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# Toxic Air Discharge and Infant Mortality: Effects of Community Size and Socioeconomics

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*Walden University*

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# Walden University

College of Health Sciences

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Walden University  
2019

Abstract

Toxic Air Discharge and Infant Mortality: Effects of Community Size and

Socioeconomics

by

Khabira H. Salter

MS, Indiana Wesleyan University, 2011

BS, Wilberforce University, 2008

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health Epidemiology

Walden University

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## Abstract

Living in counties where manufacturers release environmental toxins, such as those tracked by the Environmental Protection Agency's (EPA) toxic release inventory (TRI), may elevate infants' health risks. Because infant mortality (IM) is a strong indicator of a population's health status, it is an important topic in public health research. The purpose of this research was to examine the potential relationships between IM, community size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. The dependent variable was IM per 1,000 live births in a given community for each of the 3 years included in this analysis (1987, 1995, and 2004). The independent variables included county size and factors related to mother's SES (education, age, ethnicity, and marital status). The theoretical framework consisted of Mosley and Chen's framework for exploring child survival. Archival, publicly available data were pulled from (a) the EPA's TRI data, and (b) linked birth and infant death files from the National Center for Health Statistics. The researcher followed a quantitative, retrospective cross-sectional design and conducted 3 linear regression models to test the research questions. Results indicated that an increase in community size was significantly associated with an increase in IM. Regarding the relationships between IM and the 4 different maternal characteristics (education, age, ethnicity, and marital status) included in the analysis, findings were mixed for the 3 years examined. Despite these unexpected findings, the overall results from this investigation, when considered alongside findings from previous research on IM, indicate that policy changes and interventions are needed to reduce socioeconomic disparities in IM, and to save the lives of more infants.

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## Dedication

I dedicate my dissertation with love to my children, Michael, Reyhan, and Sabirah, for they have been my true encouragement throughout the entire doctorate program and have given me the optimum support needed to attain my doctorate degree. I love you all tremendously.

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## Chapter 1: Introduction to the Study

### **Introduction**

Infant mortality (IM) is a strong indicator of a population's health status (Organisation for Economic Co-operation and Development [OECD], 2010), making it an important topic in public health research. Exposure to toxic air pollution is correlated with an increased risks for many serious health conditions in adults, including cardiovascular disease, lung cancer, and respiratory infection (Tanaka, 2015). In addition, air pollution can create oxidative stress and chronic inflammation, which may influence pregnancy and infant outcomes (Savitz et al., 2013). The negative effects of toxic air pollution on infants and fetuses have been examined in a number of studies (Agarwal, Banterghansa, & Bui, 2010; Arceo et al., 2015; Bobak, 2000; Bowatte et al., 2015; Carbajal-Arroyo et al., 2011; Cesur, Tekin, & Ulker, 2016; Currie & Schmieder, 2009; Ezziane, 2013; Greenstone & Chay, 2003; Kaiser et al., 2004; Knittel, Miller, & Sanders, 2011; Loomis, Castillejos, Gold, McDonnell, & Borja-Aburto, 1999; MacIntyre et al., 2014; Proietti et al., 2013; Sartorius & Sartorius, 2014; Van Rossem et al., 2015; Woodruff, Darrow, & Parker, 2008; Woodruff, Grillo, & Schoendorf, 1997). Despite the quantity of scholarship on the relationship between air pollution and infant health, findings are largely inconsistent (Romieu et al., 2004). Krzysanoski and Kuna-Dibbert (2005) suggested that the reason for such inconsistencies may be differences in the ways infant exposure to toxins and health outcomes are assessed. Further, much of the existing literature lacks concurrent examination of important factors that may modify the relationship between IM and exposure to airborne toxins, such as socioeconomic status

(SES) and community size. Because IM is rarely caused by a single factor but is the result of a variety of factors working in concert (Mosley & Chen, 1984), a more dynamic approach to IM research is needed to shed new light on the relationship between IM and exposure to toxic air pollution. Implications for positive social change will inform policy makers of factors that can be addressed to reduce IM and health outcome disparities that are based on SES and county size.

In this research, I addressed shortcomings in the existing literature by focusing on the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. The Toxic Release Inventory (TRI) is an Environmental Protection Agency (EPA) database that contains data from manufacturing facilities that produce or process more than 25,000 pounds of toxic chemicals each year (EPA, 2010). Manufacturers must report releases that occur from spills and routine emissions released into land, water, or sewage treatment plants (Hendryx & Fedorko, 2011) to the EPA. In the current study, toxic air releases included any of the 595 chemicals specified by the TRI.

This chapter provides an introduction to the current investigation, beginning with a discussion of the background of the problem, followed by the problem statement and purpose of the study. Next, the research questions and theoretical framework are presented. The nature of the study is described, as are definitions, assumptions, delimitations, limitations, and significance. The chapter closes with a short summary.

### **Background**

Since the 18th century, scientists have explored links between the environment and health outcomes (Braud, Noeur, & Lamar, 2011). In the health science literature, the

general consensus is that fetuses and infants are more susceptible to toxic pollutants than are older children and adults (Agarwal et al., 2010). A significant body of research indicates that air pollution, in particular, can have a strong, negative effect on infant health (Agarwal et al., 2010; Bobak, 2000; Carbajal-Arroyo et al., 2011; Currie & Schmieder, 2009; Ezziane, 2013; Greenstone & Chay, 2003; Kaiser et al., 2004; Loomis et al., 1999; Woodruff et al., 2008; Woodruff et al., 1997). For example, Greenstone and Chay (2003) found that the quantity of total suspended particulates (TSPs) was significantly related to IM in the United States. Currie and Neidell (2005) reported a strong relationship between carbon monoxide exposure and IM. Currie and Schmieder (2009) found that exposure to TRI air releases correlated with adverse health outcomes among infants. Agarwal et al. (2010) reported that toxic air pollutants tracked in the TRI database, especially chemicals known or suspected of being carcinogenic, were strongly linked to IM and other poor health outcomes.

Living in counties where manufacturers release environmental toxins, such as those tracked by the EPA's (2010) TRI, may increase an infant's exposure to air pollutants. Research indicates that such increased proximity may elevate adverse health risks. For example, Braud et al. (2011) found that proximity to toxic release sites was related to premature delivery among a sample of women in Tennessee. In another study, Suarez, Brender, Langlois, Zhan, and Moody (2007) reported that Texan mothers living near sites of toxic air releases were more likely to give birth to infants with neural tube defects.

When examining the relationship between IM and proximity to toxic air releases, it is important to consider factors that may act as modifiers. Results from previous



investigations indicate that one such factor may be SES. For example, in a study on the relationship between exposure to air pollution and respiratory-related IM in Mexico City, Carbajal-Arroyo et al. (2011) found that increased exposure to air pollution was linked with adverse health outcomes among infants of low to medium SES. Similarly, Romieu et al. (2004) found that infants of low SES might be more vulnerable to the effects of air pollution and death from respiratory illness. In another study, Padilla et al. (2013) found that lower neighborhood SES was linked with elevated IM among populations in France. Another potential modifier is community size, but a dearth of scholarship exists on the potential effects of community size on IM related to exposure to toxic air pollutants.

IM is usually the result of many factors working together (Mosley & Chen, 1984), so it is important for researchers to examine variables that may influence the effects of exposure to airborne toxins and IM. Such a dynamic approach may shed new light on the relationship between IM and exposure to toxic air pollution and provide leaders with additional information needed to enact measures and policies to protect the lives and well-being of infants exposed to such pollutants. In the current investigation, I addressed shortcomings in the existing research, such as lack of examination of factors that may modify the relationship and focus on the potential relationships between IM, exposure to air pollution, county size, and factors related to mothers' SES.

### **Problem Statement**

The problem with existing research on the relationship between exposure to toxic air pollution and IM rates is that much of it fails to concurrently examine multiple factors that may influence IM. As Mosley and Chen (1984) explained, infant death is rarely the result of a single factor; but rather, results from a variety of factors. Thus, to provide a

more holistic understanding of the relationship between IM and exposure to toxic air pollution, I examined the potential influence of community size and factors related to mothers' SES. This allowed me to explore whether community size or SES factors modified the relationship between IM and exposure to toxic air pollution.

### **Purpose of the Study**

My purpose in this research was to examine the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. The dependent variable was IM, and independent variables included county size, with two levels of population size (250,000 to 500,000 or > 500,000), and variables related to mother's SES, including mother's (a) education level, (b) age, (c) ethnicity, and (d) marital status. Specifically, I investigated whether community size and SES-related factors had any modifying effects on the relationship between IM and residence in counties where more than 25,000 pounds of annual TRI toxic air releases occur.

### **Research Questions and Hypotheses**

**RQ1.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and community size?

**H<sub>10</sub>.** Community size (small vs. large) is not a predictor of IM in the population of counties where more than 25,000 pounds of annual toxic air releases occur.

**H<sub>1A</sub>.** Community size (small vs. large) is a predictor of IM in a population of counties where more than 25,000 pounds of annual toxic air releases occur.

**RQ2.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and the following maternal characteristics (a) education level, (b) age, (c) ethnicity, and (d) marital status?

**H<sub>20</sub>.** Maternal characteristics are not predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

**H<sub>2A</sub>.** Maternal characteristics are predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

### **Theoretical Framework**

Mosley and Chen's (1984) framework for exploring child survival served as the theoretical framework for the current study. This framework was based on the supposition that multiple factors must be considered to develop a comprehensive understanding of IM predictors. Typically, research on population mortality is examined through either a medical or social lens. Medical research may focus on the biology of disease processes, such as disease transmission, dietary practices, and environmental contamination. Social scientists, on the other hand, may examine IM via patterns of mortality across populations and commonly measured social determinants, such as income level and maternal education (Mosley & Chen, 1984). Although existing medical and social research provides important insights into IM, the disciplinary compartmentalization creates some superficiality, especially in terms of factors related to SES.

Mosley and Chen (1984) argued that different disciplines must be used to develop social policies and medical interventions to improve infant survival rates. IM research typically falls into a single discipline (social science, medical, epidemiological, or

nutrition); however, the exploration of IM through a single disciplinary lens precludes a holistic understanding of the multitude of factors that may influence IM. Thus, Mosley and Chen created a framework that incorporated multiple disciplines and methodologies into a single, coherent framework. The premise of the scholars' work that most strongly aligned with the current research is that the reduction of survival in any society is the result of a concert of social, biological, economic, and environmental forces. Because IM is rarely the result of a single, isolated factor, a cross-disciplinary approach is needed in IM research. The current investigation accomplished this by investigating the potential relationships between IM and environmental (proximity to toxic air releases), spatial (community size), and sociodemographic (mother's education level, age, ethnicity, and marital status) variables.

### **Nature of the Study**

The nature of the current investigation was quantitative, and I followed a retrospective cross-sectional design. Quantitative methods allow researchers to predict, confirm, or test theories of phenomena (Cooper & Schindler, 2003) and are appropriate for examining the statistical significance of relationships between variables (Swanson & Holton, 2005). In contrast, qualitative investigations allow themes and ideas to inductively emerge from data, rather than through statistical tests of predetermined variables. Qualitative studies are ideal when researchers have smaller samples and desire to obtain in-depth, rich data rather than statistically significant, generalizable data from larger samples. Because of the small sample sizes, qualitative findings cannot be generalized to larger populations, whereas quantitative findings can.

Because my purpose in the current study was to examine the statistical significance of relationships between predetermined variables, a quantitative method was most appropriate. This type of investigation allowed for the determination of statistical certainty while examining relationships across a larger U.S. population. In terms of design, I selected a retrospective, cross-sectional design to explore the relationships between IM, county size, and factors related to mothers' SES. The dependent variable was IM per 1,000 live births in a given community for 3 years included in this analysis (1987, 1995, and 2004). The independent variables related to mother's SES included mother's education, age, ethnicity, and marital status. Another independent variable of interest was community size, defined as population size 250,000 to 500,000 or more than 500,000.

I drew a purposive sample from data available on the EPA's website, which provides TRI database information to the public. I downloaded SES data from the website for the National Bureau of Economic Research, and obtained linked birth-death data from the NCHS website. Once I obtained data from their respective websites, I organized all information into a spreadsheet and matched up data by county. Once organized, I uploaded the data into SPSS for analysis. Data analysis consisted of a three separate linear regression models; each model tested both of the research questions for one year of data. I used IBM SPSS Statistics 25 software for both descriptive and inferential analysis. I set a 0.05 level of significance for all inferential testing.

## Definitions

***Air pollution.*** Air pollution consists of a mix of particulate matter (PM), liquids, and gases released into the atmosphere through human activities, such as combustion and manufacturing (Backes et al., 2013).

***Infant mortality.*** The number of infant deaths per 1,000 live births in a county, for a given year.

***Particulate matter.*** Particulate matter are atmospheric substances present in liquid, solid, or semivolatile form (Backes et al., 2013, p. 48). Relationships exist between exposure to PM and a variety of human health conditions, including respiratory- and cardiovascular-related mortality and morbidity (Adar et al., 2014).

***Preterm birth.*** Preterm birth describes that which takes place prior to 37 weeks of gestation (Malley et al., 2017).

***Toxic release inventory.*** A database that began under the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 (EPA, 2010), which requires manufacturers to report releases of more than 25,000 pounds of any of the 595 specified chemicals included in the TRI list. Facilities included in the TRI database include a variety of chemical, oil, gas, and paper manufacturers (Johnson, Ramsey-White, & Fuller, 2016).

## Assumptions

The main assumption inherent to the current investigation was that all study data had been accurately entered into their respective databases. I also assumed that all manufacturers that release toxic air pollution reported it honestly and accurately. It is possible that some manufacturers that release at least 25,000 pounds of toxins each year

do not report to the EPA; however, the execution of the current investigation required the assumption that they do.

### **Scope and Delimitations**

The current investigation was subject to delimitations. First, the scope was limited to an examination of the potential effects of airborne toxins. Although other types of environmental pollution, such as water and land releases, may affect IM, I examined only the effects of toxic air releases. The scope was also limited by the quantity of air pollution reported. The TRI only contains data from manufacturers that release at least 25,000 pounds of toxic chemicals into the air each year. Manufacturers that release less than 25,000 pounds are not required to report these releases, although it is certainly possible that such releases have a negative effect on the health of surrounding populations. In addition, I included only data from counties that have populations of at least 250,000; thus, smaller, more rural communities were not examined. Finally, my decisions related to the method and theoretical framework represent delimitations.

### **Limitations**

The current study had limitations. First, I conducted this research with financial constraints that prevented the execution of a cohort study, which may be a preferred design to examine the relationships between study variables. A prospective cohort study is a desirable study design for sociological and environmental investigations (Manolio, Bailey-Wilson, & Collins, 2006).

The current investigation was also limited to the accuracy of information entered into the databases from which the study dataset were compiled. Although these databases undergo rigorous quality checks, it is possible that human errors may occur during the

data entry process. The periods for which study data were available presented additional limitations. The county of residence for infants ceased to be recorded after 2004 to protect the privacy of families. In addition, county residence data were not collected between the years of 1992 and 1994. Thus, available data were limited to the years of 1987 to 1991 and 1995 to 2004.

### **Significance**

In this study, I provided an original contribution by examining variables that had not been analyzed together by previous researchers. Although an ample body of research supports the positive association between IM and exposure to toxic air pollution (Agarwal et al., 2010; Bobak, 2000; Carbajal-Arroyo et al., 2011; Currie & Schmieder, 2009; Ezziane, 2013; Greenstone & Chay, 2003; Kaiser et al., 2004; Loomis et al., 1999; Woodruff et al., 2008; Woodruff et al., 1997), researchers have failed to investigate the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur.

### **Social Change Implications**

The current research has significant social implications, because I have shed light on factors that policymakers may address to reduce IM and health outcome disparities based on SES and county size. For example, results indicated that a higher IM rate was indicated for infants born to women with certain SES characteristics; this may serve as an indication that certain infant populations need additional policies or health guidelines to protect them.



## **Summary**

My aim in this current investigation was to address shortcomings in the existing research on IM and exposure to toxic air pollution by focusing on the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. In this chapter, I provided an introduction to the current investigation, highlighting the background of the study and gaps in existing scholarship. In the following chapter, I provide a comprehensive examining of the current research related to IM and exposure to airborne toxins. I detail methods in Chapter 3, and I provide study results in Chapter 4. A discussion of findings and implications appear in Chapter 5.

## Chapter 2: Literature Review

### **Introduction**

Many scholars have studied IM and exposure to toxic air pollution, but findings on the topic are inconsistent (Romieu et al., 2004) due to differences in exposures and health outcomes assessed (Krzyzanowski & Kuna-Dibbert, 2005). Although previous investigators have explored the relationship between IM and exposure to toxic air pollutants (Agarwal et al., 2010; Bobak, 2000; Carbajal-Arroyo et al., 2011; Currie & Schmieder, 2009; Ezziane, 2013; Greenstone & Chay, 2003; Kaiser et al., 2004; Loomis et al., 1999; Woodruff et al., 2008; Woodruff et al., 1997), most have failed to concurrently examine factors that may modify the relationship, such as SES and community size. IM is rarely the result of a single factor, but the result of a variety of factors (Mosley & Chen, 1984). Thus, a more dynamic approach to IM research may shed new light on the relationship between IM and exposure to toxic air pollutants.

I focused on the relationship between IM, community size, sociodemographic factors, and residence in counties where air releases monitored in the Toxic Release Inventory (TRI; EPA, 2010) occur. My aim in this chapter is to contextualize the current study through a review and synthesis of existing research related to the topic of IM and exposure to toxic air pollution. The chapter begins with a discussion of my search strategy and theoretical framework, followed by a review of the professional and academic literature. The review of the literature begins with a discussion of air pollution, particulate matter, and strategies employed to mitigate airborne pollution. I also discuss the TRI and dedicate a significant portion of this review to research on the health effects of exposure to airborne toxins, especially PM. I discuss general human health

implications of exposure to airborne toxins, as well as those specific to children, infants, and developing fetuses. I give particular focus to IM and other infant health outcomes. The final section of the review includes a discussion of socioeconomic health disparities related to exposure to airborne toxins. I focus on four main sociodemographic factors that correlate with my research questions: maternal education, family income, race, and community size. The chapter closes with a summary.

### **Search Strategy**

I used several online databases to locate literature for this chapter, including ProQuest, EBSCO, ABI Inform, Lexis Nexis, JSTOR, FirstSearch, Academic Search Premier, and Sage. I leveraged Walden's online library as well as Google Scholar to perform searches. Most of the literature included in this chapter was published after 2012; however, I also included seminal studies or older research that was particularly relevant. Keywords included *infant mortality, infant health, infant death, air pollution, particulate matter, toxic release inventory, Toxic Substances Control Act, fetal growth, preterm birth, socioeconomic status, SES, urban versus rural, family income, maternal education, health effects, ozone, chemical releases, airborne pollution, environmental pollutants, emissions, respiratory illness, respiratory disease, and asthma.*

### **Theoretical Framework**

Mosley and Chen's (1984) framework for exploring child survival provided the foundation for the current study. This framework was based on the supposition that multiple factors must be considered to develop a comprehensive understanding of predictors of child mortality. Different disciplines must be used to develop social policies and medical interventions to improve infant survival rates. Research on IM typically falls

into one of the following disciplines: social science, medical, epidemiological, and nutrition. However, the exploration of IM through a single disciplinary lens cannot present a holistic understanding of the multitude of factors that influence mortality. Thus, Mosley and Chen created a framework that incorporated multiple disciplines and methodologies into a single coherent framework. The premise of the scholars' work that most strongly aligned with the current research is that the reduction of survival "in any society is due to the operation of social, economic, biological, and environmental forces" (Mosley & Chen, 1984, p. 27).

Rarely is a child's death the result of a single, isolated factor; thus, it is necessary to consider a variety of cross-disciplinary factors when investigating IM. I did this by investigating the relationships between IM and environmental (proximity to toxic air releases), spatial (community size), and sociodemographic (mother's education level) variables. In public health research, mother's education level is often used to gauge SES (Aaro et al., 2009; Aizer & Currie, 2014; Ansem, Schrijvers, Rodenburg, & van de Mheen, 2014; Daly, Duncan, McDonough, & Williams, 2002; Ganchimeg et al., 2014).

### **Infant Mortality**

*Infant mortality* refers to death that occurs within 1 year of birth (Padilla et al., 2013). IM rates are strong indicators of the health of a population (Haider, 2014). A variety of risk factors can contribute to elevated IM rates, including infection, sanitation, and distal factors such as SES and education (Sartorius & Sartorius, 2014). Globally, the U.S. IM rate ranks 27th among industrialized nations, with strong and persistent socioeconomic and racial disparities (Hirai et al., 2014). After a plateau in U.S. IM rates between 2000 and 2005, the mortality rate declined by 12% between 2006 and 2011 (Lu

& Johnson, 2014). During that same period, preterm births declined by 10% (Hamilton, Martin, & Ventura, 2013). However, despite these improvements, “now is not the time to declare mission accomplished” (Lu & Johnson, 2014, p. S13). Research from the National Center of Health Statistics (MacDorman, Hoyert, & Matthews, 2013) indicated the United States is still among the bottom of developed nations when it comes to IM. In addition, persistent and significant racial and ethnic disparities in IM continue to exist throughout the country (Lu & Johnson, 2014).

Rates of IM are sensitive to regional factors, such as SES and living conditions (Sartorius & Sartorius, 2014). Research also indicates IM is influenced by population density, living standards, and urban infrastructure (Sedetskaya, 2015). As Sartorius and Sartorius explained, an understanding of IM risk factors by region can help guide policymakers in making decisions to mitigate the harmful health outcomes associated with exposure to environmental toxins. Much of the focus of strategies aimed to reduce IM focus on the following five core areas: (a) smoking cessation, (b) breastfeeding, (c) family planning, (d) safe sleep for infants, and (e) immunization (Lu & Johnson, 2014). Educational campaigns designed to inform mothers of strategies for each of these five core areas include the use of short messaging services and media to deliver relevant health information.

In 2012, the U.S. Health and Human Services Secretary called for the creation of a national advisory committee tasked with addressing the country’s IM rate. A primary focus of the committee was to address social determinants that cause disparities in IM rates, such as SES, education, and access to healthcare (Lu & Johnson, 2014). The

committee revised the Healthy Start program, the largest federal program tasked with addressing IM, to include the following six goals (Lu & Johnson, 2014):

1. Improve perinatal health outcomes.
2. Improve women's health prior to pregnancy through a focus on reproductive planning and health promotion strategies.
3. Promote health services that emphasize quality improvement and care coordination.
4. Strengthen families and parenting by addressing stress and providing trauma-informed care.
5. Focus on the collective role of the community.
6. Improve accountability.

Although the revised goals of the Healthy Start program may certainly help to reduce IM, noticeably absent from the strategies is addressing the effects of fetal and natal exposure to toxic air pollution. Based on a substantial body of research that indicates strong links between IM and exposure to air toxins, such as PM and NO<sub>2</sub>, there seems to be an important gap in the goals. Although the committee emphasized the importance of smoking cessation, which can reduce fetal and infant exposure to harmful air toxins, it did not address factors in the larger environment (e.g., proximity to manufacturing plants that release airborne toxins).

### **Air Pollution**

My focus in the current study was the effect of exposure to airborne toxins on rates of IM; thus, the literature review begins with a discussion of air pollution. Across the world, increases in exposure to air pollution correlates with increased risks for many

serious health conditions, such as cardiovascular disease, lung cancer, and respiratory infection (Tanaka, 2015). Air pollution can also create oxidative stress and chronic inflammation in humans, which may influence pregnancy and infant outcomes (Savitz et al., 2013).

Air pollution consists of a mix of particulate matter (PM), liquids, and gases released into the atmosphere through human activities, such as combustion and manufacturing (Backes et al., 2013). As Backes et al. (2013) explained, accelerated population growth in countries throughout the world resulted in increased exposure to air pollution. Exposure to high levels of air pollution is often outside the power of individuals (Tanaka, 2015), especially those of low SES. Most of the research on the effects of exposure to airborne toxins focuses on a specific type of pollution called PM.

### **Particulate Matter**

Particulate matter describes diverse atmospheric substances present in liquid, solid, or semivolatile form (Backes et al., 2013, p. 48). According to Adar, Filigrana, Clements, and Peel (2014), relationships exist between exposure to PM and a variety of human health conditions, including respiratory- and cardiovascular-related mortality and morbidity. Annually, researchers link PM exposure to more than 3.7 million deaths around the world (Bell et al., 2013). Atmospheric PM is generally lower in the United States and other developed nations than it is in developing countries, due to governmental monitoring and legislation designed to limit toxic releases, such as the Environmental Protection Agency's Clean Air Act. However, even with these mitigation policies, over 74 million Americans live in areas with PM levels that exceed federal regulation (Bell et al., 2013).

Most of the PM emitted into the atmosphere is the result of human activities, such as fossil fuel combustion, demolition, industrial activities, and fires (Backes et al., 2013; Nelin, Joseph, Gorr, & Wold, 2012). The measurement of PM is typically expressed as the mass of particles, in micrograms, present within a cubic meter of air. In addition, PM is categorized as three sizes (measured as  $\mu\text{m}$ ): coarse ( $\text{PM}_{10}$ ), fine ( $\text{PM}_{2.5}$ ) and ultrafine ( $\text{PM}_{0.1}$ ) (Backes et al., 2013). As illustrated in Table 1, the size of PM particles vary by source. Fine particles ( $\text{PM}_{2.5}$ ) are those typically created through processes of combustion or phytochemical reaction in the atmosphere, and consist of carbon, sulfate, nitrate, and metals; larger particles are usually formed by the grinding and resuspension of solid materials, such as road dust and other organic debris (Adar et al., 2014).



Table 1

*Sources of Particulate Matter by Classification*

	Type of PM		
	Coarse PM (PM <sub>10</sub> )	Fine PM (PM <sub>2.5</sub> )	Ultrafine PM (PM <sub>0.1</sub> )
		Construction	Industrial processes
Source	Demolition	Agriculture	Condensation
	Dust	Combustion of wood	Volcanic emissions
	Mechanical processes	and fossil fuels	Nucleation

*Note.* Source: Backes et al. (2013).

Of the different forms of air pollution, PM is responsible for most of the poor health outcomes associated with exposure (Backes et al., 2013). Strong associations exist between PM exposure and adverse health outcomes from cardiovascular and bronchial complications, as well as increased levels of inflammation throughout the body (Hertel et al., 2010; Lee et al., 2011). Fine and ultrafine PM may pose greater health risks than larger particles because they can enter the tracheo-bronchial and alveolar regions of the human respiratory tract (Adar et al., 2014). As Larr and Neidell (2016) explained,

PM<sub>2.5</sub> penetrates deep into the lungs and passes into the bloodstream, thereby affecting both the lungs and the heart. It can reduce lung function and increase

respiratory symptoms such as airway irritation, difficulty breathing, and asthma. It can also induce heart attacks or irregular heartbeat. (p. 95)

The deep bronchial penetration of fine and ultrafine PM allows these particles to interact with immune cells and create systemic effects after entering the body's bloodstream. Because of these differences, fine and coarse PM may affect human health in different ways. According to Proietti et al. (2013), PM derived from natural sources often are larger, and thus, less harmful; however, those derived from human activities such as combustion are smaller and may be more harmful to human health.

### **PM Exposure and Infant Outcomes**

The effects of air pollution on infant health are also substantiated by significant research (Arceo et al., 2015; Bowatte et al., 2015; Carbajal-Arroyo et al., 2011; Cesur, Tekin, & Ulker, 2016; Ezziane, 2013; Knittel et al., 2011; Loomis et al., 1999; MacIntyre et al., 2014; Proietti et al., 2013; Van Rossem et al., 2015; Sartorius & Sartorius, 2014; Woodruff et al., 1997). In Woodruff et al.'s (1997) highly cited study, the researchers classified exposure to PM<sub>10</sub> as low, medium, or high in order to explore the relationship between differing levels of PM exposure and IM. Results indicated that IM increased by 10% for infants in the high exposure group. Similarly, Loomis et al. (1999) found that increases in exposure to PM<sub>2.5</sub> were associated with significant increases in IM. Carbajal-Arroyo et al. (2011) reported a correlation between increased exposure to PM<sub>10</sub> and respiratory-related IM.

Cesur et al. (2016) conducted a novel study on the relationship between IM and PM exposure in Turkey. The researchers chose Turkey as the focus of their investigation because of the country's expanding natural gas infrastructure, which resulted in a drastic

reduction to its reliance on coal. Consequently, the carbon emissions in the Republic of Turkey decreased dramatically in recent decades. Using longitudinal data, Cesur et al. were able to chart IM from 2001 to 2011 to investigate if the reduction of emissions aligned with a reduction in IM. Results indicated a significant reduction in IM that correlated with the country's reduction in carbon emissions: "Our results indicate that the expansion of natural gas services has led to a significant reduction in the rate of infant mortality" (p. 27). The researchers went on to postulate that a 1% increase in natural gas use was associated with a 4% decline in IM in 2011, alone. Cesur et al. concluded that the health benefits of increased of natural use and subsequent reduction in coal consumption are significant.

In another foreign study on the relationship between IM and emissions, Ezziane (2013) investigated the correlation between IM, low birth weight, and air pollution in Belgium, Greece, Hungary, Iceland, Japan, Mexico, Portugal, and Sweden for the period between 1990 and 2009. Archival data on the emission levels for sulphur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), low-level ozone (O<sub>3</sub>), and PM<sub>10</sub> were culled from three sources: (a) OECD statistics, (b) the Convention on Long-Range Transboundary Air Pollution, and (c) national inventory submissions. In general, results from Ezziane's analysis indicated that the global incidence of infants born with low birth weight had increased to over 8% of all births in 2010. The scholar reported that exposure to large amounts of the four pollutants investigated was associated with increased risk for adverse birth outcomes. Specifically, countries with high volumes of emissions (Portugal, Greece, Japan, and Belgium) demonstrated high risk for low birth weight and IM. In contrast, statistics from countries with lower levels of emissions (Iceland and Sweden) indicated

fewer adverse outcomes. Ezziane concluded that low birth weight and IM were linked with air pollution, explaining that mitigation strategies that reduced exposure to airborne toxins were critical to reducing IM. Of particular relevance to the current research was Ezziane's findings that regional differences existed in IM and PM exposure.

Arceo et al. (2015) also explored the relationship between exposure to air pollution and IM in the context of developing countries. The researchers conducted weekly measures of pollution and mortality across 48 Mexico City municipalities between 1997 and 2006. The researchers chose this location because it reflected the high levels of IM typically present in developing countries, as well as the ranges of pollution volume found in the United States. In this way, Mexico City provided the researchers with an opportunity to estimate the effects of pollution within a range often found in developing countries, and then compare that estimate to the effect at ranges used in U.S. estimates. Data analysis indicated that over the course of one year, a 1 part per million increase in CO led to 240 infant deaths per 100,000 births. Accordingly, a 1% increase in CO over the course of 1 year led to a 0.326% increase in IM. In terms of PM<sub>10</sub> exposure, the researchers found that a 1% increase in pollution led to a 0.40% increase in IM. Results on the effects of PM<sub>10</sub> on IM in Mexico City were comparable to the effects found in the United States.

Domestic research also indicates strong relationships between PM exposure and IM. Research conducted in southern California using data from 2002 to 2007 indicated that ambient pollution from traffic and other various pollutants, especially PM, can significantly influence rates of IM (Knittel et al., 2011). Knittel et al. (2010) posited that a 1-unit reduction in PM<sub>10</sub> could reduce IM by approximately 6% (saving 18 lives per

100,000 births). Research also indicates that prenatal exposure to air pollution can affect the blood pressure of newborns. Van Rossem et al. (2015) reviewed data from a cohort of 1,131 mother-infant pairs located in Boston, Massachusetts. The researchers estimated exposure to PM<sub>2.5</sub>, black carbon, nitrogen oxides, nitrogen dioxide, ozone, and carbon monoxide, as measured at monitoring sites proximal to participants' homes. Results indicated that higher levels of exposure to PM<sub>2.5</sub> and black carbon during the third trimester correlated with higher systolic blood pressure in infants.

### **Respiratory Illnesses**

Air pollution is linked to acute lower respiratory infections (ALRIs) in children, which account for almost 20% of worldwide deaths among young children (Mehta, Shin, Burnett, North, & Cohen, 2013). Indeed, the disease burden of respiratory infections such as pneumonia and bronchitis is high in early childhood, with pneumonia as the leading cause of childhood death in the world (Darrow et al., 2014). In a systematic review of the relationship between air pollution and acute lower respiratory infection (ALRI) in children, Mehta et al. (2013) found that each 10- $\mu$ g/m increase in PM<sub>2.5</sub> was associated with a 12% increase in risk for ALRI. The researchers also found significant regional differences in the impact that increases in PM<sub>2.5</sub> exposure had on child health. For example, the researchers reported that a 10- $\mu$ g/m increase in exposure to PM<sub>2.5</sub> would result in 66,000 additional cases of childhood ALRI in the United States, but 372,000 in Mexico. This finding was particularly salient to the current research because it suggests the protective mechanism of socioeconomic factors.

In another systematic review, Bowatte et al. (2015) explored the relationship between childhood exposure to traffic-related air pollution (TRAP) and the development

of asthma. Analysis revealed that increased, long-term exposure to PM<sub>2.5</sub> and black carbon was associated with higher risks for childhood asthma, through age 12. Similarly, Darrow et al. (2014) found significant evidence of the relationship between short-term exposure to traffic pollution, such as PM<sub>2.5</sub>, nitrogen dioxide, and carbon monoxide, and increased emergency room visits for respiratory infections for Atlanta-area children up to the age of 4. Results indicated that early life exposure to traffic pollution, especially PM<sub>2.5</sub>, can exacerbate childhood respiratory infections. Because such infections are leading cause of death among young children worldwide (Mehta et al., 2013), these findings are particularly salient to the study of the relationship between exposure to airborne toxins and IM.

MacIntyre et al. (2014) examined the relationship between outdoor air pollution and risks for respiratory infection in early childhood. The researchers utilized data from 10 European birth cohorts consisting of 16,059 individuals. Analysis revealed consistent relationships between exposure to air pollution and the risk for pneumonia during the first 2 years of life. In addition, MacIntyre et al.'s findings indicated that the effects of air pollution on risks for respiratory infection may be higher during the first year of life, a finding which "could highlight a unique period of susceptibility when children are at increased risk of respiratory infections due to air pollution" (p. 111). The researchers concluded that mitigation policies aimed at reducing exposure to air pollution may reduce the incidence of early childhood pneumonia. This finding was pertinent to the current research because, as childhood pneumonia is a leading cause of early childhood death, such mitigation measures may reduce IM rates, considerably.

## **Mitigation Scenarios**

To contextualize the scope of the health problems associated with PM exposure and the growing awareness of dangers associated with exposure, it is helpful to review some of the strategies health leaders across the world have instituted to reduce negative health outcomes related to PM. Because of the significant health risks associated with exposure to fine PM, health leaders across the world institute strategies aimed at mitigating, or reducing the poor health outcomes associated with its exposure. Much of the research on such mitigation strategies, especially those aimed at improving child and infant outcomes, is promising. For example, Larr and Neidell (2016) examined the projected effects of ozone and PM exposure on IM using vital statistics data from 2012. Calculations performed by the researchers linked PM<sub>2.5</sub> exposure to 34 infant deaths per 100,000 births (Larr & Neidell, 2016). Projecting into 2050, the researchers noted few changes in these effects on IM rates because PM exposure is expected to be largely unaffected by climate change. However, according to Larr and Neidell's (2016) projections, mitigation policies could result in a 10.5% to 21.6% decrease in IM. Similar to the potential impacts on IM, the researchers postulated that failure to mitigate the effects of exposure to air pollution would only result in small reductions in the negative effects; however, mitigation could result in significant, positive improvements in earnings and hospitalization rates. For example, without mitigation, the effects of exposure to PM and ozone could increase up to 3%; however, with a mitigation plan, respiratory-related hospitalizations could drop by as much as 8% (Larr & Neidell, 2016).

While Larr and Neidell's (2016) findings were based on future projections, Greenstone and Hanna (2014) examined the actual effects of environmental mitigation

policies on IM. The researchers pulled data from India, where PM concentrations are five times as high as those in the United States. In fact, according to the Environmental Performance Index (2013), India had the worst air pollution index out of 132 countries included in the researchers' analysis. Greenstone and Hanna specifically examined the effects of two air pollution mitigation policies: The Supreme Court Action Plans (SCAPs) and the Mandated Catalytic Converters. The SCAPs were instituted by India's Supreme Court to help mitigate the country's rapidly rising pollution levels. These action plans included policies that fueled regulations, restricted levels of industrial pollution, and developed roads to bypass areas with high levels of pollution. The other policy Larr and Niedell examined, Mandated Catalytic Converters, pertained to the mandatory use of devices (called catalytic converters) which are used to reduce vehicular pollution.

Results from Larr and Niedell's (2016) study indicated modest declines in IM from these air regulations. However, of particular importance was evidence that environmental regulations can succeed, even in governments such as India, where institutional settings are weak. The key is to ensure strong public demand and support for the mitigation policies. Essentially, Greenstone and Hanna's research suggested that by rallying the public to support mitigation policies, such policies (and their enforcement) may be more effective. One way to rally the public may be to improve education of the effects of exposure to airborne toxins, especially on infant health and mortality.

Tanaka (2015) investigated the effects of mitigation strategies for air pollution on IM rates in China. After 1998, the Chinese government imposed strict sanctions on environmental pollution which required power plants to drastically reduce air pollution. Tanaka culled data on IM from the Chinese Disease Surveillance Points system, which



included data on birth and death registrations from 1991 through 2000. Analysis revealed that IM rate dropped significantly after implementation of the regulations. Specifically, the researcher found that the regulations likely reduced IM by 3.29 deaths per 1000 live births, which corresponds to a 20% reduction in IM. Such sanctions, therefore, may drastically reduce IM in areas with high levels of toxic air pollution.

Low emission zones (LEZs) are another common mitigation strategy for reducing air pollution. Gehrsitz (2017) used data from Germany, where leaders introduced a series of LEZs at different points in time, to explore the effects of this governmental mitigation strategy on infant health. The scholar found that LEZs correlated with reductions in PM<sub>10</sub> by up to 8%. While Gehrsitz admitted that the reductions were too small to correlate with improvements in infant health, this research indicates that widespread implementation of LEZs may provide a strategy for reducing air pollution that is, in fact, linked with IM and other infant and childhood health problems. Another possible reason that Gehrsitz was unable to demonstrate a significant relationship between the institution of LEZs and improvements in infant health is that the researcher focused on PM<sub>10</sub>, which may have fewer health risks associated with exposure (Proietti et al, 2013).

It is important to note that most of the research on mitigation strategies aimed at reducing the negative health outcomes associated with PM exposure has been conducted in countries other than the United States. This may be because the United States already has environmental controls and monitoring in place to temper the human health effects of PM exposure. However, that is not to say PM exposure does not negatively affect human health in this country. Thus, the lack of research on mitigation strategies to curtail the

effects of PM exposure on human health presents a noteworthy gap in the existing PM literature.

### **Toxic Release Inventory**

In the United States, a federal strategy developed to mitigate airborne pollution is the Toxic Release Inventory (TRI). The TRI database began under the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 (EPA, 2010). This act requires reporting from manufacturing facilities that produce or process more than 25,000 pounds of specified chemicals included in the TRI list. Facilities included in the TRI database include a variety of chemical, oil, gas, and paper manufacturers (Johnson et al., 2016). Manufacturers must report any releases that occur from spills, routine emissions released into land, water, or sewage treatment plants. In addition, manufacturers must report any chemicals transferred to other locations for recycling or disposal (Hendryx & Fedorko, 2011).

As of 2016, the TRI chemical list included 595 individual chemicals (EPA, 2016). TRI is reported at the facility level, and separate reports must be filed for each monitored chemical (Agarwal et al., 2010). Information in the TRI database are classified by medium (air, water, land), and release site (on-site or off-site). Using the TRI database, I was able to determine the total number of pounds of specified chemicals released into the atmosphere for each county included in the analysis, for the specified date range of 1987 to 2015. I chose this range because although TRI data became available in 1986, they were incomplete. In addition, full reporting for 2016 was not available at the time I performed data collection and analysis.

Previous researchers have used the TRI to explore relationships between exposure to toxic pollutants and adverse health outcomes. For example, Hendryx and Fedorko (2012) used TRI data to investigate differences in general mortality rates among rural and urban areas, based on exposure to pollutants. The researchers reported significantly higher rates of general mortality in rural and urban areas with higher levels of toxic air releases. Chen, Luo, and Hendryx (2015) used data from the TRI to examine the correlation between cardiovascular disease (CVD)-related mortality and exposure to zinc compound air pollutants, and found an association between exposure to zinc air releases and increased CVD mortality. Johnson et al. (2016) used TRI data to examine the relationship between sociodemographic factors and the presence and characteristics of toxic releases and found that a greater proportion of minorities lived in areas where TRI facilities were located. Similarly, Wilson et al. (2012) investigated racial disparities in the proximity to TRI facilities. Data from the TRI have also been used to explore relationships between exposure to TRI releases and lymphoma (Moreau, Buchanich, Geskin, Akilov, & Geskin, 2014), the presence of heavy metals in the blood (Bank, 2013), and house values (Currie, Davis, Greenstone, & Walker, 2015).

### **PM Exposure and Human Health**

Exposure to PM has deleterious effects on human health. Adar et al. (2014) conducted a meta-analysis of 33 studies to explore the effects of fine PM on human health. Analysis indicated that short-term exposure to higher levels of PM<sub>2.5</sub> was correlated with higher rates of hospitalization and mortality, especially those caused by respiratory-related illnesses. The researchers hypothesized that individuals residing in urban areas may be more prone to the negative health consequences of PM exposure,

such as mortality and morbidity due to respiratory and cardiovascular diseases, due to greater concentrations of PM typically found in urban environments. However, Adar et al. (2014) admitted that further research was needed on any urban/rural discrepancies in the health effects of PM.

In addition to its effects on adult and fetal health, exposure to air pollution can also have negative health implications for children. As Larr and Neidell (2016) explained, children are particularly vulnerable to exposure to toxic pollutants, which can lead to severe illnesses, complications, and even death. The increased health risks associated with exposure to environmental toxins in early life is due to children's rapid rates of physical development. As Proeitti et al. (2013) explained, factors that present few risks in adulthood can be deleterious to the body's development and maturation during childhood. Because younger children are more affected by exposure to air pollution than are older ones, similar doses of pollution can have greater negative impacts when they occur earlier in life (Larr & Neidell, 2016). In addition, children are particularly vulnerable to air toxins because they respire at higher rates, have narrower airways, are more frequently exposed to outdoor air, and are undergoing critical phases of bronchial and immune development (Darrow et al., 2014).

### **Neonatal Outcomes**

*Neonatal* refers to the period of time surrounding birth. Because IM was the focus of the current investigation, a discussion of the neonatal outcomes associated with exposure to air toxins contextualizes some of the common risks for infant death.

**Preterm birth.** Particulate matter exposure is linked to poor fetal outcomes. As Choi et al. (2012), the developing fetus is particularly susceptible to exposure to toxins, due to high rates of cellular growth within the delicate balance of changing hormonal requirements for such growth. Interruptions to this balance can disrupt fetal growth. As Backes et al. (2013) explained, chronic exposure to such pollutants can interrupt biological growth and development. Thus, exposure to PM, via the mother, is associated with preterm births and IM (Backes et al., 2013; Rossner et al., 2011).

Preterm birth, which describes birth prior to 37 weeks of gestation (Malley et al., 2017), is a strong predictor of infant and child mortality (Johnson et al., 2016). As Rappazzo et al. (2014) explained, preterm birth is a stronger indicator of fetal underdevelopment that increases risks for other outcomes, including stunted neurodevelopment and IM. Studies indicated that maternal exposure to PM is associated with low birth weight (Stieb et al., 2012) and stillbirth (Faiz et al., 2012), and can increase risks for preterm births (Huynh, Woodruff, Parker, & Schendorf, 2006; Nieuwenhuijsen, Dadvand, Grellier, Martinez, & Vrijheid, 2013; Stieb, Chen, Exshoul, & Judek, 2012; Wu et al., 2009). Wu et al. (2009) found that risks for preterm births increased by 128% among women with high levels of PM<sub>2.5</sub> exposure.

Research also indicated that the timing of maternal exposure to PM can influence the likelihood that exposure will lead to preterm birth (Rappazzo, Daniels, Messer, Poole, & Lobdell, 2014). For example, Rappazzo et al. (2014) found that PM exposure that occurred closer to the time of implantation and birth were more strongly related with preterm births than exposure that took place during other periods. However, the

researchers cautioned that all maternal exposure to PM<sub>2.5</sub> is concerning, regardless of its magnitude and timing.

Malley et al. (2017) explored the relationship between maternal exposure to PM<sub>2.5</sub> and preterm births in 183 countries. Not only did the researchers find significant relationships between PM exposure and preterm birth around the world, but they also noticed regional differences in the relationship. For example, the scholars reported that most of the preterm births associated with PM<sub>2.5</sub> exposure took place in Southeast Asia, North Africa, the Middle East, and West sub-Saharan Africa. Although Malley et al. postulated this relationship was due to higher rates of exposure to PM in these regions, it is also possible that sociodemographic factors such as poverty increased individuals associated health risks. The scholars explained, “Our analysis emphasizes the importance of also considering its contribution to effects in utero that lead to increased postnatal mortality and lifetime morbidity” (p. 8), suggesting that efforts aimed at reducing preterm births must include the mitigation of risk factors for preterm birth, such as maternal exposure to PM.

While common sources of fine PM are the result of combustion from industrial processes, exposure to can also result from household use of solid biomass fuels, such as coal and wood. For this reason, people living in impoverished regions of the world may be at particularly elevated risks for the negative health outcomes associated with PM. In addition to PM, smoke from burning solid biomass can emit carbon monoxide, nitrogen dioxide, and other carcinogens that expose people to much higher concentrations of airborne toxins than typical exposure found in polluted, urban areas (Wylie et al., 2014). To explore the relationship between exposure to pollutants from cooking with biomass

fuels and preterm birth, Wylie et al. (2014) analyzed secondary data on two cohorts of women in India. Results indicated that infants born to women who cooked with solid biomass were an average of 112 grams lighter and more likely to be born preterm than were infants born to women who did not cook with biomass fuels. In addition, rates of stillbirths were higher in the biomass group. The researchers concluded that the effects of antenatal exposure to such airborne household toxins may be higher than anticipated.

Despite the above evidence, not all researchers have reported an association between PM exposure and preterm birth. For example, Johnson et al. (2016) investigated the relationship between exposure to PM and preterm birth using data from birth certificates and hospital records for 258,294 births that took place in New York City between 2008 and 2010. Results did not provide conclusive evidence that exposure to PM<sub>2.5</sub> was related to preterm birth. Because preterm birth raises risks for IM, Johnson et al.'s findings may challenge those of other researchers who reported relationships between IM and PM<sub>2.5</sub> exposure (Adar et al., 2014; Bowatte et al., 2015; Fleisch et al., 2015; Larr & Neidell, 2016; Mehta et al., 2013; Savitz et al., 2013).

The effects of exposure to airborne toxins on developing fetuses are particularly important in areas with higher levels of airborne pollutants that are virtually impossible to avoid (Proietti et al., 2013). As Proietti et al. (2013) explained, "Especially in these areas, strong evidence for the association between prenatal exposure to air pollution and infant mortality exists, clearly indicating the need for more stringent measures to reduce air pollution" (p. 18). Thus, the current research shed new light on spatial and regional inequities in IM rates, based on proximity to plants that produce airborne toxins.

**Fetal growth.** Another way that exposure to air pollution can affect the IM rate is through the inhibition of fetal growth. In a study on the association between exposure to PM<sub>2.5</sub> and NO<sub>2</sub>, Savitz et al. (2013) reported a strong association between reduced fetal growth and exposure to those air pollutants. The researchers used data from the New York City Community Air Survey, which provided them with linked spatial information and SES of births. The researchers utilized data on 252,967 births that took place between 2008 and 2010 in New York City. Analysis indicated correlations between exposure to the chemicals and birth weight reductions, for each phase of pregnancy. Savitz et al. estimated the effects of PM<sub>2.5</sub> exposure on birth weight to be 20 grams for exposure during the first trimester, 30 grams for exposure during the second trimester, and 40 grams for total pregnancy exposure per 10  $10\mu\text{g}/\text{m}^3$ . The effects of NO<sub>2</sub> exposure were a birth weight reduction of 18 g per 10 parts per billion. The researchers concluded that their results substantiated the effect of air pollutants such as PM<sub>2.5</sub> on fetal growth and called for further research to determine if such exposure may correlate with other outcomes, such as IM.

In another study, Fleisch et al. (2015) conducted a study to investigate the relationship between maternal exposure to traffic pollution and fetal growth. The researchers used data from a cohort of 2,115 women located in the Boston, Massachusetts area and assessed third trimester exposure to black carbon and PM<sub>2.5</sub>. Analysis revealed that in utero exposure to traffic-related pollution correlated with reduced fetal growth at birth and rapid rates of infant weight gain. Infants with the highest levels of exposure to the airborne toxins were three times as likely to have low birth weight followed by rapid



weight gain after birth. The scholars explained that the rapid weight gain could be the result of inflammation of fat tissues or neuroinflammation and altered satiety signals.

**Low birth weight.** In line with findings on the effects of PM exposure on fetal growth (Fleisch et al., 2015; Savitz et al., 2013), other researchers (Backes et al., 2013; Hao, Strosider, Balluz, & Qualters, 2016; Dadvand et al., 2013; Fleischer et al., 2014; Johnson et al., 2016; Pedersen et al., 2013) reported on the negative outcomes associated with birth weight. PM<sub>2.5</sub> is particularly linked with low birth weight because these particles can enter the airways and inhibit fetal growth through oxidative stress, vascular dysfunction, blood coagulation, inflammation, or the interruption of nutrient transfer (Fleisch et al., 2015). Low birth weight (LBW) is an important consideration in the study of IM because it is strongly related to infant morbidity and mortality (Hao et al., 2016). For example, Hao et al. (2016) explored the relationship between LBW and PM exposure using data from all counties in the contiguous United States. The dataset included information from over 3.8 million singleton births between 2001 and 2002. By using data from the entire country, the scholars were able to explore geographic variations in the relationship between LBW and PM exposure.

Of particular relevance to the current research was Hao et al.'s (2016) findings on the significant geographical variations in the relationship between PM exposure and LBW. The researchers hypothesized different reasons for these geographic variations, beginning with differences in the constituents of PM in various parts of the country. For example, the analysis revealed high levels of sulfate in the Mid-Atlantic, high nitrate concentrations in Southern California, and low concentrations of sulfate in central Mountain zones. Thus, Hao et al. posited that high concentrations of sulfate and nitrate

PM<sub>2.5</sub> may correlate with LBW in Middle Atlantic and East North Central regions of the United States, and that high concentrations of nitrate PM<sub>2.5</sub> may correlate with LBW in the West North Central region of the country.

Two other possible reasons for the regional differences described in Hao et al.'s (2016) study were co-variances with other pollutants and differences in weather and climate, such as humidity and temperature. Finally, regional differences in people's behaviors may contribute to differences in the impact that airborne PM has on their health. For example, previous research indicated that higher use of central air conditioning was associated with fewer adverse health outcomes associated with PM<sub>2.5</sub> (Bell, Ebisu, Peng, & Dominici, 2009). Such a variation could be reflective of climate (individuals in hotter climates are more likely to have and use central air conditioning) as well as SES (individuals in poorer communities may be less likely to have and afford central air conditioning).

While the current investigation focused on the potential differences in the relationship between exposure to air pollution and IM, based on community size, findings from Hao et al.'s (2016) study indicated a few variables that could be factors, if significant differences based on community size are found. Hao et al.'s research, which substantiated regional differences in the impact of PM exposure on birth weight, suggests potential reasons for variations in the relationship between IM and PM exposure. The current study expanded upon these findings by exploring how the size of a community and sociodemographic variables affected potential relationships.

### **Indicators of Socioeconomic Status**

Exposure to environmental pollutants can disproportionately affect the health of individual's bases on sociodemographic indicators, such as race and income (Gray, Edwards, Schultz, & Miranda, 2014). Accordingly, indicators of SES likely undergird demographic disparities in U.S. IM data (Hirai et al., 2014). As Backes et al. (2013) postulated, research that correlates sociodemographic factors and health outcomes provides information that leaders may use to create legislation aimed at improving public health and reducing such disparities (p. 50).

Of the various sociodemographic factors that can influence health, individuals of low SES are often more susceptible to the negative health outcomes associated with exposure to air pollution (Padilla et al., 2013). For example, Bell, Zanobetti, and Dominici (2013) found that individuals with low levels of income were at a higher risk for death associated with short-term exposure to PM. In addition, Bell et al. reported that poor education and unemployment correlated with higher risks of mortality from PM exposure. A population analysis of South Carolina conducted by Gray, Edwards, and Miranda (2013) found that low SES, indicated by income, poverty, and education, and higher percentages of minority populations were correlated with higher levels of PM<sub>2.5</sub> in communities.

Not only can an individual's SES affect his or her health risks, but so too can the SES of entire communities. For example, Padilla et al. (2013) studied the relationship between IM and exposure to air pollution (ambient NO<sub>2</sub>) in poor communities in France. The scholars used 21 variables to calculate SES, such as education, age, and employment, of the two communities included in their investigation. After calculating the deprivation

index for the two communities, the researchers created models to explore the relationships between exposure to air pollution and IM in each of the communities and found significant spatial variations. Padilla et al. found that IM was explained by spatial variations. The researchers concluded that socioeconomic characteristics can interact with spatial variations to influence health risks associated with exposure to air pollution. Accordingly, in the current study, I considered how community size interacted with the relationship between IM and exposure to air pollution.

SES is also particularly relevant to an investigation of how toxins from TRI facilities influence individuals' health. For example, Johnson et al. (2016) explored how the distribution of TRI facilities in the metro-Atlanta area related to differences in the sociodemographic characteristics of surrounding communities. Using data from the 2000 census, the researchers identified racial and SES disparities. For example, nearly 60% of the TRI facilities in the metro-Atlanta area were located in lower-middle class communities with higher percentages of minority populations. The researchers also noted an important disparity in education levels, which was particularly relevant to the current research. As the percentage of women with college degrees in an area increased, a reduction in the amount of TRI chemicals release was also noted; thus, "female education was shown to be a consistent predictor across the range of exposures evaluated" (Johnson et al., 2016, p. 9).

### **Maternal Education**

As indicated above, researchers often use maternal education as an indicator of SES. For example, Ansem (2014) used maternal education as an indicator of SES during a study on the role of SES in children's dietary patterns. Sartorius and Sartorius (2014)

found that maternal education was a significant risk factor for IM. Aarø et al. (2009) employed parent education as an SES indicator in a study on the relationship between SES and health behaviors.

In addition to the formal educational attainment used to gauge SES, targeted education for infant health may also reduce infant health risks. For example, Prince, Young, Sappenfield, and Parish (2016) conducted a study to examine the reasons for the sudden and drastic reduction in IM that occurred in Alaska between 2010 and 2011. The researchers examined birth data and stratified it by race and urban/rural residence, hypothesizing that these variables have related to the drop. Analysis revealed that race and rurality were related to IM risk, but did not explain the statewide drop in IM. The researchers concluded that statewide efforts aimed at reducing IM via an educational campaign on postnatal, sleep associated deaths may have been responsible for the drop in IM.

### **Family Income**

Income is another common SES indicator. According to Tacke and Waldmann (2013), income is the most important indicator of a region's health status (Tacke & Waldmann, 2013). People typically live longer and experience lower levels of general and IM in wealthier regions (Tacke & Waldmann, 2013). In addition to country/community income levels, family income is another common SES indicator that is strongly linked with general mortality (Daly et al., 2011).

Interestingly, the effects of income on IM rates may be influenced by income inequality. Tacke and Waldmann (2013) investigated the relationship between IM and income inequality using data from 93 countries. The researchers found statistically

significant relationships between IM and income inequality. Tacke and Waldmann postulated that one reason for this trend may be relative deprivation that occurs as people compare themselves to those in other social classes. The scholars explained,

Relative deprivation due to income distance to the rich may matter for health not because of stress or health risk taking, but if the nonrich try to emulate the consumption behavior of the rich by allocating a larger share of consumption from nonvisible goods (such as healthcare) to status goods. (p. 3241).

Third, Tacke and Waldmann suggested that the correlation between higher rates of income inequality and IM may be because income inequality is usually associated with poor public spending on factors such as education, healthcare, and sanitary infrastructures.

### **Race**

In the United States, significant racial disparities exist in rates of IM rates (Haider, 2014). Past researchers have investigated race as an SES indicator in studies on IM (Haider, 2014; Hirai et al., 2014; Wilson et al., 2012). Minorities, especially those of low SES, are more vulnerable to health risks associated with exposure to environmental pollution – an association that is influenced by social issues such as racism, classism, and segregation (Wilson et al., 2012). Similarly, research indicates that the effects of exposure to air pollution are more severe among minority populations (Gray et al., 2014).

Many researchers have investigated the relationship between race and IM. For example, Haider (2014) used U.S. census data from 2000 to 2004 to explore the associations between IM and race, maternal marital status, education, and age. The researcher included data for the following racial demographics: non-Hispanic whites,

African American, Mexicans, Puerto Ricans, Asians, and Native Americans. Data analysis indicated significant racial discrepancies in IM. The IM rate for non-Hispanic whites was 5.35, while the rates were significantly higher for African Americans (12.35), Native Americans (8.31), and Puerto Ricans (7.61). Rates among Mexicans (5.04) and Asians (4.34) were slightly lower (Haider, 2014). Haider (2014) found that the differences in IM by race were mostly attributable to the characteristics of maternal education, marital status, and age, which are all associated with household income.

In another investigation, Gray et al. (2014) explored the relationship between exposure to PM<sub>2.5</sub> and O<sub>3</sub> and birth outcomes using birth data from North Carolina between 2002 and 2006. The researchers found that both pollutants were associated with low birth weight and small size for gestational age. Importantly, neighborhood SES and race also proved to be factors. Non-Hispanic Black women with lower levels of education had a higher risk of poor birth outcomes compared to more educated non-Hispanic White mothers. The researchers explained, “Air pollution exposure contributed an additional harmful effect on pregnancy after controlling for race and individual and area-level SES” (Gray et al., 2014, p. 6).

Finally, Wilson et al. (2012) investigated the relationship between the racial and socioeconomic characteristics of communities and proximity to TRI facilities in the Charleston, South Carolina area. Similar to Johnson et al.’s (2016b) study, the scholars culled data from the 2000 census and the TRI database and found significant evidence of racial and sociodemographic disparities in individuals’ mean distance from TRI facilities. For example, in the 0.5-, 1-, and 5-mile buffer zones, the researchers discovered that 52%, 47%, and 36% of the respective populations were non-White (Wilson et al., 2012).

The scholars also found that as the percentage of Whites and college graduates increased in a census tract, the likelihood of that tract having a TRI facility decreased. Significant positive associations were noted between the number of TRI facilities in a census tract and several demographic variables, including income, education, and unemployment.

### **Community Size**

As mentioned throughout this chapter, significant regional disparities in IM exist, based on a variety of factors such as income, race, education, and access to healthcare. As Singh and Siahpush (2014) explained, “Geographical inequalities in health have long represented an important area of public health research in the USA” (p. 272). In the current study, geographic disparities were measured according to differences in community size. Vulnerability to adverse health outcomes caused by exposure to environmental toxins may be moderated by community size. For example, Hendryx and Fedorko (2012) found stronger associations between exposure to environmental toxins and general mortality in rural communities (which tend to have smaller populations) than in urban communities (which tend to be larger).

Singh and Siahpush (2014) examined rural and urban disparities in all-cause mortality using data from the U.S. vital statistics database. The researchers computed data for 12 3-year periods for major causes of death. Analysis revealed “substantial and increasing rural-urban disparities in all-cause mortality and mortality from several major causes of death over time, as both absolute and relative inequalities in mortality widened between 1969 and 2009” (p. 287). Interestingly, the general mortality rates were higher in urban and metropolitan areas; however, infant, child, and youth mortality rates were higher in rural areas. The scholars found that poverty and race helped explain



discrepancies between rural and urban mortality, but not the discrepancies between all-cause and child mortality rates. Singh and Siahpush posited that increased child mortality in rural areas relate to higher rates of obesity, chronic conditions, and exposure to secondhand smoke. Although many questions remained about the elevated rates of infant and child mortality in rural versus urban areas, this study demonstrated the importance of considering spatial characteristics, such as community size, when investigating trends in IM.

Sartorius and Sartorius (2014) examined spatial and regional disparities in IM that occurred between 1990 and 2011. The researchers culled country-level data from the World Bank Development Indicator database and found that global disparities in IM were often attributed to sanitation, lack of water, and poor maternal education. Because the current research focused only on communities in the United States, it is unlikely that potable water and sanitation will be significant factors. However, Sartorius and Sartorius' (2014) investigation does support my investigation of the influence of maternal education on IM.

Previous researchers have found that poorer access to health services can exacerbate the negative health outcomes associated with exposure air pollution (Gutierrez, 2015). Thus, it is possible that IM may be higher in smaller, more rural communities, even if the levels of airborne pollution are lower than those in larger, more populated communities. This was a fundamental question of the current research.

### **Summary**

As Hao et al. (2016) explained of current research on the relationship between exposure to air pollution and health outcomes, "Most published studies have limited

geographic areas or time periods, often with small sample size, in part due to sparsely distributed air pollution monitoring” (p. 250). The use of the TRI database, which provides information on toxic air emissions produced by facilities throughout the country, allowed me to investigate disparities in IM across the country, based on community size and proximity to facilities that emit these toxins. In addition, the use of linked birth-death records allowed me to integrate sociodemographic factors into the analysis to investigate how maternal education, income, and race may influence the relationship between IM and community size (small versus large).

Studies reviewed in this chapter indicated that significant relationships exist between IM, exposure to airborne toxins, education, income, and race. Disparities in the effects of airborne toxins on IM also exist by region. While the existing body of research on IM and exposure to airborne pollution is broad, it is also based on inconsistent methods and conceptualizations. Thus, the current research provided an original contribution by examining variables that previous scholars have not concurrently examined. Although an ample body of research supports the positive association between IM and exposure to toxic air pollution (Agarwal et al., 2010; Bobak, 2000; Carbajal-Arroyo et al., 2011; Currie & Schmieder, 2009; Ezziane, 2013; Greenstone & Chay, 2003; Kaiser et al., 2004; Loomis et al., 1999; Woodruff et al., 2008; Woodruff et al., 1997), researchers have failed to investigate how sociodemographic variables and community size may be associated with IM and in areas where significant toxic air emissions occur.

This chapter provided a review of existing research related to the topics of IM and airborne toxins. The following chapter details the methodology. Results of the analysis

are presented in Chapter 4, followed by a discussion of findings, implications, and recommendations in Chapter 5.

## Chapter 3: Research Method

### **Introduction**

Living in counties where manufacturers release environmental toxins, such as those tracked by the EPA's (2010) TRI, may increase an infant's exposure to air pollutants, both in utero and after birth. Research indicates that increased proximity to airborne toxins may elevate health risks. Because IM is a strong indicator of a population's health status (Braud et al., 2011; OECD, 2010; Suarez et al., 2007), it is an important topic in public health research.

In this chapter, I include an overview of my methods, beginning with a discussion of the study's design and rationale, followed by a description of the population, sample, and sampling strategy. Next, I present data collection and instrumentation. I provide operational definitions for variables, followed by the data analysis plan and threats to validity. I also discuss limitations and ethical assurances. The chapter concludes with a brief summary.

### **Research Design and Rationale**

#### **Study Variables**

My purpose in this quantitative investigation was to examine the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. I followed a retrospective, cross-sectional design.

**Dependent variable.** The dependent variable was IM per 1,000 live births in a given community for each of the 3 years included in this analysis (1987, 1995, and 2004).

**Independent variables.** The independent variables related to mother's SES included mother's education, age, ethnicity, and marital status. Another independent variable of interest was community size, defined as population size 250,000 to 500,000 or more than 500,000.

### **Research Design**

The two main types of methods that researchers may choose from are quantitative and qualitative. Quantitative designs allow researchers to predict, confirm, or test theories of phenomena (Cooper & Schindler, 2003) and are appropriate for examining the statistical significance of relationships between variables (Swanson & Holton, 2005). In contrast, qualitative investigations allow themes and ideas to inductively emerge from data, rather than through statistical tests of predetermined variables. Qualitative studies are ideal when researchers have smaller samples and desire to obtain in-depth, rich data rather than statistically significant, generalizable data from larger samples. Because of the small sample sizes, qualitative findings cannot be generalized to larger populations.

Because my purpose in this study was to examine the statistical significance of relationships between predetermined variables, I selected a quantitative method. In this way, I traded the depth and richness of a qualitative investigation for the statistical certainty afforded via quantitative investigation. In addition, a quantitative design allowed me to examine relationships across a larger U.S. population, which may produce more generalizable results.

I considered a number of quantitative designs for this investigation. First, I considered a systematic review, in which data from multiple, existing studies are combined and analyzed (Hoe & Hoare, 2012). This design is ideal for examining the

effectiveness of interventions; however, because the current research did not include an intervention, a systematic review was not selected. Similarly, I considered a nonrandomized trial, but because my aim in the investigation was not to explore the effects of an intervention, I did not select this design.

Another common quantitative design is a cross-sectional survey. This strategy is ideal when researchers aim to examine different factors related to study phenomena, captured for a specific timeframe (Hoe & Hoare, 2012). This is an appropriate design for gathering a large amount of numerical data across a sizeable sample, but it is methodologically weak for assessing causation. Because the current research did not require primary data collection, but used existing, archival data, a survey was not necessary. Thus, this study was a cross-sectional study because in it, I examined a point in time data by geography, which I analyzed as a secondary analysis of existing data.

Finally, I considered a quantitative, retrospective design. Panel data analysis is a form of longitudinal data analysis.

## **Methodology**

### **Population**

The population for this study included all counties (Appendix A) in the United States that were listed in the TRI database, in which the total annual release of airborne TRI toxins was more than 25,000. The three periods of analysis included 1987, 1995, and 2004. To protect families' privacy, infants' counties of residence ceased to be recorded in linked birth-death data after 2004. Including data from three separate years (in three separate analyses) allowed for discussion of the differences that might emerge in the relationships of these independent variables with IM, through time.

## **Sample and Sampling Procedures**

I collected data using purposive sampling, which is a nonprobability sampling strategy that is based on the characteristics of a population and the objectives of a study. Purposive samples are not based on participant size or any underlying theories. Instead, this sampling strategy allows researchers to specifically include participants that possess characteristics or information pertinent to the research phenomenon (Etikan, Musa, & Alkassim, 2016). A purposive sample allowed me to focus this study on individuals who reside in counties where at least 25,000 pounds of toxins are released into the atmosphere each year. I used all members of the population with available data in the sample, including all counties where TRI air releases of at least 25,000 pounds occurred during each year included in the study (1987, 1995, and 2004).

**Inclusion criteria.** To be included in the dataset, the TRI database had to indicate that total air emissions of all chemicals tracked by the TRI program for a given year were greater than 25,000 pounds. In addition, I only included counties with matching records from the linked birth-death data for a given year.

**Power analysis.** To ensure an adequate sample size was obtained to produce statistical certainty, I conducted a G\*Power analysis with OLS multiple regression criteria. The following parameters were used in the power analysis: alpha = 0.05, power = 0.80, and a medium effect size of  $f^2 = 0.15$  for the five independent variables being tested (community size, mothers' median education level, mothers' average age, percentage of mothers reporting non-Hispanic white ethnicity, and mothers' marital status). The G\*Power analysis indicated a minimum sample of 92 records were required. Because

there were approximately 200 individual county records for each of the 3 years of data, the number of records used in this study was more than sufficient to power each analysis.

### **Data Collection**

I relied on existing data. Data that I used for this investigation are available to the public, via websites for the Environmental protection Agency (EPA), the National Bureau of Economic Research, and the National Center for Health Statistics (NCHS). None of the data included in the current investigation required special permission to access or use. I downloaded data from the EPA's website, which provides TRI data for the years 1987 through 2016. For each year that I included (1987, 1995, and 2004), I downloaded and organized data into a spreadsheet. According to the EPA website, the quality of TRI data is high.

EPA works continuously to ensure that Toxic Release Inventory (TRI) data are accurate and reliable. Steps taken to promote data quality include analyzing data for potential errors, contacting TRI facilities concerning potentially inaccurate submissions, providing guidance on reporting requirements and, as necessary, taking enforcement actions against facilities that fail to comply with TRI requirements (EPA, n.d.). The online reporting tool guides facilities through the TRI reporting process and integrates a number of data quality checks to prevent errors before the submission of data.

In addition, I downloaded SES data from the website for the National Bureau of Economic Research and obtained linked birth-death data from the NCHS website. According to the NCHS (n.d.), most states routinely link birth and death data for infants. According to the website, birth and death data for infants is provided to the NCHS under



the Vital Statistics Cooperative Program, which codes data and implements quality control measures to ensure data are accurate. To process data, birth certificate numbers are obtained for all reported infant deaths that occur in a jurisdiction. From that information, the NCHS extracts final mortality records to create national, linked birth-death records. Next, any unlinked death certificates are followed up with to locate the correct birth certificate, and the link is performed. After obtaining data from their respective websites, I organized all information into a spreadsheet, which was then matched up data by county. Once organized, I uploaded data into SPSS for analysis.

### **Instrumentation**

The two main sources of data included in this study were (a) EPA's TRI data, and (b) linked birth and infant death files from the National Center for Health Statistics (NCHS). All data used in the study came from the linked birth and infant death files. The records were not linked at the individual infant level, only by cohort, and were summarized using statistical software so that the independent and dependent variables were available at the county level. The records included in the final dataset were determined by comparing these records to the EPA's TRI data. If a county were recorded as producing a total airborne TRI toxin release of more than 25,000 pounds in a given year, then that county's birth and infant death data were included in the dataset. This was done by merging the two datasets and matching records on county name; all data from counties that produced less than 25,000 pounds of airborne TRI toxins or did not appear in the TRI database were removed. The set was divided into three separate datasets: 1987, 1995, and 2004, which all contained the same variables.

### **TRI Database**

The TRI database began under the Emergency Planning and Community right-to-Know Act (EPCRA) of 1986 (EPA, 2010). This act requires reporting from manufacturing facilities that produce or process more than 25,000 pounds of specified chemicals included in the TRI list. Manufacturers must report any releases that occur from spills, routine emissions released into land, water, or sewage treatment plants. In addition, manufacturers must report any chemicals that were transferred to other locations to be recycled or disposed of (Hendryx & Fedorko, 2011).

As of 2016, the TRI chemical list included 595 individual chemicals (EPA, 2016). TRI is reported at the facility level, and separate reports must be filed for each monitored chemical (Agarwal et al., 2010). Information in the TRI database are classified by medium (air, water, land) and release site (on-site or off-site). Using the TRI database, I was able to determine the total number of pounds of specified chemicals released into the atmosphere for each county included in the analysis, for the specified date range. I chose 1987 as the first year for analysis, because although TRI data became available in 1986, it was incomplete. In addition, there were limitations on the birth-death data after the year 2004 (last year in analysis) that made it unsuitable for use following that year.

### **Linked Birth-Death Data**

Linked birth and death data for infants were obtained from the National Center for Health Statistics (NCHS), which contains linked birth and death certificate data for infants who die within the first year of life. These data were used to structure the dependent variable of IM per 1,000 live births.

Each year of the study (1987, 1995, and 2004) consisted of three separate data files (numerator, unlinked birth files, and denominator), which were offered as a numerator/denominator dataset; this provides a means for investigators to compute IM rates. The first file, which is known as the numerator file, includes linked records of live births and infant deaths for all US infants born in the given years (1987, 1998, and 2004) who died before their first birthday. The second file, also known as the unlinked file, contains information from the death certificate for all infant death records in the United States which could not be linked to their corresponding birth certificates, and the third file is the (1987, 1995, and 2004) NCHS natality file is referred as the denominator-plus file. These records are not linked at the infant level, but only by the overall cohort. Therefore, the files for each year were combined at a summary level using statistical software and county information which were available in both datasets.

Variables related to mother's SES were determined by mother's education, mother's age, race, and marital status. Any record that did not include this information was omitted from the dataset. All SES data on mothers were available via linked birth and death data from the NCHS. Annually produced NCHS natality and mortality files include statistical data from birth and death certificates that are provided by States under the Vital Statistics Cooperative Program (VSCP) to NCHS. Uniform coding specifications are used to code the data and have passed rigid quality control standards along with being edited and reviewed, and are the basis for official United States birth and death statistics (U.S. Department of Health and Human Services, 1992). The county of residence of infants ceased to be recorded in 2005, in order to protect the privacy of the families.

### **Operationalization of Variables**

The variables were operationalized as follows, through computations performed on the data set. Each variable was reported for any county that had more than 25,000 pounds of air released TRI chemicals in a specific year.

**Infant mortality (IM).** IM was a ratio variable, calculated as the number of infant deaths per 1000 live births in a county, for a given year.

**Community size.** Community size was a dichotomous (categorical) variable. This variable was coded for each county in a given year as 0 (population = 250,000 to 500,000) or 1 (population > 500,000).

**Mother's median education.** Mother's education was an ordinal variable, computed as the median level of education for each county in a given year. Education was coded as described in Table 2.

Table 2

*Coding for Mother's Education*

Code	Education level
0	No formal education
1-8	Years of elementary school
9	1 year of high school
10	2 years of high school
11	3 years of high school
12	4 years of high school
13	1 year of college
14	2 years of college
15	3 years of college
16	4 years of college
17	5 or more years of college

**Mother's average age.** Average mother's age was a ratio variable, calculated as the average age of all mothers in a county, in a given year.

**Percent non-Hispanic White mothers.** Mother's ethnicity was calculated as the percent of non-Hispanic white mothers. This ratio variable was calculated as the number of mothers in a county, for a given year, who reported being both non-Hispanic and white, divided by the total number of mothers with a known race and ethnicity.

**Percent married mothers.** Mother's marital status was a ratio variable, calculated as the number of mothers in a county, for a given year, who reported they were married, divided by the total number of mothers with a known marital status.

**County.** County was coded as a nominal categorical variable according to coding that already exists in the NCHS dataset.

**Year.** Year was coded as an interval variable according to the numerical year, with 1987 = 0, and each year following was coded as the number of years after 1987 (e.g., 1988 = 1, 1989 = 2, etc.).

Table 3

*Dependent and Independent Variables*

Variable type	Variable name	Variable source	Level of measurement
VARIABLES FROM DATA FILES			
Dependent	Infant mortality deaths per 1000 births	Linked birth and death data	Continuous
Independent	Community size	Linked birth and death data	Dichotomous
Independent	Mother's characteristics		
	1. Median mother's education	Linked birth and death data	Ordinal
	2. Percent non-Hispanic White mothers	Linked birth and death data	Continuous
	3. Percent married mothers	Linked birth and death data	Continuous

*Note.* This data set was created for data from 1987, from 1995, and 2004 (three separate data sets). The counties included in this data set for each year were determined by comparing to the counties included in the TRI database in which the total annual release of airborne TRI toxins was more than 25,000.

### Research Questions and Hypotheses

One linear regression model was tested to address the following research questions and associated statistical hypotheses:

**RQ1.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and community size?

**H1<sub>0</sub>** – Community size (small vs. large) is not a predictor of IM in the population of counties where more than 25,000 pounds of annual toxic air releases occur.

**H1<sub>A</sub>** – Community size (small vs. large) is a predictor of IM in a population of counties where more than 25,000 pounds of annual toxic air releases occur.

**RQ2.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and the following maternal characteristics (a) education level, (b) age, (c) ethnicity, and (d) marital status?

**H<sub>20</sub>** – Maternal characteristics are not predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

**H<sub>2A</sub>** – Maternal characteristics are predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

### **Data Analysis Plan**

I employed three separate linear regression models to test the research questions, one for each of the three years of the study. I used IBM SPSS Statistics 25 software for both descriptive and inferential analysis. A 0.05 level of significance was set for all inferential testing.

Prior to testing the hypotheses of this study, descriptive statistics were presented at the population level. Descriptive statistics consisted of mean, standard deviation, median, minimum, and maximum for IM, mother's median education, mother's average age, percent non-Hispanic white mothers, and percent married mothers. Summaries of frequencies (number and percent) were provided for community size. These statistics were calculated across all counties for each of the three years for which data were collected.

Assumptions for normality and homoscedasticity of the IM per 1,000 live births in a given community were performed in the form of descriptive statistics before I ran the linear regression model. I investigated multicollinearity of the independent variables before finalizing the analysis. Different combinations of assumption violations can

necessitate different approaches to treatment. Therefore, any remedies to assumption deviations were considered according to the type, number, and severity of the violation(s).

### Statistical Analyses

Research Question	Statistical Test	Variables	Interpretation	Significance
<b>RQ1:</b> In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and community size?	Linear Regression (three separate models for 1987, 1995, and 2004)	Community size	Estimated parameter for effect of community size Cohen's D	p-value < 0.05
<b>RQ2:</b> In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and the following maternal characteristics (a) education level, (b) age, (c) ethnicity, and (d) marital status?	Linear Regression (three separate models for 1987, 1995, and 2004)	Mother's median education Mother's average age Percent non-Hispanic white mothers Percent married mothers	Change in R-squared for model with and without four variables	Overall likelihood ratio test p-value <
			Individual estimated parameters for continuous variables (mother's average age, percent non-Hispanic white mothers, percent married mothers)	Individual p-values < 0.05
			Post-hoc pairwise comparisons for median education levels, if education significant overall	Overall F-test for education level p < 0.05 Tukey-adjusted p-values < 0.05

Note: both research questions were tested in the same linear regression model with all five independent variables present.

Note: three identical models was run: one for 1987, one for 1995, and one for 2004, each answering both research questions for that cohort

### Threats to Validity

It was important to acknowledge potential threats to validity. Internal validity is concerned with the validity of the claims that emerge from the analysis, while external



validity is concerned with the generalizability of those claims. Although the archival data upon which analysis was based is the result of government-mandated collection and entry, it was possible that some records were incomplete, inaccurate, or incorrectly entered into the databases. Further, when including multiple independent variables and covariant, it is possible that unaccounted for conditions may affect the relationships between the variables. Further, because the current research was not experimental and relied on purposive sampling, findings may not be generalizable to counties and years beyond the scope of the current investigation.

### **Limitations of the Research**

The study was conducted with major financial constraints; otherwise, a prospective cohort study would have been preferred. A prospective cohort study is a desirable study design for sociological and environmental investigations (Manolio et al., 2006), such as an investigation of factors that may influence IM in the United States. The distinctive quality of a prospective cohort study is that when researchers begin enrolling subjects and collecting baseline information, none of the subjects have developed any of the outcomes of interest. After baseline data are collected, longitudinal data are collected on subjects to determine if and when individuals may expire, and why. In this way, researchers can eventually use the data to answer many questions about the associations between risk factors and mortality outcomes. The hypotheses observe the relationship between IM per capita and the weighted amount of airborne toxic releases. Despite voluminous anecdotal evidence implicating air pollution on mortality, only limited epidemiologic research on an apparent association between airborne toxic releases and IM has been published.

The current investigation was also limited to the accuracy of information entered into the databases from which the study dataset were compiled. Although these databases undergo rigorous quality checks, it is always possible that human errors may occur during the data entry process. Another limitation was the timeframe for which data were available. As previously mentioned, the county of residence for infants ceased to be recorded after 2004, in order to protect the privacy of families. The TRI database was started in 1986, but was incomplete that year, making 1987 the first complete year of data collection. Consequently, three years chosen for analysis were 1987, 1995, and 2004.

### **Ethical Considerations**

Before any data were collected for the current study, I obtained study permission from Walden University's Institutional Review Board (IRB). All study data were de-identified, publicly available records; thus, issues related to confidentiality were not present. In addition, because data were retrospective and anonymous, no potential harms or threats existed for any individuals included in the sample. The anonymous nature of all records prevented the need to address ethical issues related to participant identifiers. I will store all study-related data on my personal, password-protected computer for a period of 5 years. After that period has passed, I will have all study-related data destroyed.

### **Summary**

In this chapter, I described the methods of the current investigation. The aim of this study was to examine the potential relationships between IM, county size, and factors related to mother's SES in counties where more than 25,000 pounds of annual toxic air releases occur. The study followed a retrospective, cross-sectional design. The dependent

variable was IM per 1000 live births in a given community for each of the three years included in this analysis (1987, 1995, and 2004). The independent variables included county size and factors related to mother's SES (education, age, ethnicity, and marital status). Archival, publicly available data were pulled from (a) the Environmental Protection Agency's (EPA) Toxic Release Inventory (TRI) data, and (b) linked birth and infant death files from the National Center for Health Statistics (NCHS). The research questions were tested via a single panel regression model.

This chapter included an overview of the current study's methods, including the design, population, data collection procedures, instrumentation, data analysis plan, and ethical assurances. In addition, I described study limitations and threats to validity. Study results are presented in the following chapter. A discussion of findings is provided in Chapter 5.

## Chapter 4: Results

### Introduction

My purpose in this study was to examine the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. The dependent variable was IM, and independent variables included county size, with two levels of population size (250,000 to 500,000 or > 500,000), and variables related to mother's SES, including mother's (a) age, (b) ethnicity, and (c) marital status. My goal was to investigate whether community size and SES-related factors had a modifying effect on the relationship between IM and residence in counties where more than 25,000 pounds of annual TRI toxic releases occur. The study was guided by the following research questions and hypotheses:

**RQ1.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and community size?

**H1<sub>0</sub>.** Community size (small vs. large) is not a predictor of IM in the population of counties where more than 25,000 pounds of annual toxic air releases occur.

**H1<sub>A</sub>.** Community size (small vs. large) is a predictor of IM in a population of counties where more than 25,000 pounds of annual toxic air releases occur.

**RQ2.** In populations where more than 25,000 pounds of annual toxic air releases occur, is there an association between IM and the following maternal characteristics (a) education level, (b) age, (c) ethnicity, and (d) marital status.

**H2<sub>0</sub>.** Maternal characteristics are not predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

**H2<sub>A</sub>**. Maternal characteristics are predictors of IM in counties where more than 25,000 pounds of annual toxic air releases occur.

This chapter includes a presentation of study results. It begins with a description of the data collection technique, followed by detailed findings from the analysis. I organized results by variables examined, accompanied by appropriate charts and graphs for illustration. The chapter closes with a brief summary and transition to Chapter 5.

### **Data Collection**

I used retrospective, publicly available data for this investigation. I accessed all data via websites for the EPA, the National Bureau of Economic Research, and the NCHS) None of the data used in this analysis required special permission to access or use. The primary data source for this study was linked infant birth and death data, which was pulled from the National Bureau of Economic Research (NBER) website (originally collected from the NCHS).

I pulled data for the years 1987, 1995, and 2004. I stored the original data obtained from the NBER in two separate files for each year. One file was called the *denominator* file and the other was called the *numerator* file. The denominator file contained information about all infants born in the United States during a given year, and the numerator file contained information about all infants who died within 1 year of their date of birth. Together, these files were used to calculate IM rates. The individual records were not linked across the two files; thus, data from the two files could not be easily combined at the individual record level. Instead, I combined data at an aggregate level. I aggregated information at the county level and included the following:

- IM rate, which was calculated as number of records in the numerator file divided by number of records in the denominator file, multiplied by 1,000 to represent number of deaths per 1,000 births.
- Mother's median education, as years of formal education.
- Mother's average age, in years.
- Percentage of non-Hispanic white mothers.
- Percentage of married mothers.
- Community size, categorized as 250,000 to 500,000, or more than 500,000.

### **Study Data Considerations**

A few important considerations about study data must be noted. First, not all counties recorded mother's education level, so this information was missing for some observations. In addition, ethnicity and race information was not collected consistently across all counties in 1987. Thus, the data for 1987 only includes *percentage White mothers*, without regard for ethnicity. Finally, community size data were only available for 2004; however, it should be noted that all included counties had populations greater than 250,000 for all years observed. The second data source was pulled from the EPA's TRI. I used TRI data produced for the years 1987, 1995, and 2004. I used these data only to identify the counties that recorded a total annual release of more than 25,000 pounds of airborne toxic chemicals.

I merged the two datasets to match the annual toxic release numbers to the county-level birth cohort data. Next, I identified and retained data from counties where annual toxic air releases were more than 25,000 pounds; counties with fewer than 25,000

pounds of annual toxic air releases were removed from the dataset. Because of restrictions to the individual datasets, the final dataset included records for counties with populations of 250,000 or more, and with annual toxic air releases of 25,000 pounds or more.

### Description of Data

**Infant mortality.** Summary statistics of IM data are provided in Table 4. In the table,  $n$  represents the number of observations for each year. The mean refers to the average rate of IM (calculated as the average number of infants who died per 1,000 births, across all counties). As illustrated in the table, average rates of IM decreased with time.

Table 4

#### *Summary Statistics of Infant Mortality Rates for Each Year*

Year	$n$	Mean	Median	$SD$	Minimum	Maximum
1987	173	9.85	9.86	3.29	2.77	20.03
1995	190	7.48	7.15	2.99	1.36	17.27
2004	211	7.11	6.78	3.34	0.00	19.87

**Mother's median education level.** Summary statistics of mother's median education level are provided in Table 5. In this data, each whole number represents a level of schooling, in such that 12 represents completion of high school, which consists of 12 grade levels. Accordingly, 13 represents completion of the first year of college. Observing this data then, it is evident that between 1987 and 2004, the average level of

education increased slightly, with women in 2004 having completed the first year of college, on average.

Table 5

*Summary Statistics of Mother's Median Education Level for Each Year*

Year	<i>n</i>	Mean	Median	<i>SD</i>	Minimum	Maximum
1987	142	12.23	12.00	0.541	12	14
1995	190	12.63	12.00	0.836	11	15
2004	172	13.04	13.00	1.220	11	16

**Mother's average age.** Summary statistics of mother's average age are provided in Table 6. On average, mother's age at child's birth increased slightly in the course of the years examined, from 26.47 in 1987 to 27.87 in 2004.

Table 6

*Summary Statistics of Mother's Average Age for Each Year*

Year	<i>n</i>	Mean	Median	<i>SD</i>	Minimum	Maximum
1987	173	26.47	26.35	0.981	24.38	29.29
1995	190	27.35	27.18	1.269	24.32	30.77
2004	211	27.87	27.72	1.391	24.91	31.91

**Percentage non-Hispanic White mothers.** Summary statistics of percentage non-Hispanic White mothers for 2004 are provided in Table 7. On average, the nearly 60% of mothers were non-Hispanic White for that year.



Table 7

*Summary Statistics of Percentage Non-Hispanic White Mothers for 2004*

<i>n</i>	Mean	Median	<i>SD</i>	Minimum	Maximum
211	59.70	63.57	20.275	3.7	96.8

**Percentage of married mothers.** Summary statistics of percentage married mothers for each year are provided in Table 8. On average, the rate of marriage decreased during the years examined. The average rate of marriage among mothers dropped from 76.78 in 1987 to 65.51 in 2004.

Table 8

*Summary Statistics of Percentage Married Mothers for Each Year*

Year	<i>n</i>	Mean	Median	<i>SD</i>	Minimum	Maximum
1987	173	76.78	77.22	8.558	43.3	93.7
1995	190	69.78	69.85	9.991	32.4	92
2004	211	65.51	65.29	10.103	26.1	91

**Community size.** Finally, Table 9 provides is a frequency table of the distribution of community size data for 2004. As noted previously, community data were only available for 2004. For this year, about half (51.2%) of the included communities had populations between 250,000 and 500,000, and the other half (48.8%) had populations larger than 500,000. To note, *all* counties included in the final dataset had populations of 250,000 or more.

Table 9

*Distribution of Community Size for 2004 Data*

Community Size	Frequency	Percentage
250,000 to 500,000	108	51.2
> 500,000	103	48.8
Total	211	100

**Results****Analysis of Data for 1987**

Prior to analyzing data for 1987, the outcome of *IM rate* was visually examined for an approximate normal distribution. The assumption of the multiple linear regression is that the residuals must be normally distributed; however, examining the distribution of the dependent variable prior to modeling provides insights as to whether the assumption is likely to be met. Figure 1 is a histogram of the IM rate data for 1987, across all counties.

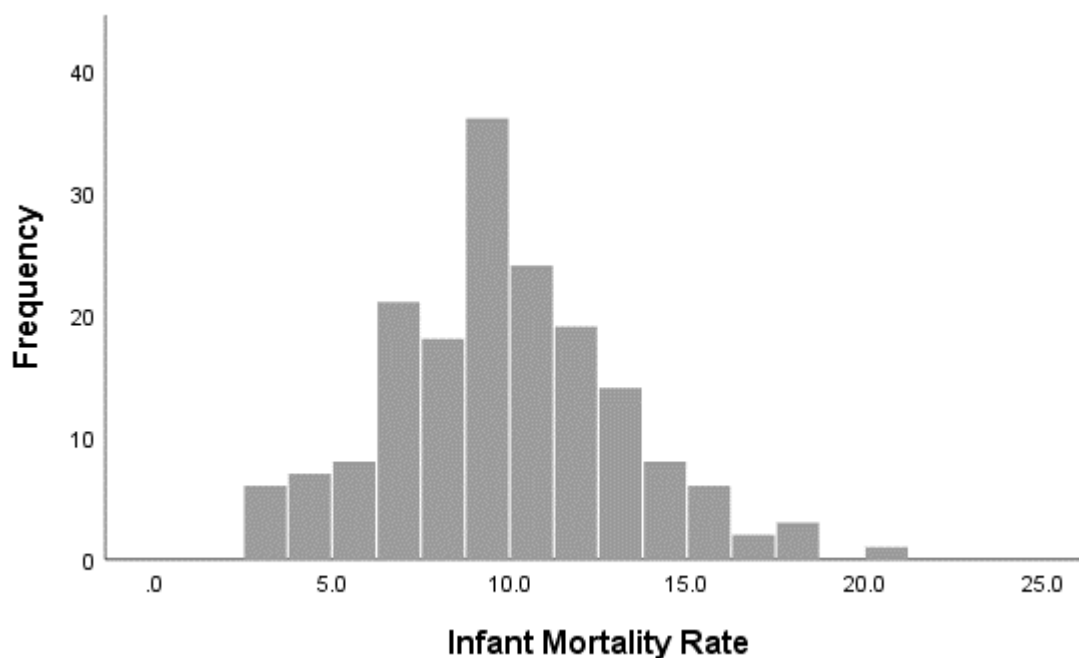


Figure 1. Distribution of infant mortality rate in 1987 across counties.

As illustrated in Figure 1, the IM rate was approximately normally distributed. As such, the researcher conducted the multiple linear regression, as planned. Note from Table 5 that only 142 of the 173 observations included a record of mothers' median education; thus, only those 142 records were used in this model.

Table 10

*Results of Multiple Linear Regression for 1987 Data*

Variable	Est. coefficient	SE	t	df	p
Intercept	39.891	6.862	5.81	137	<.001
Mothers' median education	0.955	0.597	1.60	137	.112
Mothers' average age	-1.175	0.363	-3.24	137	.002
Percentage White mothers	-0.102	0.025	-4.10	137	<.001
Percentage married mothers	-0.029	0.047	-0.61	137	.543

Results of the linear regression are provided in Table 10, including estimated coefficients, standard errors, t statistics, and *p*-values. The  $R^2$  of this model was 0.414, indicating that the independent variables explained 41.4% of the variation in IM rates, across counties. On average, IM increased by 0.995 per 1,000 live births as mother's median education level increased by 1 year. As mother's average age increased by 1 year, IM decreased by 1.175 per 1,000 live births. Decreases in IM rates were also evident for White mothers and married mothers. IM rate decreased by 0.102 per 1,000 births when percentage White mothers increased by 1; and IM rates decreased by 0.029 per 1,000 births when percentage married mothers increased by 1. Thus, overall, increases in IM rates during 1987 were associated with increased in education level, and decreases were associated with older mothers, White mothers, and married mothers.

**Statistical analysis.** T-statistics, degrees of freedom, and p-values were used to draw several conclusions about the data at the 0.05 level of significance. First, the relationship between mother's median education to IM was not significant, ( $t(137) = 1.60, p = 0.112$ ). The relationship between mother's average age and IM rate was significant, ( $t(137) = -3.24, p = 0.002$ ). The relationship between percentage White mothers and IM rate was significant, ( $t(137) = -4.10, p < 0.001$ ). Finally, the relationship between percentage married mothers and IM rate was not significant, ( $t(137) = -0.61, p = 0.543$ ).

**Assumptions.** Variance inflation factors (VIF) are a way to determine whether the correlation among the independent variables (collinearity) is too high to include all of them in the model together; these calculations are provided in Table 11.

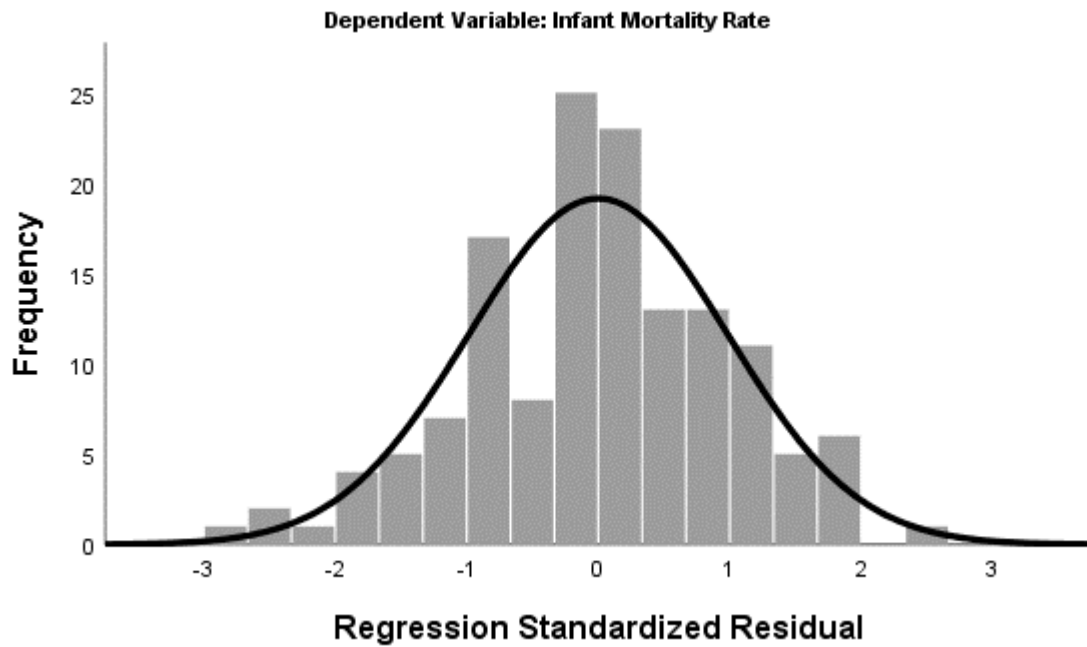
Table 11

*Variance Inflation Factors for 1987 Data*

Variable	VIF
Mothers' Median Education	2.029
Mothers' Average Age	2.530
Percentage White Mothers	2.476
Percentage Married Mothers	3.502

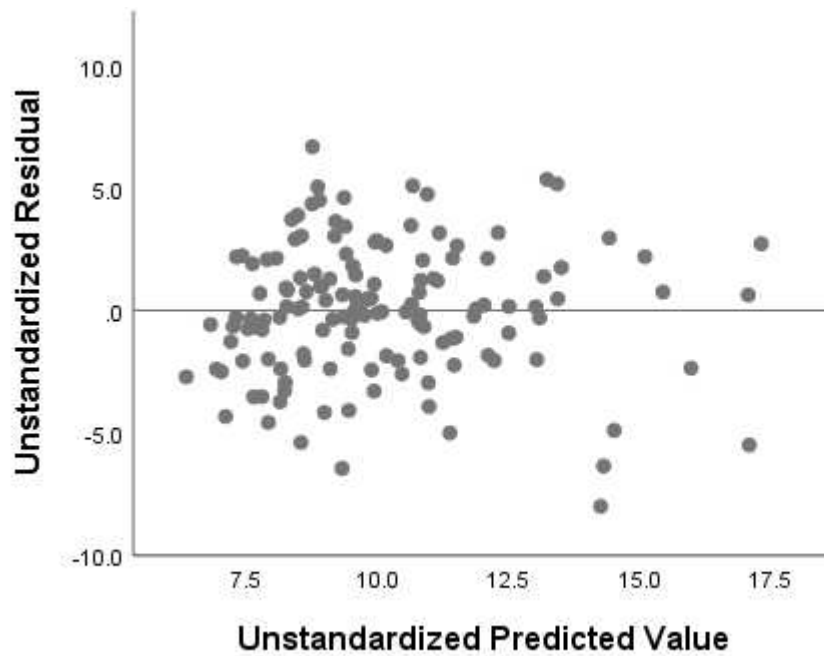
Although various authors have suggested different cutoffs to indicate collinearity of the variables that is too high to include in a multiple linear regression (Hail, Anderson, Tatham, & Black, 1995), a commonly used threshold is  $VIF = 10$ . Because none of the calculated VIFs approached this limit, the level of collinearity across the independent variables was considered acceptable.

Another assumption of multiple linear regression is that the residuals are approximately normally distributed. Figure 2 provides a histogram of the residuals, with the density curve of a normal distribution superimposed. The residuals appear to be acceptably normally distributed.



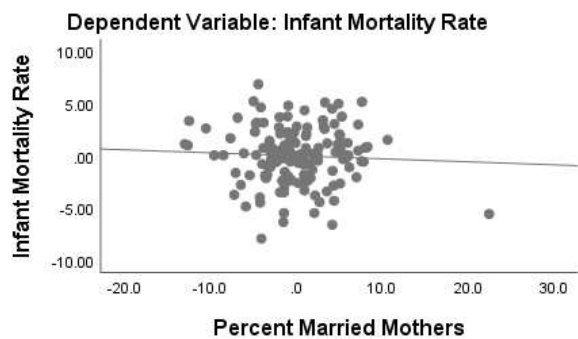
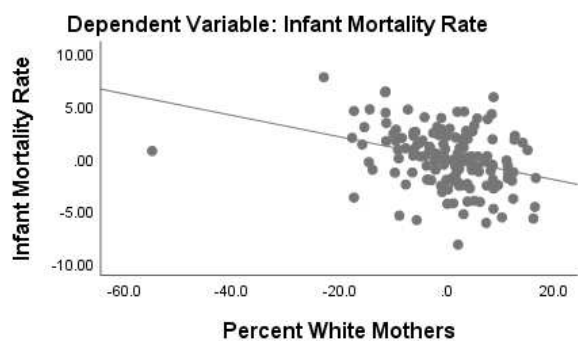
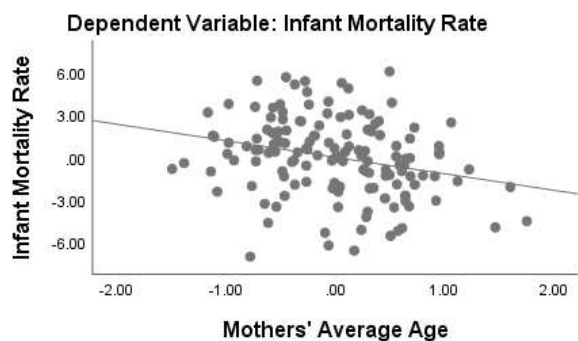
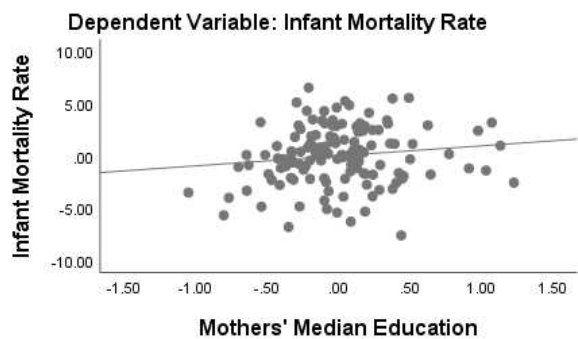
*Figure 2.* Histogram of model residuals for 1987 data.

Another assumption of linear regression is that the residuals will be homogeneous; this assumption is commonly checked via a plot of residuals versus predicted values, as illustrated in Figure 3. Based on these scatterplots, the assumption of homogeneity was met.



*Figure 3.* Plot of residuals versus predicted values for 1987 data.

A final assumption was that a linear relationship existed between the independent variables and the dependent variable. This assumption was checked via partial regression plots. These plots are provided in Figures 4a through 4d, revealing no major departures from linearity. Accordingly, the assumption of linearity was met.



Figures 4a-4d. Partial regression plots for 1987 data



**Exclusion of education variable.** Before completing analysis for the 1987 data, a second analysis was run because there were a number of counties that did not record mothers' median education. This analysis allowed the researcher to determine whether the exclusion of education as a variable (and inclusion of the previously missing observations) resulted in any changes to the model's conclusions. Results of this analysis are provided in Table 12. The  $R^2$  of this model is 0.384.

Table 12

*Results of Multiple Linear Regression for 1987 Data, Without Mothers' Median*

*Education*

Variable	Est. Coefficient	Std. Error	t	df	p
Intercept	42.31	5.723	7.39	169	< 0.001
Mothers' Average Age	-0.827	0.258	-3.21	169	0.002
Percentage White Mothers	-0.100	0.021	-4.71	169	< 0.001
Percentage Married Mothers	-0.032	0.040	-0.80	169	0.426

Comparing Table 12 to Table 10, virtually no differences exist in the p-values or the conclusions drawn from them. The model that includes education to the exclusion of some observations appears to be reasonable.

### **Analysis of Data for 1995**

Prior to analyzing data for 1987, the outcome *IM rate* was visually examined for an approximate normal distribution, Figure 5 is a histogram of these values.

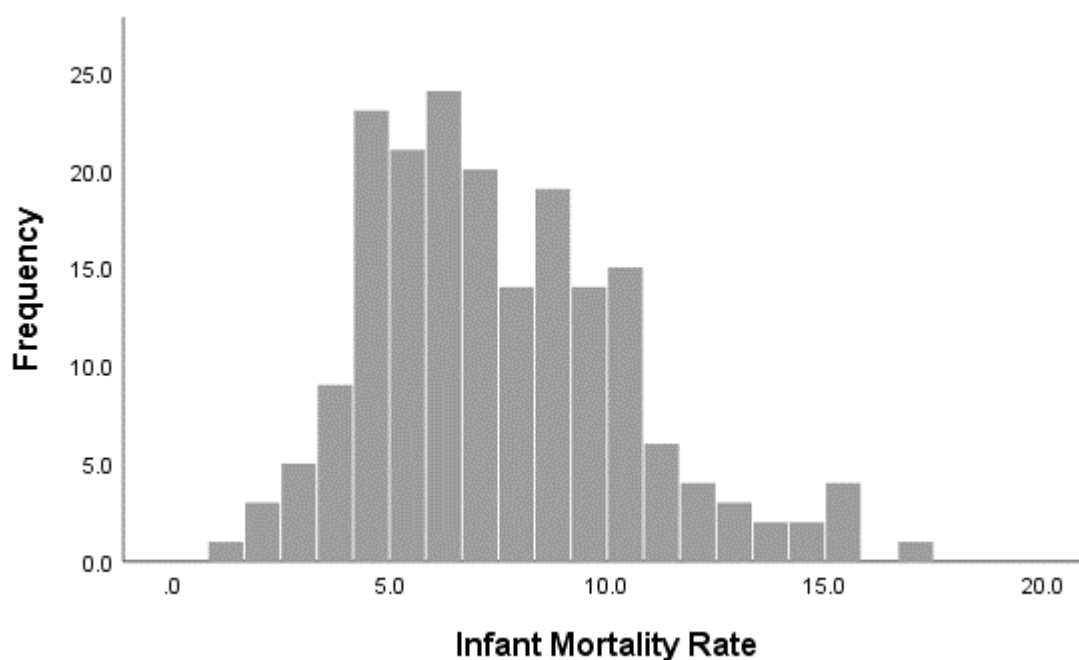


Figure 5. Distribution of IM rate in 1995 across counties.

As illustrated in Figure 5, the IM rate was approximately normally distributed. As such, the researcher conducted the multiple linear regression, as planned.

Table 13

*Results of Multiple Linear Regression for 1995 Data*

Variable	Est. Coefficient	Std. Error	t	df	p
Intercept	21.755	4.244	5.126	185	< 0.001
Mothers' Median Education	0.771	0.359	2.151	185	0.033
Mothers' Average Age	-0.459	0.244	-1.885	185	0.061
Percentage Non-Hispanic White Mothers	0.017	0.010	1.673	185	0.096
Percentage Married Mothers	-0.180	0.026	-6.814	185	< 0.001

Results of the linear regression are provided in Table 13, including estimated coefficients, standard errors, t statistics, and *p*-values. The  $R^2$  of this model is 0.321,

indicating that the independent variables explained 32.1% of the variation in IM rates, across counties. On average, IM rate increased by 0.771 per 1,000 births when mothers' median education increased by 1 year. As mother's average age increased by 1 year, IM rates decreased by 0.459 per 1,000 live births. IM rate decreased by 0.017 per 1,000 live births as percentage of non-Hispanic White mothers increased by 1. As percentage of married mothers increased by 1, IM rate decreased by 0.180 per 1,000 live births.

**Statistical analysis.** T-statistics, degrees of freedom, and p-values were used to draw several conclusions about the data at the 0.05 level of significance. First, there was a statistically significant relationship between mother's median education level and IM rate, ( $t(185) = 2.151, p = 0.033$ ). The relationship between mother's average age and IM rate was not significant, ( $t(185) = -1.885, p = 0.061$ ). The relationship between percentage non-Hispanic White mothers and IM was not significant, ( $t(185) = 1.673, p = 0.096$ ). Finally, a statistically significant relationship did exist between percentage married mothers and IM rate, ( $t(137) = -6.814, p < 0.001$ )

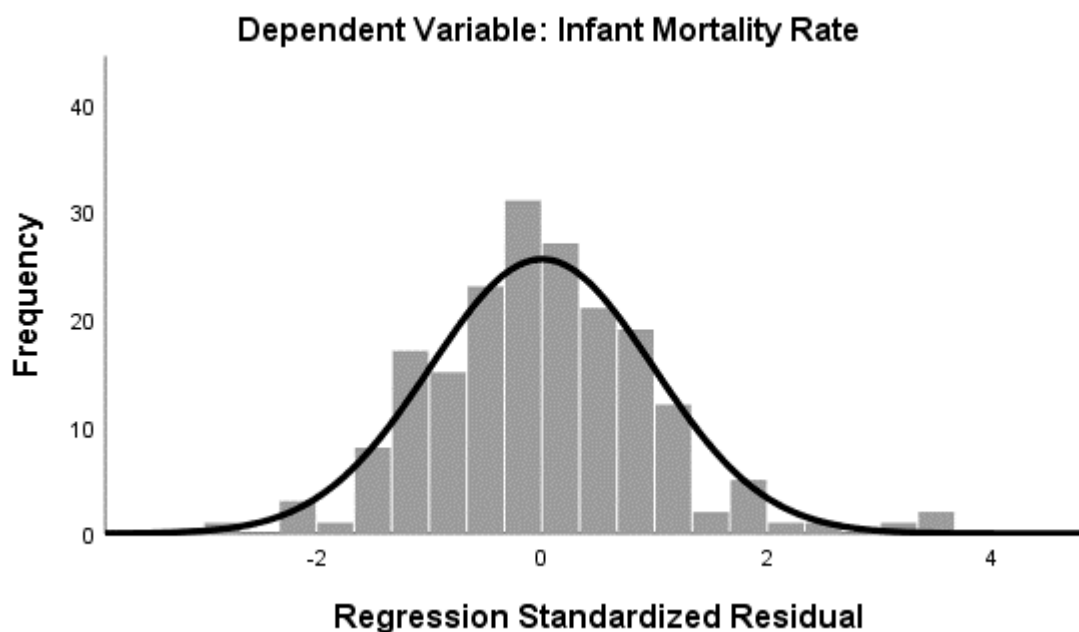
**Assumptions.** Variance of inflation factors are provided in Table 14. Because none of the calculated VIFs approached the limit of 10, the level of collinearity across the independent variables was considered acceptable

Table 14

*Variance Inflation Factors for 1995 Data*

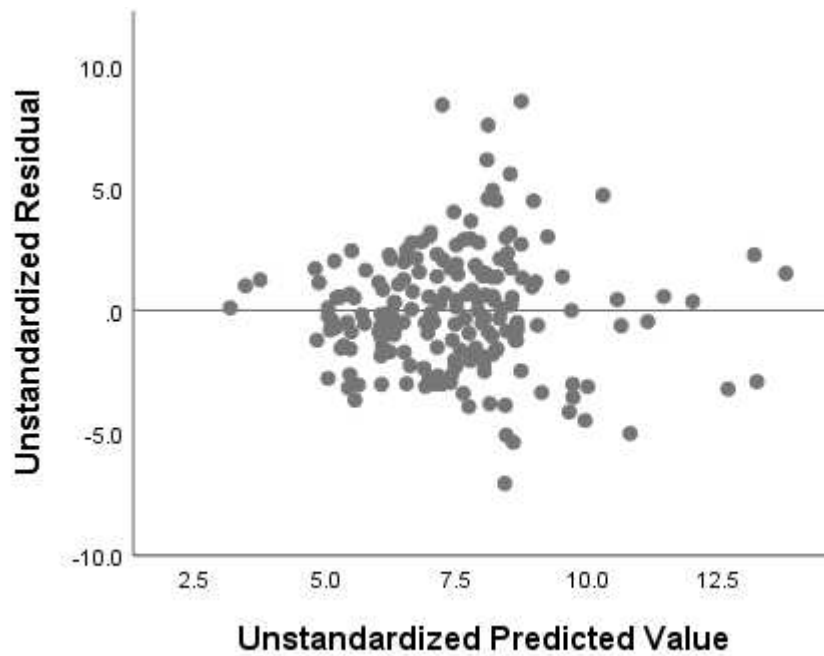
Variable	VIF
Mothers' Median Education	2.742
Mothers' Average Age	2.912
Percentage Non-Hispanic White Mothers	1.397
Percentage Married Mothers	2.135

Figure 6 provides a histogram of the residuals, with the density curve of a normal distribution superimposed. The residuals appear to be acceptably normally distributed.



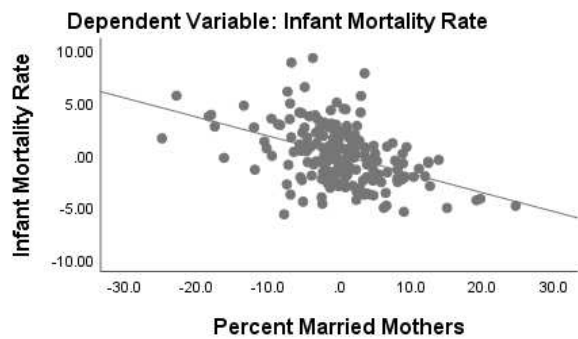
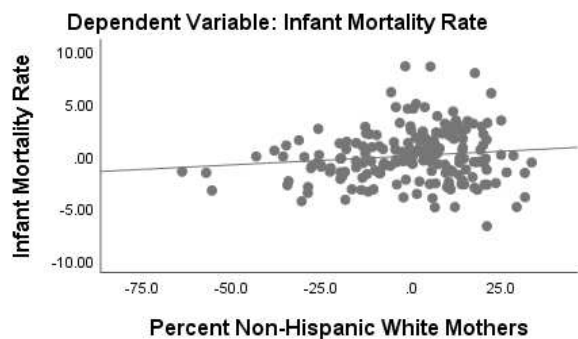
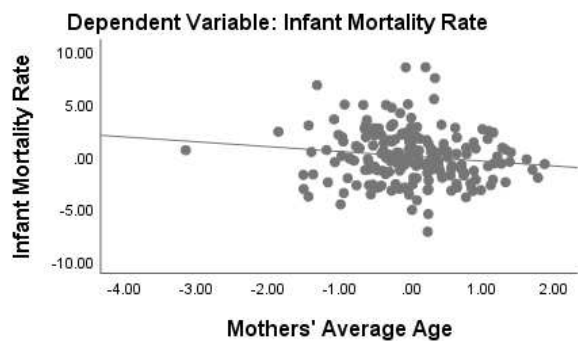
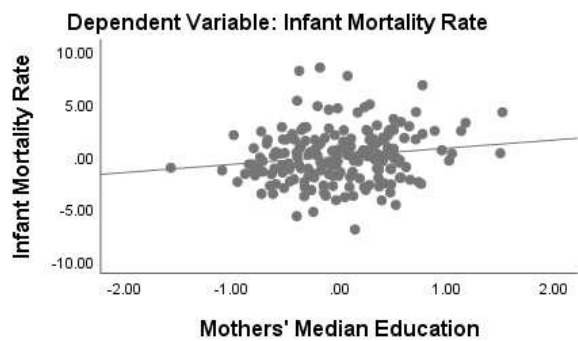
*Figure 6.* Histogram of model residuals for 1995 data.

As illustrated in Figure 7, the assumption of homogenous variance was checked via a plot of residuals versus predicted values. The variance appeared relatively homogenous for all predicted IM rates; thus, this assumption was met.



*Figure 7.* Plot of residuals v. predicted values for 1995 data.

A final assumption was that a linear relationship existed between the independent variables and the dependent variable. This assumption was checked via partial regression plots. These plots are provided in Figures 8a through 8d, revealing no major departures from linearity. Accordingly, the assumption of linearity was met.



Figures 8a-8d. Partial regression plots for 1995 data.

### Analysis of Data for 2004

As in the previous two analyses, prior to analysis of data for 2004, the outcome of *IM rate* was visually examined for an approximate normal distribution. Figure 9 provides a histogram of these values.

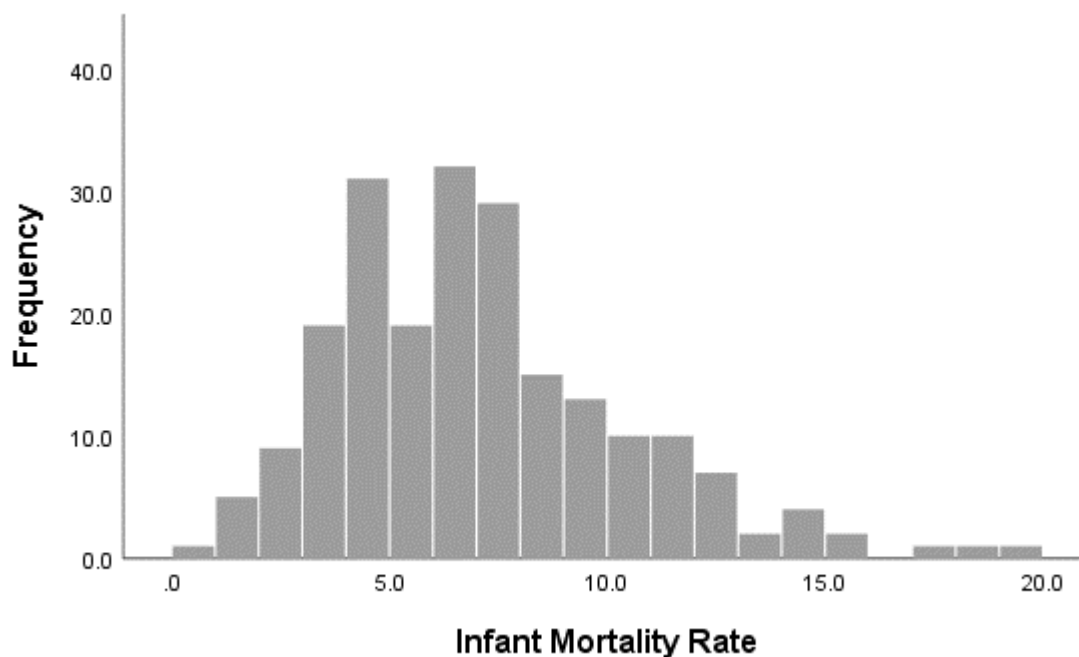


Figure 9. Distribution of IM rate in 2004 across counties.

As illustrated in Figure 9, the IM rate did not have extreme outliers. The distribution was slightly right-skewed; however, the distribution appeared close enough to normally distributed that accounting for the independent variables may result in normally distributed residuals. Thus, the researcher cautiously conducted the multiple linear regression, as planned. Note from Table 5 that only 172 of the 211 observations included a record of mothers' median education; thus, only those 172 records were used in this model.

Table 15

*Results of Multiple Linear Regression for 2004 Data*

Variable	Est. Coefficient	Std. Error	t	df	p
Intercept	29.007	5.513	5.26	166	< 0.001
Mothers' Median Education	1.006	0.368	2.74	166	0.007
Mothers' Average Age	-0.938	0.32	-2.93	166	0.004
Percentage Non-Hispanic White Mothers	0.017	0.013	1.27	166	0.206
Percentage Married Mothers	-0.164	0.035	-4.69	166	< 0.001
Community Size (> 500,000)	1.426	0.495	2.88	166	0.004

Results of the linear regression are provided in Table 15, including estimated coefficients, standard errors, t statistics, and *p*-values. The  $R^2$  of this model was 0.301, indicating that the independent variables explained 30.1% of the variation in IM rates, across counties. On average, IM increased by 1.006 per 1,000 births when mother's median education increased by 1 year. IM decreased by 0.938 per 1,000 births when mothers' average age increased by 1 year. IM increased by 0.017 per 1,000 births when percentage non-Hispanic White mothers increased by 1. An IM rate decrease of 0.164 per 1,000 births occurred as percentage married mothers increased by 1. Finally, IM rate increased by 1.426 per 1,000 births when community size increased to > 500,000 (as compared to 250,000 - 500,000).

**Statistical analysis.** T-statistics, degrees of freedom, and *p*-values were used to draw several conclusions about the data at the 0.05 level of significance. First, a statistically significant relationship existed between mother's median education level and IM rate, ( $t(166) = 2.74$ ,  $p = 0.007$ ). The relationship between mother's average age and IM rate was also significant, ( $t(166) = -2.93$ ,  $p = 0.004$ ). The relationship between



percentage non-Hispanic White mothers and IM rate was not significant, ( $t(166) = 1.27$ ,  $p = 0.206$ ). The relationship between percentage married mothers and IM rate was statistically significant, ( $t(166) = -4.69$ ,  $p < 0.001$ ). Finally, a statistically significant relationship existed between community size and IM rate, ( $t(166) = 2.88$ ,  $p = 0.004$ ).

**Assumptions.** Variance inflation factors are provided in Table 16. Again, none of these factors were close to the cutoff of 10, indicating an acceptable level of multicollinearity.

Table 16

*Variance Inflation Factors for 2004 Data*

Variable	VIF
Mothers' Median Education	4.421
Mothers' Average Age	4.539
Percentage Non-Hispanic White Mothers	1.661
Percentage Married Mothers	2.958
Community Size	1.351

Figure 10 is a histogram of the residuals to check the normal distribution assumption. The residuals appeared to be acceptably normally distributed.

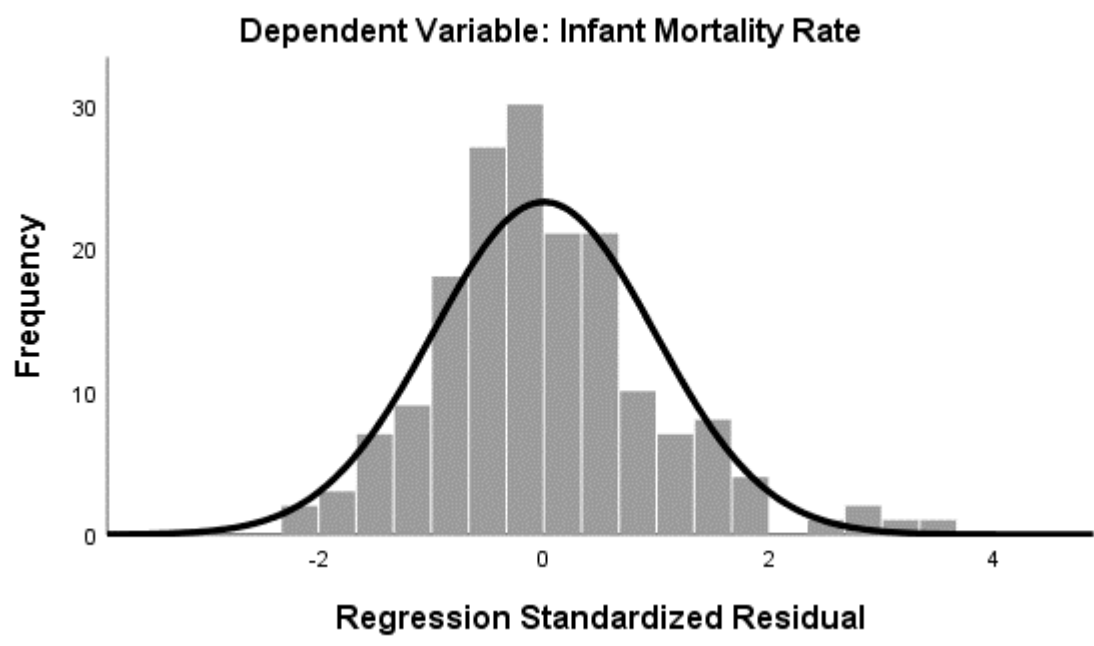
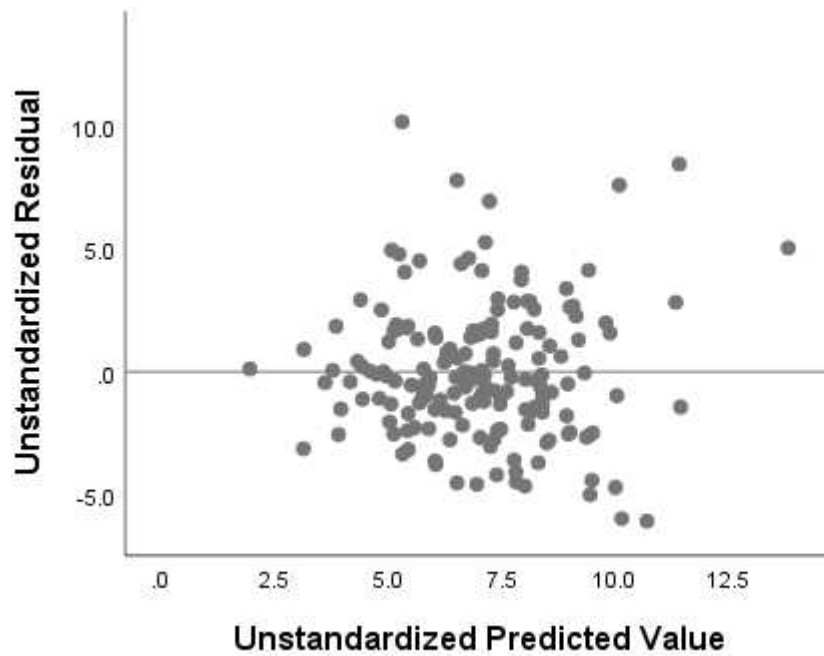


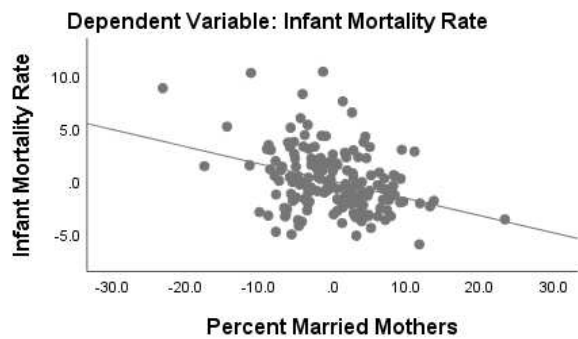
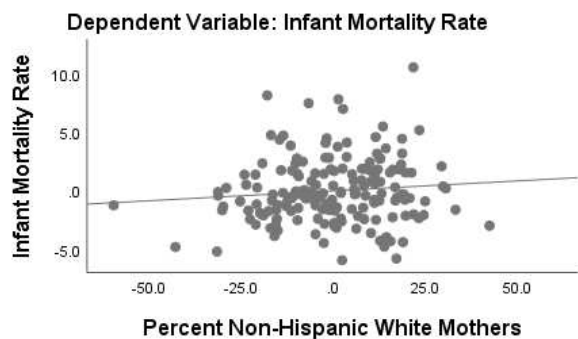
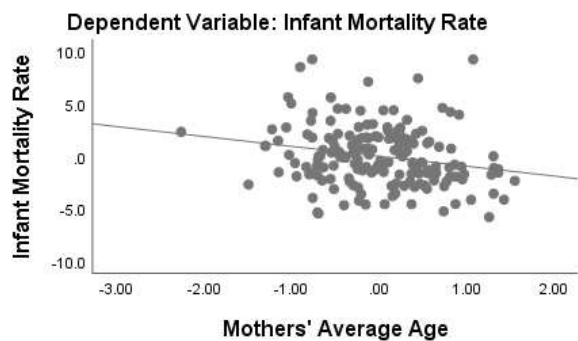
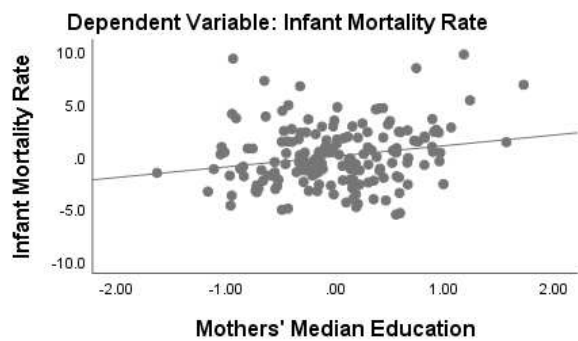
Figure 10. Histogram of model residuals for 2004 data.

The assumption of homogenous variance was checked via a plot of residuals, as illustrated in Figure 11. The variance appeared relatively homogeneous for all predicted IM rates; thus, this assumption was met.



*Figure 11.* Plot of residuals v. predicted values for 2004 data

As illustrated in Figures 12a through 12d, partial regression plots were used to check for linearity of relationships of the continuous independent variables to the dependent variable. These plots revealed no major departures from linearity. Accordingly, the assumption of linearity was met.



Figures 12a-12d. Partial regression plots for 2004 data

**Exclusion of education variable.** Before concluding analysis of the 2004 data, a second analysis was run to determine whether the exclusion of education as a variable (and inclusion of the previously missing observations) resulted in any changes to the model's conclusions. This analysis was conducted because there were a number of counties that did not record mothers' median education. The results of this model are provided in Table 17. The  $R^2$  of this model is 0.270.

Table 17

*Results of Multiple Linear Regression for 2004 Data, Without Mothers' Median*

*Education*

Variable	Est. Coefficient	Std. Error	t	df	p
Intercept	23.996	5.001	4.80	206	< 0.001
Mothers' Average Age	-0.336	0.23	-1.46	206	0.145
Percentage Non-Hispanic White Mothers	0.022	0.012	1.78	206	0.077
Percentage Married Mothers	-0.142	0.033	-4.29	206	< 0.001
Community Size	0.939	0.468	2.01	206	0.046

From the model provided in Table 17, a statistically significant effect of mothers' average age no longer existed when mothers' median education was excluded. This could mean that mothers' average age was only significant after accounting for mothers' median education. However, other effects remain significant or insignificant as in the previous model, and the directions (positive or negative) of all coefficients remain the same.

### Summary

Table 18 provides a summary of the outcomes that were statistically significant in each of the three models. As illustrated, the relationship between mother's median education and IM was not significant in 1987, but an increase in mother's median age was significantly associated with increases in IM during 1995 and 2004. The relationship between mother's average age and IM was not significant in 1995; however, a decrease in mother's age was significantly associated with increased IM during 1987 and 2004. The relationship between percentage non-Hispanic White mothers and IM rate was not significant in 1995 and 2004; however, a decrease in percentage non-Hispanic White mothers was significantly associated with an increase in IM during 1987. The relationship between percentage married mothers and IM rate was not significant in 1987; however, a decrease in percentage married mothers was significantly associated with increased IM rates in 1995 and 2004. Finally, in 2004 (the only year for which community size data were available), an increase in community size was significantly associated with an increase in IM rate.

Table 18

*Summary of Outcomes for Each Variable Analyzed*

Variable	Year		
	1987	1995	2004
Mothers' Median Education	Not sig.	Sig., +	Sig., +
Mothers' Average Age	Sig., -	Not sig.	Sig., -
Percentage [Non-Hispanic] White Mothers	Sig., -	Not sig.	Not sig.
Percentage Married Mothers	Not sig.	Sig., -	Sig., -
Community Size	-	-	Sig., +

**RQ1.** The first research question asked whether a statistically significant relationship existed between IM and community size in populations where more than 25,000 pounds of annual toxic air releases occurred. Data for 2004 (the only year for which community size data were available), revealed that an increase in community size was significantly associated with an increase in IM rate. Thus, the null hypothesis was rejected.

**RQ2.** The second research question asked whether a statistically significant relationship existed between IM and three different maternal characteristics (education level, age, ethnicity, and marital status) in populations where more than 25,000 pounds of annual toxic air releases occurred. Findings for this research question were mixed for the three years examined. Essentially, while different variables were significant during different years, the direction of significance (positive or negative) was similar. Thus, the null hypothesis may be accepted for some relationships and rejected for others, as discussed above.

This chapter provided detailed findings from the statistical analysis. Included in this chapter was a discussion of the data collection, descriptive statistics, and results of the analysis for the research questions. An in-depth discussion of study findings and implications appears in the following chapter.

## Chapter 5: Discussion

### **Introduction**

My purpose in this quantitative study was to examine the potential relationships between IM, county size, and factors related to mothers' SES in counties where more than 25,000 pounds of annual toxic air releases occur. I followed a retrospective, longitudinal design, which allowed me to explore the relationships between IM, county size, and factors related to mothers' SES. In my research, I addressed a gap in the existing body of literature, as previous scholarship on the relationship between exposure to toxic air pollution and IM rates failed to concurrently examine multiple factors that may influence IM. The holistic examination provided in this project shed new light on the relationship between IM and exposure to toxic air pollution.

For the first research question, which asked whether a statistically significant relationship existed between IM and community size in populations where more than 25,000 pounds of annual toxic air releases occurred, analysis revealed that an increase in community size was significantly associated with an increase in IM. For the second research question, which asked whether a statistically significant relationship existed between IM and three different maternal characteristics, findings were mixed for the 3 years examined.

In this chapter, I provide a discussion of study findings. It begins with my interpretation of findings, followed by a review of study limitations. I provide recommendations for future research as well as a discussion of study implications. I close the chapter with concluding remarks.



## **Interpretation of Findings**

### **Research Question 1**

The focus of the first research question was whether a statistically significant relationship existed between IM and community size in populations where more than 25,000 pounds of annual toxic air releases occurred. Data for 2004 (the only year for which community size data were available) revealed that an increase in community size was significantly associated with an increase in IM rate. Thus, the null hypothesis was rejected.

### **Research Question 2**

The focus on the second research question was whether a statistically significant relationship existed between IM and four different maternal characteristics (education level, age, ethnicity, and marital status) in populations where more than 25,000 pounds of annual toxic air releases occurred. Findings for this research question were mixed for the three years examined. Essentially, although different variables were significant during different years, the direction of significance (positive or negative) was similar. For example, for the years 1995 and 2004, an increase in mother's median education level was associated with IM, but the relationship was not significant in 1987. For the years 1987 and 2004, a decrease in mother's average age was associated with IM, but the relationship was not significant in 1995. A decrease in percent non-Hispanic White mothers was associated with IM for the 1987 data, but the relationship was not significant in 1995 or 2004. A decrease in percent married mothers was associated with IM in 1995 and 2004, but the relationship was not significant in 1987.

### **Community Size**

2004 was the only year for which community size data were available. To reiterate from Chapter 4, all communities included in this analysis had a population of at least 250,000. The analysis for 2004 revealed that IM rate increased by 1.426 per 1,000 births when community size increased to > 500,000 (as compared with 250,000 to 499,999). This relationship was statistically significant. Essentially, this analysis revealed that IM was higher in larger (population > 500,000) communities than in mid-size (population 250,000 to 499,999) communities.

This finding was particularly interesting because of its contradiction with findings reported by previous researchers. Past researchers have actually reported that IM was higher in smaller, more rural communities (Hendryx & Fedorko, 2012; Singh & Siahpush, 2014). The relationship between increased IM and small community size has been attributed to access and availability of health resources; for example, Gutierrez (2015) found that poor access to health services can exacerbate the negative effects of exposure to airborne toxins. Hendryx and Fedorko (2012) reported stronger relationships between exposure to environmental toxins and general mortality in smaller, rural communities, as opposed to larger, urban communities. Similarly, Singh and Siahpush (2014) found significant rural-urban disparities in IM, with rates higher in more rural areas.

One reason that findings from the current study may seem to counter those from previous researchers, with regard to community size, may be that none of the communities included in the dataset for the current study would be considered *small*. Because all of the included communities had populations of at least 250,000, the

categorization of any of these communities as *rural* would be questionable. Further, due to limited availability of community size data, this analysis was only able to be conducted for the year 2004. It is certainly possible that if the dataset had included smaller, rural communities, or community data for the years 1987 and 1995, findings may have differed. However, because the TRI database contains information for all communities in the U.S. that are subject to toxic air emissions of more than 25,000 pounds each year, it is reasonable to conclude that smaller, more rural communities are not typically subject to annual exposure of this size. If this is the case, despite previous researcher findings that IM was higher in rural areas, it may be that smaller communities are more vulnerable to smaller volumes of airborne pollution—as opposed to the vulnerability experienced by larger communities exposed to larger levels of airborne toxins.

### **Mother's Median Education**

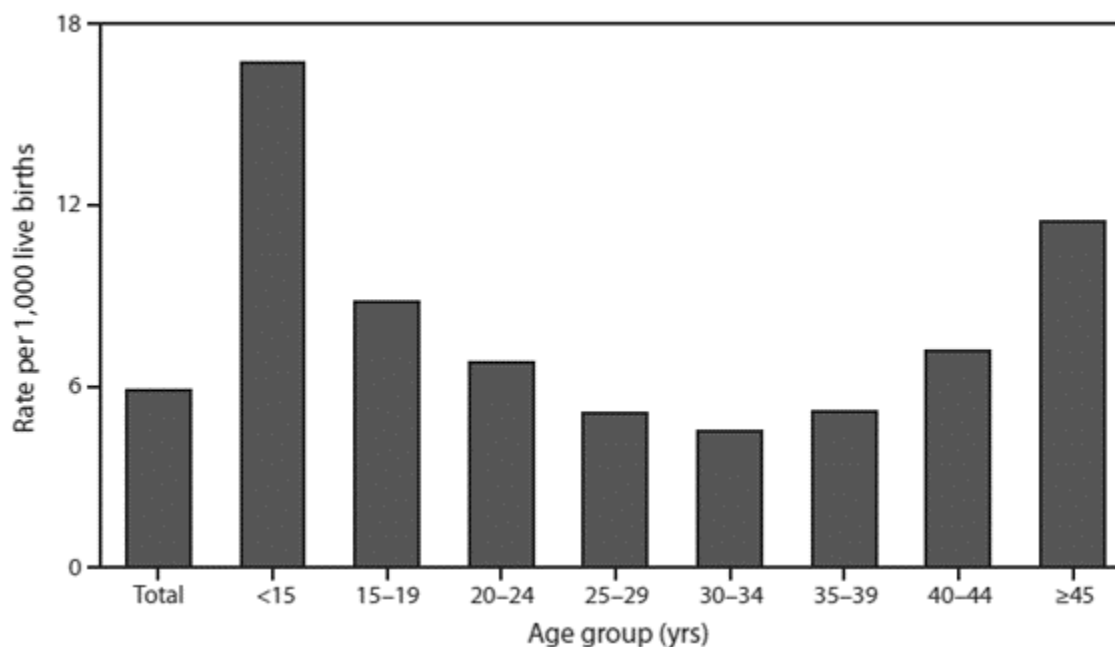
Analysis for data regarding mother's median education were mixed. For the years 1995 and 2004, an increase in mother's median education was associated with IM; however, the relationship between mother's education and IM was not significant in 1987. This finding was surprising and seems to challenge findings from previous researchers who used mother's median education as an indicator of SES (Aarø et al., 2009; Ansem, 2014; Prince et al., 2016; Sargorius, 2014). Lower levels of SES, as indicated by mother's education, has been related to a number of poor health outcomes (Aarø et al., 2009), including increased rates of IM (Prince et al., 2016; Sartorius & Sartorius, 2014).

Because of the known relationship between lower levels of education and higher rates of IM reported by previous scholars (Prince et al., 2016; Sartorius & Sartorius,

2014), findings from the current investigation were surprising. For two of the years examined (1995 and 2004), higher levels of education were actually associated with higher rates of IM. This was an interesting and surprising finding, which may relate to other variables not examined in this study. It is also possible that higher levels of education were associated with higher IM because more highly educated women are likely to be older mothers. Research indicates that more educated women often wait until later in life to have children (Lundborg, Plug, & Rasmussen, 2016). Thus, it may seem possible that the effect that seems to be the result of education may actually be the result of age. However, as discussed in the next section, this does not appear to be the case for the current analysis.

### **Mother's Average Age**

Analysis for mother's average age revealed that for 1987 and 2004, older age was significantly associated with lower rates of IM. In 1995, the relationship between mother's average age and IM was insignificant. As with many of the findings from this study, the relationship between mother's age and IM was somewhat surprising. According to data from the CDC, IM rates are highest for very young mothers (less than 15 years old).



*Figure 11.* IM rates by Mother's Age. Source: National Center for Health Statistics. Linked birth/infant death data set, 2006. Available at <http://www.cdc.gov/nchs/linked.htm>.

IM rates then decrease across age brackets and are lowest for women between the ages of 30 to 34. After that, the rates start to increase across age brackets. The average ages of mothers for each of the years examined (1987, 1995, and 2004) were 26.47, 27.35, and 27.87, respectively. Considered in the context of CDC data, it actually makes sense that an increase in age was associated with lower IM. Women in the 25 to 29 age bracket have an IM rate of 5.1 per 1,000 live births, and women in the 30 to 34 age bracket have an IM rate of 4.5 per 1,000 live births.

### **Percent Non-Hispanic White Mothers**

Analysis revealed that in 1987, a greater percentage of non-Hispanic White mothers was associated with lower rates of IM; for 1995 and 2004, the relationship was nonsignificant. Findings for 1987 align with evidence from previous research that

indicate significant racial disparities in IM rates exist. Gray et al. (2014) found that non-Hispanic White women had better birth outcomes than minority women. In another investigation, Haider (2014) found that the IM rate for non-Hispanic Whites was 5.35, while the rates were significantly higher for African Americans (12.35), Native Americans (8.31), and Puerto Ricans (7.61). However, the rate for Mexicans (5.04) was actually lower than that of Whites. This *Hispanic paradox*, as described by Haider et al., aligns with findings reported by a number of researchers that indicate Hispanics (Mexican, but not Puerto Ricans) typically experience better health outcomes than would be expected based on observable characteristics, such as income and education. Findings for this variable were thus mixed, as some aligned and others contradicted previous research. The lack of definitiveness in findings regarding the relationship between IM and race may be a function of the host of other SES factors that relate to race (such as poverty levels), which may actually create the effect on IM.

### **Percent Married Mothers**

Regarding the variable of marital status, analysis revealed that for 1995 and 2004, married mothers were less associated with IM than nonmarried mothers. For 1987, the relationship was nonsignificant. The significant association between mothers' marital status evident for 1995 and 2004 aligns with findings from previous researchers. For example, Bennett, Braveman, Egerter, and Kiely (1994) found that risks for IM were higher among single mothers than among married mothers. This association has also been linked more recently by MacDorman and Gregory (2015), who analyzed over 23,000 records of fetal deaths and found that fetal mortality rates were higher among unmarried mothers.

### **The Income Factor**

The mechanism through which mother's age and marital status influences IM rates is likely to be socioeconomic, in nature. For example, while Haider et al. (2014) identified a significant relationship between IM and marital status in his investigation of racial and ethnic disparities in IM, he also noted major differences between married and unmarried mothers, in terms of household income. Married mothers had \$30,392 more household income than did non-married mothers. Even after adjusting for other covariates, the researcher found that married mothers still had \$11,937 more annual household income than unmarried mothers. Similarly, mothers who were 35 years and older had \$26,588 more annual income than mothers between the ages of 20 and 24.

The relationship between IM and mothers' median education that emerged from the analysis is the most puzzling. If household income is the mechanism through which the socioeconomic indicators of age and marital status are linked to IM, it would reason to follow that more educated mothers would be associated with lower rates of IM. As revealed in Haider et al.'s (2014) analysis, mothers with a college degree have \$46,624 more in annual household income than do mothers without a college degree. Thus, higher levels of education should be associated with lower rates of IM, if the underlying mechanism is household income. However, results from the current investigation challenge this assumption. For the years 1995 and 2004, an increase in mother's median education was associated with an increase in IM. Consequently, it must be assumed that other variables may be at play, or other interactions may be occurring within the dataset that were not assessed for in the current investigation. This confounding finding provides an excellent opportunity for future researchers to pursue.

## **Theoretical Findings**

Mosley and Chen's (1984) framework for child survival provided the foundation for this study. This framework is based on the supposition that multiple factors must be considered to develop a comprehensive understanding of IM predictors. Much of the previous research in IM focused on IM through purely medical or social lenses. For example, medical researchers often focus on the biology of disease processes, such as disease transmission, dietary practices, and environmental contamination. In contrast, social scientists may examine IM via patterns of mortality across populations and commonly measured social determinants, such as income level and maternal education (Mosley & Chen, 1984). While existing medical and social research provides important insights into IM, the disciplinary compartmentalization creates some superficiality, especially in terms of factors related to SES. This troubling trend in the literature was addressed in the current research by examining potential relationships between IM and environmental (proximity to toxic air releases), spatial (community size), and sociodemographic (mother's age, ethnicity, and marital status) variables.

The complexity of findings from this investigation lend support to Mosley and Chen's (1984) framework, suggesting that even more variables and interaction effects may be at work, regarding the relationship between IM and exposure to toxic air pollution. This is especially true regarding the unexpected relationship between mothers' median education and IM. Indeed, this study provides an example of how important it is to examine the effects of multiple social and environmental factors when investigating health outcomes. A holistic examination of myriad factors is needed to truly understand the risk factors for negative health outcomes, such as IM. Improving such health



outcomes requires researchers to diligently employ frameworks, such as the one developed by Mosley and Chen, to identify risk factors and develop effective interventions to counter identified risks.

### **Limitations of the Study**

This study was subject to limitations. First, it was conducted under financial and time restraints that prevented the execution of a cohort study, which is a preferred design to examine the relationships between study variables (Manolio et al., 2006). The study was also limited to the accuracy of information entered into the databases from which the study data were compiled. The periods for which study data are available present additional limitations. The county of residence for infants ceased to be recorded after 2004 in order to protect the privacy of families. In addition, county residence data were not collected between the years of 1992-1994. Thus, available data are limited to the years of 1987-1991 and 1995-2004.

After data collection began, additional limitations were noted. First, not all counties recorded mother's education level, so this information was missing for some observations. In addition, ethnicity and race information was not collected consistently across all counties in 1987. Thus, the data for 1987 only includes *percent White mothers*, without regard for ethnicity. Finally, community size data were only available for 2004.

### **Recommendations**

A number of recommendations may be made to build upon findings from the current investigation. First, future researchers may replicate this study using data from other years, or analyze a larger data set consisting of data for all available years. Because

resource constraints limited the current investigation to an analysis of data from three years, follow-up studies using data from additional years may provide new insights.

Future researchers may also examine other demographic indicators. Although information regarding household income was not available in the current dataset, understanding the role of income may answer questions that emerged from the analysis, especially with regard to the relationships between age, education, and marital status. Another direction for future research might include examining the effects of pollution on IM in smaller communities. Because the current study only included communities with populations of over 250,000, it is unclear how community size relates to IM when exposure to airborne toxins are considered as moderators to the relationship. Further, it is important to understand how the ratio of pounds of annual airborne toxins to population size relates to IM. It may be that the vulnerability of smaller, more rural communities to smaller concentrations of air pollution is greater than the vulnerability of larger communities to larger concentrations of air pollution. This type of analysis may also shed additional light on the relationship between sociodemographic factors and IM by revealing the role that community size may play.

### **Implications**

A number of social change and practical implications emerged from this research, as discussed below.

#### **Positive Social Change**

Living in counties where manufacturers release environmental toxins, such as those tracked by the EPA's (2010) TRI, may increase an infant's exposure to air pollutants (Braud et al., 2011; Suarez et al., 2007). Research indicates that SES is an

important IM risk factor (Carbajal-Arroyo et al., 2011; Romieu et al., 2004); however, an understanding of the ways many SES factors interact to produce these risks is essential to reducing IM risks among vulnerable populations.

This study addressed a shortcoming in the existing IM research, which tended to focus on IM risk factors related to SES, in silos. As Mosley and Chen (1984) explained, IM is rarely the result of a single factor; but rather, results from interactions between a variety of factors. Thus, to provide a more holistic understanding of the relationship between IM and exposure to toxic air pollution, this study involved an examination of the potential influence of exposure to toxic air pollution, community size, and factors related to mothers' SES (education, age, race, and marital status) on IM. While findings were far from conclusive, they do help to illustrate the complex interactions between risk factors that may contribute to increased rates of IM. This study emphasizes the reality that much additional research is needed on this important topic – one that cannot be ignored in the quest for a reduction in health outcome disparities in this country. It is not enough to be content with the general downward trend in IM rates when significant socioeconomic disparities persist. In order to develop policies to help reduce the IM rates among vulnerable populations, a better understanding of risk factors is required. This study provided a valuable foundation for other scholars to build upon. Indeed, findings from this study and others indicate the paramount need for additional research on this topic.

### **Practical Implications**

Some practical implications can also be gleaned from this research. Because socioeconomic disparities in IM persist, policies are needed to help reduce IM among populations with SES risk factors related to IM. With regard to age, findings from this

study revealed that IM rate was positively associated with younger mothers' age. Thus, interventions may be needed to foster better prenatal education among younger mothers, especially those residing in areas exposed to significant air pollution. Policymakers may consider implementing educational programs to teach young mothers about healthy behaviors to engage in during and after pregnancy. Such education may help offset the increased risks of IM evident among younger mothers living in environments (such as those subject to significant annual air pollution) associated with higher rates of IM.

Regarding mothers' race, disparities existed in IM rates for one of the years examined (1987). Because this finding indicated racial disparities in rates of IM, efforts may be needed to reduce IM rates among racial minorities. However, in order to develop and implement the most effective policies and interventions, additional research is needed to better understand how other sociodemographic variables may influence the IM rate among minorities. New policies and interventions are needed; but first, additional research must be conducted to better understand the predictors of IM among minority populations.

Regarding marital status, findings revealed a significant relationship between IM and unmarried status. This finding is likely related to household income, rather than actual marital status. Previous researchers have reported that married women have significantly more household income than unmarried mothers (Haider et al., 2014), and household income is significantly associated with IM (Tacke & Waldmann, 2013). If this is the case, women (whether married or unmarried) who are in lower income brackets may need interventions that provide them with the healthcare resources required to reduce rates of IM. This is especially salient in communities where exposure to toxic air

pollution is higher, as research indicates that airborne pollution is significantly associated with increased risks of IM.

### **Conclusion**

In this study, the researcher examined the persistent problem of IM through a new lens. This research addressed a gap in the existing body of literature by providing a holistic examination that shed new light on the relationship between IM and exposure to toxic air pollution. Results of this analysis revealed that an increase in community size was significantly associated with an increase in IM. Regarding the relationships between IM and the four different maternal characteristics included in the analysis, findings were mixed for the three years examined. Despite these limitations and unexpected findings, overall results from this investigation, when considered alongside findings from previous research on IM, indicate that policy changes and interventions are needed to reduce socioeconomic disparities in IM, and to save the lives of more infants. A reduction in IM is not just important in terms of infants' lives, but also because IM is a reliable predictor of a community's health status (Haider, 2014). That is, when IM is a problem in a community, it is highly likely that other health issues are prevalent, as well. Findings from this study reveal that much work remains to be conducted on IM and that research is essential because of the persistent disparities in health outcomes that exist. Individuals subject to significant toxic air pollution may lack the resources to relocate to other, environmentally safer locations. Thus, it is essential that researchers understand the health risks, such as IM, in these communities – from there, policy makers may begin to create changes necessary to reduce IM.

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## Appendix A: List of Included Counties

ALEUTIANS EAST BOROUGH, AK	GENEVA, AL	CLARK, AR
DENALI, AK	GREENE, AL	CLAY, AR
FAIRBANKS NORTH STAR BORO, AK	HALE, AL	CLEBURNE, AR
KENAI PENINSULA BOROUGH, AK	HOUSTON, AL	COLUMBIA, AR
KETCHIKAN GATEWAY, AK	JACKSON, AL	CONWAY, AR
NORTHWEST ARCTIC, AK	JEFFERSON, AL	CRAIGHEAD, AR
SITKA, AK	LAMAR, AL	CRAWFORD, AR
AUTAUGA, AL	LAUDERDALE, AL	CRITTENDEN, AR
BALDWIN, AL	LAWRENCE, AL	CROSS, AR
BARBOUR, AL	LEE, AL	DALLAS, AR
BIBB, AL	LIMESTONE, AL	DESHA, AR
BLOUNT, AL	LOWNDES, AL	DREW, AR
BULLOCK, AL	MADISON, AL	FAULKNER, AR
BUTLER, AL	MARENGO, AL	FRANKLIN, AR
CALHOUN, AL	MARION, AL	GARLAND, AR
CHAMBERS, AL	MARSHALL, AL	GRANT, AR
CHILTON, AL	MOBILE, AL	GREENE, AR
CHOCTAW, AL	MONROE, AL	HEMPSTEAD, AR
CLARKE, AL	MONTGOMERY, AL	HOT SPRING, AR
CLAY, AL	MORGAN, AL	HOWARD, AR
CLEBURNE, AL	PIKE, AL	INDEPENDENCE, AR
COFFEE, AL	RANDOLPH, AL	JACKSON, AR
COLBERT, AL	RUSSELL, AL	JEFFERSON, AR
CONECUH, AL	SHELBY, AL	JOHNSON, AR
COOSA, AL	ST CLAIR, AL	LAFAYETTE, AR
COVINGTON, AL	TALLADEGA, AL	LAWRENCE, AR
CULLMAN, AL	TALLAPOOSA, AL	LEE, AR
DALE, AL	TUSCALOOSA, AL	LITTLE RIVER, AR
DALLAS, AL	WALKER, AL	LOGAN, AR
DEKALB, AL	WASHINGTON, AL	LONOKE, AR
ELMORE, AL	WILCOX, AL	MADISON, AR
ESCAMBIA, AL	WINSTON, AL	MARION, AR
ETOWAH, AL	ARKANSAS, AR	MILLER, AR
FAYETTE, AL	ASHLEY, AR	MISSISSIPPI, AR
FRANKLIN, AL	BAXTER, AR	MONROE, AR
	BENTON, AR	NEVADA, AR
	BOONE, AR	OUACHITA, AR
	BRADLEY, AR	PHILLIPS, AR
	CALHOUN, AR	POINSETT, AR
	CARROLL, AR	POLK, AR

POPE, AR  
PULASKI, AR  
RANDOLPH, AR  
SALINE, AR  
SCOTT, AR  
SEBASTIAN, AR  
SEVIER, AR  
ST FRANCIS, AR  
UNION, AR  
WASHINGTON, AR  
WHITE, AR  
WOODRUFF, AR  
YELL, AR  
EASTERN, AS  
APACHE, AZ  
COCHISE, AZ  
COCONINO, AZ  
GILA, AZ  
GREENLEE, AZ  
LA PAZ, AZ  
MARICOPA, AZ  
MOHAVE, AZ  
NAVAJO, AZ  
PIMA, AZ  
PINAL, AZ  
SANTA CRUZ, AZ  
YAVAPAI, AZ  
YUMA, AZ  
ALAMEDA, CA  
AMADOR, CA  
BUTTE, CA  
COLUSA, CA  
CONTRA COSTA, CA  
FRESNO, CA  
GLENN, CA  
HUMBOLDT, CA  
IMPERIAL, CA  
INYO, CA  
KERN, CA  
KINGS, CA  
LAKE, CA  
LASSEN, CA  
LOS ANGELES, CA  
MADERA, CA  
MENDOCINO, CA  
MERCED, CA  
MONTEREY, CA  
NAPA, CA  
NEVADA, CA  
ORANGE, CA  
PLACER, CA  
RIVERSIDE, CA  
SACRAMENTO, CA  
SAN BENITO, CA  
SAN BERNARDINO, CA  
SAN DIEGO, CA  
SAN FRANCISCO, CA  
SAN JOAQUIN, CA  
SAN LUIS OBISPO, CA  
SAN MATEO, CA  
SANTA BARBARA, CA  
SANTA CLARA, CA  
SANTA CRUZ, CA  
SHASTA, CA  
SISKIYOU, CA  
SOLANO, CA  
SONOMA, CA  
STANISLAUS, CA  
TULARE, CA  
TUOLUMNE, CA  
VENTURA, CA  
YOLO, CA  
YUBA, CA  
ADAMS, CO  
ARAPAHOE, CO  
BOULDER, CO  
BROOMFIELD, CO  
COSTILLA, CO  
DENVER, CO  
DOUGLAS, CO  
EL PASO, CO  
FREMONT, CO  
GARFIELD, CO  
GRAND, CO  
JEFFERSON, CO  
LARIMER, CO  
MESA, CO  
MOFFAT, CO  
MONTROSE, CO  
MORGAN, CO  
PROWERS, CO  
PUEBLO, CO  
ROUTT, CO  
WELD, CO  
FAIRFIELD, CT  
HARTFORD, CT  
LITCHFIELD, CT  
MIDDLESEX, CT  
NEW HAVEN, CT  
NEW LONDON, CT  
TOLLAND, CT  
WINDHAM, CT  
DISTRICT OF  
COLUMBIA, DC  
KENT, DE  
NEW CASTLE, DE  
SUSSEX, DE  
ALACHUA, FL  
BAY, FL  
BRADFORD, FL  
BREVARD, FL  
BROWARD, FL  
CITRUS, FL  
CLAY, FL  
COLLIER, FL  
COLUMBIA, FL  
DESOTO, FL  
DIXIE, FL  
DUVAL, FL  
ESCAMBIA, FL  
FLAGLER, FL  
GULF, FL  
HAMILTON, FL  
HARDEE, FL  
HENDRY, FL  
HERNANDO, FL  
HIGHLANDS, FL  
HILLSBOROUGH, FL  
INDIAN RIVER, FL

JACKSON, FL  
 LAFAYETTE, FL  
 LAKE, FL  
 LEE, FL  
 LEON, FL  
 LEVY, FL  
 MANATEE, FL  
 MARION, FL  
 MARTIN, FL  
 MIAMI-DADE, FL  
 NASSAU, FL  
 OKALOOSA, FL  
 ORANGE, FL  
 OSCEOLA, FL  
 PALM BEACH, FL  
 PASCO, FL  
 PINELLAS, FL  
 POLK, FL  
 PUTNAM, FL  
 SANTA ROSA, FL  
 SARASOTA, FL  
 SEMINOLE, FL  
 ST JOHNS, FL  
 ST LUCIE, FL  
 SUMTER, FL  
 SUWANNEE, FL  
 TAYLOR, FL  
 UNION, FL  
 VOLUSIA, FL  
 WAKULLA, FL  
 APPLING, GA  
 ATKINSON, GA  
 BALDWIN, GA  
 BARROW, GA  
 BARTOW, GA  
 BEN HILL, GA  
 BERRIEN, GA  
 BIBB, GA  
 BROOKS, GA  
 BRYAN, GA  
 BULLOCH, GA  
 CAMDEN, GA  
 CARROLL, GA

CATOOSA, GA  
 CHATHAM, GA  
 CHATTOOGA, GA  
 CHEROKEE, GA  
 CLARKE, GA  
 CLAYTON, GA  
 CLINCH, GA  
 COBB, GA  
 COFFEE, GA  
 COLQUITT, GA  
 COLUMBIA, GA  
 COOK, GA  
 COWETA, GA  
 CRISP, GA  
 DADE, GA  
 DAWSON, GA  
 DECATUR, GA  
 DEKALB, GA  
 DOOLY, GA  
 DOUGHERTY, GA  
 DOUGLAS, GA  
 EARLY, GA  
 EFFINGHAM, GA  
 ELBERT, GA  
 EMANUEL, GA  
 FAYETTE, GA  
 FLOYD, GA  
 FORSYTH, GA  
 FRANKLIN, GA  
 FULTON, GA  
 GILMER, GA  
 GLYNN, GA  
 GORDON, GA  
 GRADY, GA  
 GREENE, GA  
 GWINNETT, GA  
 HABERSHAM, GA  
 HALL, GA  
 HARALSON, GA  
 HART, GA  
 HEARD, GA  
 HENRY, GA  
 HOUSTON, GA

JACKSON, GA  
 JASPER, GA  
 JEFFERSON, GA  
 LAMAR, GA  
 LAURENS, GA  
 LEE, GA  
 LIBERTY, GA  
 LOWNDES, GA  
 MACON, GA  
 MARION, GA  
 MCDUFFIE, GA  
 MERIWETHER, GA  
 MONROE, GA  
 MORGAN, GA  
 MURRAY, GA  
 MUSCOGEE, GA  
 NEWTON, GA  
 PAULDING, GA  
 PEACH, GA  
 PICKENS, GA  
 PIERCE, GA  
 PIKE, GA  
 POLK, GA  
 PULASKI, GA  
 PUTNAM, GA  
 RABUN, GA  
 RANDOLPH, GA  
 RICHMOND, GA  
 ROCKDALE, GA  
 SCHLEY, GA  
 SCREVEN, GA  
 SPALDING, GA  
 STEPHENS, GA  
 SUMTER, GA  
 TATTNALL, GA  
 TELFAIR, GA  
 TERRELL, GA  
 THOMAS, GA  
 TIFT, GA  
 TOOMBS, GA  
 TROUP, GA  
 TURNER, GA  
 UNION, GA

UPSON, GA  
WALKER, GA  
WALTON, GA  
WARE, GA  
WARREN, GA  
WASHINGTON, GA  
WAYNE, GA  
WHITE, GA  
WHITFIELD, GA  
WILCOX, GA  
WILKES, GA  
WILKINSON, GA  
GUAM, GU  
HAWAII, HI  
HONOLULU, HI  
KAUAI, HI  
MAUI, HI  
ADAIR, IA  
ALLAMAKEE, IA  
APPANOOSE, IA  
BLACK HAWK, IA  
BOONE, IA  
BREMER, IA  
BUCHANAN, IA  
BUENA VISTA, IA  
BUTLER, IA  
CALHOUN, IA  
CARROLL, IA  
CASS, IA  
CEDAR, IA  
CERRO GORDO, IA  
CHEROKEE, IA  
CLAY, IA  
CLAYTON, IA  
CLINTON, IA  
CRAWFORD, IA  
DALLAS, IA  
DAVIS, IA  
DELAWARE, IA  
DES MOINES, IA  
DICKINSON, IA  
DUBUQUE, IA  
EMMET, IA

FAYETTE, IA  
FLOYD, IA  
FRANKLIN, IA  
GREENE, IA  
HAMILTON, IA  
HANCOCK, IA  
HARDIN, IA  
HENRY, IA  
HOWARD, IA  
IDA, IA  
IOWA, IA  
JASPER, IA  
JEFFERSON, IA  
JOHNSON, IA  
JONES, IA  
KOSSUTH, IA  
LEE, IA  
LINN, IA  
LOUISA, IA  
LYON, IA  
MADISON, IA  
MAHASKA, IA  
MARION, IA  
MARSHALL, IA  
MILLS, IA  
MONROE, IA  
MONTGOMERY, IA  
MUSCATINE, IA  
O'BRIEN, IA  
OSCEOLA, IA  
PAGE, IA  
PALO ALTO, IA  
PLYMOUTH, IA  
POCAHONTAS, IA  
POLK, IA  
POTTAWATTAMIE, IA  
POWESHIEK, IA  
SAC, IA  
SCOTT, IA  
SIOUX, IA  
STORY, IA  
TAYLOR, IA  
UNION, IA

WAPELLO, IA  
WASHINGTON, IA  
WAYNE, IA  
WEBSTER, IA  
WINNEBAGO, IA  
WOODBURY, IA  
WORTH, IA  
WRIGHT, IA  
ADA, ID  
BANNOCK, ID  
BENEWAH, ID  
BINGHAM, ID  
BUTTE, ID  
CANYON, ID  
CARIBOU, ID  
CASSIA, ID  
JEROME, ID  
KOOTENAI, ID  
MINIDOKA, ID  
NEZ PERCE, ID  
OWYHEE, ID  
PAYETTE, ID  
POWER, ID  
SHOSHONE, ID  
TWIN FALLS, ID  
ADAMS, IL  
ALEXANDER, IL  
BOONE, IL  
BUREAU, IL  
CARROLL, IL  
CASS, IL  
CHAMPAIGN, IL  
CHRISTIAN, IL  
CLARK, IL  
CLAY, IL  
CLINTON, IL  
COLES, IL  
COOK, IL  
CRAWFORD, IL  
CUMBERLAND, IL  
DE WITT, IL  
DEKALB, IL  
DOUGLAS, IL

DUPAGE, IL  
EDGAR, IL  
EFFINGHAM, IL  
FAYETTE, IL  
FORD, IL  
FRANKLIN, IL  
FULTON, IL  
GRUNDY, IL  
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IROQUOIS, IL  
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JASPER, IL  
JEFFERSON, IL  
JO DAVIESS, IL  
KANE, IL  
KANKAKEE, IL  
KENDALL, IL  
KNOX, IL  
LA SALLE, IL  
LAKE, IL  
LAWRENCE, IL  
LEE, IL  
LIVINGSTON, IL  
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MASSAC, IL  
MCDONOUGH, IL  
MCHENRY, IL  
MCLEAN, IL  
MONTGOMERY, IL  
MORGAN, IL  
MOULTRIE, IL  
OGLE, IL  
PEORIA, IL  
PIATT, IL  
PIKE, IL  
PUTNAM, IL  
RANDOLPH, IL  
RICHLAND, IL

ROCK ISLAND, IL  
SANGAMON, IL  
SHELBY, IL  
ST CLAIR, IL  
STEPHENSON, IL  
TAZEWELL, IL  
VERMILION, IL  
WARREN, IL  
WASHINGTON, IL  
WAYNE, IL  
WHITE, IL  
WHITESIDE, IL  
WILL, IL  
WILLIAMSON, IL  
WINNEBAGO, IL  
WOODFORD, IL  
ADAMS, IN  
ALLEN, IN  
BARTHOLOMEW, IN  
BLACKFORD, IN  
BOONE, IN  
CARROLL, IN  
CASS, IN  
CLARK, IN  
CLAY, IN  
CLINTON, IN  
DAVIESS, IN  
DEARBORN, IN  
DECATUR, IN  
DEKALB, IN  
DELAWARE, IN  
DUBOIS, IN  
ELKHART, IN  
FAYETTE, IN  
FLOYD, IN  
FOUNTAIN, IN  
FULTON, IN  
GIBSON, IN  
GRANT, IN  
HAMILTON, IN  
HANCOCK, IN  
HARRISON, IN  
HENDRICKS, IN

HENRY, IN  
HOWARD, IN  
HUNTINGTON, IN  
JACKSON, IN  
JASPER, IN  
JAY, IN  
JEFFERSON, IN  
JENNINGS, IN  
JOHNSON, IN  
KNOX, IN  
KOSCIUSKO, IN  
LAGRANGE, IN  
LAKE, IN  
LAPORTE, IN  
LAWRENCE, IN  
MADISON, IN  
MARION, IN  
MARSHALL, IN  
MARTIN, IN  
MIAMI, IN  
MONROE, IN  
MONTGOMERY, IN  
MORGAN, IN  
NEWTON, IN  
NOBLE, IN  
ORANGE, IN  
PERRY, IN  
PIKE, IN  
PORTER, IN  
POSEY, IN  
PULASKI, IN  
PUTNAM, IN  
RANDOLPH, IN  
RIPLEY, IN  
RUSH, IN  
SCOTT, IN  
SHELBY, IN  
SPENCER, IN  
ST JOSEPH, IN  
STEBEN, IN  
SULLIVAN, IN  
SWITZERLAND, IN  
TIPPECANOE, IN



TIPTON, IN  
 VANDERBURGH, IN  
 VERMILLION, IN  
 VIGO, IN  
 WABASH, IN  
 WARREN, IN  
 WARRICK, IN  
 WASHINGTON, IN  
 WAYNE, IN  
 WELLS, IN  
 WHITE, IN  
 WHITLEY, IN  
 ALLEN, KS  
 ANDERSON, KS  
 ATCHISON, KS  
 BARTON, KS  
 BOURBON, KS  
 BROWN, KS  
 BUTLER, KS  
 CHEROKEE, KS  
 CLAY, KS  
 COWLEY, KS  
 CRAWFORD, KS  
 DICKINSON, KS  
 DONIPHAN, KS  
 DOUGLAS, KS  
 ELLSWORTH, KS  
 FINNEY, KS  
 FORD, KS  
 FRANKLIN, KS  
 GEARY, KS  
 GRANT, KS  
 HARVEY, KS  
 JACKSON, KS  
 JOHNSON, KS  
 LABETTE, KS  
 LEAVENWORTH, KS  
 LINN, KS  
 LYON, KS  
 MARION, KS  
 MARSHALL, KS  
 MCPHERSON, KS  
 MIAMI, KS

MITCHELL, KS  
 MONTGOMERY, KS  
 NEMAHA, KS  
 NEOSHO, KS  
 OSBORNE, KS  
 OTTAWA, KS  
 PHILLIPS, KS  
 POTTAWATOMIE, KS  
 RENO, KS  
 RUSH, KS  
 RUSSELL, KS  
 SALINE, KS  
 SEDGWICK, KS  
 SEWARD, KS  
 SHAWNEE, KS  
 SHERMAN, KS  
 SMITH, KS  
 WILSON, KS  
 WYANDOTTE, KS  
 ALLEN, KY  
 ANDERSON, KY  
 BALLARD, KY  
 BARREN, KY  
 BELL, KY  
 BOONE, KY  
 BOURBON, KY  
 BOYD, KY  
 BOYLE, KY  
 BULLITT, KY  
 BUTLER, KY  
 CALDWELL, KY  
 CALLOWAY, KY  
 CAMPBELL, KY  
 CARROLL, KY  
 CHRISTIAN, KY  
 CLARK, KY  
 CLINTON, KY  
 CRITTENDEN, KY  
 DAVIESS, KY  
 FAYETTE, KY  
 FLEMING, KY  
 FRANKLIN, KY  
 GALLATIN, KY

GRANT, KY  
 GRAVES, KY  
 GRAYSON, KY  
 GREEN, KY  
 GREENUP, KY  
 HANCOCK, KY  
 HARDIN, KY  
 HARRISON, KY  
 HENDERSON, KY  
 HENRY, KY  
 HOPKINS, KY  
 JEFFERSON, KY  
 JESSAMINE, KY  
 KENTON, KY  
 KNOX, KY  
 LAUREL, KY  
 LAWRENCE, KY  
 LEWIS, KY  
 LINCOLN, KY  
 LOGAN, KY  
 LYON, KY  
 MADISON, KY  
 MARION, KY  
 MARSHALL, KY  
 MARTIN, KY  
 MASON, KY  
 MCCRACKEN, KY  
 MCCREARY, KY  
 MEADE, KY  
 MERCER, KY  
 METCALFE, KY  
 MONTGOMERY, KY  
 MUHLENBERG, KY  
 NELSON, KY  
 OHIO, KY  
 OLDHAM, KY  
 OWEN, KY  
 PENDLETON, KY  
 POWELL, KY  
 PULASKI, KY  
 ROWAN, KY  
 SCOTT, KY  
 SHELBY, KY

SIMPSON, KY  
 TAYLOR, KY  
 TRIMBLE, KY  
 UNION, KY  
 WARREN, KY  
 WEBSTER, KY  
 WHITLEY, KY  
 WOODFORD, KY  
 ACADIA PARISH, LA  
 ASCENSION PARISH,  
 LA  
 ASSUMPTION PARISH,  
 LA  
 BEAUREGARD PARISH,  
 LA  
 BIENVILLE PARISH, LA  
 BOSSIER PARISH, LA  
 CADDO, LA  
 CALCASIEU PARISH,  
 LA  
 CAMERON PARISH, LA  
 CLAIBORNE PARISH,  
 LA  
 DE SOTO PARISH, LA  
 EAST BATON ROUGE  
 PARISH, LA  
 EVANGELINE PARISH,  
 LA  
 FRANKLIN PARISH, LA  
 GRANT PARISH, LA  
 IBERIA PARISH, LA  
 IBERVILLE PARISH, LA  
 JACKSON PARISH, LA  
 JEFFERSON PARISH,  
 LA  
 LA SALLE PARISH, LA  
 LAFOURCHE PARISH,  
 LA  
 LINCOLN PARISH, LA  
 LIVINGSTON PARISH,  
 LA  
 MOREHOUSE PARISH,  
 LA

NATCHITOCHE  
 PARISH, LA  
 ORLEANS PARISH, LA  
 OUACHITA PARISH, LA  
 PLAQUEMINES  
 PARISH, LA  
 POINTE COUPEE  
 PARISH, LA  
 RAPIDES PARISH, LA  
 RICHLAND PARISH, LA  
 SABINE PARISH, LA  
 ST BERNARD PARISH,  
 LA  
 ST CHARLES PARISH,  
 LA  
 ST JAMES PARISH, LA  
 ST JOHN THE BAPTIST  
 PARIS, LA  
 ST LANDRY PARISH,  
 LA  
 ST MARTIN PARISH,  
 LA  
 ST MARY PARISH, LA  
 ST TAMMANY PARISH,  
 LA  
 TANGIPAHOA PARISH,  
 LA  
 TERREBONNE PARISH,  
 LA  
 UNION PARISH, LA  
 VERMILION PARISH,  
 LA  
 WASHINGTON PARISH,  
 LA  
 WEBSTER PARISH, LA  
 WEST BATON ROUGE  
 PARISH, LA  
 WEST FELICIANA  
 PARISH, LA  
 WINN PARISH, LA  
 BARNSTABLE, MA  
 BERKSHIRE, MA  
 BRISTOL, MA

ESSEX, MA  
 FRANKLIN, MA  
 HAMPDEN, MA  
 HAMPSHIRE, MA  
 MIDDLESEX, MA  
 NORFOLK, MA  
 PLYMOUTH, MA  
 SUFFOLK, MA  
 WORCESTER, MA  
 ALLEGANY, MD  
 ANNE ARUNDEL, MD  
 BALTIMORE, MD  
 BALTIMORE (CITY),  
 MD  
 CARROLL, MD  
 CECIL, MD  
 CHARLES, MD  
 DORCHESTER, MD  
 FREDERICK, MD  
 GARRETT, MD  
 HARFORD, MD  
 HOWARD, MD  
 KENT, MD  
 MONTGOMERY, MD  
 PRINCE GEORGE'S, MD  
 SOMERSET, MD  
 TALBOT, MD  
 WASHINGTON, MD  
 WICOMICO, MD  
 WORCESTER, MD  
 ANDROSCOGGIN, ME  
 AROOSTOOK, ME  
 CUMBERLAND, ME  
 FRANKLIN, ME  
 HANCOCK, ME  
 KENNEBEC, ME  
 KNOX, ME  
 LINCOLN, ME  
 OXFORD, ME  
 PENOBSCOT, ME  
 PISCATAQUIS, ME  
 SAGadahoc, ME  
 SOMERSET, ME

WALDO, ME  
 WASHINGTON, ME  
 YORK, ME  
 ALGER, MI  
 ALLEGAN, MI  
 ALPENA, MI  
 ANTRIM, MI  
 ARENAC, MI  
 BARAGA, MI  
 BARRY, MI  
 BAY, MI  
 BERRIEN, MI  
 BRANCH, MI  
 CALHOUN, MI  
 CASS, MI  
 CHARLEVOIX, MI  
 CHEBOYGAN, MI  
 CLARE, MI  
 CLINTON, MI  
 CRAWFORD, MI  
 DELTA, MI  
 DICKINSON, MI  
 EATON, MI  
 GENESEE, MI  
 GRAND TRAVERSE, MI  
 GRATIOT, MI  
 HILLSDALE, MI  
 HOUGHTON, MI  
 HURON, MI  
 INGHAM, MI  
 IONIA, MI  
 IOSCO, MI  
 ISABELLA, MI  
 JACKSON, MI  
 KALAMAZOO, MI  
 KALKASKA, MI  
 KENT, MI  
 LAPEER, MI  
 LENAWEЕ, MI  
 LIVINGSTON, MI  
 LUCE, MI  
 MACOMB, MI  
 MANISTEE, MI

MARQUETTE, MI  
 MASON, MI  
 MECOSTA, MI  
 MENOMINEE, MI  
 MIDLAND, MI  
 MONROE, MI  
 MONTCALM, MI  
 MUSKEGON, MI  
 NEWAYGO, MI  
 OAKLAND, MI  
 OCEANA, MI  
 OGEMAW, MI  
 ONTONAGON, MI  
 OSCEOLA, MI  
 OSCODA, MI  
 OTSEGO, MI  
 OTTAWA, MI  
 SAGINAW, MI  
 SANILAC, MI  
 SCHOOLCRAFT, MI  
 SHIAWASSEE, MI  
 ST CLAIR, MI  
 ST JOSEPH, MI  
 TUSCOLA, MI  
 VAN BUREN, MI  
 WASHTENAW, MI  
 WAYNE, MI  
 WEXFORD, MI  
 ANOKA, MN  
 BELTRAMI, MN  
 BENTON, MN  
 BLUE EARTH, MN  
 BROWN, MN  
 CARLTON, MN  
 CARVER, MN  
 CHISAGO, MN  
 CLAY, MN  
 COTTONWOOD, MN  
 CROW WING, MN  
 DAKOTA, MN  
 DODGE, MN  
 DOUGLAS, MN  
 FILLMORE, MN

FREEBORN, MN  
 GOODHUE, MN  
 GRANT, MN  
 HENNEPIN, MN  
 HOUSTON, MN  
 HUBBARD, MN  
 ISANTI, MN  
 ITASCA, MN  
 JACKSON, MN  
 KANABEC, MN  
 KANDIYOHI, MN  
 KOOCHICHING, MN  
 LAC QUI PARLE, MN  
 LAKE, MN  
 LAKE OF THE WOODS,  
 MN  
 LE SUEUR, MN  
 LYON, MN  
 MARSHALL, MN  
 MARTIN, MN  
 MCLEOD, MN  
 MEEKER, MN  
 MILLE LACS, MN  
 MORRISON, MN  
 MOWER, MN  
 NICOLLET, MN  
 NOBLES, MN  
 OLMSTED, MN  
 OTTER TAIL, MN  
 PENNINGTON, MN  
 PIPESTONE, MN  
 POLK, MN  
 POPE, MN  
 RAMSEY, MN  
 REDWOOD, MN  
 RENVILLE, MN  
 RICE, MN  
 ROCK, MN  
 ROSEAU, MN  
 SCOTT, MN  
 SHERBURNE, MN  
 ST LOUIS, MN  
 STEARNS, MN

STEELE, MN  
 SWIFT, MN  
 TODD, MN  
 WABASHA, MN  
 WADENA, MN  
 WASECA, MN  
 WASHINGTON, MN  
 WINONA, MN  
 WRIGHT, MN  
 ADAIR, MO  
 AUDRAIN, MO  
 BARRY, MO  
 BARTON, MO  
 BOONE, MO  
 BUCHANAN, MO  
 BUTLER, MO  
 CALLAWAY, MO  
 CAMDEN, MO  
 CAPE GIRARDEAU, MO  
 CARROLL, MO  
 CARTER, MO  
 CHRISTIAN, MO  
 CLAY, MO  
 CLINTON, MO  
 COLE, MO  
 COOPER, MO  
 CRAWFORD, MO  
 DAVIESS, MO  
 DENT, MO  
 DOUGLAS, MO  
 DUNKLIN, MO  
 FRANKLIN, MO  
 GASCONADE, MO  
 GREENE, MO  
 GRUNDY, MO  
 HENRY, MO  
 HOWARD, MO  
 HOWELL, MO  
 IRON, MO  
 JACKSON, MO  
 JASPER, MO  
 JEFFERSON, MO  
 JOHNSON, MO

LACLEDE, MO  
 LAFAYETTE, MO  
 LAWRENCE, MO  
 LEWIS, MO  
 LINCOLN, MO  
 LIVINGSTON, MO  
 MACON, MO  
 MADISON, MO  
 MARIES, MO  
 MARION, MO  
 MCDONALD, MO  
 MILLER, MO  
 MISSISSIPPI, MO  
 MONITEAU, MO  
 MONTGOMERY, MO  
 MORGAN, MO  
 NEW MADRID, MO  
 NEWTON, MO  
 NODAWAY, MO  
 OSAGE, MO  
 PEMISCOT, MO  
 PERRY, MO  
 PETTIS, MO  
 PIKE, MO  
 PLATTE, MO  
 POLK, MO  
 PULASKI, MO  
 RALLS, MO  
 RANDOLPH, MO  
 REYNOLDS, MO  
 SALINE, MO  
 SCOTT, MO  
 SHANNON, MO  
 ST CHARLES, MO  
 ST FRANCOIS, MO  
 ST LOUIS, MO  
 ST LOUIS (CITY), MO  
 STE GENEVIEVE, MO  
 STODDARD, MO  
 TANEY, MO  
 TEXAS, MO  
 VERNON, MO  
 WARREN, MO

WASHINGTON, MO  
 WAYNE, MO  
 WEBSTER, MO  
 WRIGHT, MO  
 ADAMS, MS  
 ALCORN, MS  
 AMITE, MS  
 BOLIVAR, MS  
 CALHOUN, MS  
 CHICKASAW, MS  
 CHOCTAW, MS  
 CLAIBORNE, MS  
 CLARKE, MS  
 CLAY, MS  
 COAHOMA, MS  
 COPIAH, MS  
 COVINGTON, MS  
 DESOTO, MS  
 FORREST, MS  
 GEORGE, MS  
 GREENE, MS  
 GRENADA, MS  
 HANCOCK, MS  
 HARRISON, MS  
 HINDS, MS  
 HOLMES, MS  
 ITAWAMBA, MS  
 JACKSON, MS  
 JASPER, MS  
 JONES, MS  
 LAFAYETTE, MS  
 LAMAR, MS  
 LAUDERDALE, MS  
 LAWRENCE, MS  
 LEAKE, MS  
 LEE, MS  
 LEFLORE, MS  
 LINCOLN, MS  
 LOWNDES, MS  
 MADISON, MS  
 MARION, MS  
 MARSHALL, MS  
 MONROE, MS

MONTGOMERY, MS	ANSON, NC	MACON, NC
NESHOBA, MS	ASHE, NC	MADISON, NC
NEWTON, MS	BEAUFORT, NC	MARTIN, NC
NOXUBEE, MS	BERTIE, NC	MCDOWELL, NC
OKTIBBEHA, MS	BLADEN, NC	MECKLENBURG, NC
PANOLA, MS	BRUNSWICK, NC	MITCHELL, NC
PEARL RIVER, MS	BUNCOMBE, NC	MONTGOMERY, NC
PERRY, MS	BURKE, NC	MOORE, NC
PIKE, MS	CABARRUS, NC	NASH, NC
PONTOTOC, MS	CALDWELL, NC	NEW HANOVER, NC
PRENTISS, MS	CARTERET, NC	NORTHAMPTON, NC
QUITMAN, MS	CATAWBA, NC	ONSLOW, NC
RANKIN, MS	CHATHAM, NC	ORANGE, NC
SCOTT, MS	CHEROKEE, NC	PASQUOTANK, NC
SMITH, MS	CHOWAN, NC	PERSON, NC
STONE, MS	CLEVELAND, NC	PITT, NC
SUNFLOWER, MS	COLUMBUS, NC	RANDOLPH, NC
TATE, MS	CRAVEN, NC	RICHMOND, NC
TIPPAH, MS	CUMBERLAND, NC	ROBESON, NC
TISHOMINGO, MS	DAVIDSON, NC	ROCKINGHAM, NC
UNION, MS	DAVIE, NC	ROWAN, NC
WARREN, MS	DUPLIN, NC	RUTHERFORD, NC
WASHINGTON, MS	DURHAM, NC	SAMPSON, NC
WAYNE, MS	EDGECOMBE, NC	SCOTLAND, NC
WEBSTER, MS	FORSYTH, NC	STANLY, NC
WINSTON, MS	FRANKLIN, NC	STOKES, NC
YALOBUSHA, MS	GASTON, NC	SURRY, NC
YAZOO, MS	GRAHAM, NC	SWAIN, NC
CASCADE, MT	GRANVILLE, NC	TRANSYLVANIA, NC
FLATHEAD, MT	GREENE, NC	UNION, NC
GALLATIN, MT	GUILFORD, NC	VANCE, NC
JEFFERSON, MT	HALIFAX, NC	WAKE, NC
LEWIS AND CLARK, MT	HARNETT, NC	WARREN, NC
MISSOULA, MT	HAYWOOD, NC	WATAUGA, NC
RICHLAND, MT	HENDERSON, NC	WAYNE, NC
ROOSEVELT, MT	HERTFORD, NC	WILKES, NC
ROSEBUD, MT	HOKE, NC	WILSON, NC
SILVER BOW, MT	IREDELL, NC	YADKIN, NC
STILLWATER, MT	JACKSON, NC	YANCEY, NC
YELLOWSTONE, MT	JOHNSTON, NC	BENSON, ND
ALAMANCE, NC	LEE, NC	BOWMAN, ND
ALEXANDER, NC	LENOIR, NC	BURLEIGH, ND
	LINCOLN, NC	CASS, ND

GRAND FORKS, ND	SARPY, NE	EDDY, NM
MCHENRY, ND	SCOTTS BLUFF, NE	GRANT, NM
MCLEAN, ND	SEWARD, NE	HIDALGO, NM
MERCER, ND	STANTON, NE	LEA, NM
MORTON, ND	THAYER, NE	MCKINLEY, NM
OLIVER, ND	THURSTON, NE	QUAY, NM
PEMBINA, ND	WASHINGTON, NE	SAN JUAN, NM
RAMSEY, ND	WAYNE, NE	SAN MIGUEL, NM
RANSOM, ND	YORK, NE	SANDOVAL, NM
RICHLAND, ND	BELKNAP, NH	VALENCIA, NM
ROLETTE, ND	CARROLL, NH	CARSON CITY (CITY),
SARGENT, ND	CHESHIRE, NH	NV
STUTSMAN, ND	COOS, NH	CLARK, NV
TRAILL, ND	GRAFTON, NH	DOUGLAS, NV
ADAMS, NE	HILLSBOROUGH, NH	ELKO, NV
BOX BUTTE, NE	MERRIMACK, NH	EUREKA, NV
BUFFALO, NE	ROCKINGHAM, NH	HUMBOLDT, NV
BUTLER, NE	STRAFFORD, NH	LANDER, NV
CASS, NE	SULLIVAN, NH	LYON, NV
CHASE, NE	ATLANTIC, NJ	MINERAL, NV
CHEYENNE, NE	BERGEN, NJ	NYE, NV
COLFAX, NE	BURLINGTON, NJ	PERSHING, NV
CUMING, NE	CAMDEN, NJ	STOREY, NV
DAKOTA, NE	CAPE MAY, NJ	WASHOE, NV
DAWSON, NE	CUMBERLAND, NJ	WHITE PINE, NV
DODGE, NE	ESSEX, NJ	ALBANY, NY
DOUGLAS, NE	GLOUCESTER, NJ	ALLEGANY, NY
GAGE, NE	HUDSON, NJ	BRONX, NY
HALL, NE	HUNTERDON, NJ	BROOME, NY
HAMILTON, NE	MERCER, NJ	CATTARAUGUS, NY
HOLT, NE	MIDDLESEX, NJ	CAYUGA, NY
JEFFERSON, NE	MONMOUTH, NJ	CHAUTAUQUA, NY
KEITH, NE	MORRIS, NJ	CHEMUNG, NY
LANCASTER, NE	OCEAN, NJ	CHENANGO, NY
LINCOLN, NE	PASSAIC, NJ	CLINTON, NY
MADISON, NE	SALEM, NJ	COLUMBIA, NY
MORRILL, NE	SOMERSET, NJ	CORTLAND, NY
NEMAHA, NE	SUSSEX, NJ	DELAWARE, NY
OTOE, NE	UNION, NJ	DUTCHESS, NY
PHELPS, NE	WARREN, NJ	ERIE, NY
PLATTE, NE	BERNALILLO, NM	ESSEX, NY
RICHARDSON, NE	CHAVES, NM	FULTON, NY
SALINE, NE	DONA ANA, NM	GENESEE, NY

GREENE, NY	ATHENS, OH	MAHONING, OH
HERKIMER, NY	AUGLAIZE, OH	MARION, OH
JEFFERSON, NY	BELMONT, OH	MEDINA, OH
KINGS, NY	BROWN, OH	MERCER, OH
LEWIS, NY	BUTLER, OH	MIAMI, OH
LIVINGSTON, NY	CHAMPAIGN, OH	MONROE, OH
MADISON, NY	CLARK, OH	MONTGOMERY, OH
MONROE, NY	CLERMONT, OH	MORGAN, OH
MONTGOMERY, NY	CLINTON, OH	MORROW, OH
NASSAU, NY	COLUMBIANA, OH	MUSKINGUM, OH
NEW YORK, NY	COSHOCTON, OH	NOBLE, OH
NIAGARA, NY	CRAWFORD, OH	OTTAWA, OH
ONEIDA, NY	CUYAHOGA, OH	PAULDING, OH
ONONDAGA, NY	DARKE, OH	PERRY, OH
ONTARIO, NY	DEFIANCE, OH	PICKAWAY, OH
ORANGE, NY	DELAWARE, OH	PIKE, OH
ORLEANS, NY	ERIE, OH	PORTAGE, OH
OSWEGO, NY	FAIRFIELD, OH	PREBLE, OH
PUTNAM, NY	FAYETTE, OH	PUTNAM, OH
QUEENS, NY	FRANKLIN, OH	RICHLAND, OH
RENSSELAER, NY	FULTON, OH	ROSS, OH
RICHMOND, NY	GALLIA, OH	SANDUSKY, OH
ROCKLAND, NY	GEAUGA, OH	SCIOTO, OH
SARATOGA, NY	GREENE, OH	SENECA, OH
SCHENECTADY, NY	GUERNSEY, OH	SHELBY, OH
SCHUYLER, NY	HAMILTON, OH	STARK, OH
SENECA, NY	HANCOCK, OH	SUMMIT, OH
ST LAWRENCE, NY	HARDIN, OH	TRUMBULL, OH
STEUBEN, NY	HENRY, OH	TUSCARAWAS, OH
SUFFOLK, NY	HIGHLAND, OH	UNION, OH
TIOGA, NY	HOCKING, OH	VAN WERT, OH
TOMPKINS, NY	HOLMES, OH	WARREN, OH
ULSTER, NY	HURON, OH	WASHINGTON, OH
WARREN, NY	JACKSON, OH	WAYNE, OH
WASHINGTON, NY	JEFFERSON, OH	WILLIAMS, OH
WAYNE, NY	KNOX, OH	WOOD, OH
WESTCHESTER, NY	LAKE, OH	WYANDOT, OH
WYOMING, NY	LAWRENCE, OH	ADAIR, OK
YATES, NY	LICKING, OH	BEAVER, OK
ADAMS, OH	LOGAN, OH	BRYAN, OK
ALLEN, OH	LORAIN, OH	CADDO, OK
ASHLAND, OH	LUCAS, OH	CANADIAN, OK
ASHTABULA, OH	MADISON, OH	CARTER, OK

CHOCTAW, OK  
 CLEVELAND, OK  
 COMANCHE, OK  
 CRAIG, OK  
 CREEK, OK  
 CUSTER, OK  
 GARFIELD, OK  
 GARVIN, OK  
 GRADY, OK  
 JOHNSTON, OK  
 KAY, OK  
 KINGFISHER, OK  
 KIOWA, OK  
 LE FLORE, OK  
 LINCOLN, OK  
 LOVE, OK  
 MARSHALL, OK  
 MAYES, OK  
 MCCURTAIN, OK  
 MCINTOSH, OK  
 MURRAY, OK  
 MUSKOGEE, OK  
 NOBLE, OK  
 NOWATA, OK  
 OKLAHOMA, OK  
 OKMULGEE, OK  
 OSAGE, OK  
 OTTAWA, OK  
 PAYNE, OK  
 PITTSBURG, OK  
 PONTOTOC, OK  
 POTTAWATOMIE, OK  
 ROGERS, OK  
 SEMINOLE, OK  
 SEQUOYAH, OK  
 STEPHENS, OK  
 TEXAS, OK  
 TILLMAN, OK  
 TULSA, OK  
 WAGONER, OK  
 WASHINGTON, OK  
 WOODS, OK  
 WOODWARD, OK

BENTON, OR  
 CLACKAMAS, OR  
 CLATSOP, OR  
 COLUMBIA, OR  
 COOS, OR  
 CROOK, OR  
 DESCHUTES, OR  
 DOUGLAS, OR  
 HARNEY, OR  
 HOOD RIVER, OR  
 JACKSON, OR  
 JEFFERSON, OR  
 JOSEPHINE, OR  
 KLAMATH, OR  
 LANE, OR  
 LINCOLN, OR  
 LINN, OR  
 MALHEUR, OR  
 MARION, OR  
 MORROW, OR  
 MULTNOMAH, OR  
 POLK, OR  
 UMATILLA, OR  
 UNION, OR  
 WASCO, OR  
 WASHINGTON, OR  
 YAMHILL, OR  
 ADAMS, PA  
 ALLEGHENY, PA  
 ARMSTRONG, PA  
 BEAVER, PA  
 BEDFORD, PA  
 BERKS, PA  
 BLAIR, PA  
 BRADFORD, PA  
 BUCKS, PA  
 BUTLER, PA  
 CAMBRIA, PA  
 CAMERON, PA  
 CARBON, PA  
 CENTRE, PA  
 CHESTER, PA  
 CLARION, PA

CLEARFIELD, PA  
 CLINTON, PA  
 COLUMBIA, PA  
 CRAWFORD, PA  
 CUMBERLAND, PA  
 DAUPHIN, PA  
 DELAWARE, PA  
 ELK, PA  
 ERIE, PA  
 FAYETTE, PA  
 FRANKLIN, PA  
 FULTON, PA  
 GREENE, PA  
 HUNTINGDON, PA  
 INDIANA, PA  
 JEFFERSON, PA  
 JUNIATA, PA  
 LACKAWANNA, PA  
 LANCASTER, PA  
 LAWRENCE, PA  
 LEBANON, PA  
 LEHIGH, PA  
 LUZERNE, PA  
 LYCOMING, PA  
 MCKEAN, PA  
 MERCER, PA  
 MIFFLIN, PA  
 MONROE, PA  
 MONTGOMERY, PA  
 MONTOUR, PA  
 NORTHAMPTON, PA  
 NORTHUMBERLAND,  
 PA  
 PHILADELPHIA, PA  
 POTTER, PA  
 SCHUYLKILL, PA  
 SNYDER, PA  
 SOMERSET, PA  
 SULLIVAN, PA  
 SUSQUEHANNA, PA  
 TIOGA, PA  
 UNION, PA  
 VENANGO, PA



WARREN, PA  
 WASHINGTON, PA  
 WESTMORELAND, PA  
 WYOMING, PA  
 YORK, PA  
 AGUADA, PR  
 AGUADILLA, PR  
 AIBONITO, PR  
 ANASCO, PR  
 ARECIBO, PR  
 BARCELONETA, PR  
 BAYAMON, PR  
 CABO ROJO, PR  
 CAGUAS, PR  
 CAROLINA, PR  
 CATANO, PR  
 CIALES, PR  
 CIDRA, PR  
 COAMO, PR  
 FAJARDO, PR  
 GUAYAMA, PR  
 GUAYANILLA, PR  
 GUAYNABO, PR  
 GURABO, PR  
 HORMIGUEROS, PR  
 HUMACAO, PR  
 ISABELA, PR  
 JAYUYA, PR  
 JUNCOS, PR  
 LAS PIEDRAS, PR  
 LUQUILLO, PR  
 MANATI, PR  
 MAYAGUEZ, PR  
 MOCA, PR  
 PENUELAS, PR  
 PONCE, PR  
 QUEBRADILLAS, PR  
 SABANA GRANDE, PR  
 SALINAS, PR  
 SAN GERMAN, PR  
 SAN JUAN, PR  
 SANTA ISABEL, PR  
 TOA ALTA, PR

TOA BAJA, PR  
 TRUJILLO ALTO, PR  
 VEGA ALTA, PR  
 VEGA BAJA, PR  
 VILLALBA, PR  
 YABUCOA, PR  
 YAUCO, PR  
 BRISTOL, RI  
 KENT, RI  
 NEWPORT, RI  
 PROVIDENCE, RI  
 WASHINGTON, RI  
 ABBEVILLE, SC  
 AIKEN, SC  
 ALLENDALE, SC  
 ANDERSON, SC  
 BAMBERG, SC  
 BARNWELL, SC  
 BEAUFORT, SC  
 BERKELEY, SC  
 CALHOUN, SC  
 CHARLESTON, SC  
 CHEROKEE, SC  
 CHESTER, SC  
 CHESTERFIELD, SC  
 CLARENDON, SC  
 COLLETON, SC  
 DARLINGTON, SC  
 DILLON, SC  
 DORCHESTER, SC  
 EDGEFIELD, SC  
 FAIRFIELD, SC  
 FLORENCE, SC  
 GEORGETOWN, SC  
 GREENVILLE, SC  
 GREENWOOD, SC  
 HAMPTON, SC  
 HORRY, SC  
 KERSHAW, SC  
 LANCASTER, SC  
 LAURENS, SC  
 LEE, SC  
 LEXINGTON, SC

MARION, SC  
 MARLBORO, SC  
 NEWBERRY, SC  
 OCONEE, SC  
 ORANGEBURG, SC  
 PICKENS, SC  
 RICHLAND, SC  
 SPARTANBURG, SC  
 SUMTER, SC  
 UNION, SC  
 WILLIAMSBURG, SC  
 YORK, SC  
 BEADLE, SD  
 BON HOMME, SD  
 BROOKINGS, SD  
 BROWN, SD  
 CODINGTON, SD  
 DAVISON, SD  
 GRANT, SD  
 KINGSBURY, SD  
 LAKE, SD  
 LAWRENCE, SD  
 LINCOLN, SD  
 MCCOOK, SD  
 MINNEHAHA, SD  
 PENNINGTON, SD  
 UNION, SD  
 YANKTON, SD  
 ANDERSON, TN  
 BEDFORD, TN  
 BLOUNT, TN  
 BRADLEY, TN  
 CAMPBELL, TN  
 CARROLL, TN  
 CARTER, TN  
 CHEATHAM, TN  
 CLAIBORNE, TN  
 CLAY, TN  
 COCKE, TN  
 COFFEE, TN  
 CROCKETT, TN  
 CUMBERLAND, TN  
 DAVIDSON, TN

DECATUR, TN	RUTHERFORD, TN	CHEROKEE, TX
DEKALB, TN	SCOTT, TN	COLLIN, TX
DICKSON, TN	SEQUATCHIE, TN	COLORADO, TX
DYER, TN	SEVIER, TN	COMAL, TX
FAYETTE, TN	SHELBY, TN	COOKE, TX
FRANKLIN, TN	SMITH, TN	CORYELL, TX
GIBSON, TN	STEWART, TN	DALLAS, TX
GILES, TN	SULLIVAN, TN	DAWSON, TX
GREENE, TN	SUMNER, TN	DEAF SMITH, TX
HAMBLEN, TN	TIPTON, TN	DENTON, TX
HAMILTON, TN	TROUSDALE, TN	EASTLAND, TX
HANCOCK, TN	UNICOI, TN	ECTOR, TX
HARDEMAN, TN	UNION, TN	EL PASO, TX
HARDIN, TN	WARREN, TN	ELLIS, TX
HAWKINS, TN	WASHINGTON, TN	ERATH, TX
HAYWOOD, TN	WAYNE, TN	FAYETTE, TX
HENDERSON, TN	WEAKLEY, TN	FLOYD, TX
HENRY, TN	WHITE, TN	FORT BEND, TX
HICKMAN, TN	WILLIAMSON, TN	FRANKLIN, TX
HUMPHREYS, TN	WILSON, TN	FREESTONE, TX
JEFFERSON, TN	ANDERSON, TX	GALVESTON, TX
KNOX, TN	ANDREWS, TX	GOLIAD, TX
LAUDERDALE, TN	ANGELINA, TX	GRAY, TX
LAWRENCE, TN	ARANSAS, TX	GRAYSON, TX
LEWIS, TN	ATASCOSA, TX	GREGG, TX
LINCOLN, TN	AUSTIN, TX	GRIMES, TX
LOUDON, TN	BASTROP, TX	GUADALUPE, TX
MACON, TN	BEE, TX	HALE, TX
MADISON, TN	BELL, TX	HARDEMAN, TX
MARSHALL, TN	BEXAR, TX	HARDIN, TX
MAURY, TN	BOWIE, TX	HARRIS, TX
MCMINN, TN	BRAZORIA, TX	HARRISON, TX
MCNAIRY, TN	BRAZOS, TX	HAYS, TX
MONROE, TN	BROWN, TX	HENDERSON, TX
MONTGOMERY, TN	BURLESON, TX	HIDALGO, TX
MORGAN, TN	CALDWELL, TX	HILL, TX
OBION, TN	CALHOUN, TX	HOCKLEY, TX
OVERTON, TN	CAMERON, TX	HOPKINS, TX
POLK, TN	CAMP, TX	HOUSTON, TX
PUTNAM, TN	CARSON, TX	HOWARD, TX
RHEA, TN	CASS, TX	HUNT, TX
ROANE, TN	CASTRO, TX	HUTCHINSON, TX
ROBERTSON, TN	CHAMBERS, TX	JACK, TX

JASPER, TX  
JEFFERSON, TX  
JOHNSON, TX  
JONES, TX  
KARNES, TX  
KAUFMAN, TX  
KERR, TX  
LAMAR, TX  
LAMB, TX  
LAVACA, TX  
LEE, TX  
LEON, TX  
LIBERTY, TX  
LIMESTONE, TX  
LIVE OAK, TX  
LUBBOCK, TX  
MARION, TX  
MATAGORDA, TX  
MCCULLOCH, TX  
MCLENNAN, TX  
MIDLAND, TX  
MILAM, TX  
MONTAGUE, TX  
MONTGOMERY, TX  
MOORE, TX  
MORRIS, TX  
NACOGDOCHES, TX  
NAVARRO, TX  
NEWTON, TX  
NOLAN, TX  
NUECES, TX  
ORANGE, TX  
PALO PINTO, TX  
PANOLA, TX  
PARKER, TX  
PARMER, TX  
POLK, TX  
POTTER, TX  
RANDALL, TX  
RED RIVER, TX  
ROBERTSON, TX  
RUNNELS, TX  
RUSK, TX

SABINE, TX  
SAN PATRICIO, TX  
SHELBY, TX  
SMITH, TX  
TARRANT, TX  
TAYLOR, TX  
TITUS, TX  
TOM GREEN, TX  
TRAVIS, TX  
TYLER, TX  
UPSHUR, TX  
VAL VERDE, TX  
VAN ZANDT, TX  
VICTORIA, TX  
WALKER, TX  
WASHINGTON, TX  
WHARTON, TX  
WICHITA, TX  
WILBARGER, TX  
WILLIAMSON, TX  
WILSON, TX  
WISE, TX  
WOOD, TX  
YOUNG, TX  
BOX ELDER, UT  
CACHE, UT  
CARBON, UT  
DAVIS, UT  
DUCHESNE, UT  
EMERY, UT  
JUAB, UT  
MILLARD, UT  
MORGAN, UT  
SALT LAKE, UT  
TOOELE, UT  
UINTAH, UT  
UTAH, UT  
WASHINGTON, UT  
WEBER, UT  
ACCOMACK, VA  
ALBEMARLE, VA  
ALEXANDRIA (CITY),  
VA

ALLEGHANY, VA  
AMHERST, VA  
APPOMATTOX, VA  
AUGUSTA, VA  
BATH, VA  
BEDFORD, VA  
BEDFORD (CITY), VA  
BLAND, VA  
BOTETOURT, VA  
BRISTOL (CITY), VA  
BUCKINGHAM, VA  
BUENA VISTA (CITY),  
VA  
CAMPBELL, VA  
CARROLL, VA  
CHARLES CITY, VA  
CHARLOTTE, VA  
CHESAPEAKE (CITY),  
VA  
CHESTERFIELD, VA  
CLARKE, VA  
COLONIAL HEIGHTS  
(CITY), VA  
COVINGTON (CITY),  
VA  
CULPEPER, VA  
DANVILLE (CITY), VA  
DINWIDDIE, VA  
ESSEX, VA  
FAIRFAX, VA  
FAUQUIER, VA  
FLUVANNA, VA  
FRANKLIN, VA  
FREDERICK, VA  
FREDERICKSBURG  
(CITY), VA  
GALAX (CITY), VA  
GILES, VA  
GRAYSON, VA  
GREENSVILLE, VA  
HALIFAX, VA  
HAMPTON (CITY), VA  
HANOVER, VA

HARRISONBURG	SCOTT, VA	KITSAP, WA
(CITY), VA	SHENANDOAH, VA	KLICKITAT, WA
HENRICO, VA	SMYTH, VA	LEWIS, WA
HENRY, VA	SOUTHAMPTON, VA	MASON, WA
HOPEWELL (CITY), VA	SPOTSYLVANIA, VA	PIERCE, WA
ISLE OF WIGHT, VA	SUFFOLK (CITY), VA	SKAGIT, WA
JAMES CITY, VA	SUSSEX, VA	SNOHOMISH, WA
KING WILLIAM, VA	TAZEWELL, VA	SPOKANE, WA
LEE, VA	VIRGINIA BEACH	STEVENS, WA
LOUDOUN, VA	(CITY), VA	THURSTON, WA
LOUISA, VA	WARREN, VA	WALLA WALLA, WA
LUNENBURG, VA	WASHINGTON, VA	WHATCOM, WA
LYNCHBURG (CITY),	WAYNESBORO (CITY),	YAKIMA, WA
VA	VA	ASHLAND, WI
MARTINSVILLE (CITY),	WINCHESTER (CITY),	BARRON, WI
VA	VA	BROWN, WI
MECKLENBURG, VA	WYTHE, VA	BUFFALO, WI
MONTGOMERY, VA	YORK, VA	BURNETT, WI
NEWPORT NEWS	ST CROIX, VI	CALUMET, WI
(CITY), VA	ST THOMAS, VI	CHIPPEWA, WI
NORFOLK (CITY), VA	ADDISON, VT	COLUMBIA, WI
NORTHUMBERLAND,	BENNINGTON, VT	CRAWFORD, WI
VA	CALEDONIA, VT	DANE, WI
ORANGE, VA	CHITTENDEN, VT	DODGE, WI
PAGE, VA	ESSEX, VT	DOOR, WI
PATRICK, VA	FRANKLIN, VT	DOUGLAS, WI
PETERSBURG (CITY),	ORANGE, VT	DUNN, WI
VA	ORLEANS, VT	EAU CLAIRE, WI
PITTSYLVANIA, VA	RUTLAND, VT	FOND DU LAC, WI
PORTSMOUTH (CITY),	WINDHAM, VT	GRANT, WI
VA	WINDSOR, VT	GREEN, WI
PRINCE GEORGE, VA	ADAMS, WA	GREEN LAKE, WI
PRINCE WILLIAM, VA	BENTON, WA	JEFFERSON, WI
PULASKI, VA	CHELAN, WA	JUNEAU, WI
RADFORD (CITY), VA	CLALLAM, WA	KENOSHA, WI
RICHMOND, VA	CLARK, WA	KEWAUNEE, WI
RICHMOND (CITY), VA	COWLITZ, WA	LA CROSSE, WI
ROANOKE, VA	FERRY, WA	LAFAYETTE, WI
ROANOKE (CITY), VA	FRANKLIN, WA	LANGLADE, WI
ROCKBRIDGE, VA	GRANT, WA	LINCOLN, WI
ROCKINGHAM, VA	GRAYS HARBOR, WA	MANITOWOC, WI
RUSSELL, VA	JEFFERSON, WA	MARATHON, WI
SALEM (CITY), VA	KING, WA	MARINETTE, WI

MARQUETTE, WI	JACKSON, WV	WASHAKIE, WY
MILWAUKEE, WI	JEFFERSON, WV	WESTON, WY
MONROE, WI	KANAWHA, WV	
OCONTO, WI	LEWIS, WV	
ONEIDA, WI	LOGAN, WV	
OUTAGAMIE, WI	MARION, WV	
OZAUKEE, WI	MARSHALL, WV	
PIERCE, WI	MASON, WV	
POLK, WI	MCDOWELL, WV	
PORTAGE, WI	MERCER, WV	
PRICE, WI	MINERAL, WV	
RACINE, WI	MONONGALIA, WV	
RICHLAND, WI	MONROE, WV	
ROCK, WI	MORGAN, WV	
RUSK, WI	NICHOLAS, WV	
SAUK, WI	OHIO, WV	
SAWYER, WI	PENDLETON, WV	
SHAWANO, WI	PLEASANTS, WV	
SHEBOYGAN, WI	POCAHONTAS, WV	
ST CROIX, WI	PRESTON, WV	
TAYLOR, WI	PUTNAM, WV	
TREMPEALEAU, WI	RANDOLPH, WV	
VERNON, WI	RITCHIE, WV	
WALWORTH, WI	ROANE, WV	
WASHINGTON, WI	TUCKER, WV	
WAUKESHA, WI	TYLER, WV	
WAUPACA, WI	UPSHUR, WV	
WAUSHARA, WI	WAYNE, WV	
WINNEBAGO, WI	WIRT, WV	
WOOD, WI	WOOD, WV	
BERKELEY, WV	ALBANY, WY	
BOONE, WV	BIG HORN, WY	
BRAXTON, WV	CAMPBELL, WY	
BROOKE, WV	CARBON, WY	
CABELL, WV	CONVERSE, WY	
CALHOUN, WV	FREMONT, WY	
FAYETTE, WV	GOSHEN, WY	
GRANT, WV	LARAMIE, WY	
GREENBRIER, WV	LINCOLN, WY	
HAMPSHIRE, WV	NATRONA, WY	
HANCOCK, WV	PLATTE, WY	
HARDY, WV	SWEETWATER, WY	
HARRISON, WV	UINTA, WY	