INVESTIGATION OF RACIAL AND SOCIOECONOMIC DISPARITIES IN ASTHMA HOSPITALIZATIONS IN METROPOLITAN DETROIT, MICHIGAN

By

Lonnie Barnes

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

Geography—Master of Science

2018

ABSTRACT

INVESTIGATION OF RACIAL AND SOCIOECONOMIC DISPARITIES IN ASTHMA HOSPITALIZATIONS IN METROPOLITAN DETROIT, MICHIGAN

By

Lonnie Barnes

Racial health disparities are a consistent problem in the United States. Compared to whites, African Americans experience worse health outcomes both in terms of morbidity and mortality from various forms of chronic and infectious disease and continue to have lower life expectancy at birth. The causes of these disparities are not always immediately apparent, but previous literature on the topic indicates that they are rooted in larger structures of inequality which render disadvantaged populations more exposed and susceptible to disease. This study investigates the racial and socioeconomic dimensions of morbidity due to asthma, an increasingly-common chronic condition of poorly-understood etiology in metropolitan Detroit, an urban area marked by high levels of racial segregation and economic inequality. Data on asthma hospitalizations from the Healthcare Cost and Utilization Project were used, along with socioeconomic indicators from the U.S. Census Bureau, to (a) ascertain the magnitude of racial disparities in asthma hospitalization, (b) identify risk factors for hospitalization, and (c) determine the extent to which these risk factors explain any racial disparities. Descriptive statistics indicate that even after controlling for ZIP code socioeconomic position, black hospitalization rates for asthma were considerably higher than white rates at all levels of the socioeconomic hierarchy. Results of logistic regression models indicate that factors such as type of insurance and having other conditions in addition to asthma affect the risk of hospitalization for the condition but are able to account for only a small portion of the increased odds of hospitalization in the study area's

African American population. The findings highlighted in this study are important, but also demonstrate the necessity for further research on this topic.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1: INTRODUCTION

Racial and socioeconomic disparities in health outcomes are a striking and persistent problem in the United States. To give just a few examples, life expectancy at birth, a measure of all-cause mortality across age groups, has increased nationally since 1929 when all states implemented a death-registration system. However, whites of both sexes reached a life expectancy of 70 years in 1954, which was 42 years earlier than that of blacks (herein referred to as black or African American) who reached the same life expectancy of 70 years in 1996. After 1954, the black-white gap started closing from a difference in life expectancy of 7.4 years to 6.6 years in 1996. By 2013, the black-white gap in life expectancy had declined to 3.5 years. The largest contributor to these disparities in life expectancy is infant mortality –i.e., infant deaths less than 1 year of age, with the probability (qx) of African American babies =0.01122 and white babies dying before their first birthday =0.005063 (equating to 1,122 African American babies vs. 506 white babies and in a population of 100,000) (National Vital Statistics Reports, 2017). These trends in black-white disparities are also observed for other cause-specific death rates (National Vital Statistics System, 2015).

Similar gradients exist in measures of infectious and chronic disease morbidity. For example, the prevalence of HIV in the United States in 2015 was 14.4 per 100,000 population (range, 13.5 to 15.2) with the prevalence rate for African Americans 49.5 (45.0 to 54.0) and whites 6.0 (5.4 to 6.6) rate ratio (RR) = 8.3 (Centers for Disease Control and Prevention (CDC), 2017). The largest black-white disparities in HIV prevalence was among females, RR = 13.5 compared to males, RR = 7.6 (CDC, 2017). Although the HIV prevalence rate decreased substantially for African Americans from 2010 to 2015—in 2010, RR = 9.2 (females, RR = 19.4 and males, RR = 9.2)—the black-white disparities persist. In terms of chronic diseases in 2014, blacks had a higher rate of emergency room visits (94.5 per 100 population) compared to whites (40.3) (CDC, 2017). Similar black-white differences in findings were observed for hypertension (African Americans, 33.9 per 100 (standard error=0.88) and whites, 26.6 (0.38), diabetes (1.34 per 100 vs. 7.6) and age-adjusted prevalence rates for selected types of cancer, in particular prostate cancer (3.0 per 100 vs. 2.3) (CDC, 2017). While these disparities are obvious and consistent, the reasons for them are complex and remain unclear.

It was argued in the past that the disparities in health are due to genetic differences between racial groups, but this theory has largely been rejected because race in the United States is not a biological construct—rather, race is a social construct, as evinced by the fact that racial classifications have changed over time and from place to place (U.S. Bureau of the Census, 2017). A second possible explanation is that racial health disparities are actually the result of racial groups being unevenly distributed across the socioeconomic spectrum. However, even when socioeconomic (SES) differences are controlled for in statistical models, racial disparities are generally still apparent (Grady & Darden, 2012). Allostatic load, a measure of the physiologic burden imposed on a person by stress, has also been shown to be higher in African Americans than in whites across all ages and may in part be explained by low SES (Geronimus, Hicken, Keene, & Bound, 2006); however, above and beyond SES there remain racial differences in health. There is a need to continue to disentangle the causes underlying these high rates of morbidity and premature mortality among African Americans to reduce the black white health disparities in the United States.

Purpose of Study

The purpose of this study is to investigate racial differences in asthma hospitalization, a common chronic condition with complex etiology. Asthma will be studied in the Detroit

Metropolitan Area, which is highly segregated, by race and SES—for this study the modified Darden-Kamel Socioeconomic Position (SEP) Index (Darden, Rahbar, Jezierski, Li, & Velie, 2010) will be used to define the neighborhood context in which children and adults with asthma reside. Another variable that will reflect the potential for ambient pollution that may exacerbate asthma leading to hospitalization will be proximity to an industrial facility emitting one or more criteria air pollutants. Furthermore, as an indirect measure of allostatic load, this study will assess the number of underlying conditions for each person hospitalized for asthma as a potential mediator of the area-level and asthma hospitalization relationship, controlling for differences in individual-level characteristics. A conceptual pathway by which these risks contributes to asthma hospitalizations among African Americans and whites will guide the statistical models to further explain factors underlying racial disparities in asthma hospitalization.

This thesis will begin with a literature review on asthma and individual and area-level risk factors for asthma followed by the gaps in the literature and the goal and aims of this study. Chapter 3 will consist of a description of the study area. Chapters 4 and 5 will present the Data and Methods for the study. The Results and Discussion of results will be presented in Chapters 6 and 7 followed by the Conclusion and Recommendations for future asthma research in Chapter 8.

CHAPTER 2: LITERATURE REVIEW

Asthma

Asthma is a chronic respiratory disorder characterized by periods of wheezing, breathlessness and chest tightness caused by inflammation of the airways (Akinbami L. J., et al., 2012). The International Classification of Disease, 9th edition (ICD-9), code for asthma is 493.xx. ICD-9 distinguishes different phenotypes of the condition: for example, 493.00 is the code for extrinsic asthma, while 493.10 is the code for intrinsic asthma. ICD-10, a more recent edition, lists asthma as J45.xx, and the subcategories are classified by severity: J45.2 is mild intermittent asthma, and J45.5 is severe persistent asthma. Both ICD-9 and ICD-10 codes are given here because the dataset used in this study contains each of the two as the patient's hospital discharge diagnosis.

There is no known cure for asthma, though both long- and short-term treatments are available which reduce the frequency and severity of asthma attacks respectively and make the condition fatal in only a small proportion of cases (NHIS, 2015). As such, primary concerns with asthma are the degree to which it limits quality of life, as well as the costs of medical care. The mean annual cost of medical treatment for adults with asthma in the U.S. was estimated to be almost $2700 per patient in 2003 (Cisternas, Blanc, Yen, Katz, & Earnest, 2003). Nonmedical costs, such as household allergy control measures and caretaking were, on average, $500 per year.

Asthma Epidemiology

The 2015 National Health Interview Survey (NHIS) estimated that the prevalence of asthma in the U.S. population was 7.8 per 100 population, meaning an estimated 24.6 million people suffered from the condition (NHIS, 2015). Its prevalence increased considerably in the final decades of the 20th century. Between 2001 and 2010, prevalence continued to increase, though at a decreased incidence (Akinbami, Simon, & Rossen, 2015). This considerable increase in the United States is consistent with a much larger, global trend—the prevalence of the condition is increasing on an international scale, with the highest rates present in developed, Anglophone countries, and rates increasing with development in developing nations (Beasley, Crane, FRACP, Lai, & Peirce, 2000).

In addition, there are notable disparities in asthma prevalence along sex, age, racial, and SES lines (NHIS, 2015). In children, the frequency of the condition more than doubled from 3.6% in 1980 to 7.5% in 1995 (Akinbami L. J., Moorman, Garbe, & Sondik, 2009) and is now among the most common chronic diseases in children (2015) prevalence rate 8.4 per 100. In children, males have a higher prevalence than females (9.9 per 100 vs. 6.9), though the opposite is true in adults—women have a prevalence rate of 9.7, while that of men is 5.4 (NHIS, 2015). This trend, whereby asthma is more common in boys than in girls, but also more common in women than in men, appears to be consistent over time, both in the U.S. and elsewhere (Subbarao, Mandhane, & Sears, 2009).

The increased prevalence of asthma since the end of the last century has coincided with the emergence of substantial disparities by race: in 1980, there was almost no gap in rates between white and African American children, but by 2010 asthma had become twice as common in African American children (Akinbami, Simon, & Rossen, 2015). Among African American and Puerto Rican children, the prevalence was 13.4% and 13.7% respectively in 2015, while this figure stood at 7.8% for white children, according to the 2015 NHIS. Disparities exist not only in prevalence, but also in the severity of the condition. For example, African American asthma patients were more likely than white patients to have severe asthma and, among those with severe asthma, were more likely to use more long-term medications (Haselkorn, Lee, Mink,

& Weiss, 2008). In addition, African American children with asthma are 53% more likely than white children to experience activity limitation due to their condition (Akinbami, LaFleur, & Schoendorf, 2002). And while African American and white patients with asthma have similar physician and outpatient visit rates, the former were also three times as likely to make an emergency department visit or to be hospitalized for their condition (Akinbami, LaFleur, & Schoendorf, 2002). Data from the 2010 National Hospital Discharge Survey indicated that African Americans were hospitalized for asthma at more than three times the rate of whites (NDHS 2010). The overall case-fatality rate for asthma is low, but it is nearly twice as high in the African American population compared to whites: 23 per 100,000 black population compared to 13 per 100,000 white population (Akinbami L. J., et al., 2012). These disparities in severity of morbidity and premature mortality have also been present longer than the disparities in prevalence (Akinbami, Simon, & Rossen, 2015).

Economic Costs

Barnett and Nurmagambetov (2011) estimate that in 2006, the total, society-wide cost of medical treatment for all people with asthma in the United States was $14.8 billion. In addition to these direct costs, people living with asthma, or their caretakers, may also be forced to miss days of work or school because of their condition, which not only results in a loss of productivity, but can also affect long- and short-term financial security. Between 2002 and 2007, workers with asthma missed almost 3 more days of work per year than those without asthma, resulting in a total annual wage loss of $1.6 billion. During the same period, school children missed an average of nearly an extra day of school per year, with a total cost of $370 million annually (Barnett & Nurmagambetov, 2011). These costs also vary greatly with the severity of the condition. In the most extreme cases, asthma can lead to death: there were 3,600 fatalities in the U.S. in 2015—a cause-specific, age-adjusted death rate of 10.3 per million (NHIS, 2015). Barnett and Nurmagambetov (2011) estimated that the cost of productivity lost due to premature death from asthma during the six-year study period was an average of $2.3 billion each year. In a different study, large variations were found in direct and indirect costs of asthma by degree of severity: the cost of medical services for people with severe asthma (on average more than $3000 annually) was more than ten times higher than for people with mild asthma, and severe asthma caused people to miss an average of nearly 9 days of work per year, while people with mild asthma generally did not miss any (Godard, Chanez, Siraudin, Nicoloyannis, & Duru, 2002). The higher costs associated with severe asthma, in combination with the fact that the condition is both more prevalent and more likely to be severe in blacks, mean that black families bear a disproportionate load of the overall asthma health and cost-burdens.

Asthma Risk Factors

It is important at this point to differentiate between the development of asthma (i.e., new cases) and exacerbation in people who already have the condition, though the two do share certain causes. As is common with many chronic diseases, determining the specific causes of asthma has proven difficult, but what seems to be clear is that development of the condition depends on interaction between genetic susceptibility and environmental factors. It is unlikely that the recent increase in the incidence and prevalence of the condition in the United States and other countries is due to an increase in genetic susceptibility, as such widespread change would take far longer than a few decades. Therefore, the rise in asthma incidence appears to be due to behavioral and environmental factors, such as exposure to tobacco smoke (Lau, et al., 2002; Jaakkola, Piipari, Jaakkola, & Jaakkola, 2003), dietary habits (Link & Phelan, 1995), allergic sensitization (Sears, et al., 1991; Palmer, et al., 2002; Riedler, Eder, Oberfeld, & Schreuer, 2000), exposure to animals (Takkouche, Gonzalez-Barcala, Etminan, & FitzGerald, 2008), and occupation-related exposures, especially in health professionals (Bakerly, et al., 2008).

Chetty (2009) identifies categories of causes of exacerbation in people with asthma—some of which are also associated with new onset of asthma. The first is infections of the respiratory tract, with which a majority of exacerbations are associated. Secondly, exposure to allergens is another important cause of exacerbation. Some of these allergens are found outside, such as pollen, but others are found inside the home, such as insects, pets or allergens in food. Environmental exposures are the third category. Occupational asthma, which is caused by exposure to dusts, gases or fumes in the workplace is an example of this. Other environmental risk factors for asthma exacerbation are cold air or exposure to ambient air pollutants (Chetty, 2009). Observational studies of the association between ambient air pollution and exacerbation tend to focus on exposure to all or some of the six criteria air pollutants—ozone, sulfur dioxide, carbon monoxide, lead, nitrogen dioxide, and particulate matter—which are commonly emitted pollutants with federally-regulated limits on ambient concentration levels. The presence of outdoor air pollutants is dependent on the presence of either stationary sources (such as factory emissions) or mobile sources (such as high-traffic roads) of pollution.

Schildcrout et al. (2006) conducted an observational study of asthma exacerbations in children in eight cities in the U.S. and Canada over a 22-month period between 1993 and 1995. Daily, citywide concentrations of five of the six criteria pollutants were collected from the U.S. EPA for the U.S. cities and from Environment Canada for Toronto. At the end of each day, the 990 children who participated in the study recorded whether they had felt symptoms of asthma and, if so, how long they had lasted that day. They also recorded whether how many times they used an inhaler each day. Controlling for race/ethnicity, family income, and day of the week, logistic and Poisson regression models were estimated to determine the relationships between severity of asthma symptoms and ambient concentrations of the criteria pollutants, both individually and two pollutants simultaneously, with a temporal lag of up to two days. Results indicate that an increase of 1 part per million (ppm) in carbon monoxide concentration and 20 parts per billion in nitrogen dioxide concentration at a two-day lag were associated with an odds ratio (OR) of symptoms = 1.08 (95% CI: 1.02, 1.15) and 1.09 (95% CI: 1.03, 1.09) respectively.

Friedman et al. (2001) examined how changes in traffic patterns during the 1996 Summer Olympic Games in Atlanta affected concentration levels of particulate matter, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone; and asthma hospitalizations among the city's residents aged one to 16 during the event. The 17 days of the Olympic Games were compared to the preceding and following four-week periods. Results indicate that the peak daily ozone concentrations decreased 27% during the Olympic Games, and there was a significant reduction in the number of Medicaid claims made for asthma during the event (RR = 0.48, 95% CI: 0.44, 0.86).

While these studies, among others, have been important in showing the association between exposure to ambient air pollution and asthma exacerbation, they have not, in most cases, accounted for how racial or socioeconomic disparities in the former may affect the latter. In fact, an extensive body of literature has shown that in both urban and rural areas of the U.S., the spatial distribution of these pollution sources is not random—they tend to cluster in and around communities of low SES and communities of color (Bullard, 2000; Bullard, 1983; Szasz & Meuser, 1997; Downey, 2006; Grineski S. E., 2007; Grineski, Collins, Chakraborty, & McDonald, 2012). This phenomenon, which has consistently been shown to be true, has come to be known in recent decades as environmental inequality. A further discussion of environmental inequality will come later in this chapter.

Racial Disparities in Asthma

The magnitude of asthma disparities has already been outlined earlier in this chapter, but, partly because of the complexity of the condition itself, the causes of the differences in prevalence, morbidity, and even mortality are not well understood. Wright and Subramanian (2007) note that research into asthma epidemiology has typically focused on individual-level risk factors, but these do not adequately explain disparities in the population. Instead, the authors stress the need for multilevel analysis which takes into account both individual factors and the larger social context. They offer a framework for understanding how different processes operating at the neighborhood level influence and interact with individual traits to produce differential health outcomes. Structural factors (like concentrated poverty), physical conditions (such as building conditions) and social processes, all of which are interrelated, combine to produce disparities in exposure to psychological and environmental stress. These sources of stress, in conjunction with individual differences in genetic susceptibility, overall health levels, and behavior, can strongly influence health outcomes. The uneven distribution of these factors across spaces which are heavily stratified by race and SES appear to contribute to the large asthma disparities that are consistent today.

Canino, McQuaid and Rand (2009) similarly argue for a multilevel approach to understanding and addressing asthma disparities and offer their own framework for this purpose. The framework includes different aspects of and interactions between health care systems and individual/community systems. The factors they identify operate at varying scales and differentially allow for racial and socioeconomic groups to effectively prevent and treat asthma;

several of these factors are consistent with the sources of health disparities more generally. Firstly, policies related to health care, such as access to, cost of, and eligibility for medical care may disadvantage minorities and people of low SES. They note, for example, that minorities are more likely to use government-sponsored health care plans which may restrict their access to specialists, preventive care or treatment. Secondly, individual bias and issues of cultural competence among health care providers and clinicians can influence the quality of care received by minorities. Factors of the community system include the social and environmental context in which people live; these different contexts have varying amounts of poverty, indoor and outdoor allergens and pollution and environmental stress. Individual factors include modifiable factors such as health literacy, beliefs and illness management.

Returning to health disparities more generally, it bears stressing that racial difference alone cannot explain these disparities—it is meaningless outside of a particular social context, and so understanding the mechanisms by which the disparities arise is key. Williams and Jackson (2005) identify three central sources of racial health disparities: SES, residential segregation and medical care, all of which are, of course, are related.

Under the domain of SES, differences in education, income, health practices and stress are some of the important drivers of health inequities between groups of differing socioeconomic position (Williams & Jackson, 2005). Centuries of institutional racism in the U.S. have led to vast differences in educational and occupational attainment, household income and wealth between African Americans and whites. But just as race alone cannot explain the large gaps in health outcomes between racial groups, neither can income, occupation or education explain these disparities. Adler and Newman (2002) posit that these measures are in fact proxies for other related determinants which differ across the range of SES, namely environmental exposures, behavior factors, biological determinants (such as infectious agents) and health care. Further complicating the possible confounding of race and SES is the fact that commonly used measures of SES—including income, occupation, and education—are often not directly comparable between racial groups. For example, educational and occupational attainment are, of course, related, yet white workers earn more and experience lower unemployment rates at every level of education than black workers. And median wealth of white people without a high school diploma is roughly equal to the median wealth of black people with a bachelor's degree (Hamilton, Darity Jr., Price, Sridharan, & Tippett, 2015). What this means is that white families that are considered poor may not experience poverty in the same way as a poor black family. A key driver of these racial SES disparities, and the ways in which poverty is differentially experienced, is residential segregation.

The history of the racist housing policies of the 20th century, such as racial zoning, racially restrictive covenants, and redlining (Massey & Denton, 1993), as well as contemporary instances of discrimination mean that many U.S. cities are still highly residentially segregated by race (Massey & Rugh, 2014). Segregation plays a large role in determining access to education and employment, and the same policies that segregated urban areas by differentially subsidizing homeownership by race continue to be a key factor in the massive racial wealth gap (Williams & Collins, 2001). This has meant that middle-class black families, as measured by income, are more likely to live in areas of low SES than even poor white households (Williams & Collins, 2001).

In addition to being a central determinant of SES, segregation can have an independent influence on health because place and health outcomes are strongly linked (Pickett & Pearl, 2001). For example, access to recreational facilities, green spaces and grocery stores can vary greatly between neighborhoods (Williams & Jackson, 2005). The concentration of poverty in segregated areas also means that municipal services in these areas are often limited, leading to low-quality housing and declining infrastructure (Williams & Collins, 2001). Grady (2016), for example, has found that African American mothers in poor, racially-segregated urban areas of Michigan are at greater risk of preterm birth than African American mothers in poor, more integrated neighborhoods.

Lastly, residential segregation is an important determinant of both access to and quality of medical care (Williams & Collins, 2001). Gaskin, Dinwiddie, Chan and McCleary (2012) provide an informative summary and a valuable study of the relationship between segregation and health care. Minorities living in segregated neighborhoods, for example, may have less access to medical services because health professionals are less likely to live in these neighborhoods. Additionally, health care providers are less likely to locate in these neighborhoods, in part because the fact that minorities are more likely to be covered by government-sponsored health care or uninsured, meaning that providers are not reimbursed to the same degree. The lower density of providers in these also increases the time spent using health care services by patients, either through travel or wait times, which may discourage people who need care from seeking it.

Importantly, though, there are inequalities in access to and quality of medical care that operate independently of segregation. Unsurprisingly, people who have health insurance are generally healthier than those who don’t, and there are important racial and ethnic differences in insurance rates (Richardson & Norris, 2010). But even in the insured population, there are differences in the quality of treatment that people receive. One way this can happen is through bias, stereotyping, or a lack of cultural competence on the part of health professionals. The belief, for example, that African Americans will not adhere to prescribed treatment may cause a health professional to not provide a prescription when it would be appropriate or necessary to do so (Richardson & Norris, 2010).

Environmental Justice and Asthma Studies in Detroit

Despite increased awareness about issues of environmental inequality in recent years, as well federal legislation meant to address these disparities, studies in recent years have continued to find instances of disproportionate health risk being placed on marginalized communities in U.S. cities (Sicotte & Swanson, 2007; Grineski, Bolin, & Boone, 2007; Collins, Grineski, & Morales, 2017).

In studies on Detroit, findings have varied, depending on the extent of the study area, the time period, and the kinds and sources of pollution. Studying all of Michigan, Saha and Mohai (2005) conducted a longitudinal study of the siting of treatment, storage and disposal facilities (TSDFs). They found that these facilities were generally located in majority-white neighborhoods until public concern over the safety of these facilities started to grow in the 1970s. After this time, siting began to concentrate in neighborhoods with low median incomes and larger minority populations. A number of these TSDFs were located in the Detroit area, suggesting that the degree of environmental inequality increased during the study period.

Wu and Batterman (2006) examined the characteristics of Wayne County schools near high-traffic roads, using data from the Michigan Department of Transportation and Michigan's Center for Educational Performance and Information in the year 2000. They found that these schools were more likely to be located in poor areas and have higher percentages of minority students.

Downey (2006), studying the demographic characteristics of neighborhoods around Toxic Release Inventory (TRI) facilities for six counties in the Detroit area—Lapeer, Monroe, St. Clair, Wayne, Macomb, and Oakland—found that a neighborhood's racial composition was a statistically significant predictor of proximity to one of these facilities, with black neighborhoods being disproportionately close to these facilities.

Smith (2007) used data from the 1970-1990 U.S. Censuses, EPA, and Michigan Department of Environmental Quality to measure the significance of race and economic deprivation in determining the location of Superfund sites and landfill. He concluded that a tract's economic deprivation, rather than its racial composition, was the most significant predictor for these two kinds of facilities.

Two studies, by Lee and Mohai (2011) and Downey (2005), yielded surprising results. Lee and Mohai (2011) used census data from 1990 and 2000 to compare the characteristics of census block groups around brownfield sites where 1) cleanup had not yet started, 2) cleanup had been initiated, and 3) cleanup had already occurred. They found that cleanup of brownfield sites was more likely to happen in disadvantaged neighborhoods.

Downey (2005) examined the racial and socioeconomic characteristics of neighborhoods around manufacturing facilities within the City of Detroit from 1970 to 1990. He found that the densest corridors of manufacturing facilities were disproportionately located in majority-white working-class neighborhoods during this time. This unexpected disparity was attributed to the city's history of racial segregation and white resistance to the influx of black residents into white neighborhoods.

All of these studies have focused on different sources and kinds of pollution during various time periods, yet none have used data from later than the turn of the century nor have they incorporated health outcomes.

Li et al. (2011a) conducted a population-based matched case-control study to investigate the association between proximity of residence to major roads and asthma exacerbation in the pediatric Medicaid population in the city of Detroit. Cases were all children having made at least one asthma claim during the period 2004-2006, while controls were randomly selected children on Medicaid without respiratory illness. All addresses were geocoded. Distance from the nearest major road, as both continuous and categorical, was the only predictor variable in the conditional logistic regression, as the cases and controls were matched by race, gender and age to avoid confounding. Results of the analysis indicated that there was a higher risk of hospitalization for asthma with decreasing distance to a major road; however, this relationship was not statistically significant.

Li et al. (2011b), in a separate study, examined the relationship between asthma hospitalizations in the same Medicaid population during the same period and daily ambient pollution concentrations using a case-crossover design. Data on air pollution came from the Michigan Department of Natural Resources and Environment and Environment Canada, and included measurements for carbon monoxide, nitrogen dioxide, sulfur dioxide, particulate matter, and ozone. For each pollutant, daily concentrations were computed by averaging hourly measurements from the monitoring sites within and around the study area. The authors used Poisson regression models for each pollutant to predict its effect on the number of hospitalizations in a particular day, with one through 5-day lags and 2-, 3-, and 5-day moving averages. Results indicated that sulfur dioxide and particulate matter were significantly associated with daily hospitalization for 3-, 4-, and 5-day lags and 5-day moving averages. Carbon monoxide and nitrogen dioxide were associated with hospitalization for 4- and 5-day lags.

These studies demonstrate the role of exposure to air pollution in the overall asthma burden in Detroit, but by only using patients on Medicaid within the City of Detroit, with a substantial portion of the metropolitan population excluded. Furthermore, there is no analysis of how exposure to these pollutants may be patterned by race and socioeconomic status or how this may contribute to the overall asthma disparity in the study area.

Environmental Health Justice Studies

The number of studies linking disparities in exposure or proximity to pollutants, hazards and waste to unequal health outcomes, while still small in comparison to the body of literature on environmental inequality, has grown in recent years, and many of these studies have used asthma or other respiratory conditions as the outcome of interest.

Maantay (2007) used GIS methods to examine the association between proximity to noxious land uses and asthma hospitalization in the Bronx, New York City. Five years of individual-level hospitalizations, from 1995-1999, were georeferenced, and buffers of half a mile, one quarter mile, and 150 meters were created around Toxic Release Inventory (TRI) sites, major stationary point sources, and major roadways respectively. Using a simple coincidence analysis, in which the characteristics of the people within the area of the buffers are compared to those outside, census data at the block group level revealed that both a higher proportion of people within the buffers were minority and a higher proportion were poor compared to the people living outside the buffers. Additionally, people living within the buffer area were 30%

more likely to be hospitalized for asthma. When looking at TRI sites and other stationary sources individually, this figure was 60% and 66% respectively.

Grineski (2007) modeled the relationship between asthma hospitalization, sociodemographic characteristics and ozone and toxic release inventory (TRI) emissions in Phoenix, Arizona. Each ZIP code in the study area was assigned three measures of pollution exposure: a TRI score based on the number of pounds of emissions from the facilities in its area, and an ordinal ozone concentration level based on a modeled emissions surface, and an indoor hazard score based on the median age of housing in the ZIP code. The results of multivariate Poisson regression analyses indicated that the ZIP-code-level asthma hospitalization rate were significantly and positively associated with all three pollution measures and proportion of the ZIP code population who were black, with ozone concentration being the strongest predictor.

Grineski, Collins, Chakraborty and McDonald (2012) studied the association between air pollution exposure, census tract sociodemographic characteristics and hospitalization for asthma and respiratory infection in children in El Paso, Texas. Data on air pollution came from the U.S. Environmental Protection Agency (EPA) National Air Toxics Assessment (NATA), and included major and small stationary and on- and off-road mobile sources of pollution. Emissions from these sources were used to calculate a respiratory hazard index (RHI) for each census tract. Census and hospitalization data are for the year 2000. Census tract sociodemographic variables included proportion Hispanic, proportion of households with a female head of household, proportion of households below the federal poverty line, and median year of home construction. Hospitalization rates for asthma and respiratory infection were calculated for each census tract. To analyze the relationship between these datasets, Pearson correlation coefficients of 1) the sociodemographic variables with RHI values from each kind of pollution sources, and 2) both the RHI values and sociodemographic indicators with the two health outcomes were calculated. Second, several ordinary least squares (OLS) regression models were estimated to predict hospitalizations using pollution sources and the sociodemographic indicators. Results indicate that all sociodemographic variables except proportion Hispanic were significantly correlated with RHI values, and asthma was significantly correlated with proportion of female-headed households, median year of home construction, and major and small stationary sources of pollution. Respiratory infection rates were significantly correlated with all pollution and sociodemographic variables. OLS models predicting asthma hospitalizations indicate that only median year of home construction was significant, while RHI, proportion Hispanic and median year of home construction were significant predictors of infection rates.

Contributions of This Study

This study attempts to make two main contributions to the understanding of asthma, asthma disparities and environmental justice. The first is to determine the association between proximity to stationary sources of air pollution and asthma hospitalizations in a social context in which both are unevenly distributed with respect to different groups. The second is to integrate the concerns of the environmental justice literature into the broader context of the study of racial health disparities, which, as previously mentioned in this chapter, has tended to focus on other important factors like segregation and medical care. Considering the size and consistency of studies of environmental inequality, it is both plausible that disparate exposures to environmental are partly responsible for the considerable health disparities in the U.S., and important that the relationship between the two is identified.

Study Goals, Aims and Hypotheses

The goal of this study is to investigate racial disparities in asthma hospitalizations in the Detroit Metropolitan Area in 2015.

This study has three specific aims.

Specific Aim 1: The first aim is to identify individual and area-level risk factors for asthma hospitalization in the Detroit Metropolitan Area (DMA).

Specific Aim 2: The second aim is to determine whether there are racial disparities in asthma hospitalization rates and how these rates vary by area-level socioeconomic position (SEP) in the Detroit Metropolitan Area.

Specific Aim 3: The third aim is to attempt to describe a pathway by which racial disparities in asthma hospitalization rates exist in the Detroit Metropolitan Area to inform future research in this area.

These aims will be investigated according to the following hypotheses:

1. Hospitalization rates will be higher in the study area's black population than the white population.
2. Hospitalization rates will be higher in areas of low SES than in areas of high SES.
3. When adjusting for neighborhood-level SES, the black-white disparity in hospitalization rates will be reduced but not eliminated.
4. Variables identified as risk factors for hospitalization will be more prevalent in black and low-SES neighborhoods.
5. In the parts of the study area that are racially residentially integrated, black and white hospitalization rates will be similar.

In the following section, a conceptual framework of environmental inequality is presented. This framework, in conjunction with the above review on drivers of racial health disparities, will help guide the analysis discussed in Chapter 5.

Conceptual Framework

Szasz and Meuser (1997) note that many early studies of environmental inequality focused only on documenting cases in which poor and minority populations were disproportionately exposed to hazardous waste sites without offering an explanation of how these situations arose with such regularity. They also note that, in some studies, intentional discrimination was used as the central criterion for determining whether the siting of facilities in minority neighborhoods was racist. At the time, this conceptualization of environmental racism, as necessarily involving discriminatory intent, was common. For example, Benjamin Chavis—the former head of the United Church of Christ Commission on Racial Justice, which published one of the foundational studies of environmental inequality—is credited with introducing the term "environmental racism," and defined it, in part, as the "deliberate targeting of communities of color for toxic and waste facilities" (Holifield, 2001). Discriminatory intent may be involved in some cases of environmental inequality, but it can be difficult to prove.

Others have argued that this framework of environmental inequality is too simplistic, arguing for a more complex formulation of racism in this context (Pulido, 1996) as well as a deeper analysis of the origins of the social and economic conditions that give rise to situations of environmental inequality (Morello-Frosch, 2002; Szasz & Meuser, 1997). Pellow (2000) argues for it to be conceptualized it as a sociohistorical process involving multiple stakeholders with varying amounts of resources and power at their disposal. Viewed in this way, environmental inequality depends on underlying processes which can be political, economic, historical and/or social in nature (Fisher, Kelly, & Romm, 2006). These processes, all inter-related, can produce the uneven spatial distribution of social groups and pollution that often result in residentially-segregated, vulnerable groups being burdened with pollutants and hazards (Eyles, et al., 2001). The central aims for studies of environmental inequality should be not only to examine the sociodemographic characteristics of neighborhoods burdened by hazardous pollutants, but also to consider how political, economic, social, and historical processes have created that spatial distribution of people and hazards.

This body of work has been integral in documenting and explaining disparities in exposure to toxic and hazardous emissions but has remained separate from otherwise-related research in some important ways. As Brulle and Pellow (2006) note, EJ studies have generally documented disparities in exposure and procedure while stopping short of finding associations with health outcomes. The assumption that differential exposure between different racial and SES groups will lead to different health outcomes seems reasonable—especially given the size of the body of literature associating exposure to pollution and health outcomes—but there is a need for further research on this connection. As has already been mentioned, there has been a relatively small number of studies that have actually attempted to measure the association between disparities in environmental exposures and health outcomes (Grineski, Collins, Chakraborty, & McDonald, 2012; Grineski S. E., 2007; Maantay, 2007). Secondly, Brulle and Pellow (2006) note that explanations for the persistent racial health disparities in the U.S. have largely excluded concerns relating to EJ. The integration of 1) health outcomes in to EJ research, and 2) EJ into the broader understanding of racial health disparities already outlined in this chapter are important steps in this field of research.

CHAPTER 3: STUDY AREA

The Detroit Metropolitan Area (DMA) is defined in this study as the area encompassed by Wayne, Oakland and Macomb Counties. The City of Detroit lies entirely in northeast Wayne County. The populations of each in 2015 were estimated to be 1,778,969 in Wayne County; 1,202,362 in Oakland County; and 840,978 in Macomb County. The DMA is marked by high levels of racial segregation—80.1% of the central city's residents are black, while this number is just 13.9% in Oakland County and 10.1% in Macomb County (U.S. Census Bureau, 2015).

Starting round World War I and continuing into the second half of the 20th century, African Americans began to flee the harsh realities of Jim Crow in the South and headed for the large cities in the North. Before that time, there had been relatively low levels of black-white segregation in these cities, mainly due to the small numbers of black residents (Massey & Denton, 1993). Once the black populations began to increase, however, whites began to leave central cities, buying houses in the growing suburbs with substantial assistance from the loan programs of the Federal Housing Administration and Veterans Administration, from which black prospective homeowners were largely and intentionally excluded (Massey & Denton, 1993). These practices not only kept racial groups segregated but allowed for the differential development of wealth between them. The discriminatory practices of blockbusting, restrictive covenants and redlining were eventually outlawed, but part of their legacy is that racial segregation remains high in large cities with large black populations, such as Detroit (Rugh & Massey, 2014).

Mid-20th century Detroit was one of the economic centers of the thriving industrial U.S. economy, providing employment opportunities for both black and white residents—though black workers were generally paid less and had to perform the toughest tasks (Sugrue, 2005).

Deindustrialization in the second half of the century saw a steady decline in manufacturing employment, and in the city's population, which began to drop from a high of around 1.8 million in the 1950s. In 1977, there was less than half the number of workers employed in manufacturing than there had been thirty years earlier. Most of these jobs relocated to smaller towns in the Midwest, South and West, but some of them moved into the suburbs of the Detroit Area—between 1947 and 1958, all 25 new facilities built by Chrysler, Ford and General Motors in the Detroit Area were in the suburbs (Sugrue, 2005). Detroit today continues to be plagued by high levels of inequality across the metropolitan area, pockets of high concentrated poverty, and racial segregation.

Asthma in Detroit

The City of Detroit also has a substantial asthma burden. While the prevalence rates nationally and for Michigan are 7.8% and 11% respectively, 15% of Detroit residents currently have the condition. A report published by the Michigan Department of Health and Human Services (MDHHS) specifically on the state of asthma in Detroit outlines not only the magnitude of the burden within the city, but also the racial disparity (DeGuire, et al., 2016). The rate of hospitalization for asthma from 2008 to 2013 was three times higher than the rest of the state, and white Detroit residents were hospitalized at a rate 35% lower than black residents. The report does not identify causal factors for either the high rates or the disparity, and it is limited to the City of Detroit.

The industrial history, degree of inequality, segregation, and racial disparities in health in Metropolitan Detroit make it a potentially important case study in the link between environmental inequality and racial health disparities.

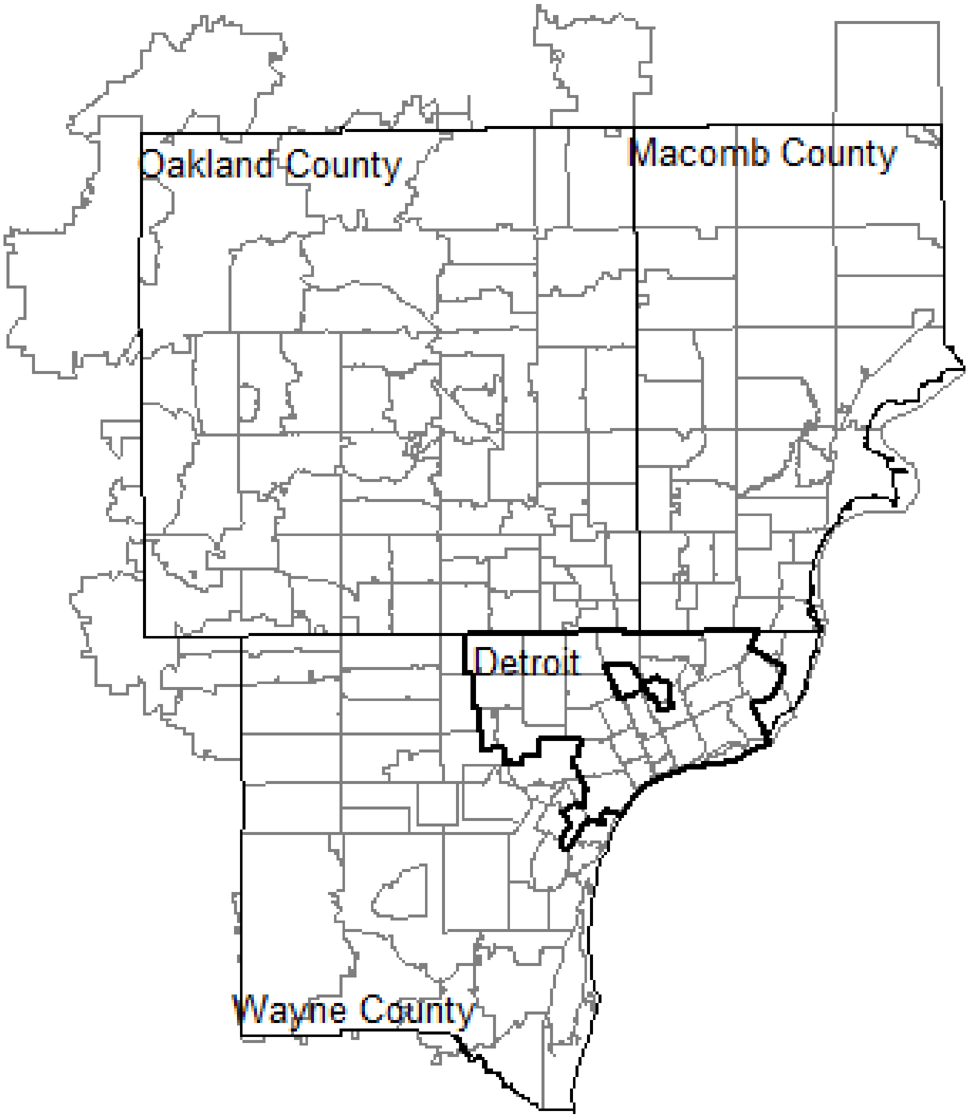

Figure 1. All ZIP codes (n=178 ZCTAs) in the study area of metropolitan Detroit.

CHAPTER 4: DATA

Socioeconomic Data

Data on the sociodemographic characteristics of the DMA were obtained from U.S. Bureau of the Census American Community Survey (ACS) 2011-2015 five-year estimates (2015). The ACS is a survey sent to a small percentage of U.S. households each year, providing data on the changing U.S. population between decennial censuses. The variables used in this study include unemployment rate; median household income; median value of owner-occupied housing units; median gross rent; percent of households below the federal poverty line; percent of households with at least one car available; percent of residents aged 25 or older with a bachelor’s degree; percent of workers employed in management, business, science or arts occupations; and percent homeownership. The data used for this study is aggregated to the ZIP Code Tabulation Area (ZCTA) level. ZCTAs are areas used by the Census Bureau which typically correspond to ZIP codes. They are used in this analysis because the hospitalization data includes a ZIP code of residence for each admission. Using the ZCTA boundaries from the 2015 ACS, the DMA contains a total of 178 ZCTAs (herein referred to as ZIP codes).

Asthma Data

Data on hospital admissions for asthma come from the Michigan State Inpatient Database (SID) for the year 2015 from the Healthcare Cost and Utilization Project (HCUP) at the Agency for Healthcare Research and Quality. These data received from HCUP were classified as a limited dataset under HIPAA, and as such the Agency did not require ethical approval for use of these data; however, a training course was mandatory prior to data use that covered safe management and how to report results for publication. For the first nine months of the year, admissions were coded using the International Classification of Disease, 9^{th} edition (ICD-9), while the 10th edition (ICD-10) was used for the last three months. Asthma hospitalizations were defined as admissions for which either 1) the admitting diagnosis was for asthma (ICD-9: 493.xx; ICD-10: J45.xx) or 2) the admitting diagnosis was for a particular symptom of asthma (including wheezing, cough, breathing difficulty or chest pain), and the second or third diagnosis was asthma, with those asthma symptoms having been marked as present upon arrival at the hospital. A total of 6,555 admissions met these criteria in the study area. The SID also contains variables such as age, race, sex, and ZIP code of residence, type of insurance, visit type, and number of other chronic conditions. Importantly, the dataset also includes a medical number for each patient, so repeat visits could be identified; however, in the majority of cases, patients made only one visit for asthma during the year in question. Because of low numbers for other racial groups, only admissions of black or white patients were included in the study.

Air Pollution

All facilities in the three-county DMA that emit at least one of the six criteria air pollutants are used for this study. The criteria air pollutants are sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), ozone, lead, and particulate matter (PM). National Ambient Air Quality Standards (NAAQS) have been established by the EPA to monitor and limit ambient concentrations of these pollutants, because they are both commonly emitted and potentially harmful to human health. Exposures to ozone, SO_2, PM and NO_2 have all been shown to be associated with respiratory symptoms which vary in severity—from chest tightness and wheezing to development of increased symptoms of asthma. Young children being exposed to lead can negatively affect their development, leading to lowered IQ and behavioral problems (Moody, Darden, & Pigozzi, 2016b). Exposure to CO can lower the blood's oxygen-carrying ability, reducing the oxygen supply to the body's tissue and organs (EPA 2013). Facility locations, as well as the pollutants emitted, were obtained from the EPA's Integrated Compliance System for Air (ICIS-AIR) database. The database contains information on the number of criteria pollutants emitted by facility, and whether the emissions of each pollutant is classified as major or minor. Emissions are classified as major if more than 100 tons of a pollutant are emitted annually (42 U.S.C § 7602 (j)). Only these facilities, of which there are 65 in the study area, are included in this study. This is because the pollutants in question are commonly-emitted, and there are subsequently many sources of pollution which are unlikely to cause harm to the public, such as universities and hospitals. The spatial distribution of these facilities is displayed in Figure 3 (page 46).

CHAPTER 5: METHODS

Darden-Kamel Composite Index

To measure socioeconomic status (SES) in the DMA, the Darden-Kamel Composite Socioeconomic Index will be used. The Darden-Kamel CSI is a nine-variable index which assigns a socioeconomic position (SEP) to every unit of analysis in the study area; a unit with a high score reflects a high SEP (Darden, Rahbar, Jezierski, Li, & Velie, 2010). The index is generally calculated at the census-tract level, but in this case, it is used at the ZCTA level. The nine variables used to calculate the index are:

1. Percentage of the population 25 years or older with at least a bachelor's degree.
2. Median household income of family members 15 years and older.
3. Percentage of workers 16 years or older in managerial or professional positions, based on the U.S. Census Bureau's occupational classification scheme.
4. Median value of owner-occupied housing units.
5. Median gross rent of dwelling.
6. Percentage of all housing units that are owner-occupied.
7. Percentage of families below the federal poverty line.
8. Percentage of civilians 16 or older unemployed but looking for a job.
9. Percentage of households with a vehicle available.

The Darden-Kamel CSI is calculated for each ZIP code as follows:

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

where CSI_i is the composite socioeconomic index for ZIP code i; k is the number of variables used to calculate the index; V_{ij} is the jth SEP variable for ZIP code i; V_{jDMA} is the mean of the jth SEP variable for the DMA; and $S(V_{jDMA})$ is the standard deviation of the jth SEP variable for the DMA. In short, a z-score for each variable in each ZIP code is calculated in order to standardize the contribution of each variable to overall CSI. For percentage below the poverty line and the unemployment rate, the z-scores are multiplied by -1 to account for their negative relationship with SES. Each ZIP code is assigned a score by summing the z-scores for all nine variables, and they are then classified into an SEP quintile based on an ordinal scale: very high (VHSEP), high (HSEP), middle (MSEP), low (LSEP) and very low socioeconomic position (VLSEP); by definition, each quintile will contain the same number of ZIP codes. This classification method has been used previously for studying socioeconomic differences within a metropolitan area (Darden, Rahbar, Jezierski, Li, & Velie, 2010) as well as for health-related studies in Detroit (Moody, Darden, & Pigozzi, 2016a; Moody, Darden, & Pigozzi, 2016b; Grady & Darden, 2012).

Proximity Measurement

Chakraborty and Maantay (2011) provide a valuable discussion and critique of the methods used to assess proximity and exposure to ambient pollution. They describe three broad methodological categories used in environmental justice studies: spatial coincidence analysis, distance-based methods, and pollutant fate and transport modeling.

Spatial coincidence analysis is the simplest of the three methods, and generally involves assigning an exposure level to people living within the boundaries of a spatial unit (census tracts, ZIP codes, etc.) in which a source of pollution lies. The exposure measurement can be as simple as presence vs. absence, the number of facilities within each spatial unit, or can actually take into account the quantity of pollutants emitted by facilities within the unit. The authors point out that whichever of these methods is used, coincidence analysis has several limitations. Most importantly, this kind of analysis assumes that pollution is confined to the unit of analysis in which it is emitted. This assumption may be especially problematic when, for example, a facility's location is near the edge of a census tract or ZIP code—in this situation, the fact that neighboring areas within which people may travel are likely to be similarly affected by pollution from the facility is not factored into the analysis.

Distance-based methods allow for improvement of some of the limitations of coincidence analysis. These methods do not assume that pollution is confined to the spatial units in which they are emitted; rather, there, in most cases, everyone within a predefined distance of a pollution source is assumed to be affected. The most commonly-used example is buffer analysis, but these methods also have their limitations. Firstly, buffers of the same size are generally drawn around all pollution sources, regardless of their size or the magnitude of their emissions. Secondly, these methods also often assume that all people within the buffer are equally affected while those who are outside are not affected at all. This second limitation can be mitigated through the use of continuous distance-based measurements, such as the distance from the centroid of the unit of analysis to the nearest facility. The most complex distance-based methods use mathematical distance-decay functions to take into account the fact that, in certain instances, those living closest to a facility may not be affected by its emissions due to, for example, the direction and height at which emissions are released into the atmosphere.

Lastly, some researchers have used pollutant modeling to determine areas exposed to hazards. This modeling process typically requires data about emissions, as well meteorological variables such as temperature, and wind speed and direction. These methods are often the most accurate for determining exposure to pollution.

To measure proximity to stationary sources of air pollution in this study, each ZIP code will be assigned a value corresponding to the number of facilities in that ZIP code and all neighboring ZIP codes. This is a relatively simple measure, but it does improve on the assumption in most spatial coincidence analyses that pollution is confined to the ZIP code in which it is emitted.

Descriptive Analysis

Using the hospitalization and ACS sociodemographic data, overall and race-specific hospitalization rates will be calculated for each ZIP code, using the underlying population as the denominator. The rates will be age-adjusted using the age distribution of the study area: 76.8% of the population are adults, and 23.2% are children (age <18). Age adjustment is commonly performed using smaller age groups (i.e., 5- or 10-year age groups) but given the relatively low numbers within each ZIP code, adjusting for just two age groups was appropriate. Because of the uneven distribution of hospitalizations across the study area, there are numerous ZIP codes with too few hospitalizations to yield a stable rate, so rates will only be reported for the ZIP codes with 20 or more hospitalizations.

Overall, race-specific, and race-age-specific hospitalization rates will also be calculated by SEP in order to allow comparison not only across different SEP levels, but also between racial groups in the same level of SEP. Aggregating the rates to the SEP level, as opposed to the ZIP code, also avoids the problem of low numbers yielding unstable rates.

Lastly, the segregated nature of the DMA means that even if black and white residents are living in neighborhoods of similar SES as measured by the Darden-Kamel Index, they may still be unlikely to be living in the same neighborhoods. To control for this fact, race-specific hospitalization rates in the ZIP codes in which there were both at least 20 black and 20 white admissions will be compared; these ZIP codes would necessarily be relatively integrated.

Statistical Analysis

Poisson Probabilities

Using race-age-sex-specific rates for each SEP level, an expected number of hospitalizations in each ZIP code will be calculated. For example, the expected number of hospitalizations for black boys in a VLSEP ZIP code is found by multiplying the overall hospitalization rate for black boys in all VLSEP ZIP codes by the total population of black boys in that particular ZIP code. The same process is repeated for the other seven race-sex-specific groups (black girls, men, and women; and white girls, boys, men and women) and the products of all seven groups are summed to yield an expected number of hospitalization for that ZIP code. The expected number of hospitalizations is then compared to the actual number of hospitalizations in each ZIP code using a Poisson distribution. The Poisson distribution is commonly used for count data and will yield the probability of the observed hospitalization count being lower than what it actually is (the area under the curve to the left of the observed value). The Poisson cumulative density function is given by:

$$p(x) = \sum_{i=0}^{x} \frac{e^{-\lambda}\lambda^{i}}{i!},$$

where $p(x)$ is the cumulative probability of the observed value, x is the observed value, λ is the scale parameter (in this case, the expected value).

This means that in ZIP codes in which the expected and observed counts are similar, the probability value will be around 0.5; in ZIP codes in which the observed count is much larger than expected, the probability will approach 1, and in ZIP codes in which the observed count is much lower than expected the probability will approach 0.

Logistic Regression

Logistic regression will be used to determine risk factors for asthma hospitalization. Logistic regression is a commonly-used technique, especially in health studies, for a categorical—often binary—dependent variable. This method models the probability of an event occurring given some set of predictor variables. The logistic regression model is given by:

$$\frac{\pi}{1-\pi} = \exp(\beta_0 + \beta_1 X_1 + \ldots + \beta_i X_i),$$

where π is the probability of success, β_0 is the intercept term, $\beta_1 \ldots \beta_i$ are the slope coefficients for independent variables $X_1 \ldots X_i$. By definition, $\frac{\pi}{1-\pi}$ is the odds of success, or the odds of the occurrence of the outcome of interest. Slope coefficients are estimated using a maximum likelihood approach. The logistic models were estimated using the Proc Logistic command in SAS software (Version 9.4, 2018).

For this study, logistic regression will be used to compare the characteristics of people hospitalized for asthma to those who were hospitalized for injuries (ICD-9: 800.xx-939.xx, 950.xx-957.xx; ICD-10: S00.xx-T14.xx). This group of hospitalizations was chosen as the comparison group because they are unlikely to be associated with the variables that are thought to be risk factors for asthma, and the racial and class distribution of the people who were hospitalized for injuries closely reflects the overall racial and class distribution of the study area. The results of these logistic regression models can then, in effect, be interpreted as a comparison of the characteristics of people hospitalized for asthma with a representative sample of the underlying population of the DMA.

A number of different models will be used. Firstly, in a more exploratory effort, bivariate logistic regression models will be estimated to investigate the relationships between asthma hospitalization and all independent variables, which are both individual- and neighborhood-level. Second, a SEP-stratified model with race (black vs. white) as the only independent variable will be estimated to observe the racial differences by SEP in asthma hospitalizations. Third, a model that contains only individual-level variables together: insurance type (public vs. private), age, sex, race, and number of chronic conditions will be estimated to investigate the effects of race on asthma hospitalization controlling for differences in sex, age and underlying susceptibility. Because almost all of those hospitalized for asthma aged 65 and older were covered by Medicare, only people aged below that threshold are included in this model. The third type of model includes only ZIP code variables: SEP, facility sum, and racial composition (percent of ZIP code residents who are black). The final model combines individual- and neighborhood-level variables and includes interaction terms.

To report the results from each of these models, the output intercept coefficients and their accompanying standard errors and chi-square tests and p-values are provided. In the same tables of results from the logistic regression are reported, specifically the odds ratio and 95% Confidence Intervals.

CHAPTER 6: RESULTS

Descriptive Results

Table 1 shows the mean values for the nine variables used to calculate the Darden-Kamel Index for all five levels of SEP. For each variable, there is a clear and substantial gradient. On average, in very low SEP ZIP codes, median household income is $25,000, more than 1 in 3 families have income below the federal poverty line, 12% of residents 25 or older have a bachelor's degree, and more than 1 in 5 working-age residents are unemployed. In very high SEP ZIP codes, by contrast, average median household income is just below $100,000, only 3% of families on average live on incomes below the FPL, more than half of residents have a bachelor's degree, and 5% of working-age residents are unemployed. The median value of owner-occupied homes in these neighborhoods is also more than five times higher than in VLSEP ZIP codes.

SEP Quintiles	% Residents with a Bachelor's Degree	Median Household Income ($)	% Below Poverty	% Residents Employed in Management, Business, Science or Arts	% Homeownership	% Unemployment	% Households with a Vehicle Available	Median Rent ($)	Median Value of Owner-Occupied Housing ($)
Very Low SEP (1)	12.64	25,767	34.91	22.05	47.91	22.55	75.40	724	48,217
Low SEP (2)	20.64	44,167	15.55	29.51	61.39	11.27	89.93	859	89,400
Middle SEP (3)	28.07	58,318	7.97	36.81	72.95	8.09	94.99	879	134,811
High SEP (4)	35.88	72,132	4.73	43.47	79.85	6.93	95.82	976	187,768
Very High SEP (5)	56.62	99,879	3.33	57.41	85.41	5.41	97.32	1,268	268,077
Overall	30.79	60,063	13.33	37.85	69.48	10.87	90.88	940	145,717

Table 1. Mean values of the nine variables used to compute the Darden-Kamel Composite Socioeconomic Index for DMA ZIP codes, 2011-2015. *Source: U.S. Bureau of the Census (2015).*

Figure 2 displays the spatial distribution of ZIP code SEP across the study area. Immediately obvious are the clustering of low and very low SEP neighborhoods in and around the central city and the general increase of SEP with distance from the city.

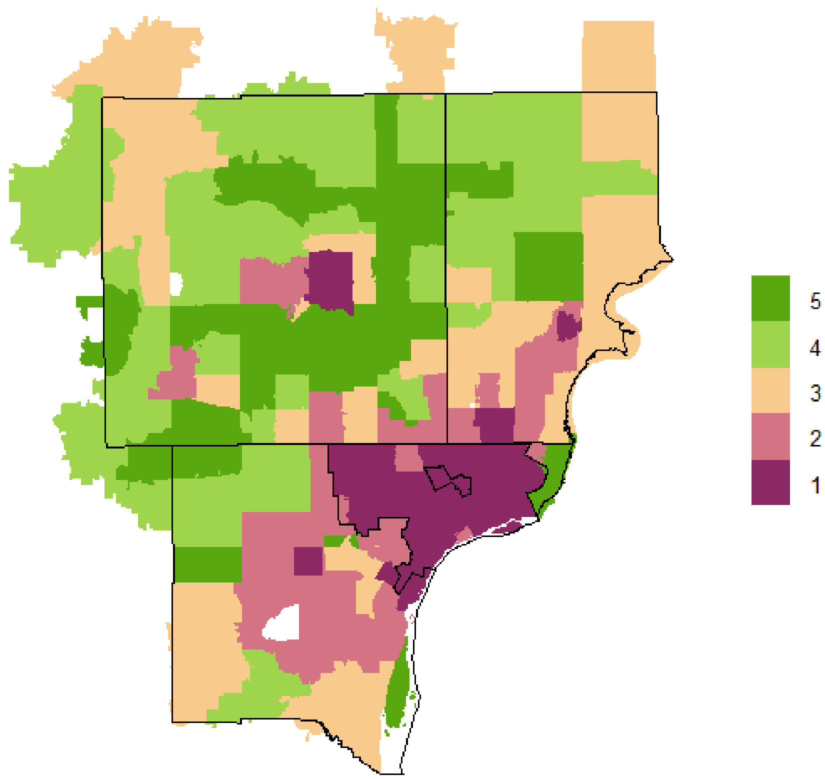

Figure 2. Spatial distribution of ZIP-code socioeconomic position as calculated by the Darden-Kamel Composite Socioeconomic Index.
1=lowest SEP, 5=highest SEP. Source: U.S. Bureau of the Census (2015).

Table 2 shows the overall hospitalization rates by SEP and race, exhibiting that there are substantial disparities by both. In VLSEP ZIP codes, the hospitalization rate is nearly 5 times higher than in VHSEP, and more than twice as high as in even LSEP ZIP codes. Within VLSEP ZIPs, the black hospitalization rate is 3.65 higher than the white rate, indicated by the risk ratio in the far-right column. This black-white risk ratio generally decreases as the SEP increases, but even at the highest SEP, African Americans are hospitalized at a rate 2.72 times higher than whites. Strikingly, the black hospitalization rate in these VHSEP ZIP codes is still considerably higher than the white rate even in VLSEP neighborhoods.

There are also notable differences when admissions are separated by age, as shown in Table 3. At every SEP level, white adults have higher rates than white children (age < 18), but the pattern is not as clear for black children and adults. Black adults have higher rates in the VLSEP and LSEP ZIP codes, but the rates for black children are higher, especially in the MSEP and VHSEP ZIP codes. The largest black-white disparities in this case are present between children, where in VLSEP and MSEP ZIP codes, the risk ratios are 4.73 and 5.67 respectively. The lowest hospitalization rate for black children is considerably higher than the highest rate in white children. In adults, the disparities are not as large, but are still substantial, ranging from 3.42 to 2.03; adults are also the case for which the lowest black hospitalization rate is lower than the highest rate for whites.

SEP	All Admissions		Black Admissions		White Admissions		RR
	N	Hospitalization Rate	N	Hospitalization Rate	N	Hospitalization Rate	
1	3,135	374.4	2,720	458.0	228	125.6	3.65
2	1,629	171.1	764	358.4	741	109.3	3.28
3	811	108.8	189	223.3	527	85.7	2.61
4	539	78.2	47	166.4	442	72.1	2.31
5	441	67.1	64	159.0	317	58.6	2.72

Table 2. Hospitalization rates by SEP and racial group.

Sources: HCUP (2015) and U.S. Bureau of the Census (2015).

SEP	Black Child Rate	White Child Rate	Child RR	Black Adult Rate	White Adult Rate	Adult RR
1	449.3	95.1	4.73	460.6	134.9	3.42
2	339.4	89.1	3.81	364.1	115.3	3.16
3	332.1	58.6	5.67	190.4	93.9	2.03
4	125.5	53.9	2.33	178.8	77.6	2.31
5	252.3*	46.9	5.38*	130.8	62.1	2.11

Table 3. Hospitalization rates by SEP and race-age group.

Sources: HCUP (2015) and U.S. Bureau of the Census (2015).

** - unstable rate due to low numbers.*

Table 4 shows the characteristics of patients admitted for asthma by race and SEP level. Immediately apparent is the uneven distribution of blacks and whites across the SEP spectrum. In VLSEP ZIP codes, for example, 76% percent of the black-white population is black, but in HSEP and VHSEP ZIP codes, just 4% and 6% are black respectively. This uneven distribution in the underlying population is partly reflected in the raw admission numbers—with a much smaller number of black admissions in VHSEP than in VLSEP ZIP codes, for example—but there are still important disproportionalities reflected in these totals: in the most extreme case, African Americans in VLSEP ZIP codes constitute just 16.2% of the study area's black and white population, but 45.0% of all asthma admissions are among African Americas. At every level of SEP, however, the percentage of black admissions is disproportionate relative to the black population proportion: in VHSEP ZIP codes, 16.0% of the admissions come from the 6.7% of the population that is black.

Table 4 also contains information about the ages, gender, number of chronic conditions, insurance type and visit type by race and SEP level. There are no substantial differences by visit type; in almost all cases, roughly 90% of the asthma admissions were admitted through the emergency room.

Table 4. Selected characteristics of the 6,039 black and white hospital admissions by ZIP code. *Source: HCUP (2015). Cells with "-" entered are not reported due to a count of 10 or fewer.*

	VLSEP		**LSEP**		**MSEP**		**HSEP**		**VHSEP**		**Total**	
	Black	White	Black	White	Black	White	Black	White	Black	White	B (%)	W (%)
Population	594,283 76.7 16.2	180,560 23.2 4.9	213,836 24.1 5.8	673,601 75.9 18.4	88,923 11.9 2.4	655,958 88.1 17.9	28,485 4.2 0.8	644,026 95.8 17.6	39,471 6.7 1.1	546,386 93.3 14.9	964,99 8 26.3	2,700,531 73.7
Admission	2,720 92.3 45.0	228 7.7 3.8	764 50.8 12.7	741 49.2 12.3	189 26.4 3.1	527 73.6 8.7	47 9.6 0.8	442 90.4 7.3	64 16.8 1.1	317 83.2 5.2	3,784 62.7	2,255 37.3
Age Group												
Children	689 94.9 51.2 25.3	37 5.1 2.7 16.2	200 62.1 14.8 26.2	122 37.9 9.1 16.5	67 48.6 5.0 35.4	71 51.4 5.3 13.5	-	68 89.5 5.0 15.4	26 30.6 1.9 40.6	59 69.4 4.4 18.6	990 73.5	357 26.5
Adults	2,031 91.4 43.3 74.7	191 8.6 4.1 83.8	564 47.7 12.0 73.8	619 52.3 13.2 83.5	122 21.1 2.6 64.6	456 78.9 9.7 86.5	39 9.4 0.8 83.0	374 90.6 8.0 84.6	38 12.8 0.8 59.4	258 87.2 5.5 81.4	2,794 59.5	1,898 40.5
Sex												
Males	891 91.8 44.8 32.8	80 8.2 4.0 35.1	251 49.7 12.6 32.9	254 50.3 12.8 34.3	61 27.0 3.1 32.3	165 73.0 8.3 31.3	17 10.8 0.9 36.2	140 89.2 7.0 31.7	19 14.8 1.0 29.7	109 85.2 5.5 34.4	1,239 62.4	748 37.6
Females	1,829 92.5 45.1 67.2	148 7.5 3.7 64.9	513 51.3 12.7 67.1	487 48.7 12.0 65.7	128 26.1 3.2 67.7	362 73.9 8.9 68.7	30 9.0 0.7 63.8	302 91.0 7.5 68.3	45 17.8 1.1 70.3	208 82.2 5.1 65.6	2,545 62.8	1,507 37.2

Table 4 (cont'd)

	VLSEP		**LSEP**		**MSEP**		**HSEP**		**VHSEP**		**Total**	
	Black	White	Black	White	Black	White	Black	White	Black	White	B (%)	W (%)
No. of Chronic Conditions												
1	540	30	139	106	57	62	-	64	19	58	761	320
	94.7	5.3	56.7	43.3	47.9	52.1		91.4	24.7	75.3	70.4	29.6
	50.0	2.8	12.9	9.8	5.3	5.7		5.9	1.8	5.4		
	19.9	13.2	18.2	14.3	30.2	11.8		14.5	29.7	18.3		
2 to 4	843	64	238	193	57	116	19	95	18	71	1,175	539
	92.9	7.1	55.2	44.8	32.9	67.1	16.7	83.3	20.2	79.8	68.6	31.4
	49.2	3.7	13.9	11.3	3.3	6.8	1.1	5.5	1.1	4.1		
	31.0	28.1	31.2	26.0	30.2	22.0	40.4	21.5	28.1	22.4		
5 to 9	983	95	287	301	54	231	18	174	20	120	1,362	921
	91.2	8.8	48.8	51.2	18.9	81.1	9.4	90.6	14.3	85.7	59.7	40.3
	43.1	4.2	12.6	13.2	2.4	10.1	0.8	7.6	0.9	5.3		
	36.1	41.7	37.6	40.6	28.6	43.8	38.3	39.4	31.3	37.9		
10+	354	39	100	141	21	118	-	109	-	68	486	475
	90.1	9.9	41.5	58.5	15.1	84.9		96.5		90.7	50.6	49.4
	36.8	4.1	10.4	14.7	2.2	12.3		11.3		7.1		
	13.0	17.1	13.1	19.0	11.1	22.4		24.7		21.5		

Table 4 (cont'd)

	VLSEP		**LSEP**		**MSEP**		**HSEP**		**VHSEP**		**Total**	
	Black	White	Black	White	Black	White	Black	White	Black	White	B (%)	W (%)
Insurance												
Medicare	716	74	215	318	57	254	17	223	22	146	1,027	1,015
	90.6	9.4	40.3	59.7	18.3	81.7	7.1	92.9	13.1	86.9	50.3	49.7
	35.1	3.6	10.5	15.6	2.8	12.4	0.8	10.9	1.1	7.1		
	26.3	32.5	28.1	42.9	30.2	48.2	36.2	50.5	34.4	46.1		
Medicaid	1,545	107	304	185	58	91	-	78	19	32	1,934	493
	93.5	6.5	62.2	37.8	38.9	61.1		90.7	37.3	62.7	79.7	20.3
	63.7	4.4	12.5	7.6	2.4	3.7		3.2	0.8	1.3		
	56.8	46.9	39.8	25.0	30.7	17.3		17.6	29.7	10.1		
Private	407	43	225	228	67	168	19	136	22	134	740	709
	90.4	9.6	49.7	50.3	28.5	71.5	12.3	87.7	14.1	85.9	51.1	48.9
	28.1	3.0	15.5	15.7	4.6	11.6	1.3	9.4	1.5	9.2		
	15.0	18.9	29.5	30.8	35.4	31.9	40.4	30.8	34.4	42.3		
Self-pay	47	-	18	-	-	11	-	-	0	-	73	31
	95.9		66.7			64.7					70.2	29.8
	45.2		17.3			10.6						
	1.7		2.4			2.1						

Table 4 (cont'd)

	VLSEP		**LSEP**		**MSEP**		**HSEP**		**VHSEP**		**Total**	
	Black	White	Black	White	Black	White	Black	White	Black	White	B (%)	W (%)
Admission Type												
ER	2,496 92.4 45.0 91.8	206 7.6 3.7 90.4	698 50.1 12.6 91.4	695 49.9 12.5 93.8	173 26.8 3.1 91.5	473 73.2 8.5 89.8	46 10.1 0.8 97.9	411 89.9 7.4 93.0	60 17.3 1.1 93.8	287 82.7 5.2 90.5	3,473 62.6	2,072 37.4
Urgent	192 91.4 45.0 7.1	18 8.6 4.2 7.9	55 57.3 12.9 7.2	41 42.7 9.6 5.5	15 23.8 3.5 7.9	48 76.2 11.2 9.1	-	27 96.4 6.3 6.1	-	26 86.7 6.1 8.2	267 62.5	160 37.5
Elective	-	-	-	-	-	-	0	-	0	-	19 46.3	22 53.7
Other	21 100.0 84.0 0.8	0	-	0	0	-	0	0	0	0	24 96.0	-

Figure 3 displays the crude hospitalization rate in ZIP codes in which there were at least 20 asthma admissions (i.e., minimum numerator by which to calculate a stable rate) (Grady & Enander, 2009). The figure shows how the high counts of asthma hospitalization are concentrated in LSEP ZIP codes.

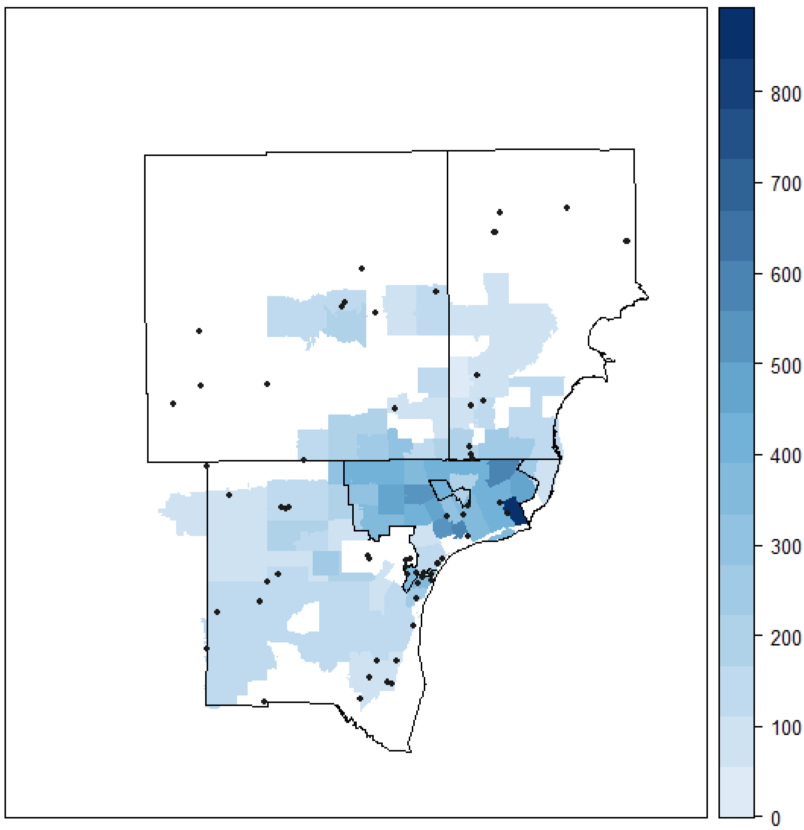

Figure 3. Crude hospitalization rate by ZIP code.
ZIP codes with fewer than 20 admissions excluded. Points displayed on the map are the locations of the air-polluting facilities included in this study.

Figure 4 shows a scatterplot of the crude hospitalization rate in each of the 86 ZIP codes with at least 20 hospitalizations by the proportion of residents who are black, stratified by SEP. Not only does this figure show how the hospitalization rate tends to increase with decreasing SEP, but also that rates tend to be higher rates in ZIP codes with higher proportions of black residents. Because there is less variation in both hospitalization rates and racial composition in the HSEP (SEP=4) ZIP codes, this positive relationship between hospitalization rate and proportion of black residents is most obvious in the LSEP (SEP=2) ZIP codes.

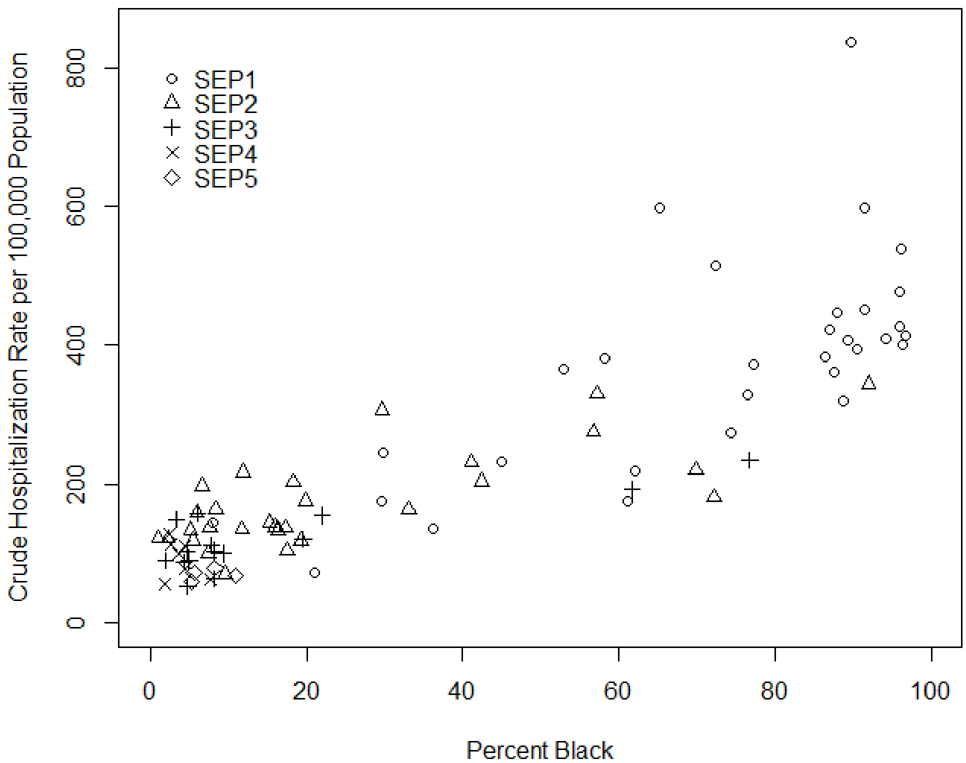

Figure 4. Scatterplot of proportion of black residents vs. crude hospitalization rate by ZIP code. *ZIP codes are grouped by SEP. Source: HCUP (2015) and U.S. Bureau of the Census (2015).*

The degree of racial segregation in metropolitan Detroit means that of the 178 ZIP codes in the study area, there are just 9 in which there were at least 20 black and 20 white asthma hospital admissions. Each of these 9 ZIP codes was classified as either LSEP (SEP=2) or VLSEP (SEP=1), and all were majority white, ranging from 52.2% to 77.8% white. All were located just near the boundary of the city of Detroit. In these ZIP codes, the mean black-white hospitalization rate ratio was RR=3.01 (range, RR=1.85 to 5.84).

Analytic Results

Figure 5 shows a scatterplot of the observed number of hospitalizations in a ZIP code vs. the expected number, given the age-sex-race-class distribution of the underlying population. The points that are along the diagonal line have the same number of expected and observed counts. Points below the line have fewer observed hospitalizations than were expected, while points above the line had more observed hospitalizations than expected. In most cases, once the structure of the population is taken into account, the observed hospitalization count is fairly close to that expected.

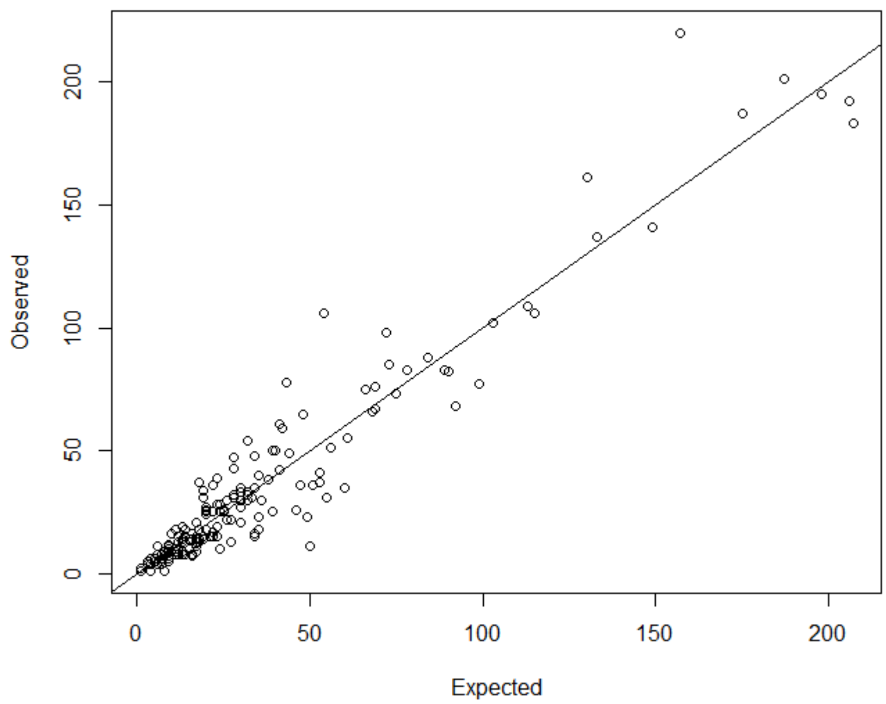

Figure 5. Scatterplot of expected vs. observed hospitalization counts by ZIP code.
HCUP (2015) and U.S. Bureau of the Census (2015).

Figure 6 shows the spatial distribution of the probability of being hospitalized with asthma—based on a Poisson distribution with mean and variance equal to the expected hospitalization count. The bluer the ZIP code, the higher the observed was compared to the expected hospitalizations, while the greener the ZIP code, the lower the observed was compared to the expected hospitalizations. The bluer ZIP codes are located in all three counties and scattered throughout the study area not limited to Detroit.

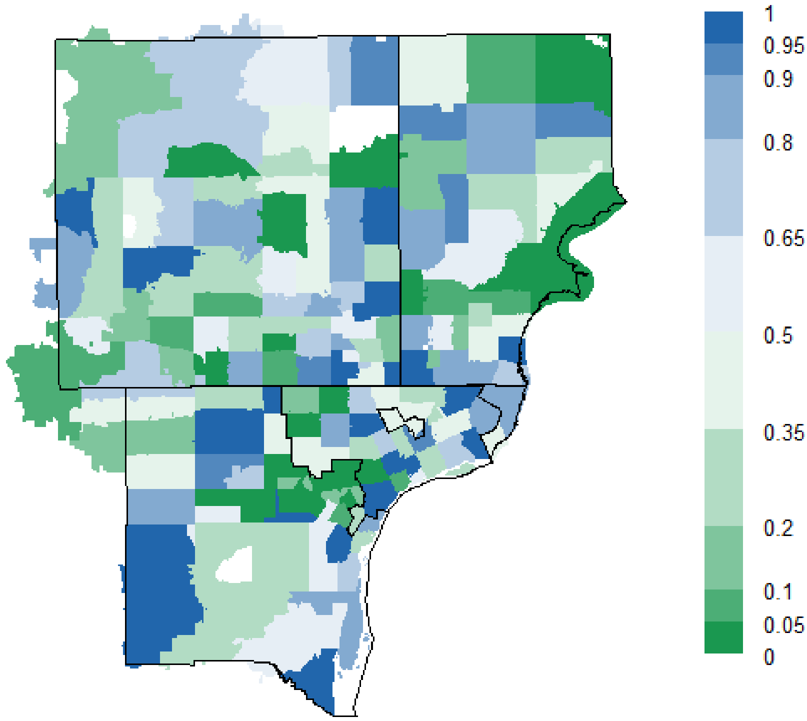

Figure 6. Spatial distribution of probability of observed hospitalization count based on Poisson distribution with a mean of the expected hospitalization count.
Source: HCUP (2015) and U.S. Bureau of the Census (2015).

Logistic Regression Results

Results of the logistic regression models are shown in Tables 5 to 10. Table 5 reports the odds ratios for the bivariate models. In every case, the odds ratios are significant. As compared with those hospitalized for injuries (used here as a representative population sample), adults and children hospitalized for asthma are 3.3 times more likely to be black than white. Those living in VLSEP (SEP=1) and LSEP (SEP=2) ZIP codes are significantly more likely to hospitalized for asthma, and people in HSEP (SEP=4) and VHSEP (SEP=5) ZIP codes are significantly less likely to be hospitalized, relative to similar adults and children in MSEP (SEP=3) ZIP codes. When stratified by age, children are overrepresented among those hospitalized for asthma. There is also a demographic skew by sex, where males are significantly less likely to be hospitalized for asthma than females. Furthermore, having public insurance instead of private insurance appears to imply elevated risk of asthma hospitalization. Having other chronic conditions in addition to asthma is also suggestive of elevated risk for asthma hospitalization. Lastly, the presence of facilities with stationary sources of pollution in and around the ZIP code of residence has an odds ratio below 1 but is only slightly significant.

Variable	R^2	Odds Ratio (95% CI)
Race[1]	0.081	3.316 (3.056, 3.599)
SEP1 vs. 3	0.0472	2.343 (2.061, 2.663)
SEP2 vs. 3		1.515 (1.320, 1.739)
SEP4 vs. 3		0.704 (0.591, 0.839)
SEP5 vs. 3		0.743 (0.614, 0.898)
Facilities	0.0012	0.974 (0.959, 0.989)
Adult[2]	0.0472	0.314 (0.283, 0.349)
Sex[3]	0.0711	3.039 (2.809, 3.300)
Insurance[4]	0.0423	2.370 (2.184, 2.572)
Conditions[5]	0.005	1.340 (1.211, 1.482)

Table 5. Exploratory bivariate logistic regression models.
1: Black vs. White; 2: Adult vs. Child; 3: Female vs. Male;
4: Public vs. Private; 5: 2-4 conditions vs. 1 (asthma only).

The SEP-stratified models shown in Table 6 indicate significantly elevated odds of hospitalization for African Americans compared to whites at every level of SEP. In fact, the odds ratio estimate for VHSEP is the highest of the five levels at OR=3.43.

Predictor	β	SE	Wald's χ^2	*df*	*p*	OR (95% CI)
Race, SEP1	0.5098	0.0486	109.89	1	<.0001	2.772 (2.291, 3.354)
Race, SEP2	0.5831	0.0421	192.18	1	<.0001	3.210 (2.722, 3.785)
Race, SEP3	0.4930	0.0702	49.3689	1	<.0001	2.680 (2.036, 3.529)
Race, SEP4	0.4508	0.1227	13.5015	1	0.0002	2.463 (1.523, 3.985)
Race, SEP5	0.6164	0.1096	31.6157	1	<.0001	3.431 (2.232, 5.273)

Table 6. Bivariate SEP-stratified logistic regression models.
β-*values and chi-square statistics are for different estimates than odds ratios.*

The regression model shown in Table 7 estimates the relationships between all individual characteristics and risk of asthma hospitalization. Results indicate that having chronic conditions in addition to asthma is, again, a risk factor for hospitalization. Having public insurance carries elevated odds of hospitalization relative private insurance, and there is still a strong interaction between adult status and sex. Relative to bivariate model including race, once these individual factors are considered, the odds of African Americans being hospitalized for asthma is slightly reduced from OR=3.316 to OR=2.822 (95% CI 2.483-3.207). This finding suggests that not all but a substantial portion of the racial disparities in asthma hospitalization is explained by individual-level characteristics. Table 7 also includes diagnostic statistics for this model. The likelihood ratio, score, and Wald tests compare the fit of this model to a model which only includes the intercept. The significantly-low p-values indicate that the model is an improvement over the intercept-only model (Peng, Lee, & Ingersoll, 2002). The Hosmer & Lemeshow statistic tests the goodness-of-fit of the model. The null hypothesis for this test, which has a chi-square distribution, is that the model fits the data well. The p-value means that, in this case, the null hypothesis would not be rejected.

Predictor	β	SE	Wald's χ^2	*df*	*p*	OR (95% CI)
Constant	-0.1760	0.0339	26.89	1	<.0001	NA
Race[1]	0.5187	0.0379	252.48	1	<.0001	2.822 (2.483, 3.207)
Sex[2]	0.4465	0.0335	177.22	1	<.0001	--
Chronic[3]	0.6019	0.0379	252.92	1	<.0001	3.333 (2.873, 3.866)
Adult[4]	-1.1822	0.0407	843.54	1	<.0001	--
Insurance[5]	0.2288	0.0324	50.02	1	<.0001	1.580 (1.392, 1.794)
Adult*Sex[2]	0.2930	0.0335	76.52	1	<.0001	
Adult						4.388 (3.785, 5.088)
Child						1.359 (1.094, 1.689)
Test			χ^2	*df*	*p*	
Overall model evaluation						
Likelihood ratio test			2,161.27	6	<.0001	
Score test			1,893.42	6	<.0001	
Wald test			1,413.71	6	<.0001	
Goodness-of-fit test						
Hosmer & Lemeshow			13.05	8	0.1101	

Table 7. Logistic regression for asthma hospitalizations. Only individual variables included. *Source: HCUP (2015).* 1: Black vs. White; 2: Female vs. Male; 3: 2-4 conditions vs. 1 (asthma only); 4: Adult vs. Child; 5: Public vs. Private. β-*values and chi-square statistics are for different estimates than odds ratios.*

The model in which only neighborhood-level variables are included in shown in Table 8. Race continues to be highly significant (OR=2.936, 95% CI 2.635, 3.272), while there are some notable differences when high and low levels of SEP are compared to the middle SEP level—with living in low SEP (OR=1.244, 95% CI 1.078-1.435) the most significant risk factor controlling for the number of polluting facilities. Interestingly, facility sum is not significant. Diagnostics for this model indicate a significantly better fit than the intercept-only model, and the large p-value for the Hosmer & Lemeshow statistic indicates that the model fits the data well.

Predictor	β	SE	Wald's χ^2	*df*	*p*	OR (95% CI)
Constant	-0.3138	0.0319	96.9009	1	<.0001	
Race	0.5386	0.0277	378.6359	1	<.0001	2.936 (2.635, 3.272)
SEP				1		
1 vs. 3	0.1504	0.0462	10.5921		0.0011	1.149 (0.990, 1.333)
2 vs. 3	0.2299	0.0414	30.8698		<.0001	1.244 (1.078, 1.435)
4 vs. 3	-0.1874	0.0612	9.3725		0.0022	0.819 (0.685, 0.980)
5 vs. 3	-0.2048	0.0678	9.1302		0.0025	0.805 (0.663, 0.978)
Facilities	-0.00625	0.00797	0.6150	1	0.4329	0.994 (0.978, 1.009)
Test			χ^2	*df*	*p*	
Overall model evaluation						
Likelihood ratio test			902.82	6	<.0001	
Score test			883.07	6	<.0001	
Wald test			848.83	6	<.0001	
Goodness-of-fit test						
Hosmer & Lemeshow			6.5432	8	0.5866	

Table 8. Logistic regression analysis of asthma hospitalization and area-level characteristics.
1: Black vs. White; 2: Female vs. Male; 3: 2-4 conditions vs. 1 (asthma only); 4: Adult vs. Child; 5: Public vs. Private. Source: HCUP (2015). β-values and chi-square statistics are for different estimates than odds ratios.

The next model is shown in Table 9 and adds ZIP code SEP to the model shown in Table 7. Odds ratios for most of the individual variables remain almost the same compared to the previous model, but the significance of race is once again reduced from OR=2.822 to OR=2.634; 95% CI: 2.239, 3.099. In this model, LSEP becomes not significant although the odds of asthma hospitalization remain high for adults and children living in those areas.

Predictor	β	SE	Wald's χ^2	*df*	*p*	OR (95% CI)
Constant	-0.2394	0.0414	33.43	1	<.0001	NA
Race[1]	0.4843	0.0415	136.46	1	<.0001	2.634 (2.239, 3.099)
Sex[2]	0.4531	0.0337	180.31	1	<.0001	--
Chronic[3]	0.5983	0.0380	247.82	1	<.0001	3.309 (2.851, 3.840)
Adult[4]	-1.1900	0.0410	842.43	1	<.0001	--
Insurance[5]	0.2107	0.0337	39.11	1	<.0001	1.524 (1.335, 1.739)
Adult*Sex[2]	0.2912	0.0337	74.83	1	<.0001	
Adult						4.431 (3.820, 5.139)
Child						1.383 (1.111, 1.720)
SEP						
1 vs. 3	0.0914	0.0709	1.66	1	0.1974	1.021 (0.816, 1.277)
2 vs. 3	0.2588	0.0617	17.62	1	<.0001	1.207 (0.976, 1.493)
4 vs. 3	-0.2070	0.0921	5.05	1	0.0246	0.758 (0.580, 0.990)
5 vs. 3	-0.2136	0.0944	5.12	1	0.0237	0.753 (0.573, 0.989)
Test			χ^2	*df*	*p*	
Overall model evaluation						
Likelihood ratio test			2,185.83	10	<.0001	
Score test			1,907.27	10	<.0001	
Wald test			1,422.82	10	<.0001	
Goodness-of-fit test						
Hosmer & Lemeshow			13.36	8	0.1001	

Table 9. Logistic regression for asthma hospitalizations and individual-level variables, SEP included. *1: Black vs. White; 2: Female vs. Male; 3: 2-4 conditions vs. 1 (asthma only); 4: Adult vs. Child; 5: Public vs. Private. Source: HCUP (2015). β*-values and chi-square statistics are for different estimates than odds ratios.

Table 10 displays results of the final regression model, which includes all variables, both individual and neighborhood-aggregated. The model has very similar betas and odds ratios for the individual characteristics as the previous model. Similarly, having public insurance appears to be a have higher odds of hospitalization than private insurance, there is a large difference by adult status and age, and having additional conditions elevates the odds of hospitalization for asthma. Despite the incorporation of additional neighborhood level variables, race remains a highly significant risk factor for asthma hospitalizations in the study area (OR = 2.602; 95% CI: 2.208, 3.067).

Predictor	B	SE	Wald's χ^2	*df*	*p*	OR (95% CI)
Constant	-0.2150	0.0480	20.09	1	<.0001	NA
Race[1]	0.4782	0.0419	130.20	1	<.0001	2.602 (2.208, 3.067)
Sex[2]	0.4526	0.0337	179.90	1	<.0001	--
Adult[3]	-1.1909	0.0410	843.24	1	<.0001	--
Chronic[4]	0.5994	0.0380	248.51	1	<.0001	3.316 (2.857, 3.849)
Insurance[5]	0.2107	0.0337	39.11	1	<.0001	1.524 (1.336, 1.739)
Adult*Sex[2]	0.2910	0.0337	74.74	1	<.0001	
Adult						4.425 (3.815, 5.133)
Child						1.382 (1.110, 1.719)
SEP						
1 vs. 3	0.0989	0.0713	1.92	1	0.1654	1.026 (0.820, 1.284)
2 vs. 3	0.2627	0.0618	18.08	1	<.0001	1.209 (0.977, 1.496)
4 vs. 3	-0.2100	0.0922	5.19	1	0.0227	0.754 (0.577, 0.985)
5 vs. 3	-0.2245	0.0950	5.58	1	0.0181	0.743 (0.564, 0.977)
Facilities	-0.0117	0.0116	1.0147	1	0.3138	0.988 (0.966, 1.011)
Test			χ^2	*df*	*p*	
Overall model evaluation						
Likelihood ratio test			2,186.85	11	<.0001	
Score test			1,908.03	11	<.0001	
Wald test			1,423.43	11	<.0001	
Goodness-of-fit test						
Hosmer & Lemeshow			14.05	8	0.0626	

Table 10. Logistic regression analysis, all variables included.

1: Black vs. White; 2: Female vs. Male; 3: 2-4 conditions vs. 1 (asthma only); 4: Adult vs. Child; 5: Public vs. Private. Source: HCUP (2015). β-values and chi-square statistics are for different estimates than odds ratios.

CHAPTER 7: DISCUSSION

This chapter will begin with a review and discussion of the hypotheses listed toward the end of Chapter 1, including what the findings summarized in the previous chapter indicate about the hypotheses, and how the findings complement the previous literature.

<u>Hypothesis 1: Hospitalization rates will be higher in the study area's black population than the white population.</u>

This hypothesis has, unfortunately yet unsurprisingly, been shown to be correct. It was expected that black hospitalization rates for asthma would be higher than white rates because this has consistently been shown to be the case at the national level. In this study area, the age-adjusted hospitalization rate for African Americans was 394.08 per 100,000 population, while the white rate was 85.78 per 100,000—a rate ratio of 4.59. This figure is somewhat consistent with previous findings in other studies of asthma severity: Akinbami, LaFleur and Schoendorf (2002) found that, nationally, African Americans with asthma are three times as likely as whites to be hospitalized because of their condition. The report on asthma in Detroit published by the MDHHS (2016) states that in 2013, the rate of hospitalization for black residents of Detroit was about 1.5 times higher than the rate for white residents. A possible explanation for the discrepancy in these rate ratios is that the MDHHS report was confined to the city of Detroit. The rates are lower in the surrounding suburbs, and this is also where the majority of the white residents—and a minority of the black residents—of Metropolitan Detroit live. Therefore, when the suburbs are included in the analysis, the disparities between the white and black hospitalization rates widen.

Hypothesis 2: Hospitalization rates will be higher in areas of low SES than in areas of high SES.

This has also proven true, based on the descriptive statistics displayed in Table 2. For the adults and children, there is a clear gradient in hospitalization rates, with a decrease for every increasing level of SEP. The VLSEP rate is nearly 5 times higher than the VHSEP rate. Similar gradients are also evident in the race-specific SEP rates: from VLSEP to VHSEP ZIP codes, the white rate decreases by about 40%, while the black rate remains almost 3 times higher in VLSEP ZIP codes compared to VHSEP ZIP codes. In both cases, the rate continually, but not linearly, decreases with increasing SEP. Figures 1 and 2 illustrate the spatial pattern of hospitalization rates decreasing with increasing SEP. About half of the ZIP codes in the study area had too few hospitalizations to be able to calculate a stable rate; however, the concentration of these low-count ZIP codes in high-SES areas is revealing in regard to the patterning of hospitalization risk by SES. This relationship between asthma risk and SES is consistent with what has been found previously in Boston (Litonjua, Carey, Weiss, & Gold, 1999), and New York City (Claudio, Tulton, Doucette, & Landrigan, 1999), and current national prevalence statistics (NHIS, 2015); however, this is the first instance in which the Darden-Kamel Index has been used to illustrate these differences across several levels of socioeconomic position.

Hypothesis 3: When adjusting for neighborhood-level SES, the black-white disparity in hospitalization rates will be reduced but not eliminated.

While the black and white rates decline similarly with increasing SEP, it must, of course, be pointed out that the actual rates are markedly different between the two groups. While the black-white rate ratio is reduced relative to the overall rate ratio mentioned above (4.59), at every level of SEP, the black hospitalization rate is much higher than the white rate. Therefore, this hypothesis is accepted. Strikingly, the black hospitalization rate in VHSEP ZIP codes is higher still than the white rate in VLSEP ZIP codes. It is clear from these descriptive statistics alone that while accounting for differences in SES between the black and white population of metropolitan Detroit does account for part of the racial disparity in hospitalization rates, there is still substantial residual inequality which SES cannot explain.

Furthermore, the interaction between SES and race means that the population most adversely affected by severe asthma is the black population in low SES areas. VLSEP ZIP codes are not only the most disproportionately black, but of all groups, the black population in these ZIP codes has, by far, the highest hospitalization rate, accounting for almost half (45%) of all hospitalizations despite constituting just 16% of the total population.

<u>Hypothesis 4: Factors that increase risk of asthma hospitalization will explain a large amount of any black-white variation in hospitalization rates.</u>

Based on the variables included in this analysis, this hypothesis should be rejected. Exploratory bivariate logistic regression models indicate that, without controlling for other factors, African Americans have odds of being hospitalized for asthma 3.3 times higher than whites. Once type of insurance, additional chronic conditions, differences in age and sex, as well neighborhood-level factors like SEP and facility proximity are included in the analysis, the black-white odds ratio decreased to 2.9. This means that these factors explain only a small part of the black-white disparity.

<u>Hypothesis 5: In the parts of the study area that are racially residentially integrated, black and white hospitalization rates will be similar.</u>

Because of the degree of racial segregation in metropolitan Detroit, testing this hypothesis was slightly more difficult than expected. Of the 178 ZIP codes in the study area, only 9 had a sufficient number of black and white hospitalizations (at least 20) to be able to directly compare stable, race-specific rates. Of these 9 ZIP codes, 2 were VLSEP, while the remaining 7 were LSEP. They were also all majority-white and located close to or just outside of the edge of the city of Detroit. In every single one these ZIP codes, the black hospitalization rate was considerably higher than the white hospitalization rate. The lowest rate ratio was 1.85, and the highest was 5.84.

Interpretation of Results

The results of both the descriptive and statistical analyses indicate that race has—in the sense of statistical association—a highly significant effect on risk of asthma hospitalization, even when individual characteristics, such as insurance or having additional chronic conditions, and neighborhood characteristics, such as socioeconomic position or proximity to stationary sources of air pollution, are taken into account. But it is worth reiterating, at this point, that race has no biological basis, and therefore, even though asthma hospitalizations are more likely to occur in African Americans and in people who are sensitive to allergens, being black cannot be considered a risk factor for asthma in the same way that allergic sensitivity might be, because the former carries no inherent risk. Racial difference in this case should be considered a proxy for differences in exposure to certain features of the social and physical environment which can both induce and exacerbate asthma. Since the odds of asthma hospitalization for African Americans remain much higher than for whites even after the aforementioned variables are accounted for means that there are additional factors which, in this study area, are unknown to explain the racial disparities. Their absence is one of the limitations of this study.

Before a discussion of additional limitations, it is also important to note that, while having public health insurance tended to elevate the risk of hospitalization in these statistical analyses, this does not mean that private health insurance is more protective than public insurance in regard to asthma. The quality of public insurance depends heavily upon both the degree to which the government, either state or federal, is willing to cover the costs of asthma treatment for people who are already more likely to be in a vulnerable social position, and the willingness of health professionals to treat people who use this kind of insurance. While it may be preferable to public health insurance in some instances, the private health insurance system in the United States is far from perfect, and the rising costs associated with it mean that a substantial portion of the country goes without insurance—notably, a study by Wilper et al. (2009) found that risk of death among the uninsured is 40% higher than for the rest of the population. In other countries with universal public health insurance systems, there is no such population at elevated risk of death due to lack of insurance.

Given the complex—and, to some degree, still poorly understood—etiology of racial disparities in asthma, it is not possible to include all factors that may contribute to asthma exacerbation in an analysis of this kind; however, information on factors that are known to increase the risk of asthma hospitalization, including certain behavioral factors such as smoking or diet; factors related to quality of medical care, such as access to a primary care physician, or distance to the nearest health service facility; adherence to medication; or housing quality; among others, may have provided a more complete picture of the nature of this issue.

Another possible limitation is that the Darden-Kamel Index used in this study may not fully capture the way in which SES differs between black and white residents. As was mentioned in Chapter 1, the commonly-used measures of SES, including income, occupation, and education, often do not carry the same meaning between racial groups. Education, for example, is widely thought of as an equalizing force between the disadvantaged and advantaged, and while it is certainly necessary, it appears not to be sufficient in this regard: African Americans, as measured by unemployment, are worse off than whites, roughly by a factor of 2, at every level of educational attainment. Differences in wealth illustrate the degree of inequality between African Americans and whites, and, due its intergenerational nature, can provide a sense of the cumulative effect of anti-black racism throughout U.S. history. A 2015 report indicates that after the Great Recession, which hit black families disproportionately hard, the typical black household had about 6 cents of wealth for every dollar owned by the typical white household—this racial wealth gap remains substantial across all levels of education and income (Hamilton, Darity Jr., Price, Sridharan, & Tippett, 2015). These inequities mean that poor white households may be more economically secure than black households with more income and higher educational attainment. By not incorporating this into the analysis, the within-SEP comparisons used here may not accurately capture the economic precarity uniquely experienced by some black households. This may explain part of the residual disparity in hospitalization rates.

There are also several further limitations to this project, some of which stem from only having the ZIP code of residence, as opposed to a census tract or even an address, for the study subjects. This, in turn, necessitated the aggregation of certain variables to the same level. The Darden-Kamel Index, for example, is typically calculated by census tract. Performing the calculations by ZIP code instead captures the same general pattern of increasing SES with distance from the central city; however, for each ZIP code in the study area, there are an average of 6.5 census tracts, and a considerable amount of variation by census tract is lost in the process of aggregation. Additionally, while the terms “neighborhoods” and “ZIP codes” have generally been used interchangeably in this study, ZIP codes often do not always reflect the size of neighborhoods throughout a study area; census tracts are typically believed to more appropriately do this. Additionally, other studies on asthma in Detroit mentioned in Chapter 2 used geocoded addresses for each case of hospitalization, but this data was only available for people on Medicaid. This method provides a more spatially accurate analysis for this population, but, by only using Medicaid records, fails to account for large-scale differences by SES across the study area.

Chapter 3 included a brief discussion of different methods of estimating proximity to sources of ambient air pollution and exposure to actual concentrations of pollution. The method used in this study was simple and did not include actual emissions data or any emissions modeling. The fact that the analysis performed here generally yielded a weak association between asthma hospitalization and the number of facilities surrounding the ZIP code of residence does not mean exposure to certain kinds of air pollution does not increase the risk of asthma hospitalization, since a large body of literature has already shown this to be the case (Guarnieri & Balmes, 2014; Kim, Jahan, & Kabir, 2013; Dick, Doust, Cowie, Ayres, & Turner, 2014). Unfortunately, the degree to which disparities in exposure to pollution—which has been shown to occur in Detroit by multiple other studies—may contribute to disparities in health outcomes in metropolitan Detroit remains unclear. Estimating proximity to pollution is also made more difficult by having to aggregate to the ZIP code: doing so assumes that everybody in a particular ZIP code is equally proximate to the facilities, which, depending on the size of the ZIP code, may be implausible. For example, exposure to pollution emitted by vehicles has been shown to be linked with asthma exacerbation, but this association is typically only present within a few hundred meters of a major road (Gowers, et al., 2012). Because the only geographic identifier in the Healthcare Cost and Utilization Project data about place of residence for each admission was the ZIP code, the relationship between mobile sources of pollution and exacerbation was unlikely to show up in a ZIP-code-level analysis, and for this reason was not included in the analysis.

An additional difficulty with this study is the degree to which the sociodemographic structure of the DMA complicates analysis. In a study area that was less stratified by SES and less racially segregated, it would be easier to make direct comparisons of hospitalization rates by SES and race within a unit of analysis. But even when controlling for ZIP-code SEP the high levels of segregation in metropolitan Detroit mean that white and black residents in ZIP codes that receive the same classification according to the Darden-Kamel Index generally do not live in the same ZIP codes. This complicates the analysis to a certain degree because it means there are differences in social and environmental conditions between ZIP codes which may be, at best, difficult to control for. A within-ZIP-code assessment was attempted in this study, but just 9 of metropolitan Detroit's 178 ZIP codes were sufficiently integrated for such a comparison. Again, a smaller unit of analysis would have also provided a more accurate comparison, as social and environmental conditions will also vary less across a smaller area.

Another limitation of this study was the absence of data on the prevalence of asthma. Because not every person who has asthma needs to visit the hospital, a comparison of the characteristics of the people who were hospitalized to those who were not would be a potentially informative analysis. Additionally, based on this study, it is uncertain whether the large disparities in hospitalization rates are the result of higher prevalence or greater severity of the condition in the African American population. Using previously reported national statistics, it is likely that it is due to a combination of both: African Americans are both more likely to have asthma and, if they do, are more likely to have a severe form of the condition.

A final limitation has to do with the representativeness of the group used for comparison in the logistic regression. People hospitalized for injuries did reflect the race and class structure of the study area population but did not in other ways. For example, a disproportionate share of people from this group were adult and male, so the odds ratios estimated for age and sex in the logistic regression models may be slightly skewed. This is shown specifically by the fact that the hospitalization rate for girls is lower than for boys, but the odds ratio for this category was consistently higher than 1, indicating that relative to those hospitalized for injuries, girls were at higher risk than boys of asthma hospitalization. The odds ratios of women to men may also be slightly higher than it truly is for the same reason, but the descriptive analysis shows that women have a higher risk of hospitalization for asthma than men. In fact, a striking statistic that has not been noted until this point is the magnitude of the hospitalization rate for black women in low-SES areas. As was noted earlier in this study, asthma is more common in boys than in girls, but also more common in women than in men. Because sex, unlike race or SES, does have a biological basis, it is more plausible that these differences occur at least in part due to biological differences associated with development. But this fact also means that in a social context such as Detroit—or much of the United States, for that matter—in which black people and people of low SES have elevated risk of asthma, it is black women in areas of low SES who carry the greatest asthma burden. As has already been mentioned, the overall, age-adjusted black hospitalization rate in the study area is 394.08 per 100,000 population. In VLSEP ZIP codes, this figure increases to 458.0 per 100,000, but for black women specifically, it is 639 per 100,000. Even in LSEP ZIP codes, the hospitalization rate for black women is 477 per 100,000.

Policy Implications

Whenever such large disparities are shown to exist, it is important to know if effective policy measures and public health interventions are in place to reduce or eliminate the inequities. There is certainly evidence that certain strategies can help to improve outcomes for both children and adults who have asthma. According to a systematic review by Bravata et al. (2009), strategies such as self-management, patient education and provider education have been shown to decrease the severity of pediatric asthma, as measured by decreased number of days with symptoms, fewer days of missed school, and/or fewer uses of health care facilities. Additionally, in situations in which more than one of these strategies were implemented, statistically significant improvements were more likely to occur. In a separate systematic review of the efficacy of educational strategies, Guevara, Wolf, Grum & Clark (2003) found that these strategies tended to be most effective among children and adolescents with moderate to severe asthma. These kinds of strategies also appear to be the preferred method for Healthy People, a U.S. government initiative that sets ten-year objectives for improved health outcomes. Among its objectives for the year 2020 specifically relating to asthma are the reduction of asthma-related premature deaths and hospitalizations and emergency department visits that result from this condition. Some of the interventions that are highlighted in the Healthy People's 2020 objectives for asthma and other respiratory diseases also tend to focus on strategies like patient education (Peytremann-Bridevaux, Arditi, Gex, Bridevaux, & Burnand, 2015), exercise training (Dale, McKeough, Troosters, Bye, & Alison, 2015), and culture-specific programs for minority patients (McCallum, Morris, Brown, & Chang, 2017). These programs certainly have a role in lowering the overall asthma burden not only in Detroit or the United States, but also in many other parts of the world as the global prevalence of asthma continues to rise and as our understanding of the condition improves.

But intervention strategies like patient education or self-management which focus on individual factors can only do so much. This is because, as had been discussed at length earlier in this thesis, chronic conditions like asthma have complex etiology, and are affected not only by individual behaviors, but also by structural and environmental factors. Health disparities are rooted in various forms of structural inequality, which ultimately leave certain segments of the population more vulnerable to negative health outcomes compared to others who are not exposed to these structures. As long these social inequalities remain, individual-level intervention strategies, especially those that primarily focus on disease management rather than prevention, can only reduce asthma prevalence to a certain extent but will probably not eliminate the racial, ethnic and geographic disparities.

CHAPTER 8: CONCLUSION

This study had several aims. The first was to determine the magnitude of racial inequality in hospitalization rates for asthma in metropolitan Detroit, both before and after accounting for differences in socioeconomic status. The second was to identify risk factors for hospitalization and the degree to which they help to explain the racial disparity in hospitalization rates. It has been shown that there are substantial disparities in the frequency of hospitalization for asthma between African Americans and whites in the study area, and that differences in the type of insurance, the presence of additional chronic conditions, neighborhood-level SES and proximity to stationary sources of air pollution are able to explain only a small part of this disparity.

Contributions of This Study

This study has contributed to the research on racial health disparities by attempting to integrate concerns of environmental justice into the larger set of possible explanatory factors. Connecting environmental justice concerns to health outcomes is a second valuable contribution of this study. Both of these contributions are potentially important avenues for future research on the ways in which social inequality leads to disparate health outcomes.

Future Research

Future research in this area should improve upon some of the limitations listed in the previous chapter. Perhaps most importantly, the use of data on asthma prevalence in addition to hospitalizations would be a valuable future step by revealing risk factors for asthma exacerbation in people who are known to have the condition. Secondly, the use of more sophisticated methods for determining exposure to air pollution would provide a more intricate understanding of the link between environmental inequality and health outcomes. This improvement would also necessitate a finer geographical scale of analysis.

Racial health disparities are rooted in larger structures of racial hierarchy which render black Americans, as well as other people of color, more exposed and susceptible to disease. This study has only focused on black-white differences in metropolitan Detroit because these two groups constitute the overwhelming majority of the study area's population, but it is also certainly necessary to address the poor health outcomes which occur in other nonwhite groups, including the Latino and Native populations of the United States. For example, as was mentioned in Chapter 1, the prevalence of asthma in Puerto Ricans is 13.7%—slightly higher than even the prevalence in African Americans. Future research should also study the interactions between previous chronic conditions as lower SEP may cause these conditions among blacks; whereas behavioral factors may explain the higher chronic disease burden among whites.

Despite the lofty ideals offered in its founding documents, structures of discrimination and disadvantage in the United States have been reinforced and reinvented since its inception. It should go without saying that these disparities are deeply unjust and unacceptable, yet their consistency and magnitude indicate the constant necessity of this assertion.

BIBLIOGRAPHY

BIBLIOGRAPHY

(2015). *2011-2015 American Community Survey Five-Year Estimates.* U.S. Census Bureau.

Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: pathways and policies. *Health Affairs, 21*(2), 60-76.

Akinbami, L. J., LaFleur, B. J., & Schoendorf, K. C. (2002). Racial and income disparities in childhood asthma in the United States. *Ambulatory Pediatrics, 2*(5), 382-287.

Akinbami, L. J., Moorman, J. E., Bailey, C., Zahran, H. S., King, M., Johnson, C. A., & Liu, X. (2012). *Trends in asthma prevalence, health care use, and mortality in the United States, 2001-2010.* Hyatsville, MD: National Center for Health Statistics.

Akinbami, L. J., Moorman, J. E., Garbe, P. L., & Sondik, E. J. (2009). Status of Childhood Asthma in the United States, 1980-2007. *Pediatrics*, S131-S145.

Akinbami, L. J., Simon, A. E., & Rossen, L. M. (2015). Changing trends in asthma prevalence among children. *Pediatrics, 137*(1).

Bakerly, N. D., Moore, V. C., Vellore, A. D., Jaakkola, M. S., Robertson, A. S., & Burge, P. S. (2008). Fifteen-year trends in occupational asthma: data from the Shield surveillance scheme. *Ocupational Medicine*, 169-174.

Barnett, S. L., & Nurmagambetov, T. A. (2011). Costs of asthma in the United States: 2002-2007. *Journal of Allergy and Clinical Immunology*, 145-152.

Beasley, R., Crane, J., FRACP, Lai, C. K., & Peirce, N. (2000). Prevalence and etiology of asthma. *Journal of Allergy and Clinical Immunology*, S466-S472.

Brulle, R. J., & Pellow, D. N. (2006). Environmental justice: human health and evironmental inequliaties. *Annual Review of Public Health, 27*, 103-124.

Bullard, R. D. (1983). Solid waste sites and the Black Houston Community. *Sociological Inquiry, 53*(2-3), 273-288.

Bullard, R. D. (2000). *Dumping in Dixie: Race, Class and Environmental Inequality (3rd ed.).* Boulder, CO: Westview Press.

Canino, G., McQuaid, E. L., & Rand, C. S. (2009). Addessing asthma health disparities: a multilevel challenge. *Journal of Allergy and Clinical Immunology, 123*(6), 1209-1217.

Chakraborty, J., & Maantay, J. (2011). Proximity Analysis for Exposure Assessment in Environmental Health Justice Research. In J. Maantay, & S. McLafferty (Eds.), *Geospatial Analysis of Environmental Health* (pp. 111-138). Springer.

Chetty, N. (2009). Asthma exacerbations--a review. *Current Allergy & Clinical Immunology, 22*(2), 75-78.

Cisternas, M. G., Blanc, P. D., Yen, I. H., Katz, P. P., & Earnest, G. (2003). A comprehensive study of the direct and indirect costs of adult asthma. *Journal of Allergy and clinical Immunology*, 1212-1218.

Claudio, L., Tulton, L., Doucette, J., & Landrigan, P. J. (1999). Socioeconomic Factors and Asthma Hospitallization Rates in New York City. *Journal of Asthma*, 343-350.

Collins, T. W., Grineski, S. E., & Morales, D. X. (2017). Sexual orientation, gender, and environmental injustice: unequal carcinogenic air pollution risks in Greater Houston. *Annals of the Association of American Geographers, 107*(1), 72-92.

Dale, M., McKeough, Z., Troosters, T., Bye, P., & Alison, J. (2015). Exercise training to improve exercise capacity and quality of life in people with non-malignant dust-related respiratory diseases. *Cochrane Database of Systematic Reviews*(11).

Darden, J. T., Rahbar, M., Jezierski, L., Li, M., & Velie, E. (2010). The measurement of neighborhood socioeconomic characteristics and black and white residential segregation in Metropolitan Detroit: implications for the study of social disparities in health. *Annals of the Association of American Geographers, 100*(1), 137-158.

DeGuire, P., Cao, B., Wisnieski, L., Strane, D., Wahl, R., Lyon-Callo, S., & Garcia, E. (2016). *Detroit: The Current Status of the Asthma Burden.* Michigan Department of Health and Human Services.

Dick, S., Doust, E., Cowie, H., Ayres, J. G., & Turner, S. (2014). Associations between environmental exposures and asthma control and exacerbations in young children: a systematic review. *BMJ Open*, 4.

Downey, L. (2005). The unintended significance of race: environmental racial inequality in Detroit. *Social Forces, 83*(3), 937-952.

Downey, L. (2006). Environmental racial inequality in Detroit. *Social Forces, 85*(2), 771-796.

Eyles, J., Finkelstein, N., Kanaroglou, P., Brook, J. R., Jerrett, M., Giovis, C., & Burnett, R. T. (2001). A GIS-environmental justice analysis of particulate air pollution in Hamilton, Canada. *Environment and Planning A*, 955-973.

Fisher, J. B., Kelly, M., & Romm, J. (2006). Scales of environmental justice: combining GIS and spatial analysis for air toxics in West Oakland, California. *Health and Place*, 701-714.

Friedman, M. S., Powell, K. S., Hutwagner, L., Graham, L. M., & Teague, G. W. (2001). Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *Journal of the American Medical Association, 285*(7), 897-905.

Gaskin, D. J., Dinwiddie, G. Y., Chan, K. S., & McCleary, R. (2012). Residential segregtaion and disparities in health care utilization. *Medical Care Research and Review, 69*(2), 158-175.

Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). “Weathering” and Age Patterns of Allostatic Load Scores. *American Journal of Public Health, 96*(5), 826-833.

Godard, P., Chanez, P., Siraudin, L., Nicoloyannis, N., & Duru, G. (2002). Costs of asthma are correlated with severity: a 1-yr prospective study. *European Respiratory Journal*, 61-67.

Gowers, A. M., Cullinan, P., Ayres, J. G., Anderson, H. R., Strachan, D. P., Holgate, S. T., . . . Maynard, R. L. (2012). Does outdoor air pollution indice new cases of asthma? Biological plausibility and evidence; a review. *Respirology, 17*, 887-889.

Grady, S. C. (2016). Sociospatial Epidemiology: Segregation. In A. B. Lawson , S. Banerjee, R. P. Haining, & M. D. Ugarte, *Handbook of Spatial Epidemiology* (pp. 609-626). Boca Raton, FL: CRC Press.

Grady, S. C., & Darden, J. T. (2012). Spatial methods to study local racial residential segregation and infant health in Detroit, Michigan. *Annals of the Association of American Geographers, 102*(5), 922-931.

Grady, S. C., & Enander, H. (2009). Geographic analysis of low birthweight and infant mortality in Michigan using automated zoning methodology. *International Journal of Health Geographics, 8*(10).

Grineski, S. E. (2007). Incorporating health outcomes into environmental justice research: the case of children's asthma and air pollution in Phoenix, Arizona. *Environmental Hazards, 74*, 360-371.

Grineski, S. E., Collins, T. W., Chakraborty, J., & McDonald, Y. J. (2012). Environmental health injustice: exposure to air toxics and children's respiratory hospital admissions in El Paso, Texas. *The Professional Geographer, 65*(1), 31-46.

Grineski, S., Bolin, B., & Boone, C. (2007). Criteria air pollution and marginalized populations: environmental inequity in metropolitan Phoenix, Arizona. *Social Science Quarterly, 88*(2), 535-554.

Guarnieri, M., & Balmes, J. (2014). Outdoor air pollution and asthma. *The Lancet, 383*, 1581-1592.

Hamilton, D., Darity Jr., W., Price, A. E., Sridharan, V., & Tippett, R. (2015). *Umbrellas don't make it rain: why studying and working hard isn't enough for Black Americans.* New York: The New School.

Haselkorn, T., Lee, J. H., Mink, D. R., & Weiss, S. T. (2008). Racial disparities in asthma-related health outcomes in severe or difficult-to-treat asthma. *Annals of Allergy, Asthma & Immunology, 101*, 256-263.

Holifield, R. (2001). Defining environmental justice and environmental racism. *Urban Geography*, 78-90.

Jaakkola, M. S., Piipari, R., Jaakkola, N., & Jaakkola, J. K. (2003). Environmental tobacco smoke and adult-onset asthma: a population-based incident case-control study. *American Journal of Public Health*, 2055-2060.

Kim, K., Jahan, S. A., & Kabir, E. (2013). A review on human health perspective of air pollution with respect to allergies and asthma. *Environment International, 59*, 41-52.

Lau, S., Nickel, R., Niggemann, B., Gruber, C., Sommerfeld, C., Illi, S., . . . MAS Group. (2002). The development of childhood asthma: lessons from the German Multicentre Allergy Study (MAS). *Paediatric Respiratory Reviews*, 265-272.

Lee, S., & Mohai, P. (2011). Racial and socioeconomic assessments of neighborhoods adjacent to small-scale brownfield sites in the Detroit region. *Environmental Practice, 13*(4), 340-353.

Li, S., Batterman, S., Wasilevich, E., Elasaad, H., Wahl, R., & Mukherjee, B. (2011a). Asthma exacerbation and proximity of residence to major roads: a population-based matched case-control study among the pediatric Medicaid population in Detroit, Michigan. *Environmental Health, 10*(34).

Li, S., Batterman, S., Wasilevich, E., Wahl, R., Wirth, J., Su, F., & Mukherjee, B. (2011b). Association of daily asthma emergency department visits and hospital asmissions with ambient air pollutants among the pediatric Medicaid population in Detroit: time-series and time-stratified case-crossover analyses with threshold effects. *Environmental Research, 111*, 1137-1147.

Litonjua, A. A., Carey, V. J., Weiss, S. T., & Gold, D. R. (1999). Race, Socioeconomic Factors, and Area of Residence Are Assoicated With Asthma Prevalence. *Pediatric Pulmonology*, 394-401.

Maantay, J. (2007). Asthma and air pollution in the Bronx: methodological and data considerations in using GIS for environmental justice and health research. *Health & Place, 13*, 32-56.

Massey, D. S., & Denton, N. A. (1993). *American Apartheid.* Harvard University Press. Retrieved 3 12, 2018

Massey, D. S., & Denton, N. A. (1993). *American apartheid: segregation and the making of the underclass.* Cambridge, MA: Harvard University Press.

Massey, D. S., & Rugh, J. S. (2014). Segregation in Post-Civil Rights America: Stalled Integration or End of the Segregated Century? *Du Bois Review, 11*(2), 205-232. Retrieved 3 12, 2018, from https://ncbi.nlm.nih.gov/pubmed/26966459

McCallum, G. B., Morris, P. S., Brown, N., & Chang, A. B. (2017). Culture-specific programs for children and adults from minority groups who have asthma. *Cochrane Database of Systematic Reviews*(8).

Moody, H. A., Darden, J. T., & Pigozzi, B. W. (2016a). The Relationship of neighborhood socioeconomic differences and racial residential segregation to childhood blood lead levels in Metropolitan Detroit. *Journal of Urban Health, 93*(5), 820-839.

Moody, H. A., Darden, J. T., & Pigozzi, B. W. (2016b). The racial gap in childhood blood lead levels related to socioeconomic position of residence in Metropolitan Detroit. *Sociology of Race and Ethnicity, 2*(2), 200-218.

Morello-Frosch, R. (2002). Discrimination and the political economy of environmental inequality. *Environment and Planning C: Government and Policy*, 447-496.

(2015). *National Health Interview Survey.* Baltimore, MD: National Center for Health Statistics.

Palmer, L. J., Celedon, J. C., Weiss, S. T., Wang, B., Fang, Z., & Xu, X. (2002). Ascaris lumbricoides Infection is Assocatied with Increased Risk of Childhood Asthma and Atopy in Rural China. *American Journal of Respiratory and Critical Care Medicine*, 1489-1493.

Pellow, D. (2000). Environmental inequality formation--toward a theory of environnmental injustice. *American Behavioral Scientist*, 581-601.

Peng, C. J., Lee, K. L., & Ingersoll, G. M. (2002). An introduction to logistic regression analysis and reporting. *The Journal of Educational Research, 96*(1), 3-14.

Peytremann-Bridevaux, I., Arditi, C., Gex, G., Bridevaux, P., & Burnand, B. (2015). Chronic disease management programmes for adults with asthma. *Cochrane Database of Systematic Reviews*(5).

Pickett, K. E., & Pearl, M. (2001). Multilevel analyses of neighborhood socioeconomic context and health outcomes: a critical review. *Journal of Epidemiology and Community Health, 55*, 111-122.

Pulido, L. (1996). A critical review of the methodology of environmental racism research. *Antipode*, 142-159.

Richardson, L. D., & Norris, M. (2010). Access to health and health care: how race and ethnicity matter. *Mount Sinai Journal of Medicine, 77*, 166-177.

Riedler, J., Eder, W., Oberfeld, G., & Schreuer, M. (2000). Austrian children living on a farm have less hay fever, asthma and allergic sensitization. *Clinical and Experimental Allergy*, 194-200.

Rugh, J., & Massey, D. (2014). Segregation in post-Civil Rights America: stalled integration or end of the segregated century? *Du Bois Review: Social Science Research on Race, 11*(2), 205-232.

Saha, R., & Mohai, P. (2005). Historical context and hazardous waste facility siting: understanding temporal patterns in Michigan. *Social Problems, 52*(4), 618-648.

Schildcrout, J. S., Sheppard, L., Lumley, T., Slaughter, J. C., Koenig, J. Q., & Shapiro, G. G. (2006). Ambient air pollution and sthma exacerbations in children: an eight-city analysis. *American Journal of Epidemiology, 164*(6), 505-517.

Sears, M. R., Burrows, B., Flannery, E. M., Herbison, G. P., Hewitt, C. J., & Holdaway, M. D. (1991). Relation between airway responsiveness and serum IgE in children with asthma and in apparently normal children. *The New England Journal of Medicine*, 1067-1071.

Sicotte, D., & Swanson, S. (2007). Whose risk in Philadelphia? Proximity to unequally hazardous industrial facilities. *Social Science Quarterly*, 515-534.

Smith, C. L. (2007). Economic deprivation and environmental inequality in postindustrial Detroit: a comparison of landfill and superfund site locations. *Organization and Environment, 20*(1), 25-43.

Subbarao, P., Mandhane, P. J., & Sears, M. R. (2009). Asthma: epidemiology, etiology and risk factors. *Canadian Medical Association Journal, 181*(9), 598-604. Retrieved 10 2, 2017, from http://cmaj.ca/content/181/9/e181.full.pdf+html

Sugrue, T. J. (2005). *Origins of the urban crisis: race and inequality in postwar Detroit.* Princeton, NJ: Princeton University Press.

Szasz, A., & Meuser, M. (1997). Environmental inequalities: literature review and proposals for new directions in research and theory. *Current Sociology*.

Takkouche, B., Gonzalez-Barcala, F.-J., Etminan, M., & FitzGerald, M. (2008). Exposure to furry pets and the risk of asthma and allergic rhinitis. *Allergy*, 857-864.

Williams, D. R., & Collins, C. (2001). Racial residential segregation: a fundamental cause of disparities in health. *Public Health Reports, 116*, 404-416.

Williams, D. R., & Jackson, P. B. (2005). Social sources of racial disparities in health. *Health Affairs, 24*(2), 325-334.

Wilper, A. P., Woolhandler, S., Lasser, K. E., McCormick, D., Bor, D. H., & Himmelstein, D. U. (2009). Health insurance and mortality in US adults. *American Journal of Public Health, 99*(12), 2606-2610.

Wright, R. J., & Subramanian, S. V. (2007). Advancing a multilevel framework for epidemiologic research on asthma disparities. *Chest, 132*(5), 757S-763S.

Wu, Y., & Batterman, S. (2006). Proximity of schools in Detroit, Michigan to automobile and truck traffic. *Journal of Exposure Science and Environmental Epidemiology, 16*(5), 457-470.