq 30^{kg}/_{m^2}$	-0.47*	1.00							
(12)	Alcohol use	-0.01	-0.18*	1.00						
(13)	>High school	0.02	0.06	-0.15*	1.00					
(14)	=High school	0.02	0.06	-0.04	-0.55*	1.00				
(15)	<High school	-0.04	-0.12*	0.19*	-0.25*	-0.66*	1.00			
(16)	Poverty status	-0.03	0.10*	-0.20*	0.29*	0.02	-0.28*	1.00		
(17)	High dep symp	-0.03	0.09	-0.03	0.11	0.02	-0.11	0.21*	1.00	

Notes: Pearson's r reported for continuous variables; Spearman's ρ reported for dichotomous variables. Abbreviations: BP, blood pressure; AH meds, anti-hypertensive medication use; pk-yrs, pack-years; BMI, body mass index; dep symp, depressive symptoms. Bonferroni-adjusted test for difference from zero, *p<0.01; [a] High depressive symptoms are only available in the 2005-2006 wave.

Table 4. 3a Black-white disparities in mean systolic blood pressure, NHANES 2001-2006

	Men (N=3220)			Women (N=3170)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	5.2***	5.4***	5.0***	6.1***	5.0***	4.8***
	(0.6)	(0.6)	(0.6)	(0.8)	(0.8)	(0.8)
Intercept	114.1	113.8	113.9	99.7	99.9	99.9
	(2.1)	(2.1)	(2.5)	(2.1)	(2.2)	(2.5)
F-statistic (df)	138.21 (4)	56.27 (10)	42.29 (13)	671.44 (4)	226.40 (10)	178.21 (13)
R^2	0.14	0.16	0.16	0.35	0.37	0.37

Notes:
All results are weighted to account for complex survey design.
Models sequentially add:
(1) Age (age and age^2); anti-hypertensive medication use;
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
*p<0.05; **p<0.01; ***p<0.001

Table 4.3b Black-white disparities in mean diastolic blood pressure, NHANES 2001-2006

	Men (N=3220)			Women (N=3170)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	1.6**	1.6**	1.4**	1.6**	1.2*	1.2*
	(0.5)	(0.5)	(0.6)	(0.5)	(0.5)	(0.6)
Intercept	44.9	45.0	43.3	40.9	40.5	40.6
	(2.4)	(2.4)	(2.6)	(1.6)	(1.6)	(1.6)
F-statistic (df)	40.32 (4)	22.06 (10)	18.33 (13)	108.07 (4)	22.06 (10)	32.47 (13)
R^2	0.11	0.13	0.13	0.13	0.13	0.14

Notes:
All results are weighted to account for complex survey design.
Models sequentially add:
(1) Age (age and age^2); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, 25-29.9 $^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
*p<0.05; **p<0.01; ***p<0.001

Table 4. 4 Blood lead-related biomarker characteristics, NHANES 2001-2006

	Men			Women		
	Black (N=842)	White (N=2378)	p[a]	Black (N=860)	White (N=2310)	p
Blood lead						
Blood lead, $^{\mu g}/_{dl}$	1.9	1.8		1.4	1.2	**
	(2.1)	(1.5)		(1.9)	(1.5)	
Serum biomarkers						
Hematocrit, %	44.3	45.5	***	38.5	40.7	***
	(4.1)	(2.2)		(3.7)	(2.0)	
Phosphorus, $^{mg}/_{dl}$	3.8	3.7	+	3.8	3.8	
	(0.6)	(0.4)		(0.5)	(0.3)	
Vitamin D, $^{ng}/_{ml}$	15.0	25.4	***	13.9	25.6	***
	(7.6)	(5.4)		(7.1)	(6.2)	
Calcium, $^{mg}/_{dl}$	9.6	9.5	***	9.5	9.5	
	(0.4)	(0.2)		(0.4)	(0.2)	
Iron, $^{\mu g}/_{dl}$	86.0	96.6	***	69.3	85.0	***
	(38.6)	(24.1)		(34.1)	(22.8)	
GFR, $^{ml}/_{min}$	112.7	117.7		119.3	101.1	***
	(44.0)	(25.6)	*	(46.6)	(24.0)	

Notes:
All results are weighted to account for complex survey design.
All results are means with standard deviations, with the exception of blood lead, which is the geometric mean.
[a] Tests for black-white difference different from zero: $^{+}p<0.10$; *p<0.05; **p<0.01; ***p<0.001

Table 4. 5a Lead-related health and demographic correlation matrix for men (N=3220), NHANES 2001-2006

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Blood lead	1.00								
(2)	Systolic BP	0.21*	1.00							
(3)	Diastolic BP	0.03	0.33*	1.00						
(4)	Age	0.43*	0.37*	-0.10	1.00					
(5)	Hematocrit	-0.07	-0.11*	0.17*	-0.29*	1.00				
(6)	Phosphorous	-0.08*	-0.12*	-0.03	-0.21*	-0.01	1.00			
(7)	Vitamin D	0.00	-0.10*	-0.08*	0.05	0.12*	0.01	1.00		
(8)	Calcium	-0.07*	-0.06	0.01	-0.20*	0.22*	0.20*	0.08	1.00	
(9)	Iron	0.01	-0.08*	0.03	-0.08	0.25*	-0.07*	0.07*	0.10*	1.00
(10)	Pack-yrs smoked	0.28*	0.15*	-0.08	0.37*	-0.09	-0.06	0.07*	-0.06	0.00
(11)	Never smoker	-0.32*	-0.06	0.05	-0.18*	-0.02	0.00	-0.05	0.03	-0.03
(12)	Former smoker	0.17*	0.14*	-0.05	0.40*	-0.13*	-0.10*	0.10*	-0.09*	0.00
(13)	Current smoker	0.19*	-0.08*	-0.01	-0.21*	0.17*	0.10*	-0.05	0.06	0.04
(14)	BMI 25-29.9 $^{kg}/_{m^2}$	0.02	0.03	0.01	0.10*	-0.02	-0.05	0.06	-0.03	0.00
(15)	BMI ≥ 30 $^{kg}/_{m^2}$	-0.04	0.11*	0.11*	0.01	0.02	-0.02	-0.12*	-0.07	-0.09*
(16)	Alcohol use	0.26*	0.07*	0.01	0.13*	0.00	0.01	0.08*	-0.01	0.14*
(17)	GFR	-0.36*	-0.19*	0.14*	-0.68*	0.22*	0.11*	-0.06	0.04	0.04
(18)	=High school	-0.01	-0.02	0.03	-0.12*	0.08*	0.01	-0.01	0.03	0.00
(19)	<High school	-0.18*	-0.06	0.02	0.04	-0.03	-0.02	0.07	-0.04	0.03
(20)	Poverty status	0.14*	0.02	0.00	-0.05	0.02	0.01	-0.12*	0.04	-0.03
(21)	High dep symp	0.05	-0.05	0.08	-0.07	0.04	0.06*	-0.03	0.00	0.03

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: BP, blood pressure; pk-yrs, pack-years; BMI, body mass index; GFR, glomerular filtration rate; dep symp, depressive symptoms.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 4.5a, cont'd Lead-related health and demographic correlation matrix for men (N=2121), NHANES 2001-2004

		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
(10)	Pack-yrs smoked	1.00								
(11)	Never smoker	-0.90*	1.00							
(12)	Former smoker	0.60*	-0.59*	1.00						
(13)	Current smoker	0.40*	-0.52*	-0.37*	1.00					
(14)	BMI 25-29.9 $^{kg}/_{m}{}^{2}$	0.01	0.01	0.07*	-0.09*	1.00				
(15)	BMI ≥30 $^{kg}/_{m}{}^{2}$	-0.02	0.04*	0.04	-0.08*	-0.54*	1.00			
(16)	Alcohol use	0.14*	-0.21*	0.09*	0.14*	0.07	-0.12*	1.00		
(17)	GFR	-0.19*	0.11	-0.25*	0.14*	-0.09*	0.36*	0.00	1.00	
(18)	=High school	0.04	-0.04*	-0.03	0.08*	0.01	0.05	-0.01	0.14*	1.00
(19)	<High school	-0.18*	0.18*	0.03	-0.24*	0.05	-0.03	0.07*	-0.03	-0.66*
(20)	Poverty status	0.08*	-0.09	-0.08*	0.19*	-0.09*	-0.03	-0.10*	-0.04	-0.01
(21)	High dep symp	0.11	-0.12*	0.01	0.13*	-0.04	0.03	0.01	0.06	0.04

		(19)	(20)	(21)
(19)	>High school	1.00		
(20)	Poverty status	-0.25*	1.00	
(21)	High dep symp	-0.11	0.12	1.00

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: BP, blood pressure; pk-yrs, pack-years; BMI, body mass index; GFR, glomerular filtration rate; dep symp, depressive symptoms.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 4.5b Lead-related health and demographic correlation matrix for women (N=3170), NHANES 2001-2006

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Blood lead	1.00								
(2)	Systolic BP	0.30*	1.00							
(3)	Diastolic BP	0.06*	0.28*	1.00						
(4)	Age	0.48*	0.57*	-0.08	1.00					
(5)	Hematocrit	0.12*	0.01	0.14*	0.05	1.00				
(6)	Phosphorous	0.11*	-0.01	-0.04	0.07	-0.01	1.00			
(7)	Vitamin D	-0.10*	-0.09*	-0.07	0.01	0.15*	0.06	1.00		
(8)	Calcium	0.14*	0.10*	0.01	0.14*	0.19*	0.18*	0.10*	1.00	
(9)	Iron	0.00	-0.09*	-0.01	-0.04	0.32*	-0.03	0.20*	0.12*	1.00
(10)	Pack-yrs smoked	0.22*	0.08	-0.04	0.19	0.09*	0.07	-0.04	0.01	0.01
(11)	Never smoker	0.19*	0.02	0.05	0.05	0.03*	0.01	0.12	-0.01	0.14
(12)	Former smoker	0.14*	0.11*	0.01	0.22*	-0.01	0.00	0.07	0.01	0.02
(13)	Current smoker	0.14*	-0.16*	-0.02	-0.22*	0.16*	0.08*	-0.06	-0.03	0.03
(14)	BMI 25-29.9 $^{kg}/_{m}{}^{2}$	0.08*	0.06	0.00	0.12*	0.03	-0.02	0.02	0.06	0.02
(15)	BMI ≥ 30 $^{kg}/_{m}{}^{2}$	-0.05	0.15*	0.10*	-0.01	-0.04	-0.06	-0.31*	-0.11*	-0.19*
(16)	Alcohol use	0.19*	0.02	0.05	0.05	0.03	0.01	0.12*	-0.01	0.14*
(17)	GFR	-0.38*	-0.30*	0.13*	-0.68*	-0.04	-0.12*	-0.22*	-0.20*	-0.13*
(18)	=High school	-0.04	0.01	0.01	-0.02	0.03	-0.01	0.01	-0.01	0.00
(19)	<High school	-0.07*	-0.11*	0.02	-0.09*	0.01	0.01	0.13*	0.01	0.06
(20)	Poverty status	0.09*	0.03	-0.07	-0.02	-0.08*	-0.02	-0.18*	0.00	-0.12*
(21)	High dep symp	0.04	0.03	0.04	-0.02	-0.04	0.00	-0.13*	-0.04	-0.06

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: BP, blood pressure; pk-yrs, pack-years; BMI, body mass index; GFR, glomerular filtration rate; dep symp, depressive symptoms.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 4.5b, cont'd Lead-related health and demographic correlation matrix for women (N=3170), NHANES 2001-2006

		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
(10)	Pack-yrs smoked	1.00								
(11)	Never smoker	0.12*	1.00							
(12)	Former smoker	0.62*	-0.63*	1.00						
(13)	Current smoker	0.54*	-0.59*	-0.25*	1.00					
(14)	BMI 25-29.9 $^{kg}/_{m}{}^{2}$	0.00	0.01	0.03	-0.04	1.00				
(15)	BMI ≥30 $^{kg}/_{m}{}^{2}$	-0.01	0.03	0.00	-0.03	-0.47*	1.00			
(16)	Alcohol use	0.12*	-0.23*	0.14*	0.14*	-0.01	-0.18*	1.00		
(17)	GFR	-0.02	-0.02	-0.13*	0.15*	-0.11*	0.45*	0.03	1.00	
(18)	=High school	0.06	-0.06	0.01	0.06	0.02	0.06	-0.04	0.07	1.00
(19)	<High school	-0.12*	0.11*	0.01	-0.16*	-0.04	-0.12*	0.19*	-0.01	-0.66*
(20)	Poverty status	0.04	-0.04	-0.11*	0.17*	-0.03	0.10*	-0.20*	0.04	0.02
(21)	High dep symp	0.08	-0.10	-0.03	0.16*	-0.03	0.09	-0.03	0.05	0.02
		(19)	(20)	(21)						
(19)	>High school	1.00								
(20)	Poverty status	-0.28*	1.00							
(21)	High dep symp	-0.11	0.21*	1.00						

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: BP, blood pressure; pk-yrs, pack-years; BMI, body mass index; GFR, glomerular filtration rate; dep symp, depressive symptoms.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 4. 6 Black-white disparities in mean blood lead, NHANES 2001-2006

	Men (N=3220)			Women (N=3170)		
	(1) Age, bio	(2) + Risk	(3) + Risk, SES	(1) Age, bio	(2) + Risk	(3) + Risk, SES
	exp(b)[b] (SE)	exp(b) (SE)	exp(b) (SE)	exp(b) (SE)	exp(b) (SE)	exp(b) (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	1.2***	1.1***	1.1***	1.3***	1.3***	1.3***
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
Intercept	0.3	0.3	0.3	0.1	0.1	0.1
	(1.5)	(1.5)	(1.4)	(1.4)	(1.3)	(1.3)
F-statistic (df)	89.85 (9)	99.63 (15)	93.24 (18)	107.11 (9)	101.22 (15)	81.24 (18)
R^2	0.18	0.30	0.33	0.28	0.38	0.39

Notes:
All results are weighted to account for complex survey design.
Models sequentially add:
(1) Age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, phosphorous, iron); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, 25-29.9 $^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week);
(3) Family poverty-to-income ratio: ≤ 1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
[b] All coefficients are exponentiated to provide the geometric mean values of the original ln-transformed blood lead.
*p<0.05; **p<0.01; ***p<0.001

Table 4. 7a Black-white disparities in the association between blood lead and systolic blood pressure, NHANES 2001-2006

	Men (N=3220)			Women (N=3170)		
	(1) Age, bio	(2) + Risk	(3) + Risk, SES	(1) Age, bio	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	2.5*	2.6**	2.2*	4.2***	3.6***	3.4***
	(1.0)	(0.9)	(1.0)	(1.0)	(0.9)	(1.0)
Blood lead	0.6	0.4	0.2	-0.8	-0.4	-0.4
	(0.7)	(0.7)	(0.7)	(0.8)	(0.8)	(0.8)
Interaction						
Black race*lead	2.5*	3.0*	3.0*	3.5**	3.7***	3.6***
	(1.2)	(1.1)	(1.1)	(1.1)	(1.0)	(1.1)
Intercept	102.2	98.7	98.8	89.9	86.5	86.2
	(7.9)	(7.7)	(8.1)	(10.4)	(10.1)	(10.3)
F-statistic (df)	56.60 (11)	39.40 (17)	37.67 (20)	238.46 (11)	137.42 (17)	108.21 (20)
R^2	0.15	0.17	0.17	0.36	0.37	0.39

Notes: All results are weighted to account for complex survey design.
Models sequentially add:
(1) Age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, phosphorous, iron); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, 25-29.9 $^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week);
(3) Family poverty-to-income ratio: $\leq$1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
*p<0.05; **p<0.01; ***p<0.001

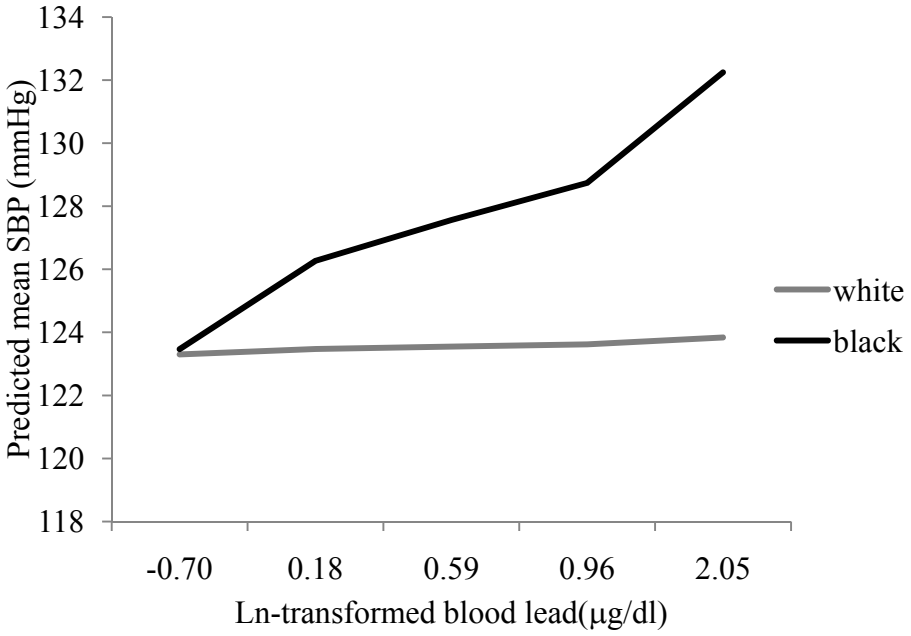
Table 4.7b Black-white comparisons in the association between blood lead and diastolic blood pressure, NHANES 2001-2006

	Men (N=3220)			Women (N=3170)		
	(1) Age, bio	(2) + Risk	(3) + Risk, SES	(1) Age, bio	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	0.2	0.1	0.0	2.5**	2.0*	2.0*
	(0.8)	(0.9)	(0.9)	(0.7)	(0.8)	(0.8)
Blood lead	0.3	1.1*	1.0*	0.8^{+}	1.4**	1.4**
	(0.4)	(0.5)	(0.6)	(0.5)	(0.5)	(0.5)
Interaction						
Black race*lead	1.0	1.3	1.4	-0.8	-0.7	-0.7
	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)	(0.8)
Intercept	20.2	17.7	16.4	22.0	19.5	19.2
	(9.3)	(9.1)	(9.6)	(7.1)	(7.0)	(7.0)
F-statistic (df)	25.92 (11)	21.68 (17)	18.66 (20)	64.21 (11)	45.08 (17)	39.00 (20)
R^2	0.14	0.16	0.16	0.15	0.17	0.17

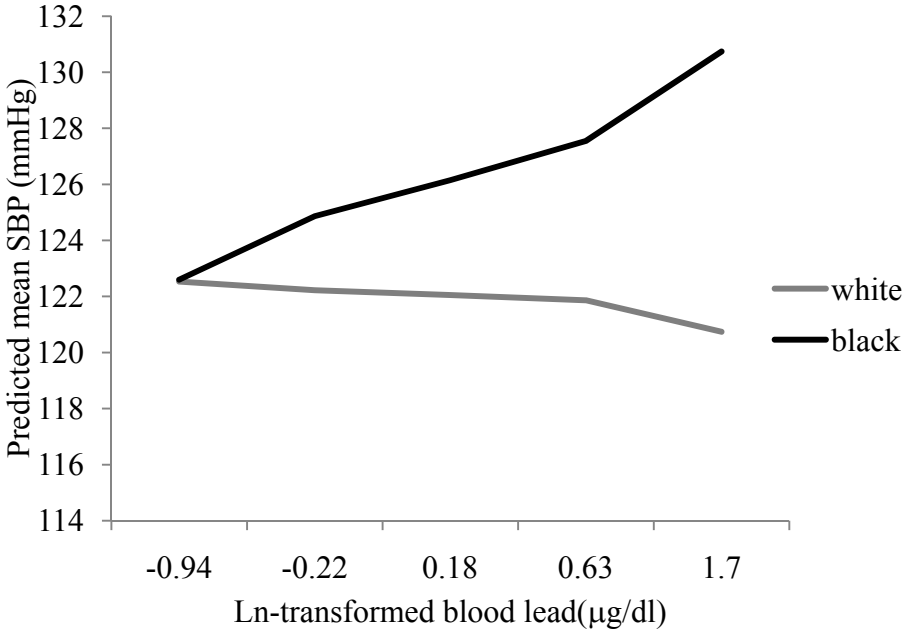
Notes: All results are weighted to account for complex survey design.
Models sequentially add:
(1) Age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, phosphorous, iron); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, $25\text{-}29.9^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
*p<0.05; **p<0.01; ***p<0.001

Figure 4. 1 Black-white disparities in the association between blood lead and systolic blood pressure (SBP) in men and women, NHANES 2001-2006

4.1a Men



4.1b Women



Notes: All estimates are weighted for NHANES complex survey design.
Slopes are adjusted for age, age^2, hematocrit, calcium, vitamin D, iron, phosphorous, and anti-hypertensive medication use, body mass index, smoking, family poverty-to-income ratio, and education.
Blood lead level, on the x-axis, is that for each of the following percentiles: 1st, 25th, 50th, 75th, and 99th, within each gender group.

Table 4. 8 Decomposition of black-white disparities in systolic blood pressure (SBP), NHANES 2001-2006

	Men (N=3220)		Women (N=3170)	
	b	(se)	b	(se)
Systolic blood pressure				
Mean, white adults	123.6	(0.5)	123.2	(0.5)
Mean, black adults	127.5	(0.6)	125.2	(0.7)
Black-white difference[a]	-3.9***	(0.6)	-3.0***	(0.8)
Level (endowments)	0.0	(0.8)	-0.2	(1.3)
Effect (coefficients)	-4.1***	(0.7)	-5.5***	(1.1)
Change in SBP for black adults if they had *level* of blood lead of white adults:	-0.1	(0.1)	-0.3*	(0.1)
Change in SBP for black adults if they had the *effect* of blood lead of white adults:	-0.7	(0.9)	-1.2**	(0.4)

Notes:

All estimates are weighted for NHANES complex survey design.

Model covariates include: age, age^2, hematocrit, calcium, vitamin D, iron, phosphorous, and anti-hypertensive medication use.

Test for difference greater than zero, * $p<0.05$; **$p<0.01$; *** $p<0.001$.

[a]The black-white differences in mean systolic blood pressure are slightly different here than those reported in Tables 6.10 and 6.11 because they are unadjusted here.

Table 4. 9a Race-educational attainment group disparities in the blood lead-systolic blood pressure association, NHANES 2001-2006

	Men (N=3220)		Women (N=3170)	
	(1) Age, bio	(2) + Risk	(1) Age, bio	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	3.1**	3.2***	4.4***	3.8***
	(0.9)	(0.9)	(1.1)	(1.1)
Education				
(≥HS; High school)	--	--	--	--
<High school	1.4	1.7	0.5	0.5
	(1.7)	(1.5)	(1.3)	(1.4)
Blood lead	0.7	0.4	-0.9	-0.5
	(0.7)	(0.7)	(0.8)	(0.8)
Interactions				
Black race*<HS	-3.3	-3.2	-1.4	-1.5
	(2.4)	(1.2)	(1.9)	(2.0)
Black race*lead	1.4	1.8	3.3*	3.4*
	(1.2)	(1.2)	(1.5)	(1.4)
<HS*lead	-0.8	-0.6	0.6	0.7
	(1.7)	(1.6)	(1.7)	(1.7)
Black race*<HS*lead	3.6	3.5	0.6	0.8
	(2.8)	(2.7)	(3.1)	(3.0)
Intercept	101.9	98.2	89.6	86.1
	(7.7)	(7.7)	(10.5)	(10.2)
F-statistic	42.47 (15)	37.91 (21)	196.84 (15)	114.32 (21)
R^2	0.15	0.17	0.36	0.37

Notes:
All results are weighted to account for complex survey design. Results are unstandardized linear regression coefficients.
Models sequentially add:
(1) Age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, iron, phosphorous); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, 25-29.9 $^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week).

$^{+}p<0.10$; *p<0.05; **p<0.01; ***p<0.001

Table 4.9b Race-poverty status group disparities in the blood lead-systolic blood pressure association, NHANES 2001-2006

	Men (N=3220)		Women (N=3170)	
	(1) Age, bio	(2) + Risk	(1) Age, bio	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	2.5*	2.8**	4.1***	3.7***
	(0.9)	(0.9)	(1.0)	(1.0)
Poverty status				
(≥1.85; nonpoor)	--	--	--	--
<1.85; poor	0.1	0.6	0.3	0.2
	(1.0)	(1.0)	(0.9)	(0.9)
Blood lead	0.8	0.5	-1.0	-0.6
	(0.8)	(0.8)	(0.9)	(0.9)
Interactions				
Black race*poor	0.2	-0.4	0.1	-0.2
	(2.1)	(2.1)	(1.3)	(1.3)
Black race*lead	2.0	2.4*	3.7+	3.7+
	(1.2)	(1.2)	(1.9)	(1.9)
Poor*lead	-0.5	-0.2	0.4	0.8
	(1.3)	(1.6)	(1.4)	(1.4)
Black race*poor*lead	1.0	1.1	-0.7	-0.5
	(2.2)	(2.3)	(.7)	(2.6)
Intercept	102.0	98.4	89.2	85.8
	(7.8)	(7.9)	(10.7)	(10.3)
F-statistic	43.09 (15)	30.54 (21)	171.16 (15)	109.52 (21)
R^2	0.15	0.17	0.36	0.37

Notes:
All results are weighted to account for complex survey design. Results are unstandardized linear regression coefficients.
Models sequentially add:
(1) Age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, iron, phosphorous); anti-hypertensive medication use;
(2) BMI (body mass index: $<25^{kg}/_{m^2}$, $25\text{-}29.9^{kg}/_{m^2}$, $\geq 30^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week).

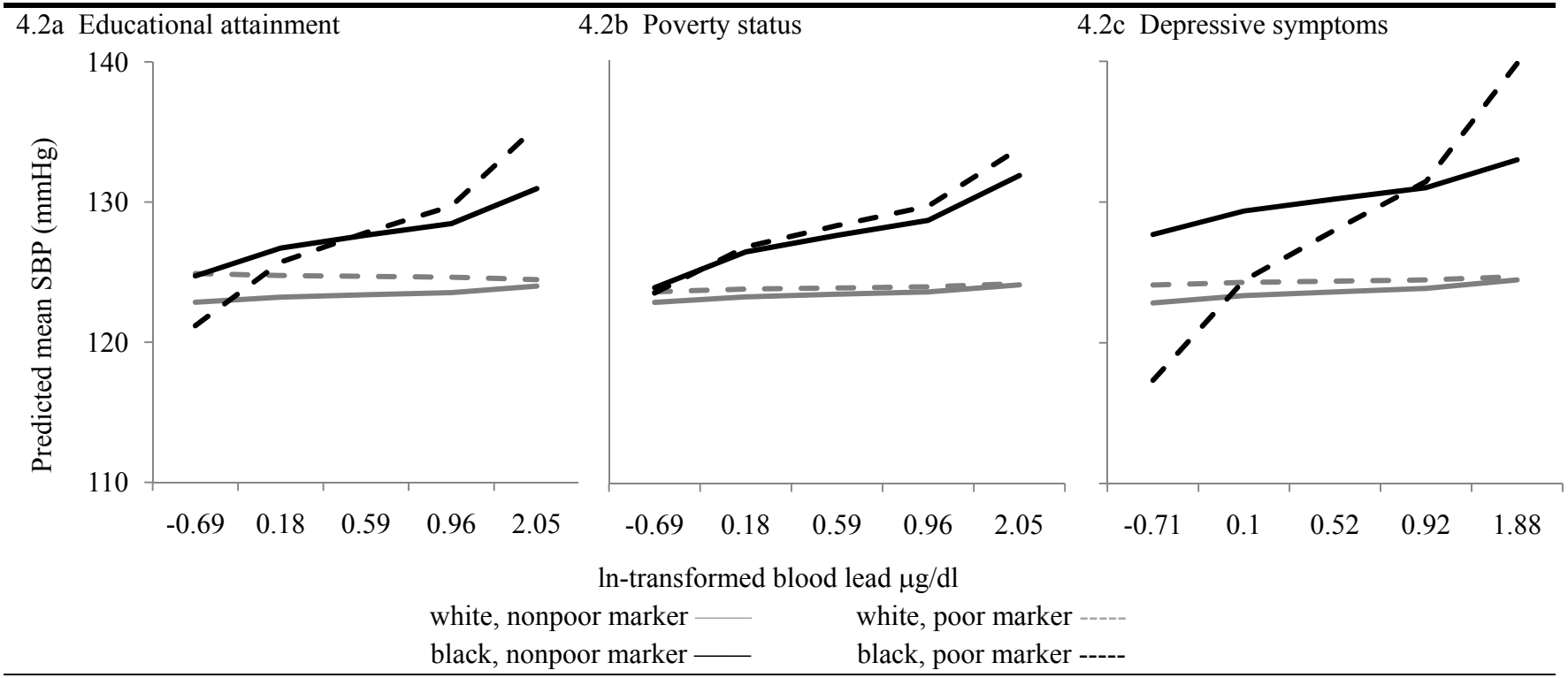
+p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 4.9c Race-depressive symptoms group disparities in the blood lead-systolic blood pressure association, NHANES 2001-2006

	Men (N=1097)	Women (N=1070)
	b (SE)	b (SE)
Race		
(White)[a]	--	--
Black	2.5*	2.8**
	(0.9)	(0.9)
Poverty status		
(≥1.85; nonpoor)	--	--
<1.85; poor	0.1	0.6
	(1.0)	(1.0)
Blood lead	0.8	0.5
	(0.8)	(0.8)
Interactions		
Black race*poor	0.2	-0.4
	(2.1)	(2.1)
Black race*lead	2.0	2.4*
	(1.2)	(1.2)
Poor*lead	-0.5	-0.2
	(1.3)	(1.6)
Black race*poor*lead	1.0	1.1
	(2.2)	(2.3)
Intercept	102.0	98.4
	(7.8)	(7.9)
F-statistic	43.09 (15)	30.54 (21)
R^2	0.15	0.17

Notes:
All results are weighted to account for complex survey design. Results are unstandardized linear regression coefficients.
Models adjusted for: age (age and age^2); lead-related biomarkers (hematocrit, calcium, vitamin D, iron, phosphorous); anti-hypertensive medication use;
$^+$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Figure 4. 2 Race-social marker disparities in the association between blood lead and systolic blood pressure (SBP) in men (N=3220), NHANES 2001-2006



Notes: All estimates are weighted for NHANES complex survey design.

Models adjusted for: age (age and age^2); bio (hematocrit, calcium, vitamin D, phosphorous, iron); anti-hypertensive medication use.

Models with educational attainment and poverty status further adjusted for: BMI (body mass index: <25 $^{kg}/_{m}^{2}$, 25-29.9 $^{kg}/_{m}^{2}$, ≥30 $^{kg}/_{m}^{2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week).

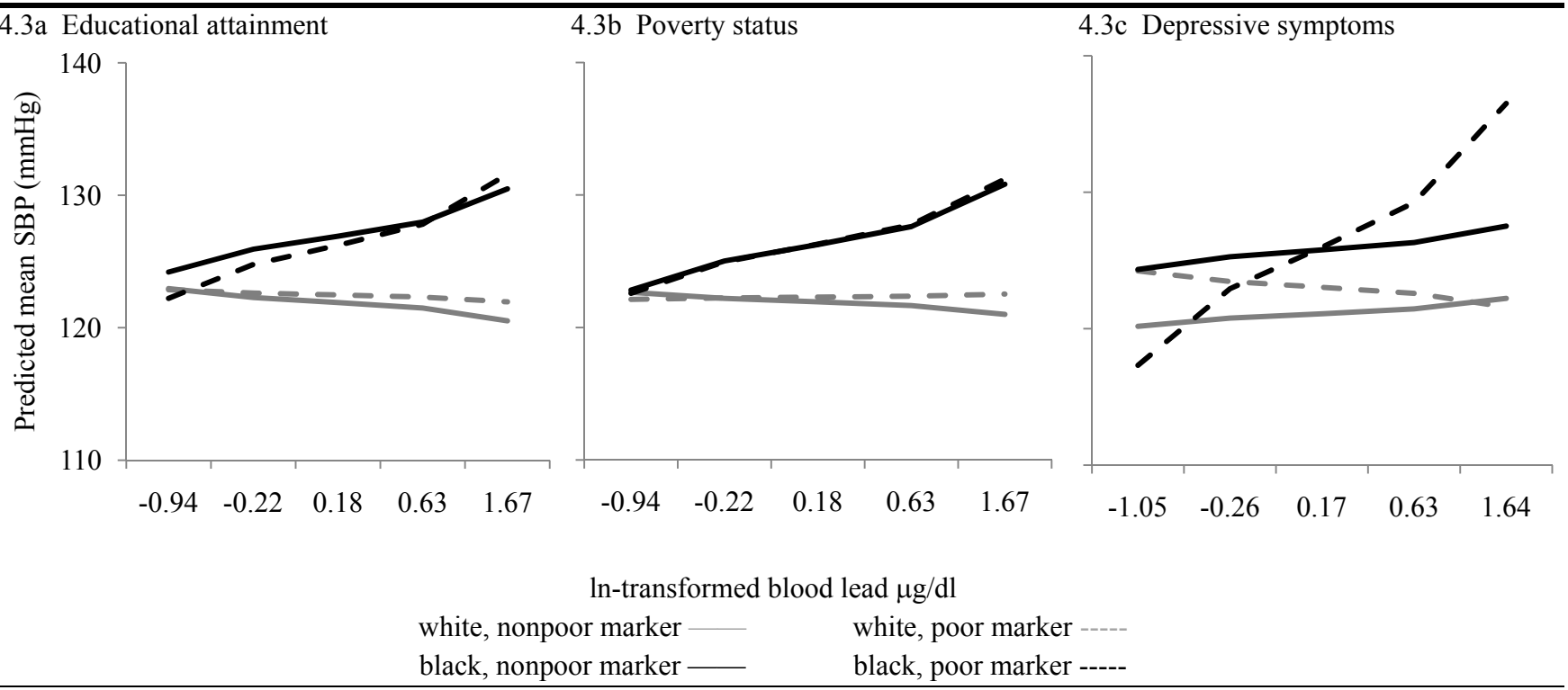
Nonpoor markers are: ≥high school education; ≥1.85 poverty-to-income ratio; or highest quartile of depressive symptoms.

Poor markers are: <high school education; <1.85 poverty-to-income ratio; or lower three quartiles of depressive symptoms.

Depressive symptoms models include only the 2005-2006 wave.

Ln-transformed blood lead level, on the x-axis, is that for each of the following percentiles: 1st, 25th, 50th, 75th, and 99th.

Figure 4. 3 Race-social stress disparities in the association between blood lead and systolic blood pressure (SBP) in women (N=3170), NHANES 2001-2006



Notes: All estimates are weighted for NHANES complex survey design.

Models adjusted for: age (age and age^2); bio (hematocrit, calcium, vitamin D, phosphorous, iron); anti-hypertensive medication use.

Models with educational attainment and poverty status further adjusted for: BMI (body mass index: $<25^{kg}/_{m}{}^{2}$, 25-29.9 $^{kg}/_{m}{}^{2}$, $\geq 30^{kg}/_{m}{}^{2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week).

Nonpoor markers are: ≥high school education; ≥1.85 poverty-to-income ratio; or highest quartile of depressive symptoms.

Poor markers are: <high school education; <1.85 poverty-to-income ratio; or lower three quartiles of depressive symptoms.

Depressive symptoms models include only the 2005-2006 wave.

Ln-transformed blood lead level, on the x-axis, is that for each of the following percentiles: 1st, 25th, 50th, 75th, and 99th.

CHAPTER FIVE

RESULTS, PART 2: ESTIMATED BONE LEAD AND HYPERTENSION

In this chapter, results from my analyses on black-white disparities in bone lead and hypertension and the potential role of education and poverty status are presented. There are two threads of results presented in sequence before they are tied together in the interaction models. Hypertension is discussed first and then bone lead; for each, descriptive characteristics, bivariate associations, and additive multivariate models are presented. With this information, black-white comparisons in the association between bone lead and hypertension (the two-way interaction models) are presented. Finally, race-education and race-poverty status comparisons in this association (the three-way interaction models) are presented.

Black-white disparities in hypertension

The hypertension-related characteristics of the National Health and Nutrition Examination Survey (NHANES) 2001-2004 are shown in Table 5.1, by gender. Means and standard deviations for continuous variables and percentages of categorical variables are reported.

First, black men and women have higher crude rates of hypertension compared to white men and women, with rates for black men and women at roughly 40% and those for white men and women at roughly 32% ($p<0.10$ for men, $p<0.001$ for women). In general, black men and women have similar or *lower* rates of some hypertension-related risk factors such as smoking, alcohol use, and obesity (in men), compared to white men and women. For example, 70% of black women have never smoked cigarettes, compared to 57% of white women ($p<0.001$). For example, black men and women are more likely to have never smoked. In fact, 70% of black women report never smoking, compared to only 57% of white women ($p<0.001$). Although they are less likely to quit once they have started smoking (i.e., be "former smokers"), overall, black men and women have smoked about half the amount of cigarettes, measured as the number of pack-years,[4] as white men and women (5.5 and 10.6 pack-years for black and white men, respectively, $p<0.001$; 2.8 and 6.3 pack-years for black and white women, respectively, $p<0.001$). There are some risk factors for which black adults have higher rates, particularly black women. For example, black women have the higher rates of obesity compared to white women (51% and 29%, respectively, $p<0.001$). Perhaps related is that black women are more likely to report no physical activity than white women (19% and 13%, respectively, $p<0.05$) Overall, black adults have higher levels of some risk factors and lower levels of others.

Black adults uniformly have lower levels of SES. Black adults are more likely to lack a high school education and live in poverty compared to white adults. Poverty rates

[4] A pack-year is a measure of the amount of cigarette smoking over time, determined by multiplying the number of packs a person has smoked per day by the number of years a person has smoked this amount. One pack-year is equivalent to one pack of cigarettes smoked per day for one year. One pack-year is also equivalent to smoking one-half pack per day for six months.

are high for black adults at roughly 40-50% compared to 20-25% for white men and women ($p<0.001$). Black adults are also less likely to have post-secondary education or hold white-collar jobs.

Tables 5.2a and 5.2b show the correlation matrices for hypertension-related health and demographic factors for men and women, respectively. Bonferroni-adjusted tests for significance are reported at the $p<0.01$ level. In general, hypertension is correlated with hypertension risk factors. It is most strongly correlated with age for both men ($r=0.42$, $p<0.01$) and women ($r=0.52$, $p<0.01$). Hypertension is moderately correlated with risk factors such as smoking, obesity, physical activity, and alcohol use (for women only). One unexpected result is that hypertension is moderately *negatively* correlated with current smoking for women ($r=-0.16$, $p<0.01$) and positively correlated with past smoking for men ($r=0.19$, $p<0.01$).

On the other hand, hypertension is generally not correlation with the SES variables. The exception is that it is weakly correlated with a lack of high school education for men ($r=0.10$, $p<0.01$) and women ($r=0.11$, $p<0.01$). It is also negatively correlated with post-secondary education, but only for women ($r=0.12$, $p<0.01$).

So far, the results show that black adults have higher crude rates of hypertension compared to white adults. Black and white adults differ with regards to some of the hypertension risk factors, raising the question of whether these factors account for the black-white hypertension disparities.

Table 5.3 shows the black-white disparities in hypertension as odds ratios, adjusted for these factors. Results are stratified by gender. Tests for significance are for difference from zero. Black men have a roughly 80% greater odds of hypertension than

white men (Model 1, $p<0.001$). The disparity does not change with the addition of hypertension risk factors (Model 2, OR=1.83), but decreases slightly with the addition of SES (Model 3, OR=1.63).

Black women have a roughly 200% greater odds of hypertension than white women (Model 1, $p<0.001$). The disparity decreases the disparity decreases with the addition of the hypertension risk factors (Model 2, OR=2.32). The disparity for decrease further with the addition of SES (Model 3, OR=2.28). These results show that black-white disparities in hypertension generally are not explained by hypertension risk factors or SES. The exception is that hypertension risk factors explains a small portion of the disparity in women.

Black-white disparities in estimated bone lead

Bone lead is estimated from blood lead and other health and sociodemographic variables using an algorithm from the literature (S. Park et al., 2009). Table 5.4 shows the estimated bone lead and the biomarker components of the algorithm, by race and gender. The other components of the algorithm are shown in Table 5.1 and include: age, educational attainment, pack-years smoked, smoking status, and white-collar occupation.

In general, there are no racial differences in the unadjusted means of any of the markers, including bone lead. For example, estimated tibia lead levels for black and white men are $1.4^{\mu g}/_{g}$ and $2.2^{\mu g}/_{g}$, respectively (p=NS). A few markers show statistically-significant differences, but the means are still within a healthy range. For example, hematocrit levels differ only by 1.2% between races (44.4% and 45.6%, for blacks and whites, respectively). Although they are statistically different, both means are

within the clinically normal range. There is a difference in total cholesterol for women, with levels of roughly $196^{mg}/_{dl}$ and $204^{mg}/_{dl}$ for black and white women, respectively ($p<0.001$). Overall, though, if there are differences in biomarkers, they are small.

The correlation matrices for the lead-related variables for men and women are shown in Tables 5.5a and 5.5b, respectively. These lead-related variables include risk factors and SES from Table 5.1, as these are risk factors for lead exposure as well as hypertension. In general, estimated tibia and patella lead are correlated with their algorithm components, as expected. Both estimated tibia and patella lead are strongly related to age for both men and women ($r\approx0.90$, $p<0.0001$).[5] Estimated tibia and patella lead levels are also strongly correlated with blood lead ($r\approx0.63$, $p<0.01$ for men; $r\approx0.56$, $p<0.01$ for women). They are also correlated with lead exposure risk factors such as smoking and SES. For example, estimated tibia and patella lead are moderately correlated with pack-years smoked for men ($r\approx0.45$, $p<0.01$) and women ($r\approx0.26$, $p<0.01$). They are also negatively correlated with post-secondary education for men ($r\approx-0.18$, $p<0.01$) and women ($r\approx-0.27$, $p<0.01$).

In sum, there are not large black-white differences in the biomarkers components of the bone lead algorithm, but there are disparities in the sociodemographic components. Estimated tibia and patella lead are correlated with most of the algorithm components. How do these factors affect black-white disparities in adjusted bone lead? The results in Tables 5.6a and 5.6b address this question for tibia and patella lead, respectively.

[5] To address potential multicollinearity problems with age and lead, I regressed estimated tibia and patella lead, separately, on age. I then calculated the tolerances and variance inflation factors (VIFs). All tolerance values were between 0.13 and 0.23; all VIFs were between 4.35 and 7.99, which indicate that the high correlation will not result in multicollinearity.

The disparity in age-adjusted mean tibia lead is 2.90 $^{\mu g}/_{g}$ and 3.23 $^{\mu g}/_{g}$ for men and women, respectively (Model 1, $p<0.001$ for men and women). The addition of the lead-related risk factors, slightly increased the disparity for men to 3.03 $^{\mu g}/_{g}$ and decreased it slightly for women to 2.77 $^{\mu g}/_{g}$ (Model 2, $p<0.001$ for men and women). The addition of SES, however, substantially attenuated the disparity substantially. For men, the disparity decreased to 1.01 $^{\mu g}/_{g}$ (Model 3, $p<0.001$). For women, the disparity decreased 1.44 $^{\mu g}/_{g}$ (Model 2, $p<0.001$). The decrease in disparity is due primarily to the reduction in bone lead for black adults as opposed to white adults.

The disparities are a slightly larger with patella lead, but the patterns when adding covariates is similar. The age-adjusted disparity is 3.12 $^{\mu g}/_{g}$ for men (Table 5.6b, Model 1, $p<0.001$) and 4.03 $^{\mu g}/_{g}$ for women ($p<0.001$). After adjusting for risk factors, the disparity increases slightly for men to 3.65 $^{\mu g}/_{g}$ (Model 2, $p<0.001$) and decreases for women to 3.11 $^{\mu g}/_{g}$ ($p<0.001$). After further adjusting for SES, the disparity decreases substantially for both men and women to 1.28 $^{\mu g}/_{g}$ and 1.61 $^{\mu g}/_{g}$, respectively (Model 1, $p<0.001$ for men and women). In sum, hypertension risk factors and SES, in particular, appear to explain much of the black-white disparities in estimated tibia and patella lead.

Racial comparisons in the association between estimated bone lead and hypertension

The results from the previous section show that black adults have higher levels of bone lead than white adults. Does this higher level of bone lead result in a stronger effect of bone lead on hypertension for black adults? In order to address this question, I interact race and bone lead within logistic regression models for hypertension. This results in

race-specific effects of bone lead on hypertension. Tables 5.7a and 5.7b contain the results from these regressions, for tibia and patella lead, respectively. Results are stratified by gender. Only the focal coefficients of race, tibia lead, the race*lead interaction, and intercept are presented. The change in the odds of hypertension due to lead is estimated by the following model:

$$\text{logit(hypertension)} = \beta_1 \text{race} + \beta_2 \text{lead} + \beta_3 \text{race*lead} \qquad \text{(Equation 5.1)}$$

White adults are the reference category for race. Therefore, β_2 represents the percent change in the odds of hypertension due to lead for white adults. The sum of β_2 and β_3 represents the percent change in odds of hypertension due to lead for black adults.

In general, the results show that there is a positive association between estimated tibia lead and hypertension. For white men, the tibia lead coefficient of 0.04 (Table 5.7a, Model 1, $p<0.001$) means that there is a four percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead. Among mean, the relation between lead and hypertension does not vary by race. The race*tibia lead interaction coefficient of 0.00 (Model 1, p=NS) means that there is no difference between black and white men. In other words, both black and white men experience a four percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead. The addition of lead exposure risk factors and then SES attenuates the association between tibia lead and hypertension for men. With all covariates in the model, there is a two percent increase in the odds of hypertension for $^{\mu g}/_{g}$ increase in tibia lead (Model 3, $p<0.05$).

For white women, there is a five percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead (Model 1, $p<0.001$). Among women, there is a different pattern in the age-adjusted model (Model 1, Table 5.7). Black women have a *weaker* association between tibia lead and hypertension than white women. The race*tibia lead interaction coefficient is -0.02 (Model 1, $p<0.05$), meaning that while white women experience a five percent increase, black women experience only a three percent increase in the odds of hypertension with each $^{\mu g}/_{g}$ increase in tibia lead. The addition of lead exposure risk factors eliminates the interaction between race and lead and also attenuates the main effect of lead on hypertension (Model 2). The addition of SES further attenuates the association, resulting in a one percent increase for each $^{\mu g}/_{g}$ increase in tibia lead (Model 3, p=NS) for both black women.

Race-SES comparisons in the association between estimated bone lead and hypertension

Although there are no racial disparities in the association between bone lead and hypertension for men and a *weaker* association for black compared to white women, I continue to examine the possibility that the lead may interact with SES to produce more complex patterns. In order to do this, I interact race, SES, and lead within logistic regression models. This results in race-SES-specific effects of bone lead on hypertension. Tables 5.8a and 5.9a contain the results for tibia lead. Tables 5.8b and 5.9b contain the results for patella lead. Tables 5.8a and 5.8b contain the results using educational attainment. Tables 5.9a and 5.9b contain the results using poverty status. In each table, I present the coefficients for only the focal variables of race, SES, lead, the

interaction terms, and the intercept. All models are stratified by gender. All models have white adults and the higher level of SES (e.g., nonpoor adults) as the omitted terms.

Overall, there is no clear pattern in the effect of bone lead on hypertension by race, SES, or gender. Because results differ depending on the SES and bone lead measure that is used and by gender, I outline the *net effects* of the bone lead-hypertension association in Tables 5.10a and 5.10b. The net effects I present are adjusted for all covariates and correspond to Model 2 in Tables 5.8a, 5.8b, 5.9a, and 5.9b. I calculated these net effects after I estimated each logistic regression model. The results are based on the following equation:

$$\begin{aligned}\text{logit(Htn)} = {} & \beta_0 + \beta_1\text{race} + \beta_2\text{lead} + \beta_3\text{social} \\ & + \beta_4\text{race*lead} + \beta_5\text{race*social} + \beta_6\text{social*lead} \\ & + \beta_7\text{race*social*lead}\end{aligned} \qquad \text{(Equation 5.2)}$$

where: Htn = hypertension
race = black race (white=0, black=1)
lead = estimated tibia lead ($^{\mu g}/_{g}$) *or*
estimated patella lead ($^{\mu g}/_{g}$)
social = educational attainment (≥high school=0, <high school=1) *or*
poverty status (nonpoor=0, poor=1)

Percent increase in odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead for:

black adults, low SES = $\beta_2 + \beta_4 + \beta_5 + \beta_7$
white adults, low SES = $\beta_2 + \beta_5$
black adults, not low SES = $\beta_2 + \beta_4$
white adults, not low SES = β_2

I begin by outlining a single example to show the interpretation of coefficients and how the net effects are calculated. Then I discuss the overall results.

I take an example using tibia lead and educational attainment in men (Table 5.8a for the regression coefficients and Table 5.10a for the net effects). The effect of bone lead on hypertension for white men with at least a high school education (I will call "high education" from here), is β_2 in equation 5.1, or the "tibia lead" term in Table 5.8a. The term is 0.05 ($p<0.001$), meaning for white men white high education, there is a five percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead.

The effect of bone lead for white men without a high school education (I will call "low education from here) is the sum of $\beta_2$and β_6 in Equation 5.1, or the sum of the "tibia lead" and "education*lead" terms in Table 5.8a. The sum is 0.02 (p=NS), meaning that for white men with low education, there is no association between tibia lead and hypertension.

The effect of bone lead for black men with high education is the sum of $\beta_2$and β_4 in Equation 5.1 or the sum of the "tibia lead" and "race*education" terms in Table 5.8a. The sum is 0.04 ($p<0.05$), meaning that there is a four percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead.

Finally, the effect for black men with low education is the sum of β_2, β_4, β_6 and β_7 in Equation 5.1 or the sum of the "tibia lead", "race*lead", "education*lead", and "race*education*lead" terms in Table 5.8a. The sum is 0.05 ($p<0.001$), meaning that for black men with low education, there is a five percent increase in the odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead.

Overall, the results differ by the SES and bone measure used, by the covariates included, and by gender. *There is no clear pattern of the association between bone lead and hypertension*. For example, in Model 1 in Table 5.8a, all men show a four to five

percent increase in the odds of hypertension due to tibia lead – with the exception of white men with low education. They show no statistically significant increase (b=0.02, p=NS) However, after adjusting for hypertension risk factors, the odds of hypertension is attenuated in all men – with the exception of black men with low education. This pattern is shown in Table 5.10a in the top left quadrant. Black men with low education show a four percent increase in the odds of hypertension each $^{\mu g}/_{g}$ increase in tibia lead ($p<0.05$), while there is no statistically-significant increase for other men.

Figure 5.2 depicts Model 2 for men in Table 5.8a. Along the x-axis is the distribution of tibia lead for men at the 1st, 25th, 50th, 75th, and 99th, percentile. The y-axis is the log odds of hypertension. The black lines represent black men – the solid black line for black men with high education and the dashed black line for black men with low education. The gray lines represent white men is a similar fashion. The black dashed line, representing black men with low education, is steeper than the other three lines. This means that the effect of tibia lead on hypertension is stronger for this group than the other three.

Tables 5.10a and 5.10b provide the net effects so that it is easier to see if there are any patterns in the association between bone lead and hypertension. These tables show that the results differ for men. For example, when using poverty status, all men show an association between tibia lead and hypertension. When using patella lead, black men with low education and white men with high education show an association between patella lead and hypertension. For women, there are no statistically-significant race-SES group differences in the association between bone lead and hypertension.

Summary

Results show that stark black-white disparities in hypertension are not explained by hypertension risk factors. I also provide the first detailed black-white comparison of the association between estimated bone lead and hypertension, using a new algorithm to estimate bone lead from blood lead and other covariates. Although black men and women have higher bone lead levels, this does not translate into a stronger association between bone lead and hypertension. Both black and white adults show a three to five percent increase in the odds of hypertension with every $^{\mu g}/_{g}$ increase in either tibia or patella lead. Finally, there is no clear pattern in race-SES group with regard to the association between bone lead and hypertension. Results differ by SES and bone lead measure, covariates included, and gender. Hypotheses are not supported with regard to black-white disparities in the association between bone lead and hypertension.

Table 5. 1 Hypertension-related health and demographic characteristics, NHANES 2001-2004

	Men			Women		
	Black (N=518)	White (N=1603)	p[a]	Black (N =537)	White (N=1575)	p
Hypertension, %	40	32	+	42	33	***
Hypertension risk factors						
Age, years, mean (SD)	41.1 (15.8)	46.3 (10.4)	***	42.9 (15.6)	48.5 (10.9)	***
Smoking, pack-years, mean (SD)	5.5 (12.9)	10.6 (13.0)	***	2.8 (8.1)	6.3 (8.8)	***
Smoking status, %						
never	54	47	*	70	57	***
former	14	29	***	11	22	***
current	32	24	**	18	21	
BMI category, %						
<25 $^{kg}/_{m}^{2}$ (lean)	33	29		21	43	***
25-29.9 $^{kg}/_{m}^{2}$ (overwt)	36	42	**	26	26	
≥30 $^{kg}/_{m}^{2}$ (obese)	30	28		51	29	***
No physical activity, %	13	10	+	19	13	*
Alcohol, drinks/week, mean (SD)	1.6 (2.3)	1.7 (1.4)		0.5 (1.1)	0.9 (1.0)	***
Diabetes, %	8	6		10	6	**
Socioeconomic status						
Education, %						
<High school	30	10	***	25	11	***
High school diploma	55	56		58	61	
>High school	15	33	***	17	28	***
White-collar occup, %	33	45	**	54	70	***
Family PIR<1.85, %	41	19	***	51	24	***

Notes:
All results are weighted to account for complex survey design.
Abbreviations are: SD, standard deviation; BMI, body mass index; overwt, overweight; occup, occupation; PIR, poverty-to-income ratio.
[a] Tests for black-white difference different from zero: +p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5. 2a Hypertension risk factor correlation matrix for men (N=2121), NHANES 2001-2004

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Hypertension	1.00								
(2)	Age	0.42*	1.00							
(3)	Pack-yrs smoked	0.15*	0.32*	1.00						
(4)	Never smoker	-0.09*	-0.19*	-0.90*	1.00					
(5)	Former smoker	0.19*	0.40*	0.60*	-0.59*	1.00				
(6)	Current smoker	-0.09*	-0.21*	0.40*	-0.52*	-0.37*	1.00			
(7)	Lean BMI	-0.18*	-0.14*	0.00	-0.04	-0.13*	0.18*	1.00		
(8)	Overweight BMI	0.03	0.12*	0.02	0.00	0.08	-0.08	-0.55*	1.00	
(9)	Obese BMI	0.16*	0.01	-0.03	0.04	0.05	-0.09*	-0.42*	-0.53*	1.00
(10)	No physical act	0.11*	0.10*	0.09*	-0.07	0.01	0.07	-0.02	-0.05	0.08
(11)	Alcohol use	0.00	0.01	0.20*	-0.21*	0.08	0.16*	0.04	0.07	-0.11*
(12)	Diabetes	0.18*	0.21*	0.09*	-0.04	0.09*	-0.04	-0.09*	-0.06	0.15*
(13)	>HS education	0.10*	0.12*	0.17*	-0.15*	0.02	0.16*	0.07	-0.03	-0.04
(14)	HS education	-0.03	-0.14*	0.04	-0.05	-0.04	0.10*	-0.05	0.00	0.06
(15)	<HS education	-0.05	0.05	-0.19*	0.19*	0.03	-0.25*	-0.01	0.04	-0.03
(16)	WC occupation	-0.02	0.09*	-0.17*	0.17*	0.02	-0.22*	0.00	0.07	-0.07
(17)	Poverty status	0.02	-0.05	0.07	-0.08	-0.09*	0.19*	0.12*	-0.09*	-0.03
		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	
(10)	No physical act	1.00								
(11)	Alcohol use	-0.06	1.00							
(12)	Diabetes	0.14*	-0.12*	1.00						
(13)	>HS education	0.12*	-0.05	0.07	1.00					
(14)	HS education	-0.06	-0.01	-0.03	-0.52*	1.00				
(15)	<HS education	-0.04	0.05	-0.03	-0.30*	-0.66*	1.00			
(16)	WC occupation	-0.05	0.04	-0.01	-0.29*	-0.22*	0.50*	1.00		
(17)	Poverty status	0.13*	-0.10	0.05	0.31*	-0.01	-0.27*	-0.25*	1.00	

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: pack-yrs, pack-years; lean BMI, body mass index<25 $^{kg}/_{m^2}$; overweight BMI, 25-29.9 $^{kg}/_{m^2}$; obese BMI, ≥30 $^{kg}/_{m^2}$; act, activity; HS, high school; WC, white collar
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5.2b Hypertension risk factor correlation matrix for women (N=2112), NHANES 2001-2004

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Hypertension	1.00								
(2)	Age	0.54*	1.00							
(3)	Pack-yrs smoked	0.02	0.08	1.00						
(4)	Never smoker	0.02	-0.02	-0.95*	1.00					
(5)	Former smoker	0.12*	0.25*	0.63*	-0.64*	1.00				
(6)	Current smoker	-0.16*	-0.24*	0.53*	-0.58*	-0.25*	1.00			
(7)	Lean BMI	-0.20*	-0.11*	0.02	-0.04	-0.01	0.05	1.00		
(8)	Overweight BMI	0.03	0.14*	0.01	0.00	0.04	-0.04	-0.48*	1.00	
(9)	Obese BMI	0.17*	-0.02	-0.02	0.04	-0.03	-0.01	-0.56*	-0.46*	1.00
(10)	No physical act	0.13*	0.13*	0.05	-0.02	0.00	0.03	-0.11*	-0.01	0.13*
(11)	Alcohol use	-0.15*	-0.18*	0.19*	-0.22*	0.11*	0.15*	0.19*	-0.02	-0.17*
(12)	Diabetes	0.21*	0.18*	0.00	0.02	0.03	-0.05	-0.15*	0.01	0.14*
(13)	>HS education	0.11*	0.14*	0.06	-0.06	0.00	0.07	-0.10*	0.03	0.07
(14)	HS education	0.02	-0.02	0.06	-0.06	0.00	0.08	-0.07	0.03	0.04
(15)	<HS education	-0.12*	-0.10*	-0.13*	0.12*	0.01	-0.16*	0.17*	-0.06	-0.12*
(16)	WC occupation	-0.03	0.01	-0.02	0.04	0.06	-0.11*	0.11*	0.00	-0.10*
(17)	Poverty status	0.04	-0.04	0.03	-0.04	-0.11*	0.17*	-0.08	-0.01	0.09*

		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	
(10)	No physical act	1.00								
(11)	Alcohol use	-0.12*	1.00							
(12)	Diabetes	0.13*	-0.16*	1.00						
(13)	>HS education	0.16*	-0.15*	0.08	1.00					
(14)	HS education	-0.04	-0.04	0.01	-0.56*	1.00				
(15)	<HS education	-0.10*	0.18*	-0.08	-0.25*	-0.67*	1.00			
(16)	WC occupation	-0.09*	0.15*	-0.10*	-0.34*	0.01	0.29*	1.00		
(17)	Poverty status	0.13*	-0.19*	0.09*	0.29*	0.02	-0.29*	-0.32*	1.00	

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: pk-yrs, pack-years; lean BMI, body mass index<25 $^{kg}/_{m}{}^{2}$; overweight BMI, 25-29.9 $^{kg}/_{m}{}^{2}$; obese BMI, ≥30 $^{kg}/_{m}{}^{2}$; act, activity; HS, high school; WC, white collar.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5. 3 Black-white disparities in hypertension, NHANES 2001-2004

	Men (N=2121)			Women (N=2112)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Race						
(White)[a]	--	--	--	--	--	--
Black	1.79*** (1.41, 2.26)	1.83*** (1.42, 2.35)	1.63** (1.26, 2.12)	3.04*** (2.23, 4.14)	2.32*** (1.65, 3.27)	2.28*** (1.64, 3.17)
F-statistic (df)	100.88 (3)	72.79 (8)	48.73 (11)	159.20 (3)	65.01 (8)	63.14 (11)

Notes:
All results are weighted to account for complex survey design. Age-adjusted hypertension prevalences are: black men, 42%; white men, 28%; black women, 49%; white women 24%.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status; df, degrees of freedom
[a] Omitted category in parentheses.
***p<0.001

Table 5. 4 Estimated bone lead-related characteristics, NHANES 2001-2004

	Men			Women		
	Black (N=518)	White (N=1603)	p[a]	Black (N=537)	White (N=1575)	p
Estimated bone lead						
Estimated tibia lead, $^{\mu g}/_{g}$	1.4 (14.5)	2.2 (8.4)		0.9 (13.0)	1.6 (8.3)	
Estimated patella lead, $^{\mu g}/_{g}$	-0.6 (20.0)	1.3 (12.0)		-1.4 (18.4)	0.3 (11.8)	*
Algorithm components						
Blood lead, $^{\mu g}/_{dl}$	2.1 (2.1)	1.8 (1.5)	*	1.4 (1.9)	1.3 (1.5)	**
BMI, $^{kg}/_{m^2}$	28.2 (7.0)	28.1 (3.6)		31.3 (7.8)	27.4 (4.3)	***
Calcium, $^{mg}/_{dl}$	9.6 (0.4)	9.5 (0.2)	***	9.5 (0.4)	9.5 (0.2)	
HDL, $^{mg}/_{dl}$	51.9 (15.9)	47.1 (8.5)	***	58.7 (16.8)	59.4 (10.9)	
Hematocrit, %	44.4 (3.8)	45.6 (2.2)	***	38.2 (3.6)	40.7 (2.0)	***
Phosphorus, $^{mg}/_{dl}$	3.8 (0.6)	3.7 (0.4)	*	3.8 (0.5)	3.8 (0.3)	
Serum creatinine, $^{mg}/_{dl}$	1.2 (0.8)	1.0 (0.2)	***	0.8 (0.5)	0.8 (0.1)	
Total cholesterol, $^{mg}/_{dl}$	198.5 (48.2)	201.9 (28.7)		195.8 (40.3)	204.2 (26.5)	***
Uric acid, $^{mg}/_{dl}$	6.1 (1.5)	6.1 (0.8)		4.9 (1.3)	4.7 (0.8)	**

Notes:

All results are weighted to account for complex survey design.

All results are means with standard deviations, with the exception of blood lead, which is the geometric mean.

Abbreviations are: BMI, body mass index; HDL, high density lipoprotein.

[a] Tests for black-white difference different from zero: *p<0.05; **p<0.01; ***p<0.001

Table 5. 5a Lead-related health and demographic correlation matrix for men (N=2121), NHANES 2001-2004

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Est tibia lead	1.00								
(2)	Est patella lead	0.99*	1.00							
(3)	Hypertension	0.44*	0.44*	1.00						
(4)	Age	0.90*	0.92*	0.43*	1.00					
(5)	Blood lead	0.63*	0.61*	0.20*	0.41*	1.00				
(6)	Total cholesterol	0.14*	0.09*	0.03	0.08	0.11*	1.00			
(7)	HDL	0.09*	0.10*	-0.03	0.04	0.17*	0.07	1.00		
(8)	Serum creatinine	0.10*	0.11*	0.13*	0.11*	0.11*	-0.03	0.00	1.00	
(9)	Hematocrit	-0.29*	-0.26*	-0.14*	-0.28*	-0.05	0.11*	-0.04	-0.11*	1.00
(10)	Uric acid	0.14*	0.11*	0.16*	0.01	0.08	0.11*	-0.16*	0.12*	-0.01
(11)	Calcium	-0.20*	-0.19*	-0.06	-0.20*	-0.07	0.12*	0.11*	-0.02	0.21*
(12)	BMI	0.09*	0.11*	0.21*	0.04	-0.09*	0.11*	-0.29*	-0.01	-0.01
(13)	Pack-yrs smoked	0.45*	0.48*	0.15*	0.37*	0.29*	0.02	0.00	0.03	-0.08
(14)	Former smoker	0.41*	0.40*	0.19*	0.40*	0.16*	0.06	0.03	0.04	-0.13*
(15)	Current smoker	-0.05	-0.04	-0.09*	-0.21*	0.21*	-0.01	0.00	-0.17*	0.17*
(16)	HS education	-0.08	-0.06	-0.03	-0.14*	-0.02	0.00	-0.06	-0.08	0.10*
(17)	>HS education	-0.18*	-0.17*	-0.05	0.05	-0.18*	0.03	0.05	0.06	-0.04
(18)	WC occupation	-0.15*	-0.14*	-0.02	0.09*	-0.19*	-0.01	0.02	0.07	-0.02
(19)	Poverty status	0.07	0.06	0.02	-0.05	0.13*	-0.03	-0.03	-0.03	0.03

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: est, estimated; HDL, high density lipoprotein cholesterol; BMI, body mass index, $^{kg}/_{m}{}^{2}$; pk-yrs, pack-years, HS, high school; WC, white collar.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5.5a, cont'd Lead-related health and demographic correlation matrix for men (N=2121), NHANES 2001-2004

		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
(10)	Uric acid	1.00								
(11)	Calcium	0.02	1.00							
(12)	BMI	0.27*	-0.11*	1.00						
(13)	Pack-yrs smoked	0.04	-0.04	0.02	1.00					
(14)	Former smoker	0.05	-0.09*	0.11*	0.60*	1.00				
(15)	Current smoker	-0.05	0.07	-0.18*	0.40*	-0.37*	1.00			
(16)	HS education	0.03	0.04	0.07	0.04	-0.04	0.10*	1.00		
(17)	>HS education	-0.02	-0.04	-0.01	-0.19*	0.03	-0.25*	-0.66*	1.00	
(18)	WC occupation	-0.04	-0.03	-0.04	-0.16*	0.02	-0.22*	-0.22*	0.50*	1.00
(19)	Poverty status	-0.03	0.06	-0.09*	0.07	-0.09*	0.19*	-0.01*	-0.27*	-0.25*

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: est, estimated; HDL, high density lipoprotein cholesterol; BMI, body mass index, $^{kg}/_{m^2}$; pk-yrs, pack-years, HS, high school; WC, white collar.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5.5b Lead-related health and demographic correlation matrix for women (N=2112), NHANES 2001-2004

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1)	Est tibia lead	1.00								
(2)	Est patella lead	1.00*	1.00							
(3)	Hypertension	0.55*	0.55*	1.00						
(4)	Age	0.93*	0.94*	0.54*	1.00					
(5)	Blood lead	0.56*	0.55*	0.24*	0.46*	1.00				
(6)	Total cholesterol	0.36*	0.33*	0.20*	0.33*	0.18*	1.00			
(7)	HDL	0.13*	0.14*	0.00	0.14*	0.09*	0.19*	1.00		
(8)	Serum creatinine	0.24*	0.24*	0.21*	0.23*	0.16*	0.07	-0.05	1.00	
(9)	Hematocrit	0.01	0.05	0.00	0.07	0.12*	0.17*	-0.01	-0.09*	1.00
(10)	Uric acid	0.44*	0.43*	0.34*	0.33*	0.22*	0.12*	-0.18*	0.34*	0.04
(11)	Calcium	0.12*	0.13*	0.12*	0.16*	0.13*	0.16*	0.10*	0.11*	0.18*
(12)	BMI	0.14*	0.16*	0.22*	0.01	-0.05	0.03	-0.31*	0.02	-0.04
(13)	Pack-yrs smoked	0.26*	0.28*	0.02	0.20*	0.23*	0.09*	-0.04	0.08*	0.08*
(14)	Former smoker	0.28*	0.27*	0.12*	0.25*	0.16*	0.10*	0.07	0.08	0.00
(15)	Current smoker	-0.15*	-0.14*	-0.16*	-0.24*	0.13*	-0.04	-0.17*	-0.13*	0.15*
(16)	HS education	0.00	0.01	0.02	-0.02	-0.02	0.02	-0.08	-0.03	0.03
(17)	>HS education	-0.27*	-0.25*	-0.12*	-0.10*	-0.08	-0.05	0.14*	-0.03	-0.01
(18)	WC occupation	-0.17*	-0.15*	-0.03	0.01	-0.03	0.01	0.10*	-0.01	0.06
(19)	Poverty status	0.08	0.07	0.04	-0.04	0.09*	-0.05	-0.15*	0.01	-0.08

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: est, estimated; HDL, high density lipoprotein cholesterol; BMI, body mass index, $^{kg}/_{m}{}^{2}$; pk-yrs, pack-years, HS, high school; WC, white collar.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5.5b, cont'd Lead-related health and demographic correlation matrix for women (N=2112), NHANES 2001-2004

		(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
(10)	Uric acid	1.00								
(11)	Calcium	0.13*	1.00							
(12)	BMI	0.36*	-0.12*	1.00						
(13)	Pack-yrs smoked	0.13*	0.01	0.02	1.00					
(14)	Former smoker	0.10*	0.03	0.00	0.63*	1.00				
(15)	Current smoker	-0.07	-0.04	-0.07	0.53*	-0. 25*	1.00			
(16)	HS education	0.03	0.00	0.07	0.06	0.00	0.08	1.00		
(17)	>HS education	-0.13*	0.00	-0.16*	-0.13*	0.00	-0.16*	-0.67*	1.00	
(18)	WC occupation	-0.04	-0.03	-0.12*	-0.02	0.06	-0.11*	0.01	0.29*	1.00
(19)	Poverty status	0.08	-0.01	0.10*	0.03	-0.11*	0.17*	0.02	-0.29*	-0.31*

Notes: Pearson's r is reported for continuous variables; Spearman's ρ is reported for dichotomous variables. Abbreviations are: est, estimated; HDL, high density lipoprotein cholesterol; BMI, body mass index, $^{kg}/_{m}{}^{2}$; pk-yrs, pack-years, HS, high school; WC, white collar.
Bonferroni-adjusted test for difference from zero, *p<0.01.

Table 5. 6a Black-white disparities in mean estimated tibia lead, NHANES 2001-2004

	Men (N=2121)			Women (N=2112)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	2.90***	3.03***	1.01***	3.23***	2.77***	1.44***
	(0.45)	(0.37)	(0.25)	(0.28)	(0.26)	(0.22)
Intercept	-32.04***	-37.57***	-34.02***	-32.76***	-37.63***	-33.45***
	(1.22)	(0.91)	(0.85)	(0.91)	(0.90)	(0.86)
F-statistic (df)	2378.01 (3)	1420.71 (8)	1712.30 (11)	2967.21 (3)	1379.75 (8)	1603.30 (11)
R^2	0.78	0.83	0.89	0.86	0.90	0.94

Notes:
All results are weighted to account for complex survey design. Results are unstandardized linear regression coefficients.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m}{}^2$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status
[a] Omitted category in parentheses.
***p<0.001

Table 5.6b Black-white disparities in mean estimated patella lead, NHANES 2001-2004

	Men (N=2121)			Women (N=2112)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)	--	--	--	--	--	--
Black	3.12***	3.65***	1.28***	4.03***	3.11***	1.61***
	(0.59)	(0.44)	(0.30)	(0.36)	(0.31)	(0.28)
Intercept	-47.09***	-56.74***	-52.97***	-50.62***	-58.81***	-54.16***
	(1.57)	(1.17)	(1.07)	(1.03)	(0.98)	(0.94)
F-statistic (df)	3290.32 (3)	2044.73 (8)	3851.54 (11)	3588.53 (3)	2059.39 (8)	2302.59 (11)
R^2	0.81	0.88	0.92	0.88	0.93	0.96

Notes:
All results are weighted to account for complex survey design. Results are unstandardized linear regression coefficients.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status
[a] Omitted category in parentheses.
***p<0.001

Table 5. 7a Black-white comparison of the association between tibia lead and hypertension, NHANES 2001-2004

	Men (N=2121)			Women (N=2112)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	0.46***	0.44***	0.41**	1.03***	0.86***	0.87***
	(0.11)	(0.12)	(0.12)	(0.14)	(0.16)	(0.15)
Tibia lead	0.04***	0.03**	0.02*	0.05***	0.02^{+}	0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)
Interaction						
Race*Tibia lead	0.00	0.01	0.01	-0.02*	-0.02	-0.02
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Intercept	-2.89***	-4.80***	-4.92***	-6.35***	-9.29***	-9.21***
	(0.69)	(0.89)	(0.92)	(0.98)	(1.31)	(1.40)

Notes: All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%);
(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school
Abbreviations are: SE, standard error; SES, socioeconomic status
[a] Omitted category in parentheses.
$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5.7b Black-white comparison of the association between patella lead and hypertension, NHANES 2001-2004

	Men (N=2121)			Women (N=2112)		
	(1) Age	(2) + Risk	(3) + Risk, SES	(1) Age	(2) + Risk	(3) + Risk, SES
	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)	b (SE)
Race						
(White)[a]	--	--	--	--	--	--
Black	0.43***	0.45***	0.41**	1.00***	0.91***	0.93***
	(0.11)	(0.12)	(0.12)	(0.13)	(0.14)	(0.14)
Patella lead	0.04***	0.02**	0.02*	0.04***	0.02	0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)
Interaction						
Race*patella lead	0.00	0.00	0.00	-0.01^{+}	-0.01	-0.01
	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)
Intercept	-2.48***	-4.42***	-4.74***	-5.54***	-10.02***	-10.46***
	(0.76)	(0.90)	(1.00)	(1.09)	(1.55)	(1.91)

Notes: All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:

(1) Age (age and age^2);

(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%);

(3) Family poverty-to-income ratio: ≤1.85, >1.85; educational attainment: <high school, high school diploma, >high school

Abbreviations are: SE, standard error; SES, socioeconomic status

[a] Omitted category in parentheses.

$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5. 8a Estimated tibia lead-hypertension association by race and educational attainment, NHANES 2001-2004

	Men (N=2121)		Women (N=2112)	
	(1) Age	(2) + Risk	(1) Age	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	0.53***	0.51***	1.06***	0.89***
	(0.12)	(0.14)	(0.14)	(0.16)
Education				
(≥High school)	--	--	--	--
<High school	0.28	0.62*	-0.05	0.10
	(0.28)	(0.27)	(0.24)	(0.25)
Tibia lead	0.05***	0.03***	0.06***	0.03*
	(0.01)	(0.01)	(0.01)	(0.02)
Interactions				
Race*education	-0.61	-0.83^{+}	-0.32	-0.40
	(0.44)	(0.42)	(0.32)	(0.33)
Race*lead	-0.01	-0.01	-0.02	-0.02
	(0.01)	(0.01)	(0.01)	(0.01)
Education*lead	-0.03*	-0.03*	-0.02	-0.02
	(0.01)	(0.01)	(0.02)	(0.02)
Race*education*lead	0.04^{+}	0.06*	0.02	0.03
	(0.02)	(0.02)	(0.02)	(0.02)
Intercept	-2.85***	-5.19***	-5.91***	-9.18***
	(0.76)	(0.99)	(1.08)	(1.47)

Notes:
All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).

$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5.8b Estimated patella lead-hypertension association by race and educational attainment, NHANES 2001-2004

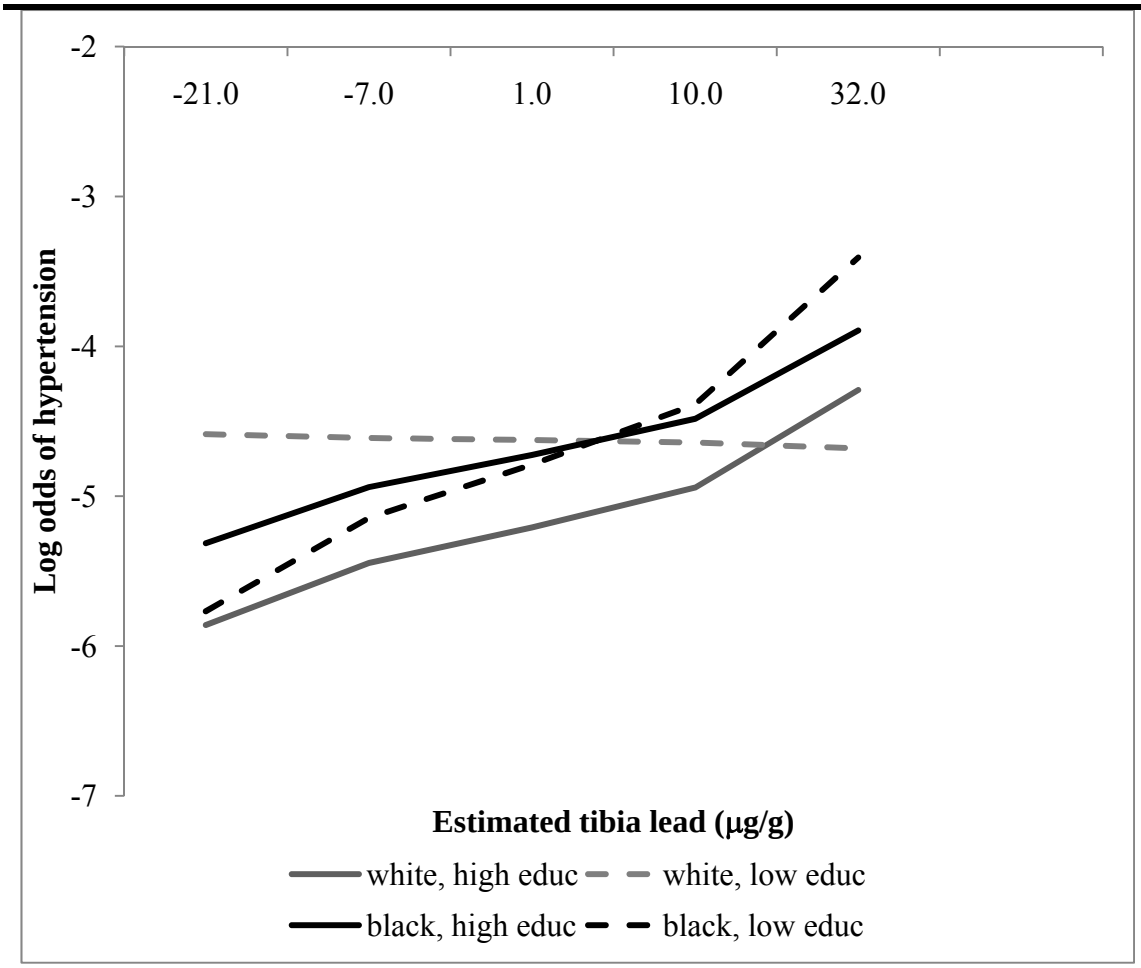
	Men (N=2121)		Women (N=2112)	
	(1) Age	(2) + Risk	(1) Age	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	0.47***	0.48***	1.07***	0.95***
	(0.13)	(0.15)	(0.13)	(0.15)
Education				
(≥High school)	--	--	--	--
<High school	0.21	0.57*	-0.05	0.14
	(0.26)	(0.26)	(0.24)	(0.25)
Patella lead	0.04***	0.02*	0.05***	0.02
	(0.01)	(0.01)	(0.01)	(0.01)
Interactions				
Race*education	-0.38	-0.57	-0.48	-0.48
	(0.42)	(0.40)	(0.36)	(0.38)
Race*lead	0.00	0.00	-0.01	-0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Education*lead	-0.02^{+}	-0.02*	-0.01	-0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Race*education*lead	0.02	0.03^{+}	0.02	0.02
	(0.02)	(0.02)	(0.02)	(0.02)
Intercept	-2.45***	-4.66***	-5.16***	-9.39***
	(0.86)	(1.07)	(1.18)	(1.67)

Notes:
All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:

(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).

$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Figure 5. 1 Black-white differences in the association between estimated tibia lead and hypertension by race and educational attainment in men, NHANES 2001-2004



Notes:

Slopes are weighted to account for complex survey design. Slopes are adjusted for age, age^2, BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).

Table 5. 9a Estimated tibia lead-hypertension association by race and poverty status, NHANES 2001-2004

	Men (N=2121)		Women (N=2112)	
	(1) Age	(2) + Risk	(1) Age	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	0.52***	0.54***	0.84***	0.68***
	(0.13)	(0.16)	(0.17)	(0.16)
Poverty status				
(≥1.85; nonpoor)	--	--	--	--
<1.85; poor	0.16	0.24	-0.17	-0.14
	(0.18)	(0.19)	(0.20)	(0.19)
Tibia lead	0.04***	0.03***	0.05**	0.03
	(0.01)	(0.01)	(0.01)	(0.02)
Interactions				
Race*poverty	-0.22	-0.36	0.50^{+}	0.56^{+}
	(0.28)	(0.32)	(0.25)	(0.24)
Race*lead	0.00	0.00	0.00	0.00
	(0.02)	(0.01)	(0.01)	(0.01)
Poverty*lead	-0.01	-0.01	0.00	0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Race*poverty*lead	0.00	0.01	-0.03*	-0.03^{+}
	(0.02)	(0.02)	(0.01)	(0.02)
Intercept	-3.01***	-5.05***	-6.27***	-9.44***
	(0.74)	(0.91)	(1.04)	(1.41)

Notes:
All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:
(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).

$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5.9b Estimated patella lead-hypertension association by race and poverty status, NHANES 2001-2004

	Men (N=2121)	Women (N=2112)

	(1) Age	(2) + Risk	(1) Age	(2) + Risk
	b (SE)	b (SE)	b (SE)	b (SE)
Race				
(White)[a]	--	--	--	--
Black	0.47**	0.49**	0.88***	0.77***
	(0.13)	(0.16)	(0.15)	(0.15)
Poverty status				
(≥1.85; nonpoor)	--	--	--	--
<1.85; poor	0.06	0.20	-0.17	-0.13
	(0.17)	(0.19)	(0.18)	(0.17)
Patella lead	0.04***	0.02**	0.04***	0.02
	(0.01)	(0.01)	(0.01)	(0.01)
Interactions				
Race*poverty	-0.13	-0.20	0.35	0.36
	(0.29)	(0.31)	(0.24)	(0.24)
Race*lead	0.00	0.00	0.00	0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Poverty*lead	0.00	0.00	0.00	0.00
	(0.01)	(0.01)	(0.01)	(0.01)
Race*poverty*lead	0.00	0.00	-0.02	-0.01
	(0.01)	(0.01)	(0.01)	(0.01)
Intercept	-2.53***	-4.42***	-5.37***	-9.51***
	(0.81)	(0.94)	(1.16)	(1.61)

Notes:
All results are weighted to account for complex survey design. Results are logit model coefficients.
Models sequentially add:

(1) Age (age and age^2);
(2) BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).

$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5. 10a: Net effects of the association between tibia lead and hypertension for race-SES groups (calculated from coefficients in Tables 5.8a and 5.9a)

	Men		Women	
	Black b (SE)	White b (SE)	Black b (SE)	White b (SE)
Educational attainment				
<High school	0.04*	0.00	0.03^{+}	0.02
	(0.02)	(0.02)	(0.02)	(0.02)
≥High school	0.03	0.03	0.01	0.03
	(0.02)	(0.01)	(0.02)	(0.02)
Poverty status				
<1.85 (poor)	0.04**	0.03**	0.00	0. 03^{+}
	(0.01)	(0.01)	(0.01)	(0.01)
≥1.85 (nonpoor)	0.04*	0.03	0.02	0.03
	(0.02)	(0.01)	(0.02)	(0.02)

Notes:
All results are weighted to account for complex survey design.
Results are calculated from the following equation:
$\text{Hypertension} = \beta_0 + \beta_1\text{race} + \beta_2\text{lead} + \beta_3\text{ses} + \beta_4\text{race*lead} + \beta_5\text{ses*lead} + \beta_6\text{race*ses} + \beta_7\text{race*ses*lead}$
Percent increase in odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead for:
black men, low SES = $\beta_2 + \beta_4 + \beta_5 + \beta_7$
white men, low SES = $\beta_2 + \beta_5$
black men, not low SES = $\beta_2 + \beta_4$
white men, not low SES = β_2
Models are adjusted for Age (age and age^2); BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).
$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

Table 5.10b: Net effects of the association between patella lead and hypertension for race-SES groups (calculated from coefficients in Tables 5.8b and 5.9b)

	Men		Women	
	Black b (SE)	White b (SE)	Black b (SE)	White b (SE)
Educational attainment				
<High school	0.03*	0.00	0.02	0.01
	(0.01)	(0.01)	(0.01)	(0.02)
≥High school	0.02	0.02*	0.01	0.02
	(0.02)	(0.01)	(0.02)	(0.01)
Poverty status				
<1.85 (poor)	0.03*	0.02*	0.00	0.02
	(0.01)	(0.01)	(0.01)	(0.01)
≥1.85 (nonpoor)	0.03^{+}	0.02**	0.01	0.02
	(0.01)	(0.01)	(0.02)	(0.01)

Notes:
All results are weighted to account for complex survey design.
Results are calculated from the following equation:
$\text{Hypertension} = \beta_0 + \beta_1\text{race} + \beta_2\text{lead} + \beta_3\text{ses} + \beta_4\text{race*lead} + \beta_5\text{ses*lead} + \beta_6\text{race*ses} + \beta_7\text{race*ses*lead}$
Percent increase in odds of hypertension for each $^{\mu g}/_{g}$ increase in tibia lead for:
black men, low SES = $\beta_2 + \beta_4 + \beta_5 + \beta_7$
white men, low SES = $\beta_2 + \beta_5$
black men, not low SES = $\beta_2 + \beta_4$
white men, not low SES = β_2
Models are adjusted for Age (age and age^2); BMI (body mass index, $^{kg}/_{m^2}$); smoking (status: never, former, current; amount: pack-years); alcohol use (drinks/week); no physical activity; diabetes diagnosis; hematocrit (%).
$^{+}$p<0.10; *p<0.05; **p<0.01; ***p<0.001

CHAPTER SIX

DISCUSSION

Black-white disparities in hypertension are particularly large and persistent (Ong et al., 2007; Wolf-Maier et al., 2003). Although the causes of these disparities are not completely understood, factors from the social environment appear to play an important role (Adler & Rehkopf, 2008; Geronimus et al., 2006; Sapolsky, 2005; Williams, 1999). One mechanism by which social factors increase hypertension disparities may be through alterations in susceptibility to environmental pollutants (Clougherty & Kubzansky, 2009; Gee & Payne-Sturges, 2004; Morello-Frosch & Lopez, 2006).

I tested this notion using education and poverty as proxies of constraints and resources in the social environment and depressive symptoms as a proxy of psychosocial stress. I took advantage of an unexplained pattern in the association between lead and blood pressure whereby black but not white adults show this positive association (Den Hond et al., 2002; Vupputuri et al., 2003). In other words, there is a black-white disparity in the *effect* of lead on blood pressure. To my knowledge, no one has examined the reason for this disparity.

In this chapter, I discuss the results presented in Chapters Four and Five. First, I discuss the results in detail, including potential reasons for null findings and alternate explanations for the hypothesized findings. I divide this discussion by chapter, discussing blood lead and blood pressure first and then estimated bone lead and hypertension. Second, I outline the strengths and limitations of my work. I end this discussion with implications for intervention and policy, and next research steps.

Black-white disparities in the effect of blood lead on systolic blood pressure

The results of my research show that blood lead levels have decreased for both black and white adults since the last report in the literature. In the NHANES III (1988-1994), geometric mean blood lead levels for black and white men were $5.4^{\mu g}/_{dl}$ and $4.4^{\mu g}/_{dl}$, respectively (Den Hond et al., 2002). Using NHANES 2001-2006, I report that current levels are $2.1^{\mu g}/_{dl}$ and $1.7^{\mu g}/_{dl}$, respectively. There are similar decreases for black and white women. In the NHANES III, levels were $3.4^{\mu g}/_{dl}$ and $1.4\ ^{\mu g}/_{dl}$, respectively (Den Hond et al., 2002). Here, I document that they are $1.2^{\mu g}/_{dl}$ and $1.5^{\mu g}/_{dl}$, respectively. There is still a small disparity, but blood lead levels for both black and white adults are at their lowest point in the NHANES.

Notably, however, I find that there has *not* been a change in the association between blood lead and systolic blood pressure for black men or women. Den Hond et al. (2002) found a three mmHg increase in systolic blood pressure for each doubling of blood lead for black men and a four mmHg increase in systolic blood pressure for each doubling of blood lead for black women. These associations were not found among whites. The results from my analyses nearly perfectly replicates Den Hond et al.'s (2002)

systolic blood pressure results, despite the reduction in mean blood lead levels across the decade.

It is not likely that the small disparity in blood lead levels explains the persistent disparity in the effect of lead. The results from my analyses using the Oaxaca-Blinder decomposition show that even if black adults had the lead levels of white adults, there would be no change in their blood pressure. These results are consistent with the literature using other techniques to account for the black-white disparities in blood lead level (Martin et al., 2006; Muntner et al., 2005; Sorel et al., 1991) In other words, the slightly higher *level* of blood lead in black compared to white adults does not explain the stronger *effect* of lead on blood pressure for black adults.

Depressive symptoms and susceptibility to the effects of blood lead

I tested the notion that social factors interact with lead to increase blood pressure and contribute to black-white disparities in the effect of lead on blood pressure. The results from my analyses are mixed. Regarding education, the results from my analyses show that black men with low education have a stronger association between blood lead and systolic blood pressure compared to black men with high education. This is not the case for black women –both low- and high-educated black women have the same positive association between blood lead and systolic blood pressure. Regarding poverty, the results are the same for black men and women. Both poor and nonpoor black men and women show the same positive association between blood lead and blood pressure. While mixed, these results suggest that low SES may be important for the effect of lead.

The results from my analyses are particularly striking when comparing black adults who report high and low depressive symptoms. Black men who report high depressive symptoms show an 8.6mmHg increase in systolic blood pressure for each doubling of blood lead while black men who report low depressive symptoms show only a 2.0mmHg increase. Similarly, black women who report high depressive symptoms show a 7.2mmHg increase in systolic blood pressure for each doubling of blood lead while black women who report low depressive symptoms show only a 1.2mmHg increase.

Depressive symptoms are often viewed as symptomatic of stress, and sometimes used as a proxy for psychosocial stress (J. S. Jackson, Knight, & Rafferty, 2010; Latkin & Aaron, 2003; R. J. Turner, Wheaton, & Lloyd, 1995). Furthermore, research suggests that dysregulation of the biological stress systems is an important link between psychosocial stress and depressive symptoms (McEwen, 2003, 2005). In other words, depressive symptoms may be a proxy for dysregulation of the biological stress systems more broadly rather than psychosocial stress, *per se*.

Because factors other than psychosocial stress can result in the dysregulation of the biological stress systems, there may be factors other than psychosocial stress that increase susceptibility to the harmful effects of lead on blood pressure. These other susceptibility factors range from fetal nutritional environment to environmental pollution (McMillen et al., 2008; Meaney, Szyf, & Seckl, 2007; Virgolini et al., 2006; Xu, Umezawa, & Takeda, 2009). Notably, as with psychosocial stress, these other susceptibility factors have also been shown to be socially-patterned (Brown, 1995; Hobel & Culhane, 2003). In other words, my results suggest that there may be numerous

socially-patterned factors that increase susceptibility to the harmful effects of lead on blood pressure.

Unexpected results in the effect of blood lead

Gender, education, blood lead, and systolic blood pressure

The results from my analyses show that black women with high education show a similar association between blood lead and systolic blood pressure as black women with low education. In contrast, black men with high education show a stronger association between blood lead and systolic blood pressure compared to black men with low education. This suggests that there are gender differences in the constraints and resources associated with low education. It may be that black women, who have a long history in the work force particularly in clerical and sales occupations, are able to find employment without a high school education (Jones, 1986). On the other hand, black men without a high school education are particularly unlikely to find employment (Holzer, Offner, & Sorensen, 2005a). In other words, when using education as a proxy for constraints and resources, gender differences in the returns to education should be considered. In fact, preliminary analyses using a higher educational level for the threshold of high constraints/low resources for women results in the hypothesized pattern. Black women with up to a high school education show a stronger association between lead and systolic blood pressure compared to black women with more than a high school education.

Poverty status, blood lead, and systolic blood pressure

The results from my analyses show that both poor and nonpoor black adults show similar associations between blood lead and systolic blood pressure. In other words, poor black adults do not show the hypothesized stronger effect of lead compared to nonpoor black adults. This may be due to the threshold of the poverty income ratio (PIR) used to define "poor". The threshold of 1.85 is used as it is the threshold used for several means-tested government assistance programs (US Department of Agriculture, 2008).

However, there may be a more appropriate threshold when using poverty status as a proxy for resources and constraints. For example, middle-income black Americans are often constrained to similar neighborhoods as low-income black Americans (Pattillo, 1999, 2005). Neighborhoods composed primarily of low-income or black residents pay higher prices for many goods and services such as home and auto insurance and banking services (Fellowes, 2006). Neighborhoods composed primarily of black Americans often lack amenities such as fully-stocked grocery stores and pharmacies (Morrison et al., 2000; Zenk et al., 2005). Therefore, a higher PIR threshold may be more appropriate. In fact, preliminary analyses using a PIR threshold that has been used in the literature to represent middle income results in the hypothesize pattern (Alaimo, Olson, & Frongillo Jr, 2001). Black women who have a PIR <3.00 show a stronger association between lead and systolic blood pressure compared to black women who have a PIR ≥3.00.

Alternatively, a polytomous variable may be appropriate. Research shows that higher-income black Americans often experience greater exposure to interpersonal and institutional racial discrimination as they integrate into the dominant white social environment (P. B. Jackson & Stewart, 2003; Pettigrew, 1999). This means that black-

white disparities in social stress (and health) would be large at the upper end of the SES distribution as well as the lower end. Research shows that black adults cannot translate upward mobility into better health outcomes to the extent that white adults can (Bond, Krueger, Rogers, & Hummer, 2003; Colen, Geronimus, Bound, & James, 2006). It may be that a PIR variable with three categories would capture such a nonlinear nature of the black-white disparities in stress and hypertension.

White adults, social factors, blood lead, and blood pressure

It is curious that white adults did not show the same interaction between lead and social factors that was seen for black adults. That is, white adults with low education, high poverty, or high depressive symptoms do not show a positive association between blood lead and systolic blood pressure. This may be due to my analyses, which include adults over the entire adult age range, coupled with the literature showing that blood lead levels at younger ages in white adults are very low. For example, geometric mean blood lead levels in NHANES III were $3.53^{\mu g}/_{dl}$ for women 50 years of age and older but only $2.09^{\mu g}/_{dl}$ for women under 50 years of age (S. K. Park et al., 2009). Therefore, it is likely that blood lead levels in the NHANES 2001-2006 dataset are nearly unmeasureable for women under 50 years of age. However, in order for social factors to exert some amplifying effect on lead, there must be some minimum level of blood lead. Lead levels increase with age, meaning that there should be amplifying effect of the social factors at older ages, when lead levels are higher – which can be tested by either using an older dataset or by including only older adults, who have higher blood lead levels, as lead levels increase with age. In fact, preliminary analyses restricted to those 50 years of age

and older show that white men with high depressive symptoms show a stronger association between lead and systolic blood pressure compared to white men with low depressive symptoms.

Blood lead and diastolic blood pressure

In contrast to the results from my analyses on systolic blood pressure, the results on diastolic blood pressure show little relationship with lead. The results show black-white disparities in adjusted blood lead and adjusted diastolic blood pressure. However, there are no disparities in the association between blood lead and diastolic blood pressure. These results are not consistent with the literature using the older NHANES III (1988-1994). In those studies, black but not white adults show a positive association between blood lead and diastolic blood pressure (Den Hond et al., 2002; Vupputuri et al., 2003) Notably, the results from those two studies, which use the same dataset, are also inconsistent. In only one of the studies, white men show a strong, *negative* association between blood lead and diastolic blood pressure (Table 4: $b= -1.98$; $p<0.001$) (Den Hond et al., 2002). In other words, there are inconsistencies among all studies using the NHANES examining lead and diastolic blood pressure over the entire adult lifespan.

It may be that diastolic blood pressure is not an appropriate marker of the effects of lead on the cardiovascular system over the entire adult lifespan. The primary mechanism by which lead results in hypertension is through oxidative stress (Vaziri, 2008; Vaziri & Sica, 2004). More broadly, oxidative stress is an important mechanism in aging and the development aging-related cardiovascular diseases, including hypertension (Harrison & Gongora, 2009; Khansari, Shakiba, & Mahmoudi, 2009). However,

diastolic blood pressure has a nonlinear (∩-shape) association with age (Franklin et al., 2001). In fact, diastolic blood pressure appears to mark an increased risk of cardiovascular disease (of which hypertension is one type) – but only for adults under 50 years of age (Franklin et al., 2001). This suggests that aging-related processes such as oxidative stress are not the predominant mechanisms involved in diastolic blood pressure changes over the lifespan. In other words, diastolic blood pressure may not be suitable to mark the cardiovascular effects of lead throughout the lifespan.

Estimated bone lead and hypertension

In addition to systolic and diastolic blood pressure, I also examined hypertension. In general, bone lead rather than blood lead is associated with hypertension. It may be that chronic and/or long-term lead exposure, marked by bone as opposed to blood lead, is necessary for the development of hypertension. However, bone lead is not collected in the NHANES. Instead, I used a newly-developed marker of bone lead that is estimated from blood lead and other sociodemographic and health variables. The estimation algorithm was developed from data in the Normative Aging Study (NAS), a sample of older white men in the Northeastern US (S. K. Park et al., 2009).

The results from my analyses show that black adults have higher levels of estimated bone lead, which is consistent with only study in the literature using this algorithm in the NHANES III (S. K. Park et al., 2009). However, black and white adults experience similar effects of lead on hypertension. For example, both black and white men experience a two percent increase in the odds of hypertension for each one $^{\mu g}/_{g}$ increase in estimated tibia lead (Table 5.7a, Model 3: $b=0.02$, $p<0.05$). These results are

similar to those in the literature using the NHANES III (S. K. Park et al., 2009). In other words, although black adults have higher estimated bone lead levels compared to white adults, they do not show a stronger effect of bone lead on hypertension compared to white adults.

It may be that the association between estimated bone lead and hypertension is nonlinear – and that there are black-white disparities in the nature of the nonlinear association. Figure 6.1 depicts a *hypothetical* diagram showing both the measured and latent association between tibia lead and hypertension. The solid black and gray lines represent the measured association between lead and hypertension for black and white adults, respectively. The lines are parallel, meaning that there is no black-white disparity in the effect of lead on hypertension. This is what the results from my analyses actually show. The dashed black and gray lines represent the latent, nonlinear association between lead and hypertension for black and white adults, respectively. Beginning at low lead levels and continuing through most of the lead distribution, black adults experience a positive association between lead and hypertension that levels off at higher lead levels. This leveling off may be due to a ceiling effect. For white adults, there is no association between lead and hypertension until higher lead levels.

This theory would support the notion that, at lower ends of the lead distribution, a susceptibility factor is required for the effect of lead on hypertension and that black adults experience the poorer social conditions that increase this susceptibility. At higher ends of the lead distribution, the level of lead begins to exert its influence on hypertension for white adults. If this theoretical representation is true, then black adults would actually show a stronger association between lead and hypertension in nonlinear models

compared to white adults. Preliminary analyses suggest that this may be the reason for the lack of black-white disparity in the association between bone lead and hypertension.

One caveat on the use of the bone lead algorithm is that estimated bone levels are likely underestimated for black adults. This means that the black-white disparities in actual bone lead are likely larger. This is because the factors important to the accumulation of lead in bone – which is the result of the complex interrelations among environmental lead exposure, absorption, and maintenance in bone – differ between the NAS sample (older white men) and other social groups.

For example, in the NAS, having a college degree reduced estimated tibia lead levels by $7.362^{\mu g}/_{g}$ (see Chapter 3 for details). This suggests that, for white men, a college degree results in substantial reduction in the risk of exposure somewhere in the pathway linking environmental lead exposure to bone lead deposition and maintenance. Research suggests that upward social and economic mobility is not translated into better health outcomes to the same extent for black and white adults (Colen et al., 2006). Middle and low income black adults often live in the same residential areas (Pattillo, 1999). Areas composed primarily of black residents are more likely to contain environmental pollutants compared to areas composed of predominantly white residents (Brown, 1995). Taken together, the literature suggests that increases in education do not result in the same reduction in environmental lead exposure for black adults as it does for white adults. In other words, there are black-white disparities in the meaning of education – and this difference in meaning impacts the accuracy of the algorithm in predicting actual bone lead levels for black adults.

Alternate explanations

Black-white differences in lead-related genetic composition

There may be genetic differences between the races that explain the difference in the effect of lead. Three genes have been shown to modify either blood lead levels or the effect of lead: δ-aminolevulinic acid dehydratase (ALAD), the vitamin D receptor (VDR), and a gene involved with hemochromatosis (HFE).

ALAD is the second enzyme in the heme production pathway of red blood cells. It has a high affinity for metal, including lead. When lead replaces the zinc that should be bound to ALAD, the effect of the enzyme decreases (Onalaja & Claudio, 2000). Furthermore, a certain type of the ALAD gene[6] may result in a stronger effect of lead on systolic blood pressure without altering blood lead levels (Scinicariello et al., 2009). However, NHANES III data show that 16% of non-Hispanic white adults carry this type of the gene, while only about 3% of non-Hispanic black adults do (Scinicariello et al., 2009). In other words, it is unlikely that ALAD, with its lower prevalence in black adults, explains the stronger association between lead and blood pressure in black compared to white adults.

Another gene, one associated with the receptor for vitamin D (VDR), may affect bone lead levels. One result of VDR activity is that lead is more readily carried from the blood to bone and increases the association between age and tibia lead (B. S. Schwartz et

[6] There are two co-dominant alleles of the ALAD gene – ALAD1, which is the more common, and ALAD2. These two alleles result in three isoforms of the enzyme with an allele number-effect association: ALAD1-1 (homozygous wild type), ALAD1-2 (carrier), and ALAD2-2 (homozygous) (Onalaja & Claudio, 2000).

al., 2000).[7] This means that VDR may affect the lead-blood pressure association simply by increasing the pool of lead stored in bone. To my knowledge, there are no reports of VDR modifying the *effect* of lead on blood pressure independent of lead level. As with ALAD, there are different versions with different levels of effect. If black Americans were more likely to have this effective version compared white Americans, then VDR may explain any modifying effects of race on the lead-blood pressure association – if the modifying effect is simply due to the *level* of bone lead. However, researchers report that black Americans are not more likely to have this effective version of VDR important in bone lead accumulation (Bell, Morrison, Nguyen, Eisman, & Hollis, 2001; C. F. James, Susan, Richard, & Bess, 1995).

Finally, mutations in the gene involved in the disease hemochromatosis, HFE, increase the intestinal absorption of iron and may also increase absorption of lead (Onalaja & Claudio, 2000). It may be that higher iron levels increase oxidative stress, which adds to the oxidative stress caused by lead. In other words, HFE may increase the harmful effect of lead on blood pressure through additive effects of oxidative stress. However, all models control for iron levels and yet, there were black-white disparities. Furthermore, black adults had lower iron levels compared to white adults in this sample. In sum, it is not likely that there are any genetic differences between black and white adults that are important for the effect of lead on blood pressure.

[7] Vitamin D binds with the VDR and ultimately increases the synthesis of calcium-binding proteins. Because lead and calcium are divalent ions with similar binding properties, these calcium-binding proteins also bind lead, facilitating its transport to bone.

Black-white disparities in cumulative lead exposure

The association between blood lead and systolic blood pressure may be due to greater cumulative exposure to lead by black compared to white adults over the lifecourse rather than an increased susceptibility due to social factors. In other words, the association seen in black adults may be due purely to the higher levels of cumulative lead exposure. This means that bone lead, a marker of cumulative lead exposure, should predict blood pressure over and above any effects of blood lead. Black adults have higher bone lead levels compared to white adults (Martin et al., 2006). However, it is not known if this higher bone lead results in a stronger association between bone lead and blood pressure for black compared to white adults.

Even if black-white disparities in cumulative lead exposure explain the black-white disparities in the association between blood lead and systolic blood pressure, social factors likely still play an important role. For instance, lead exposure may be higher in black compared to white adults, but once lead is sequestered in bone, it can remain there and do little damage to tissue if there is little release of that lead into the bloodstream. This means that the black-white disparities in lead exposure would not result in black-white disparities in lead effect, without some other factor, such as poor bone health.

However, racial disparities in bone health may be complex, particularly with regard to gender. For example, black women have higher body mass index (BMI) compared to white women (Flegal, Carroll, Ogden, & Johnson, 2002). BMI is negatively related to osteoporosis, which, in turn, is associated with blood lead (Campbell & Auinger, 2007; Lan-Juan et al., 2008; Nash et al., 2004; Silbergeld, Schwartz, & Mahaffey, 1988; Tsaih et al., 2001). Taken together, this means that black compared to

white women may have higher bone lead levels, but lower lead release from bone lead due to better bone health related to BMI. This suggests that, for women at least, the stronger association between blood lead and systolic blood pressure may not be simply due to the greater accumulation of lead by black compared to white women. This supports the notion that there are susceptibility factors important for the harmful effects of lead that vary by race.

Limitations of this research

Although these analyses were conducted with the best available data to examine black-white disparities in the association between lead, blood pressure, and social factors, there are several important limitations.

Direct or more traditional measures of the social environment (e.g., neighborhood poverty) and of psychosocial stress (e.g., Perceived Stress Scale) are not in the NHANES. Although my research has improved on previous work by acknowledging the incommensurate nature of traditional measures of SES across black and white race by interacting race with SES. Nonetheless, individual-level educational attainment and poverty status are still crude measures of the social environment.

Depressive symptoms are also a crude proxy of psychosocial stress, and may better reflect dysfunction of the biological stress systems. Depressive symptoms were only available in one wave at the time of data analysis. However, the NHANES 2007-2008 also contains these measures, allowing for replication of my analyses on the larger 2005-2008 dataset.

Blood lead, the lead biomarker used in most of these analyses, has several important limitations. Because nearly all blood lead is bound to red blood cells and therefore not available for effect, it is an indirect marker of the lead that is actually available for action in the body. Furthermore, blood lead marks short-term levels and may not provide accurate information about the long-term lead exposure and its effects. Bone lead, on the other hand, would better provide this information; however, bone lead is expensive to collect. Because of the expense involved in bone lead measurement, the prediction of bone lead from blood lead using a recently developed algorithm may be useful for future population-level studies.

NHANES is a cross-sectional study, precluding the examination of temporal ordering among lead, social stress, and blood pressure changes. There is also limited attention given to gender and age patterns of lead and hypertension, as this is the first test of my conceptual model.

Strengths and contributions of this research

My research addresses important gaps in the literature on black-white health disparities. Furthermore, it answers the call for interdisciplinary research on the interaction between the social and physical environments as fundamental cause of the poor health of black Americans (Gee & Payne-Sturges, 2004). At the core, my research is built on a strong foundation of social and biological literature. I approach black-white hypertension disparities with the understanding that US society sorts black and white Americans into profoundly different social and physical environments (Geronimus & Thompson, 2004; Morello-Frosch & Lopez, 2006; Williams & Collins, 2001). The

disparate environments are the fundamental determinants of black-white health disparities (Link & Phelan, 1996; Schulz et al., 2002; Williams & Collins, 2001). Through this "fundamental causes" lens, I integrate biologically-plausible mechanisms linking the social environment and health.

My research is one of only a handful of studies that examines the notion that social factors can increase the susceptibility to the harmful effects of environmental pollutants. It is the only research to my knowledge that specifically examines the harmful effects of social factors on the association between lead and blood pressure. Although there are earlier reports showing that black but not white adults experience this hypertensive effect of lead, no one has examined this disparity using statistical interactions rather than stratification (Den Hond et al., 2002; Vupputuri et al., 2003). Importantly, no one has examined the *reason* for the disparities in the effect of lead.

I use the most recent NHANES dataset available; there are no reports of blood lead levels in the literature using this recent dataset. The NHANES is the only population-based dataset with a large sample of black and white adults over a large age range that includes markers of lead and blood pressure. I also use a recently developed algorithm to estimate bone lead from blood lead and other health and demographic variables. In other words, I use the most up-to-date data available with markers of lead and the social factors in black and white adults.

Policy and intervention implications

The most direct implications of my research are that social factors, such as education and the underlying causes of depressive symptoms, may amplify the effects of

lead on blood pressure. Because blood lead levels are very low, it may be that even the amplified effect of lead experienced by black adults may not be important clinically. This is suggested by the fact that blood lead levels have dropped dramatically while hypertension has increased over the past decades. However, my research tests the more fundamental ideas in my conceptual model, particularly the broader notion that social factors can alter susceptibility to environmental pollutants. More specifically, it tests the notion that black-white disparities in social factors can results in black-white disparities in susceptibility to pollutants.

If social factors increase susceptibility to the effects of environmental pollutants, then policies that target these social factors as well as the environmental pollutants, simultaneously, may be most effective in reducing black-white hypertension disparities. For example, black men with low education show a stronger effect of lead on systolic blood pressure compared black men with higher education. From these results, policies that address primary and secondary education, as education at this level serves as an early point in the trajectory of access to resources and exposures to constraints over the lifecourse, may be most effective in reducing health disparities. Furthermore, these policies should target not only increasing racial parity in years of education, but also in the quality of education (Maxwell, 1994).

Next research steps

There are several analyses that can be completed in NHANES. First, all analyses should be rerun using imputed data using multiple imputation techniques. Second, the next wave of NHANES (2007-2008) was released in January 2010. Analyses involving

depressive symptoms should be repeated with the addition of this new wave to increase sample size and stability of estimates. Third, pulse pressure, the difference between systolic and diastolic blood pressures, as an outcome should be examined as this may be a better indicator of oxidative stress processes and a better predictor of cardiovascular events than systolic or diastolic blood pressure (Franklin et al., 2001). Fourth, a structural equation modeling (SEM) approach to testing my analytic model may provide more information about the interconnection associations among the variables. Fifth, the associations between lead – particularly estimated bone lead –and blood pressure may not be linear; exploration of non-parametric methods, including smoothing plots, may be helpful in clarifying associations. Sixth, it would be informative to know the extent to which nutritional markers, particularly the calcium/vitamin D system, explain the stronger effect of lead on blood pressure. Although this examination would not clarify the role of psychosocial stress, it may provide information regarding racial disparities in the susceptibility to the effects of lead. Finally, it may be possible to acquire access to geocoded information on NHANES respondents, as part of its non-public use files, to link them to US Census block and tract information. This would add important markers of the social environment, such as area-level poverty.

Regarding primary data collection, all analyses conducted as part of this project should be repeated with better measures of the social environment, such as residential area resources or SES or individual-level wealth. There should also be a more appropriate measure of psychosocial stress, which can be marked either with neighborhood environment, psychometric scales, or biological measures. While the former requires substantially fewer resources than the latter, a major challenge with using

psychometric scales is the ability to capture stress in both black and white adults. Most traditional scales (e.g., perceived stress, life events, etc) do not contain questions about racial discrimination, which is argued by some researchers, to be the most relevant form of stress in the lives of black Americans (Clark, Anderson, Clark, & Williams, 1999; Williams et al., 2003) Without including racial discrimination questions, the stress of black men and women will likely be underestimated.

There is also reason to consider biological measures of stress – one could capture the biological response to stress bypassing moderating psychological factors (e.g., personality, racial identity, stress perceptions, etc) and racial differences in stressors. However, stress research using biomarker has also proved inconsistent -- or most often yielded no racial differences in stress hormone levels (Chong, Uhart, McCaul, Johnson, & Wand, 2008; Cohen et al., 2006; Giannopoulou, Carhart, Sauro, & Kanaley, 2003). However, it may be that stress hormones collected in saliva, urine, and serum may not be the correct way to measure dysfunction of the biological stress systems (Miller, Chen, & Cole, 2009). This is because high levels of stress hormones do not actually reflect how well these hormones are working on their target systems in the body (Sapolsky, Romero, & Munck, 2000). Therefore, it may be beneficial to use biological measures of stress, but there is a need to develop and test biologically-plausible measures.

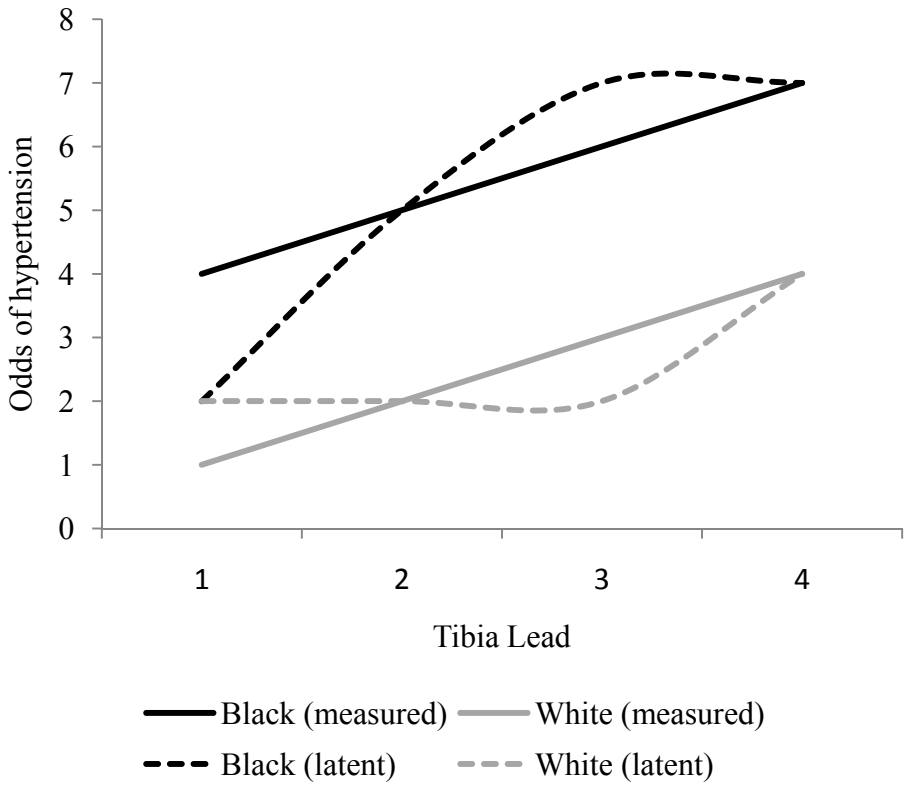
Another major step is to analyze the models with longitudinal and intergenerational data. Lead has been shown to be passed from mother to fetus (Gump et al., 2007; Rothenberg, Williams et al., 1996). Lead may be an important factor in the intergeneration transmission of poor birth outcomes and hypertension, and the association between poor birth outcomes and later hypertension development. For example,

umbilical cord and early childhood blood lead is positively associated with cardiovascular functioning, including blood pressure in children (Gump et al., 2007; Gump et al., 2005). Lead may serve as an important link between childhood environment and later adult health.

Conclusions

The results from my research partially support the notion that social factors can increase the susceptibility to the harmful effects of environmental pollutants. Even though policies have effectively reduced lead exposure in the general population, there are still fundamental factors that alter susceptibility to low lead levels, yielding increases in blood pressure even when lead levels are abated. Black-white disparities in social factors may yield disparities in susceptibility to the harmful effects of lead. These disparities in susceptibility may then result in disparities in blood pressure and hypertension.

Figure 6. 1 Theoretical diagram of the black-white disparities in the latent, nonlinear tibia lead-hypertension association



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