

The Racial Gap in Childhood Blood Lead Levels Related to Socioeconomic Position of Residence in Metropolitan Detroit

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Abstract

Childhood lead poisoning in the United States remains a persistent, prevalent environmental public health problem, especially for children living in central-city neighborhoods. These neighborhoods typically are racially segregated, are in proximity to current and/or legacy lead emission sources, consist of older housing, and contain disproportionately African American or black children of low-income families. This research had two aims: (1) to determine whether average blood lead levels (BLLs) in children in the Detroit metropolitan area are related to the socioeconomic characteristics of the neighborhoods where they live and (2) to determine the estimated effect residential differences in the socioeconomic characteristics of neighborhoods have on average BLLs in non-Hispanic black and non-Hispanic white children. Data on pediatric BLLs were obtained from the Michigan Department of Community Health, and racial and socioeconomic data were obtained from the U.S. Census Bureau's American Community Survey (2006–2010). The modified Darden-Kamel Composite Socioeconomic Index, multiple regression, and difference-of-means tests were used to determine the effect residential socioeconomic characteristics of neighborhoods have on average BLLs. Black segregated neighborhoods with lower socioeconomic characteristics were predictors of higher average BLLs in the children who lived there. When black and white children resided in neighborhoods of similar socioeconomic characteristics, the black-white gap in BLLs lessened. Significantly, after stratifying black and white children by age, living in the same neighborhoods of the lowest socioeconomic characteristics negated the black-white racial gap in BLLs entirely, but increasing levels of socioeconomic characteristics exacerbated the divide.

Keywords

lead poisoning, Detroit, race, socioeconomic position, neighborhoods

Childhood lead poisoning is a critical environmental inequality issue with racial and spatial overtones. Lead poisoning in children is due primarily to environmental conditions of the children's residences. The most significant sources are chips, dust, and soil incorporated with lead-based paint used in and on housing structures. Children subsequently ingest and/or inhale these sources (Meyer et al. 2003; U.S. Agency for Toxic Substances and Disease Registry 2007; U.S. Department of Housing and Urban Development 1990). Regulations eliminating

leaded paint, gasoline, and other lead-based products in the United States have reduced the

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prevalence of childhood lead poisoning (U.S. Agency for Toxic Substances and Disease Registry 2007). However, lead continues to be emitted from industrial processes and remains in paint and pipes in older, nonrehabilitated housing, becoming incorporated in air, soil, dust, and drinking water. Other contributors of airborne lead are legacy roadside lead dust emitted from leaded gasoline (banned in 1996) and abandoned industrial sites. Lead residence time varies widely depending on a number of variables, such as temperature and pH (U.S. Agency for Toxic Substances and Disease Registry 2007). These various contributions mean that childhood lead poisoning continues to be a major public health concern (Centers for Disease Control and Prevention 2012), and affected children are most likely to live in minority neighborhoods (Haley and Talbot 2004; Oyana and Margai 2010); in neighborhoods of very low socioeconomic status (SES), with the oldest housing (Bernard and McGeehin 2003; Haley and Talbot 2004; Kaplowitz, Perlstadt, and Post 2010; Krieger et al. 2003; Miranda, Dolinoy, and Overstreet Galeano 2002; Oyana and Margai 2010; Vivier et al. 2010); and in geographic proximity to existing or abandoned lead-emitting industries or freeways (Centers for Disease Control and Prevention 1991; U.S. Agency for Toxic Substances and Disease Registry 2007).

In the United States, exposure to a variety of environmental hazards has produced a similar spatial pattern as supported by the environmental inequality literature. This literature is complex and accompanied with a variety of stakeholders and mechanisms (Pellow 2000) but consistently finds that depressed and high-unemployment communities, typically minority communities, are at greater risk for geographic proximity to hazardous wastes and polluting industries. Some of the causal mechanisms are lowered land values, lack of oppositional power (even in the guise of protest and community organizing), acceptance of pollutants with the promise of jobs, a lack of mobility due to poverty and housing discrimination (Bryant and Mohai 1992; Bullard 2007; Bullard and Wright 1987; United Church of Christ Commission for Racial Justice 1987), and discriminatory zoning and land-use practices (Bullard 2007; Bullard and Wright 1987; Pellow 2000; United Church of Christ Commission for Racial Justice 1987). Despite substantial resistance, a similar spatial pattern of environmental hazards has been found in the Detroit metropolitan area (DMA). Minorities and/or low-income residents are burdened with increased proximity to polluting industrial facilities, commercial hazardous waste

facilities, and abandoned or uncontrolled hazardous waste sites (Mohai and Bryant 1998), traffic particulate matter (Keeler et al. 2002), toxic releases (Downey 2006), auto and especially commercial truck traffic emissions (Wu and Batterman 2006), landfills and Superfund sites (Smith 2007), and Brownfield sites (Lee and Mohai 2011). As with these environmental hazards, there is evidence to suggest siting preference of lead-emitting industries within low-income and/or minority communities. Studies also find that there is unequal enforcement of air emission regulations in these neighborhoods (Kuehn 2012; Mohai, Pellow, and Roberts 2009; O'Rourke and Connolly 2003).

PAST NEIGHBORHOOD-EFFECTS RESEARCH

Neighborhood-effects research suggests that social disparities of neighborhoods significantly affect health, more than individual or family household variables (Guest, Almgren, and Hussey 1998). Neighborhood-effects studies have well established the link between income inequality and health; declining gradients of income are directly related to declining gradients of health (Kawachi 2000). Consistent with the earlier ideas of William Julius Wilson (1987), Acevedo-Garcia and Lochner (2003) showed that in the United States, minority racial/ethnic groups (especially African Americans) live in lower quality or substandard housing in urban neighborhoods with high unemployment, lower wages, teen pregnancy, joblessness, and higher crime rates. As a result, urban areas experience higher black mortality rates. Ross and Mirowsky (2008) investigated the relationship between concentrated poverty and adverse health outcomes, demonstrating that within Illinois neighborhoods, SES more significantly predicted adverse health outcomes in the form of physical impairment than individual or household socioeconomic variables. As neighborhood SES increased, physical functioning improved. In terms of neonatal health specifically, Grady and Darden (2012) showed in metropolitan Detroit that mothers living in low-income neighborhoods were more likely to experience preterm births. As such, when controlling for neighborhood socioeconomic characteristics, racial differences in health outcomes should be insignificant. However, social scientists and health researchers have concluded that higher incomes do not necessarily guarantee that blacks, for example, will live in neighborhoods with the same quality schools, housing, health care

facilities, and so on, as whites with comparable incomes (Darden and Kamel 2000; Massey and Denton 1993), leading to health disparities by race regardless of neighborhood SES (Diez Roux 2007).

Children exposed to lead are more susceptible than adults because they absorb lead more readily, and their developing nervous system puts them at increased risk for learning disabilities (U.S. Agency for Toxic Substances and Disease Registry 2007). Lead exposure in children shows a pronounced linear dose-effect relationship, and blood lead level (BLL) concentrations as low as $<5 \mu\text{g/dL}$ can cause permanent neurological impairment. At higher concentrations, asthma, bone loss, kidney damage, brain damage, and even death can result (U.S. Agency for Toxic Substances and Disease Registry 2007). The most recent statement from the U.S. Environmental Protection Agency (2000:38) notes that “there currently is no demonstrated safe concentration of lead in blood” for children aged one to five years. Many other studies have found no safe level of blood lead concentration without doing irreparable neurological, neurobehavioral, and physiological damage (Canfield et al. 2003; Chiodo, Jacobson, and Jacobson 2004; Lanphear et al. 2000; Lidsky and Schneider 2003; Miranda et al. 2007; Schnaas et al. 2000; Surkan et al. 2007; Téllez-Rojo et al. 2006; U.S. Agency for Toxic Substances and Disease Registry 2007). This means at the very least, populations of children living in close proximity to airborne or deposited lead are likely to experience decreased academic achievement compared with their unexposed counterparts, as found by Miranda et al. (2007) and Zhang et al. (2013).

As a result of this research, in January 2013, the Centers for Disease Control and Prevention (CDC) lowered the $\geq 10 \mu\text{g/dL}$ “level of concern” to $\geq 5 \mu\text{g/dL}$ (on the basis of the 97.5th percentile of BLLs in children one to five years old), designating this the “elevated BLL” or the “reference level.” At this level, retesting, follow-up evaluations, and services are provided by Medicaid and all other primary health care providers. The CDC is expected to lower this $\geq 5 \mu\text{g/dL}$ reference value and has agreed to seek additional research directed toward developing intervention capable of maintaining children’s BLLs lower than $5 \mu\text{g/dL}$ (Centers for Disease Control and Prevention 2012, 2013c).

Nationally, it was estimated that 535,000 children aged one to five years had BLLs at or above $5 \mu\text{g/dL}$ on the basis of the 2010 census population of this age group (Centers for Disease Control and Prevention 2013a). During that same year, there

were 12 million children with BLLs greater than $1 \mu\text{g/dL}$ (Centers for Disease Control and Prevention 2013b). These numbers are unquestionably much higher because generally, only 15 percent to 35 percent of children <72 months of age are tested nationally. Michigan ranks fifth in the nation in terms of childhood lead poisoning, and more than half the children currently in the Detroit public school system have had BLLs of concern ($\geq 10 \mu\text{g/dL}$) at some point (Centers for Disease Control and Prevention 2013b). Zhang et al. (2013) assessed the effect of early childhood lead exposure on long-term academic achievement of Detroit public school children from 2008 through 2010. They found that children with BLLs $> 10 \mu\text{g/dL}$ before six years of age were more than twice as likely to score “less than proficient” on academic achievement tests in math, science, and reading compared with children with BLLs $< 1 \mu\text{g/dL}$. Similarly, Miranda et al. (2007) found that early childhood exposure to lead, even at $2 \mu\text{g/dL}$, was found to be associated with significantly lower end-of-grade scores in North Carolina children. At these levels, any other childhood health dilemma, especially one causing permanent damage, would be treated as an urgent, national crisis.

OBJECTIVES OF THE STUDY

The objectives of this study were to investigate independently a neighborhood socioeconomic index and the extent of lead poisoning disparities among non-Hispanic white and non-Hispanic black children located in metropolitan Detroit from 2006 through 2010. Efforts were made to better understand whether health disparities (in this case lead poisoning) by race exist when controlling for neighborhood socioeconomic characteristics. In other words, are black-white racial BLL disparities better explained by the characteristics of the neighborhoods in which these children live or by independent racial inequalities? We also examined the relationship of these BLLs and location within the central city compared with the inner and outer suburbs of the DMA.

This research is unique with regard to lead studies of Detroit or other metropolitan areas for the following reasons: (1) We used continuous BLL data as opposed to dichotomizing or grouping levels (e.g., elevated vs. not elevated); (2) we linked race data with Medicaid data to achieve a 90 percent report on race, as opposed to assuming that the race of the child equates to the majority of those living in the geographic unit of study; (3) we used

exploratory regression on individual measures of socioeconomic position (SEP) to establish a more predictive composite index; (4) we transformed BLL data appropriately, as dictated by methodological guidance from the U.S. Department of Health and Human Services, as opposed to deleting or manufacturing low BLL values; (5) we rejected regression results with unacceptable distributions of residuals; (6) we mapped the geographic distribution of BLLs in relation to the SEPs of neighborhoods (census tracts) in the DMA; and (7) we used neighborhood-level analyses as opposed to individual analyses that allowed comparison of BLLs of black versus white children within and across neighborhoods of similar and differing socioeconomic characteristics. This uncovered significant findings in racial disparities of BLLs within increasing levels of SEP.

STUDY AREA: METROPOLITAN DETROIT

For the purpose of this study, the DMA includes three counties: Macomb, Oakland, and Wayne. The DMA is sharply divided by race, place of residence, and socioeconomic characteristics of neighborhoods. Blacks and the poor reside disproportionately in the central city, while whites and the affluent reside disproportionately in the suburbs (Farley, Danzinger, and Holzer 2000; Darden et al. 2010). Even though racial residential segregation has declined since the 1950s (Darden and Thomas 2013), this does not necessarily mean that the gap between blacks and whites in neighborhood socioeconomic characteristics has also declined. Nor does it suggest that the gap in health, or in this case BLLs, has declined. The research shows that even when blacks achieve higher incomes than whites, these incomes do not automatically translate into higher quality housing (a harbinger of reduced BLLs), improved neighborhood characteristics (Darden 2009), or better health outcomes (Diez Roux 2007). The pattern of extreme class and racial geographic disparity makes metropolitan Detroit an ideal area to understand racial and socioeconomic disparities in health generally and childhood lead poisoning in particular.

Within the DMA, this study defined inner suburbs as incorporated places (cities) that bordered the central city of Detroit and/or were located less than five miles from the Detroit border. The outer suburbs were located five miles or greater from Detroit's border. Percentage population change (2000–2010) was calculated for the city and

suburbs in an attempt to see whether growth and decline in housing were associated with pediatric BLLs.

THE USE OF CENSUS TRACTS FOR NEIGHBORHOOD ANALYSIS

Census tracts were used as the level of analysis because this areal unit has been found to successfully capture sociodemographic characteristics of a neighborhood at a small scale (Darden et al. 2010; Krieger 2006; Krieger et al. 2003) while also consistently capturing socioeconomic gradients in health (Grady 2006; Subramanian et al. 2005). Using smaller geographic units yields a much smaller number of cases per area. Furthermore, the Michigan Department of Community Health (MDCH) prohibits individual addresses or census block group designations associated with BLL reports as a result of institutional review board policy due to a breach in patient confidentiality of children's BLLs and associated addresses. Because pediatric blood lead medical records lack information on the SES of the patients, census tract socioeconomic data were used as a quantitative proxy representing these characteristics and their relationships to disparities in health. For the purpose of this research, census tracts were most useful because of the expanse of data available and the ability to map the spatial distribution of these characteristics.

DATA

Pediatric Lead Data

Individual pediatric BLLs, reported in micrograms per deciliter, were obtained with institutional review board approval from the MDCH Childhood Lead Poisoning Prevention Program's statewide database. All Michigan Medicaid-enrolled children are required to be tested for lead at 12 and 24 months of age or between 36 and 72 months of age if not previously tested at the earlier ages. All blood lead test results (Medicaid or non-Medicaid) are reported, by law, to the MDCH (Michigan Childhood Lead Poisoning Prevention and Control Commission 2007; Michigan Department of Community Health 2004, 2013).

Between 2006 and 2010, there were 277,676 children less than one month to 16 years of age who underwent BLL testing in the DMA. These BLL reports were submitted to the MDCH and made

available for this study. Of these 277,676 case reports, 8,391 records were missing census tract identifiers, leaving 269,285 records for subsequent analyses. Then an additional 53,184 records were eliminated from the data set because they lacked appropriate composite socioeconomic index (CSI) values (described below). This left a total of 216,101 records of all races/ethnicities in which BLLs were analyzed and mapped in later sections of this study.

Most of the laboratory BLL collection units were capable of detecting levels of 1.0 $\mu\text{g}/\text{dL}$ or less. However, a small number of laboratories had higher detection limits. As a result, testing sites coded all nondetect values, and any value 1.4 $\mu\text{g}/\text{dL}$ or lower, as 1.0 $\mu\text{g}/\text{dL}$. Otherwise, test results were rounded to the nearest whole number. Portable blood lead analyzers had 3.3 $\mu\text{g}/\text{dL}$ detection limits that either detected no lead (recorded as 1 $\mu\text{g}/\text{dL}$) or 3.3 $\mu\text{g}/\text{dL}$ (recorded as 3.0 $\mu\text{g}/\text{dL}$) and greater. The "level of concern" was ≥ 10 $\mu\text{g}/\text{dL}$ for the entire data collection period.

The MDCH receives these BLL tests from the laboratories identifying the case child by name, address, gender (male $n = 110,788$ [51.54 percent]; female $n = 104,181$ [48.46 percent]; no report $n = 1,132$), birthdate, Medicaid recipient status ($n = 153,890$ [71.21 percent]) or non-Medicaid status ($n = 62,211$ [28.79 percent]), and parental self-reported race/ethnicity. These data were obtained from the MDCH for 2006 through 2010. Each child was assigned an ID, and 100 percent of the addresses were geocoded by the MDCH to the accompanying census year 2010 census tract. The sample bias of Medicaid children was overcome to some extent by gathering five years of BLL surveillance data and thus capturing more non-Medicaid cases as well. Also, Michigan tests BLLs of approximately 90 percent of all DMA children by the age of six years, ensuring that a large number of non-Medicaid children were represented in this study (Michigan Department of Community Health 2013). There were 45,202 (21.07 percent) and 169,365 (78.93 percent) children who were tested for BLL using the capillary and venous methods, respectively (no report $n = 1,534$). If an elevated BLL was present in the first capillary or venous test (≥ 10 $\mu\text{g}/\text{dL}$ during the years covered by this study), the child was administered a second venous test, and only this new venous BLL was entered, overriding the prior result.

The birthdate report rate was 100 percent, and this date was converted to age in months as a continuous variable. The number of children aged less than one month to 2 years equaled 101,617 (47.02

percent). Those aged over 2 and up to 6 years included 88,260 (40.84 percent) children, and those greater than 6 and up to 16 years consisted of 26,224 (12.14 percent) children. Controlling for age is important to this study because children typically produce the highest BLLs at age 2, as a result of hand-to-mouth behavior and playing on floors (U.S. Agency for Toxic Substances and Disease Registry 2007). Furthermore, blood lead tests reveal fairly current exposure conditions at the time of testing because blood lead travels to the soft tissues and organs, where it is stored only for a period of weeks. It is later expelled either through urine or feces or travels to teeth and bones for long-term storage (U.S. Agency for Toxic Substances and Disease Registry 2007).

Racial and ethnic groups designated within the database include non-Hispanic black or African American ($n = 97,344$ [53.75 percent]), non-Hispanic white ($n = 68,515$ [37.83 percent]), Hispanic ($n = 5,408$ [2.99 percent]), Native Hawaiian or other Pacific Islander ($n = 99$ [0.05 percent]), Asian ($n = 4,701$ [2.60 percent]), Arab ($n = 3,887$ [2.15 percent]), American Indian or Native Alaskan ($n = 569$ [0.31 percent]), and mixed race ($n = 594$ [0.33 percent]). If Hispanic and another race were both reported, the child was coded as Hispanic. The MDCH (2013) finds that tested children's parents report race approximately 50 percent of the time. This resulted in "no-report" race designations. Importantly, the MDCH linked the BLL reports of children on Medicaid to their Medicaid case records to achieve an 80 percent to 90 percent complete report on race in the database. In total there were 34,984 cases (16.19 percent) entered as "no report" on race. These no-reports were not eliminated from the mapping portion of this study, but they were eliminated from the data set in subsequent black-white-only analyses, along with an additional 15,258 Asian, Hispanic, and other ethnicities, cases. This provided a large sample size of 165,859 records encompassing non-Hispanic black and non-Hispanic white race designations only.

Neighborhood Data

Using the 2010 census tract boundaries, a total of 1,046 DMA tracts were mapped and used in analyses of this study. The U.S. Census Bureau's American Community Survey provides data by census tract on a variety of socioeconomic variables, presented below. The five-year (2006–2010) estimate data of these variables were used in analyses with corresponding years of BLL data. The five

years of MDCH blood lead data provided per census tract achieved a 100 percent match rate with the Census Bureau's census geography for the same five-year estimate data.

METHODOLOGY

Blood Lead Data

Only BLLs recorded as 1.0 $\mu\text{g}/\text{dL}$ ($n = 91,104$ [42.16 percent]) by the testing laboratories and having original values of nondetect but up to 1.4 $\mu\text{g}/\text{dL}$ (and even up to $<3.3 \mu\text{g}/\text{dL}$ for portable units) were first divided by the square root of 2. Methodological guidance from the Centers for Disease Control and Prevention (2009) recommends dividing those concentrations that could be less than the limit of detection by the square root of 2 before log transformation or calculation of geometric means. Individual-level analyses used these log-transformed values, while other analyses required aggregation of the BLLs at the census tract level. As such, the geometric means of the transformed BLLs were calculated per census tract (Centers for Disease Control and Prevention 2009).

SEP Index

We used area-based indicators of SEP applied as proxies for individual-level measures of SEP by using the modified Darden-Kamel Composite Socioeconomic Index (Darden et al. 2010). This index, used successfully in other social science studies, measures SES and assigns a higher score to census tracts with high SEP. A high score reflects better socioeconomic quality of neighborhood characteristics, and a low score reflects poor-quality neighborhood characteristics. The modified Darden-Kamel Composite Socioeconomic Index incorporates nine variables to calculate a CSI, defined as follows. The percentage below poverty is the percentage of all occupied households whose incomes in the past 12 months were below the U.S. poverty level. The poverty thresholds vary depending on size of family, number of related children, and, for one- and two-person families, age of householder by the Census Bureau. The unemployment rate is the percentage of civilians 16 years and older who were neither at work nor with a job but not at work during the reference week and who were actively seeking work during the past four weeks and available to start a job. Median

household unit income is the median income of all family members 16 years and older, including those without incomes. Percentage of households with vehicles is the percentage of occupied housing units with vehicles available. Percentage of residents with management, business, science, and arts occupations is the percentage of workers 16 years and older who hold 1 of 194 positions codified by the Census Bureau for 2006 to 2010 five-year estimates. Percentage of residents with bachelor's degrees or higher is the percentage of the total population 25 years and older with at least a bachelor's degree (e.g., 4 or more years of schooling beyond a high school education). Median value of dwelling in dollars is the median value of owner-occupied housing in which the respondent estimated how much it would sell for if it were for sale. Median gross rent of dwelling in dollars is the contract rent value plus the estimated average monthly cost of utilities. Percentage homeownership is the percentage of owner-occupied housing units regardless of mortgage status.

To standardize the contribution of each census variable included in the index, a z score was created for each of the nine census tract variables by subtracting the mean from the grand mean for the DMA and dividing by the standard deviation of the respective variables for the DMA as a whole. To ensure that each variable contributed appropriately when calculating this index, the z scores for two depreciating variables, percentage unemployment and percentage of the population below poverty, were multiplied by -1 before they were added to the remainder variables (Darden et al. 2010). The formula for the index is as follows:

$$CSI_i = \sum_{j=1}^k \frac{V_{ij} - V_{j\text{DMA}}}{S(V_{j\text{DMA}})},$$

where CSI_i is the composite socioeconomic z -score index for census tract i , is the sum of z scores for the SES variables j , relative to the DMA's SES; DMA is the three counties of Wayne, Oakland, and Macomb; k is the number of variables in the index; V_{ij} is the j th SEP variable (z score) for a given census tract i ; $V_{j\text{DMA}}$ is the mean of the j th variable in the three-county DMA; and $S(V_{j\text{DMA}})$ is the standard deviation of the j th variable in the three-county DMA.

For portions of this study, we used CSI scores directly. In other analyses, we divided the DMA into five levels (i.e. ranges) of SEP with boundaries at the

20th, 40th, 60th, and 80th percentiles (quintiles) of CSI frequency distribution. This categorization allowed a division of DMA census tracts of residence into five approximately equal proportions of the population in each group of SES: very high SEP, high SEP, middle SEP, low SEP, and very low SEP.

Using the CSIs (summed *z* scores of each of the nine aforementioned variables) and/or their subsequent SEP quintiles required removal of 116 census tracts from the population above, providing a total of 1,046 (originally 1,162) census tracts. Census tracts were excluded from the index if the Census Bureau's five-year estimates yielded fewer than 100 people, housed only juvenile institutions, or had uncharacteristic Arab or Hispanic ethnic populations (consisting of more than 10 percent of the census tract population). Large Arab and Hispanic populations were excluded from analyses because in this study we mainly compared non-Hispanic black and non-Hispanic white populations, ensuring that adequate numbers of these two groups were available for analyses. This left an individual sample size of 216,101 children and their BLLs, as described earlier. The black-versus-white race aims of this study removed all children of a race/ethnicity other than non-Hispanic white or non-Hispanic black, leaving a total individual sample size of 165,859 children, their census tracts, and BLLs.

Analysis

Using ArcGIS 10.1 (Environmental Systems Research Institute 2011), census tract geometric mean BLLs of all race/ethnicity children were mapped per SEP neighborhood from 2006 through 2010. These BLL patterns were related to the growth or decline of the city and the inner and outer suburbs of the DMA. Other geographic factors were also examined as possible contributors to unexplained variance between BLLs and neighborhood SEP. We hypothesized that the highest mean BLLs would be found in very low SEP neighborhoods located primarily in the central city of Detroit, followed by lower mean BLLs in middle SEP neighborhoods located in the inner suburbs, and that the lowest mean BLLs would be located in very high SEP neighborhoods of the outer suburbs.

Multiple regression analyses at the individual level was used to test the BLL gap in black versus white children (0 = white, 1 = black), controlling for individual age (months), gender (0 = male, 1 = female), Medicaid status (0 = non-Medicaid, 1 = Medicaid), and neighborhood housing age

(median year structure built). This last variable is available only for both vacant and occupied housing.

$$\begin{aligned} \text{Individual ln (BLL)} = & a + b_1 \text{ AGE} + b_2 \text{ GENDER} \\ & + b_3 \text{ MEDICAID} \\ & + b_4 \text{ HOUSING} \\ & + b_5 \text{ RACE.} \end{aligned}$$

A cross-level comparison included CSI as a control variable:

$$\begin{aligned} \text{Individual ln (BLL)} = & a + b_1 \text{ AGE} + b_2 \text{ GENDER} \\ & + b_3 \text{ MEDICAID} \\ & + b_4 \text{ HOUSING} \\ & + b_5 \text{ RACE} + b_6 \text{ CSI.} \end{aligned}$$

Difference of geometric mean BLLs in black and white children were calculated by neighborhood SEP and stratified over age groups as a control measure. Neighborhood-effects research has established a direct link between gradients of income and gradients of health (Kawachi 2000). As such, there should be no significant difference in black-white BLLs when stratifying by neighborhood CSI or SEP. Additionally, as neighborhood socioeconomic conditions improve, there should be a decline in BLLs regardless of race. Accordingly, we hypothesized that at all levels of CSI or SEP (controlling for economic position), black and white children would have the same average BLLs, and mean BLLs would be significantly higher in white children living in the lowest CSI and SEP neighborhoods compared with black children living in the highest CSI and SEP neighborhoods across all age groups. To investigate the gap in BLLs of black and white children, we plotted geometric mean BLLs separately against the CSI (across all census tracts within the DMA). Bivariate regression analyses were used to quantitatively examine black BLL residual patterns as a way to further investigate this gap.

RESULTS

Table 1 defines the incorporated cities (U.S. Census Bureau 2013; verified by "Incorporated Cities" list from the Michigan Office of the Great Seal, Secretary of State) fully within the three counties of the DMA and provides their percentage changes in population from 2000 to 2010 (U.S. Census Bureau 2000, 2010, 2013). Only incorporated cities were

Table 1. Growth and Decline of Detroit and Inner and Outer Suburbs from 2000 to 2010.

DMA Place	Total Population, 2000	Total Population, 2006–2010, 5-year Estimate	Percentage Change
City of Detroit	951,270	713,777	-25.00
Inner suburbs			
Allen Park City	29,376	28,542	-2.84
Berkley City	15,531	15,063	-3.01
Center Line City	8,531	8,374	-1.84
Dearborn City	97,775	98,392	0.63
Dearborn Heights City	58,264	58,066	-0.34
Eastpointe City	34,077	32,944	-3.32
Ecorse City	11,229	9,845	-12.33
Farmington City	10,423	10,380	-0.41
Farmington Hills City	82,111	80,191	-2.34
Ferndale City	22,105	20,286	-8.23
Flat Rock City	8,488	9,666	13.88
Fraser City	15,297	14,739	-3.65
Garden City	30,047	28,199	-6.15
Gibraltar City	4,264	4,601	7.90
Grosse Pointe City	5,670	5,478	-3.39
Grosse Pointe Farms City	9,764	9,561	-2.08
Grosse Pointe Park City	12,443	11,755	-5.53
Grosse Pointe Woods City	17,080	16,357	-4.23
Hamtramck City	22,976	22,594	-1.66
Harper Woods City	14,254	14,296	0.29
Hazel Park City	18,963	16,876	-11.01
Highland Park City	16,746	12,714	-24.08
Huntington Woods City	6,151	6,209	0.94
Inkster City	30,115	26,311	-12.63
Lathrup Village City	4,236	4,101	-3.19
Lincoln Park City	40,008	38,595	-3.53
Livonia City	100,545	97,915	-2.62
Madison Heights City	31,101	29,954	-3.69
Melvindale City	10,735	10,759	0.22
Oak Park City	29,793	29,892	0.33
Pleasant Ridge City	2,594	2,556	-1.46
River Rouge City	9,917	8,299	-16.32
Riverview City	13,272	12,640	-4.76
Rockwood City	3,442	3,323	-3.46
Roseville City	48,129	47,830	-0.62
Royal Oak City	60,062	57,741	-3.86
St. Clair Shores City	63,096	60,776	-3.68
Southfield City	78,296	72,949	-6.83
Southgate City	30,136	30,160	0.08
Taylor City	65,868	63,833	-3.09
Trenton City	19,584	19,051	-2.72
Village of Grosse Pointe Shores City	2,823	2,976	5.42
Warren City	138,247	135,791	-1.78

(continued)

Table 1. (continued)

DMA Place	Total Population, 2000	Total Population, 2006–2010, 5-year Estimate	Percentage Change
Westland City	86,602	84,832	-2.04
Woodhaven City	12,530	12,862	2.65
Wyandotte City	28,006	26,368	-5.85
Total	1,585,173	1,544,329	-2.58
Outer suburbs			
Auburn Hills City	19,837	21,162	6.68
Belleville City	3,997	4,000	0.08
Birmingham City	19,291	19,962	3.48
Bloomfield Hills City	3,940	3,879	-1.55
Clawson City	12,732	11,995	-5.79
Keego Harbor City	2,769	2,929	5.78
Lake Angelus City	326	294	-9.82
Memphis City	1,129	1,132	0.27
Mount Clemens City	17,312	16,616	-4.02
Northville City	6,459	6,063	-6.13
Novi City	47,386	53,823	13.58
Orchard Lake Village City	2,215	2,127	-3.97
Plymouth City	9,022	9,136	1.26
Pontiac City	66,337	60,982	-8.07
Richmond City	4,897	5,630	14.97
Rochester City	10,467	12,312	17.63
Rochester Hills City	68,825	70,606	2.59
Romulus City	22,979	23,874	3.89
South Lyon City	10,036	11,072	10.32
Sterling Heights City	124,471	129,687	4.19
Sylvan Lake City	1,735	1,803	3.92
Troy City	80,959	80,987	0.03
Utica City	4,577	4,757	3.93
Walled Lake City	6,713	6,941	3.40
Wayne City	19,051	17,924	-5.92
Wixom City	13,263	13,456	1.46
Total	580,725	593,149	2.14

Sources: U.S. Census Bureau (2000, 2010).

Note: DMA = Detroit metropolitan area.

included in this summary, because unincorporated places hold little control over the construction of new housing. Growing cities should be associated with new housing construction and a subsequent reduced risk for exposure to lead-based paint for the children living there. Conversely, children living in cities experiencing population decline, legacy sources of lead, and little new construction and renovation are more likely to have greater exposure to lead soils and dust.

Figure 1 displays these incorporated cities and their designated inner or outer suburban status, including their percentage changes in population. All bordering suburbs each had at least 50 percent of their housing stock built before 1969. Nonbordering inner suburbs less than five miles from the city also had greater than 50 percent of their housing stock built before 1969, with the exception of three places: Flat Rock City, Woodhaven City, and Farmington Hills City (U.S.

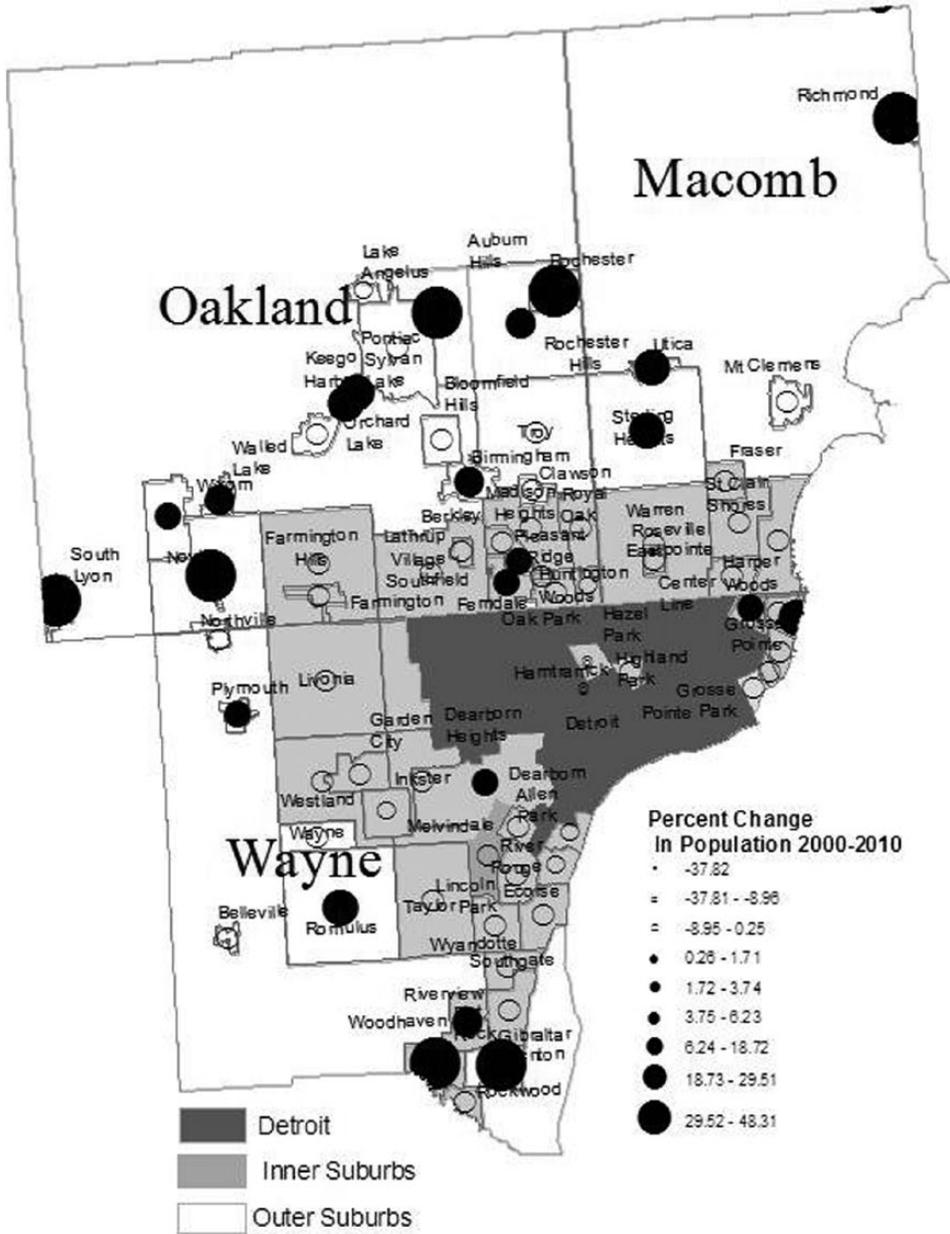


Figure 1. Growth and Decline of Detroit and Inner and Outer Suburbs. Sources: U.S. Census Bureau (2000, 2010).

Census Bureau 2010). Table 1 and Figure 1 indicate that the City of Detroit had a 25 percent decrease in population from 2000 to 2010. The inner suburbs experienced a total population decline of 2.58 percent, and the outer suburbs experienced a total population growth of 2.14 percent.

Maps (Figures 2–6) of geometric mean BLLs for all racial/ethnic groups are plotted by census tract at each level of SEP (Figures 3–5 are available online). The highest mean BLLs were found in very low SEP neighborhoods located primarily in the central city of Detroit, followed by lower mean BLLs in middle SEP neighborhoods, located in the

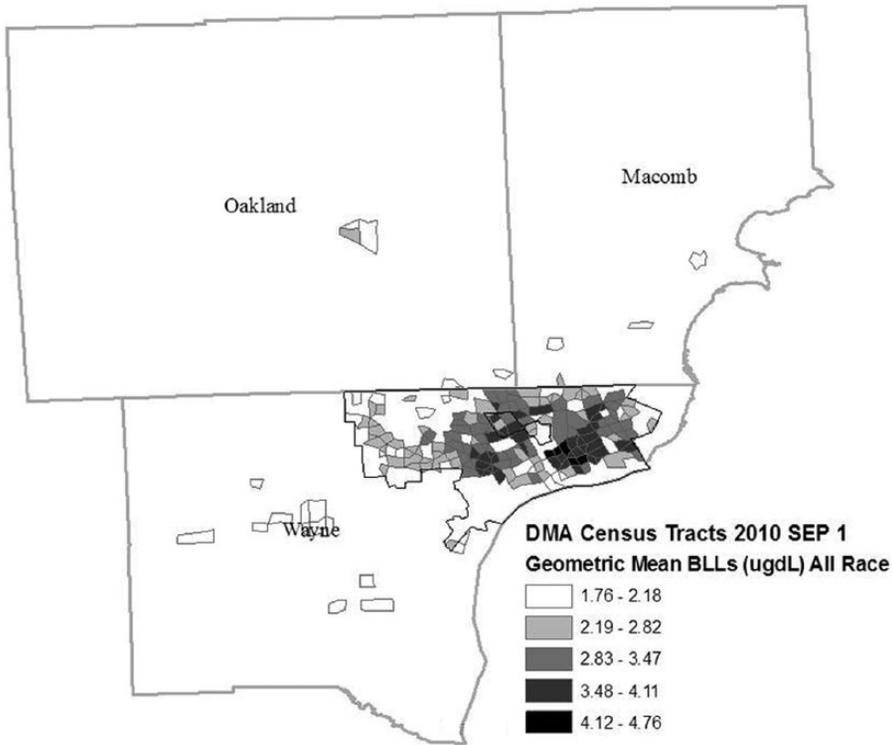


Figure 2. Geometric Mean Blood Lead Levels (BLLs), All Races, in Census Tracts of Socioeconomic Position (SEP) 1 of the Detroit Metropolitan Area (DMA).
Source: Michigan Department of Community Health, 2006 to 2010.

first suburbs (inner suburbs), and the lowest mean BLLs were located in very high SEP neighborhoods of the outer suburbs. Maps of SEP 3 (Figure 4) and SEP 5 (Figure 6) neighborhoods contained only the two lowest quintile categories of BLL, as depicted in the legends.

Controlling for individual age, gender, Medicaid status, and neighborhood housing age, the multiple regression race slope coefficient estimate of .210 ($p = .000$, $SE = .003$, tolerance = .673) suggests that individual BLLs are positively associated with black race ($p < .05$) in neighborhoods across the DMA. A cross-level comparison adding neighborhood CSI as a control variable yielded a significant multiple regression (standardized) race slope coefficient estimate of .142 ($p = .000$, $SE = .003$, tolerance = .577). Controlling for neighborhood socioeconomics had a neutralizing effect. Furthermore, regression coefficients of CSI and median housing age showed that CSI was more predictive, with a standardized regression slope coefficient of $-.015$ ($p = .000$, $SE = .000$, tolerance = .509), than housing age, at $-.006$ ($p = .000$, $SE = .000$, tolerance = .601). This is in

apparent support of some previous research finding that neighborhood poverty or socioeconomic variables are more predictive than housing age when regressed against BLLs (Krieger et al. 2003; Oyana and Margai 2010; Vivier et al. 2010). Both models' independent variables accounted for a significant part of the overall variation in lead exposure ($R^2 = .15$ and .17), but upon examination of the residuals, the influence of other variables is suggested at this scale of geographic analysis, making the use of hierarchical linear models and other hierarchical models inappropriate. Other studies attempting to predict BLLs using multiple variables while controlling for age and other characteristics have examined residuals and speak to the complicated exposure nature of lead (Bailey, Sargent, and Blake 1998; Haley and Talbot 2004; Sargent et al. 1997; Vivier et al. 2010; Zahran et al. 2013). Other research models are accompanied with issues of colinearity often dealt with by eliminating controls and degrading individual BLL data into dichotomous groups.

Differences in geometric mean BLLs for black and white children at each level of SEP and age

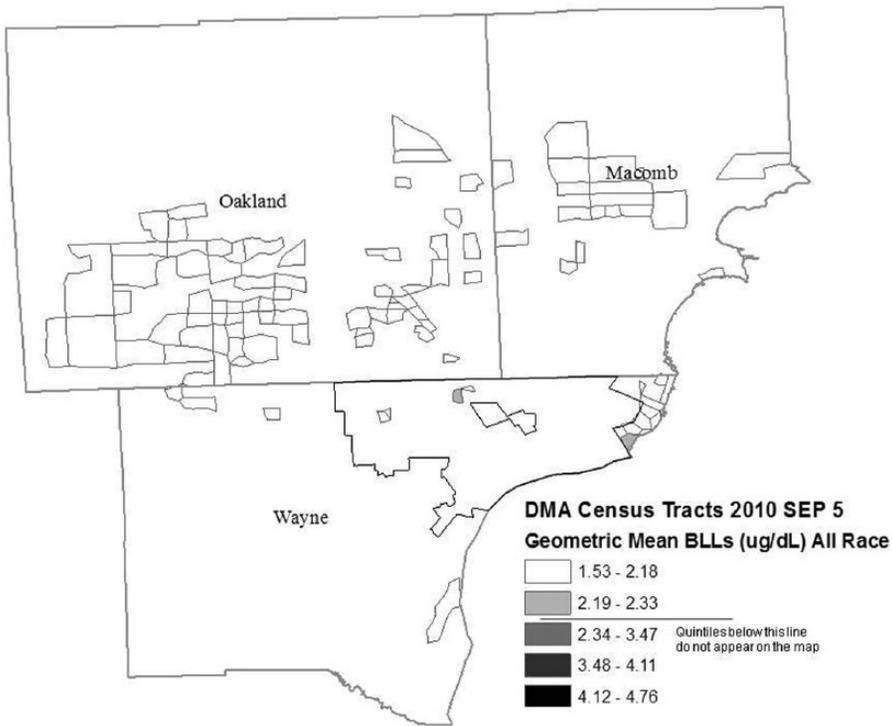


Figure 6. Geometric Mean Blood Lead Levels (BLLs), All Races, in Census Tracts of Socioeconomic Position (SEP) 5 of the Detroit Metropolitan Area DMA.
 Source: Michigan Department of Community Health, 2006 to 2010.

were calculated. Numbers of both black and white children in each level and age were sufficient, even at the very high and low ends of the spectrum (Table 2).

At all levels, black children had higher average BLLs than white children. Black children were a majority of the population in very low and low SEP neighborhoods, and white children were a majority of the population in the middle, high, and very high SEP neighborhoods. Importantly, children of black race had higher predicted geometric mean BLLs across all neighborhood SEP levels. The higher BLLs presented in children were related to the neighborhoods in which they lived. If black children lived in higher SEP neighborhoods, their collective BLLs were lower. Regardless of the SEP quintile used, black and white children BLL tested were segregated (i.e., unevenly distributed) across neighborhood SEP levels in the DMA.

For children in age groups zero to two years and greater than two to six years, the black-white differences in mean BLL were significantly different in low, medium, high, and very high SEP neighborhoods. Neighborhoods of very low SEP showed

that mean BLLs were not significantly different from each other. For children greater than six years old, the black-white differences in mean BLLs were significantly different in low, medium, and high SEP neighborhoods compared with neighborhoods of very low and very high SEP; these differences in mean BLL were not significantly different. To summarize, black and white children in all age groups living in the very low SEP neighborhoods had the same mean BLLs ($t = 0.91, p = .36$; $t = 0.85, p = .39$; and $t = 0.83, p = .41$). Black and white children greater than six years old living in very high SEP neighborhoods also had the same mean BLLs ($t = 1.92, p = .06$). Black children living in all other levels of SEP (low, medium, and high), regardless of age, had significantly greater mean BLLs than white children (no p value was greater than .036). The mean differences in black-white BLLs for children aged zero to two and greater than two to six years increased as SEP levels improved. Black-white BLL disparity in children greater than six years old increased from SEP 1 to SEP 2 but then decreased from there. Younger children are more likely to inhale and

Table 2. Differences between Mean BLLs for Black and White Children by SEP Quintile Stratified by Age Group, Detroit Metropolitan Area, 2006 to 2010.

Group	Census		BLL GM	SD	t	Two-tailed	
	Tract Count	Children n				t	df
SEP 1, ages 0–2 years							
Black	208	22,610	2.90	0.69	0.91	206.76	.362
White	168	1,801	2.76	1.79			
SEP 2, ages 0–2 years							
Black	207	11,219	2.13	0.40	2.40	312.86	.017*
White	204	8,182	1.99	0.73			
SEP 3, ages 0–2 years							
Black	200	3,469	2.00	0.64	3.23	294.92	.001*
White	208	10,782	1.83	0.33			
SEP 4, ages 0–2 years							
Black	189	1,977	1.94	0.48	4.49	234.24	.000*
White	208	9,240	1.77	0.20			
SEP 5, ages 0–2 years							
Black	170	1,011	1.98	0.75	4.10	180.70	.000*
White	209	6,587	1.74	0.16			
SEP 1, ages >2–6 years							
Black	209	25,589	3.23	0.86	0.85	198.78	.394
White	163	1,687	3.07	2.25			
SEP 2, ages >2–6 years							
Black	208	11,756	2.20	0.69	2.62	398.99	.009*
White	193	6,142	2.02	0.64			
SEP 3, ages >2–6 years							
Black	196	3,242	2.10	1.30	3.42	200.10	.001*
White	206	7,398	1.79	0.15			
SEP 4, ages >2–6 years							
Black	175	1,720	2.03	0.81	3.74	211.62	.000*
White	205	5,894	1.79	0.29			
SEP 5, ages >2–6 years							
Black	169	956	2.07	0.68	5.61	198.64	.000*
White	210	3,923	1.76	0.23			
SEP 1, age > 6 years							
Black	209	8,664	2.42	0.47	0.83	122.06	.41
White	113	463	2.55	1.64			
SEP 2, age > 6 years							
Black	189	3,422	1.93	0.43	4.91	332.86	.000*
White	167	1,740	1.74	0.29			
SEP 3, age > 6 years							
Black	154	959	1.85	0.62	3.02	189.30	.003*
White	203	1,875	1.69	0.25			
SEP 4, age > 6 years							
Black	117	470	1.93	0.93	2.12	145.41	.036*
White	203	1,576	1.73	0.43			
SEP 5, age > 6 years							
Black	931	280	1.85	0.82	1.92	107.28	.057
White	96	1,225	1.68	0.34			

Sources: Computed by authors from data obtained from the U.S. Census Bureau (2010) and the Michigan Department of Community Health, 2006 to 2010.

Note: N = 165,859 children, count = 1,046 census tracts. BLL = blood lead level; GM = geometric mean; SEP = socioeconomic position.

*p < .05.

ingest lead because they play on floors and exhibit hand-to-mouth behavior, breathing and eating more lead-laden dust than adults or older children (U.S. Agency for Toxic Substances and Disease Registry 2007). Perhaps the oldest age group is showing a reduced gap in black-white mean BLLs because the older children are no longer exhibiting these behaviors, reducing their exposure to lead-laden dust and soils.

In an attempt to investigate the seemingly widening BLL disparity with increasing SEP, Figure 7 displays geometric mean BLLs of black and white children plotted separately against the CSI for each census tract of residence in the DMA. Regression slope coefficient ($p < .05$) estimates for bivariate regressions of these two lines were $-.035$ ($p = .000$, $SE = .002$) for black children and $-.036$ ($p = .000$, $SE = .002$) for white children, indicating that CSI explains a small but significant and similar variation between black children's and white children's geometric mean BLLs in neighborhoods across the DMA ($R^2 = .222$ and $.196$). Mean BLLs for black children were at least approximately 0.2 units higher than mean BLLs for white children across all CSI levels, not surprising considering the difference-of-means tests conducted previously. The slightly different slope values appear to magnify as they approach greater levels of SEP (greater CSI). However, a difference-of-means test was applied to the two intercepts and the two slope coefficients; the results indicate that the two equations are not significantly different from each other (Runyon and Haber 1980):

$$\text{Geometric mean BLL, black race by census tract} \\ = 2.11(0.015) + -0.035 \times \text{CSI}(0.002),$$

$$\text{Geometric mean BLL, white race by census tract} \\ = 1.95(0.015) + -0.036 \times \text{CSI}(0.002).$$

The bivariate regression analysis did, on the other hand, allow us to investigate residuals that influenced the greater y -intercept slope value as black children approached higher levels of SEP (Figure 7).

Partitioning children by SEP and age provided a clearer picture of the increasing disparity of BLLs with increasing SEP, which seemingly disappeared when the children were grouped together in the bivariate regressions.

The most significant exposure to childhood lead is from lead-based paint in and on older homes, which are typically located in inner areas of central cities that are also more racially segregated, poor,

and minority (Centers for Disease Control and Prevention 2005). Given the results, housing age, measured in the multiple regression model and captured in part by measures embedded within the Darden-Kamel Socioeconomic index, was considered in relation to SEP. During this study, the inner suburbs and especially the city of Detroit experienced declining populations while also containing older housing stock, much of which was unlikely to have experienced significant renovation. Consequently, childhood exposure to lead may have become a greater risk geographically in these areas. Housing stock was younger in the suburbs, but older, non-renovated housing of the high and very high SEP neighborhoods is still an exposure risk and a possible explanation for the greatest black BLL residual of SEP 4. Then again, cities with histories of industrial processes and leaded roadways could contribute to airborne lead in these neighborhoods, a possible explanation for the black BLL residual of SEP 5. In fact, Zahran et al. (2013) found in the City of Detroit that resuspended soil contaminated with lead was a significant contributor of atmospheric lead and children's BLLs from 2001 to 2009. The upward mobility of black families into high and very high SEP neighborhoods may not have completely translated into the newest or renovated lead-free homes away from outdoor sources, explaining higher BLLs of younger black children.

DISCUSSION

We documented, using the modified Darden-Kamel Composite Socioeconomic Index, that the racial concentration of black children in very low SEP neighborhoods has contributed to the gap in childhood black-white mean BLLs in metropolitan Detroit. We further demonstrated the importance of differences in neighborhood characteristics on the BLLs of children. Findings reveal that when non-Hispanic white and non-Hispanic black children live in neighborhoods of similar socioeconomic characteristics, the gap in BLLs between the two groups decreases overall. Thus neighborhood characteristics are factors in the racial disparity of BLLs in children. However, after stratifying these children by age and SEP and using difference-of-means tests on BLLs, this gap was nonexistent in the lowest SEP neighborhoods and, importantly, became exacerbated in the high and very high SEP neighborhoods. This is an important finding, contributing to the latest health disparity and childhood lead poisoning research. This finding also supports Darden's (2009) study,

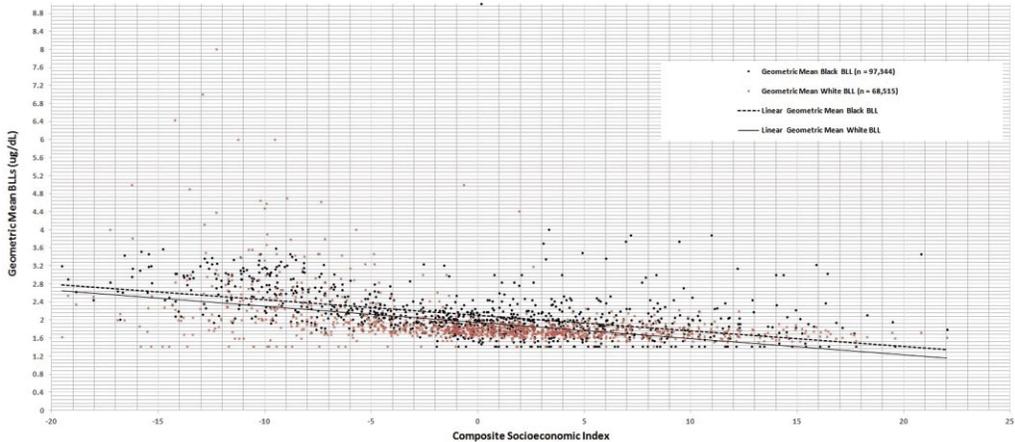


Figure 7. CSI and Geometric Mean Blood Lead Levels (BLLs), 2006 to 2010.

Sources: U.S. Census Bureau (2010) and Michigan Department of Community Health, 2006 to 2010.

which concluded that even when blacks achieve higher incomes, their status does not automatically translate into higher quality housing and neighborhood characteristics.

Areas for Further Study

More research is needed to explain the gap in BLLs in high SEP neighborhoods. Census or MDCH housing age data would be ideal at the block group or individual level, but neither are available at this time to draw any definitive conclusions. When children are no longer playing on floors, the BLLs of black and white children greater than six years old become more similar, an indication of depositional exposure to lead paint dust.

Review of other lead studies finds an attempt to include housing age as a predictive variable to BLLs, as did this study, and consistently, poverty-type variables were stronger predictors. Housing age may not be a detectable independent variable at the census tract level. Greater mean black BLLs related to exposure to lead-based paint in and on older housing would not be detected unless an individual-level analysis were used, such as examination of our residuals. A variety of other possible outside environmental contributors provide opportunity for future research, such as lead-laden dust of historic freeways, existing coal-burning plants, steel production, and so on. Examining these sources may also require individual-level analysis, because lead is a relatively heavy inorganic compound that accumulates at the surface in close geographic proximity to emitting sources.

The Unique Contribution of This Research

Using a CSI, this study found that the level of neighborhood SEP in which children lived was an important factor in predicting mean BLLs; the highest mean BLLs existed in the central city, with a declining population and disproportionately older housing stock. This scenario was true of the inner suburbs but to a lesser extent. The outer suburbs housed the lowest mean BLLs, with a growing population and newer housing.

Segregation of black and white children across the DMA means that being a black child predicted higher mean BLLs across all neighborhood SEP levels, contributing to the neighborhood effects literature that establishes a link between neighborhood inequality and declining health outcomes.

Other studies have also found that race is a factor in lead poisoning. Bernard et al. (2003) and Haley and Talbot (2004) both found increases in BLLs with increases in non-Hispanic black children. This study makes a unique contribution to our understanding of the racial disparity in BLLs, however, by revealing that children's BLL differences increased with increasing SEP. This indicates that although black children reside in neighborhoods with similar SEP as white children, they may still be relegated into unequal housing characteristics or locations and thus exposed to higher levels of lead at a lower geographic scale than the neighborhood or census tract. In this study, being black was a predictor of greater mean BLLs compared with white children, but being black and living in the same

high SEP neighborhoods as whites exacerbated the gap in mean BLLs.

Policy Implications of the Research

If racial disparities in health are to be effectively addressed, health policy and housing policy should be more closely aligned. For example, national policy ought to reinstate and/or increase funding to the Department of Housing and Urban Development lead hazard grants, allocate increased and consistent funding for healthy homes, and provide the Environmental Protection Agency with the necessary regulatory and enforcement power to ensure that children are protected from outdoor emissions of lead.

As of this writing, the MDCH seeks approval from its institutional review board to provide individual-level housing age values to researchers and government agencies to foster research designed to develop or contribute to generalizable knowledge of children's BLL exposure risk. Future research should be translated into national health policies, mandating the abatement of lead-contaminated housing before children become poisoned, especially in disparate low-income and segregated communities. Finally, a coordinated fair housing and environmental protection public policy is necessary to address this preventable public health crises.

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