The Spatial Relationships among Neurotoxicant Exposure, Child Admissions, and Mental Health Assessment Scores: How do they Interact in the State of Ohio?

Dissertation

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Abstract

In 2007, billions of pounds of known and suspected neurological toxicants were released into the air and water of the United States. Ohio had the dubious distinction of being one of the worst offenders because it was in the top-five emitters nationally (U.S. EPA, 2009a). While environmental toxicants are harmful to everyone, children are especially vulnerable because of the rapid growth and development that occurs throughout infancy and childhood (NRC, 1993). Acute or chronic exposure to these chemicals can cause a variety of psychiatric symptoms in children because they have the capacity to bypass or degrade the blood-brain barrier (Gallo, 2008).

This ecologic study investigated spatial relationships between neurotoxicant exposure and mental illness. Theories related to deep ecology and children’s increased vulnerability informed the research agenda and suggested that children would be negatively impacted by neurotoxicants. Two basic questions guided the study: 1) How do neurotoxicant hazard quotients/indices impact child admissions, and 2) How do neurotoxicant hazard quotients/indices impact mental health assessment scores? The research design controlled for several macro-level (e.g., child poverty) and micro-level (e.g., race) variables based upon prior research.

Data from the Ohio Department of Mental Health, the National-scale Air Toxics Assessment, and American Community Survey were united and modeled with geostatistical software packages (e.g., ArcMap 10.0). Address data were successfully
geocoded for 93 percent of all child records. Almost 68,000 admission records for youth, ages 5-17, were aggregated to the census tract level. After developing census tract admission rates, Empirical Bayes smoothing was used to correct for census tract with small numbers of admissions. Ordinary least squares regression was used to develop global parsimonious models and geographic weighted regression (GWR) was used to examine local relationships.

GWR models explained a large percentage of the variance in child admissions (46%) and attention deficit-hyperactivity disorder (AD-HD) admissions (40%); however, the total hazard quotient for neurotoxicants was not statistically significant in either model. The GWR model for pervasive developmental disorder only explained five percent of the variation in admissions, and the model for AD-HD with the manganese hazard index only explained 15 percent of the variation in admissions. GWR models associated with mental health assessment scores explained a moderate percentage of the variance in hopefulness (39%) and problem severity (39%) assessment scores. Child functioning was the only outcomes variable associated with a neurotoxicant (i.e., solvents). While this model explained 58 percent of the variation in child functioning, it must be interpreted with caution because of the high degree of local multicollinearity on the western edge of the state.

Findings from this study suggest neurotoxicants may play a role in child AD-HD admissions and child functioning scores, but more research needs to be conducted to
validate these findings. Future researchers could focus on smaller geographic areas instead of the state or use different statistical techniques like spatial dependence modeling or hierarchical linear modeling. Finally, they could conduct similar research other populations to expand the generalizability of the findings.
Dedication

Dedicated to my wonderful wife, Kasey
Acknowledgements

This work would have not been possible without the continuous support and assistance from numerous individuals. First and foremost, I would like to thank my wife for giving me the time to complete this grandiose project. She managed to keep her sanity despite having to take over a majority of the child care for our twin daughters and accomplish the never ending series of tasks necessary to run a household. Had she not given me most weekends off for several months, this project never would have come to fruition. Special thanks also go out to her parents for babysitting the twins while I was working. I would also like to thank my committee for their support and feedback on this project. Drs. Theresa Early, Desheng Liu, Jay Wilkins, Keith Warren and Carla Curtis were always happy to provide the answers I needed to complicated questions. Somehow, they even made it through a draft of this dissertation, which in and of itself deserves a special award.

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Innovations also deserves special thanks because he wrote the SQL code that allowed me to query the clients of interest. Staff at the U.S. EPA provided me with countless answers about the nuances of their data when I was considering incorporating it into my dissertation. Tim Antisdel was my initial contact at the U.S. EPA, and he helped me to understand the strengths and limitations of information contained in the Toxic Release Inventory. Dr. Richard Engler provided information about Risk-screening Environmental Indicators software program and gave me a deeper understanding of the calculation of toxicity weights. Tom McMullen gave me the list of toxicants classified as solvents in the National Emissions Inventory. Pat Garvey, Sally Dombrowski, and Betsy Metcalf all helped me understand the limitations of the data provided to the EPA, especially data on seasonal emissions. Finally, thanks goes out to individuals at academic institutions who provided advice. Dr. John Graham, of Pennsylvania State University, and Dr. Elizabeth Stasny, of The Ohio State University, both provided guidance about multiple imputation for which I am deeply grateful. Dr. Tom Welton, of the Imperial College of London, helped me to understand the difficulty in classifying solvents. The ResearchGate.net community also provided assistance, especially Dr. Robert Brennan of Harvard, with where the project can go in the future.
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Chapter 1: Introduction

In 2004, 826 million pounds of known and suspected neurological toxicants were released into the air and water of the United States. Ohio had the dubious distinction of being one of the worst offenders (NJPIRG Law & Policy Center, 2007); the state represented 20 percent of all neurotoxicant releases (fifth in the nation). Cronin and Kennedy (1999) say industry leaders claim that the limits imposed by current environmental regulation (e.g., Clean Air Act of 1990, Clean Water Act of 1977) are high enough to make the effects of the pollution negligible. However, this conclusion is merely speculation because the toxicology related to a vast majority of the chemicals either has not been reported or minimally studied (Environmental Defense Fund, 2005a; Johnson, 1995), leaving many unanswered questions about the cumulative toxic burden placed on people exposed to these chemicals.

While environmental toxicants are harmful to everyone, children are especially vulnerable because of the rapid growth and development that occurs throughout infancy and childhood (NRC, 1993). As Mott, Fore, Curtis, and Solomon (1997) explain, “pound for pound, children breathe more air, drink more water, and consume more food than adults. This higher rate of intake means that children will receive higher doses of whatever contaminants are present in the air, water, or food” (p. 2). Neurotoxicants in particular are extremely dangerous because of their impact on the nervous system. Acute or chronic exposure to these chemicals can cause a variety of psychiatric symptoms in
children because they have the capacity to bypass or degrade the blood-brain barrier (Gallo, 2008). Although most exposure to neurotoxicants is short-term in nature with acute effects, effects from long-term exposure can be devastating and last a lifetime (Brown, 2002).

Understanding that children are at risk from neurotoxicants is the first step in changing the status quo. Children are being poisoned without their knowledge or consent, and it does not make sense for our society (and the social work profession in particular) to idly stand by while our children are suffering. True, many children do not display the symptoms to qualify for clinical diagnosis, but the subclinical effects may be quite severe. As Grandjean and Landrigan (2006) reflect on the scientific research they conclude, “neurodevelopmental disorders caused by industrial chemicals has created a silent pandemic in our society” (p 8). Just as our forefathers bequeathed us a healthy environment, we owe the same to the newest arrivals and to future generations. Anything less would ensure a modicum of adverse health effects for children in the best-case scenario and a complete environmental catastrophe in the worst-case scenario.

One of the topics that remain to be explained is the spatial relationship between toxicant release and child mental health admissions and assessment scores at intake. Logically, one could assume children’s mental health assessment scores would decrease as the concentration of environmental toxicants increase; however no such scientific literature exits that either validates or invalidates this assumption. The scarcity of information on this topic is regrettable because the information could be beneficial to policy makers, industry leaders, and other stakeholders. Historically, the regulation of
chemicals released into the environment has been controversial, and new information could inform the ongoing debate about the appropriate levels of neurological toxicants.

*Problem Statement*

The purpose of this ecologic study is twofold: first, exposure to neurotoxicants is compared to child admissions for Ohio’s public behavioral health system, and second, exposure to neurotoxicants is compared to mental health assessment scores. More specifically, spatial analyses are used to analyze whether a proximity relationship exists between the independent and dependent variables at the census tract level for the 2007 calendar year. The primary independent variables are hazard quotients/indices that indicate exposure to neurological toxicants derived by the National-scale Air Toxics Assessments (NATA) division of the EPA (U.S. EPA, 2009b). All neurological toxicant hazard quotients are summed in the analyses unless there is a well-developed literature that associates particular toxicants with mental illness (e.g., lead). Dependent variables are obtained from the Ohio Department of Mental Health (ODMH). The first dependent variable, mental health assessment scores, is defined generally as the three major psychiatric outcomes acquired with the Ohio Scales questionnaires: hopefulness, functioning, and problem severity (ODMH, 2010). The second dependent variable, child admissions, is the crude admissions rate per census tract that is smoothed with poisson gamma empirical Bayes smoothing. In order to examine the unique contribution of pollution to mental health outcome scores and admissions, several macro-level (e.g., neighborhood poverty) and micro-level (e.g., race) control variables are obtained from the American Community Survey.
Chapter 2: Literature Review

Chapter 2 reviews the theories and research that are used to guide this research study. This chapter begins with a discussion of the theoretical foundation of this research and then moves into the macro and micro theories. Next, a literature review is provided for each of the independent, dependent, and control variables. Finally, the research objectives specify the research questions investigated for this study.

Theoretical Foundation

Environmental issues are becoming increasingly important as people recognize the profound impact their lifestyles are having on the world around them. Newspaper headlines, television news reports, and the internet have been powerful tools in raising awareness about the relationship between effects of pollution on health and well-being. Frequent investigations on local environmental issues (e.g., pollution of waterways) and global environmental issues (e.g., climate change) have led to a “global awakening” in which people understand that Earth’s entire biosphere as a closed system in which all species, great and small alike, have an important stake (Korten, 2006). Faced with the realities of environmental degradation and destruction, people have begun to demand a new cultural paradigm that moves away from anthropocentric theories toward a new theory that emphasizes the interdependence between humanity and the Earth.
Macro Theory: Deep Ecology

Deep ecology is a theoretical tradition that speaks to humanity’s connectedness to the environment, and it calls for dramatic social changes to correct the imbalance in the contemporary relationship between humanity and the planet (Naess, 1973). Values within deep ecology speak to the importance of harmony and reverence for nature, the inherent worth of all beings, and human responsibility toward the environment. Each of these values is built upon the tenet that all beings are interdependent. Van Wormer, Besthorn, and Keefe (2007) explain that deep ecology offers the realization that “humanity is part of a complex totality of interconnected relationships and that these connections among both human and non-humans are the very essence of existence” (p. 247).

Proponents of this paradigm believe people will accept these values once they engage in activities designed to raise an ecological consciousness like deep questioning, deep empathy, and holistic inquiry. These processes are believed to help stimulate this awareness through the realization of the privileges humans have as the dominant species, questioning the underlying values of societies that encourage material wealth, and establishing a profound sense of connection with the environment (Besthorn & Canada, 2002). Once consciousness is raised, advocates of this theory suggest that people will engage in activities that promote concepts like sustainability and environmental justice. No longer will people see themselves as disconnected from the environment, but rather they will see themselves as collaborators in creation of life (Coates, 2003), given the responsibility to be environmental stewards for the planet.

The premises and the conclusions drawn from deep ecology rest on several main assumptions: 1) all life is interrelated, 2) the environment has been so exploited that all
planetary life is threatened, and 3) humanity has the ability to change its paradigm on a
global scale. The assumption that all life is interrelated is a basic notion of systems
ecology, which seeks to understand the interaction between biological and ecological
systems (Kitching, 1983). Every living organism on the planet is made of the same basic
elements and has evolved together into the magnificent web of life (Mary, 2008).
Because organisms all share the same basic biology, life on Earth also shares the same
vulnerabilities to natural and man-made chemicals, a concept which has been thoroughly
demonstrated in the environmental sciences literature (Pachauri & Reisinger, 2007;

Deep ecology also assumes the current health of the planet is of paramount
importance. Humanity is one of the few species that can easily thrive in every ecosystem;
however, it is the ability to develop technology that has put the planet at greatest risk.
While humanity has made great strides in areas like medicine, transportation, and
communication, some cultures have dramatically changed, becoming obsessed with the
products or “stuff” of modernity. Coates (2003) points out that this new attitude has led to
an environmental paradox:

The ecological crisis is a long-term threat because our economy is and
extractive economy: instead of living off the natural production and
reproduction of Earth (the interest on nature’s capital), we are depleting
non-renewable resources and exploiting renewable resources frequently
beyond their capacity to survive (p. 20).

The final assumption of deep ecology is that humanity has the capability to
transform the dominant paradigm to one that is in harmony with the global ecosystem.
Coates (2003) points out that humanity lived peacefully with the earth for the last 10,000 years of its existence. If one considers the age of the universe, then the fraction of time during which humanity has caused environmental destruction is fairly small. Using Carl Sagan’s (1977) cosmic clock, he calculates that “the vast majority of the unprecedented destruction caused by human occurred in only the past half second” (p. 68). Bearing that in mind, one could speculate it is definitely possible to return to a simpler lifestyle that respect all forms of life.

Deep ecology is applied to this investigation in several ways. This theoretical paradigm serves as a useful foundation to understand the nature of the relationship between neurotoxicant exposure and its impact on mental health admissions and intake assessment scores. From this theory, a proximity relationship is predicted to exist among the variables of interest, and that any increase in neurotoxicants would bring about a corresponding decrease in mental health intake assessment scores or vice versa. Deep ecology also provides significant insights into the implications for this research study. If a significant relationship is found among the variables, then this theory offers advice on helping people understand the impact their actions have on the environment and also presents a way to question the dominant paradigm of anthropocentrism.

*Micro Theory: Children’s Increased Vulnerability*

Micro theory about the interaction between toxicants and children’s physiology are also important to this study. Environmental toxicants are harmful to infants, children, and adolescents (hereafter children) because they have an increased biological vulnerability based on three key characteristics distinctive to children (Mott et al., 1997; NRC, 1993). One such characteristic, differential intake, relates to the greater exposure
children have to chemicals based on their physiology. For example, young children may eat seven and a half times the number of fruits and drink between 11 and 21 times the amount of juice as adults, leading children to experience dramatically higher exposure than adults (Galvez, Forman, & Landrigan, 2005). Another reason the level of exposure differs between adults and children is their distinctive behavior. Young children, especially infants, are known to explore the world around them by putting objects in their mouths; they simply do not understand that some substances may be harmful. To make matters even more complicated, children usually spend most of their time at the ground level, which is where airborne chemicals settle. Mott et al. (1997) explains children’s close proximity to the ground enables them to “receive greater inhalation and dermal exposure to chemicals present on floors, carpet, grass, or dirt” (p. 5). Formaldehyde, a chemical preservative found on most new carpets, is one good example of a neurotoxicant to which young children may be exposed. Were children to play on these carpets, they may be exposed through inhaling the chemical or ingesting the chemical when they place their hands in their mouths.

Rapid growth and development is another factor that increases the biological vulnerability of children. In utero exposures to toxic chemicals are frequently linked with developmental abnormalities and miscarriage (Landrigan & Garg, 2002). Until the late 1950’s, doctors believed that the placenta protected the developing fetus from all toxins; however, they learned all too late that transplacental absorption occurred when mothers were given thalidomide. What physicians thought was an effective treatment for morning sickness turned out to be something quite different when infants were born with severe birth defects (Bearer, 1995). Polychlorinated biphenyls (PCBs) are another example of
chemicals that can interfere with growth and development because of their ability to disrupt endocrine production (Landrigan & Garg, 2002). Once ingested, PCBs stay in the body for many years, allowing mothers to pass along the dangerous chemicals to their children—even if they only eat small amounts of contaminated food per month (Mott et al., 1997).

The last major factor responsible for the increased biological vulnerability of children is the child’s distinctive means of absorption, metabolism, and excretion of substances (Mott et al., 1997). Children have increased chemical vulnerability because chemicals in their digestive tract are absorbed at a different rate than chemicals in adult digestive tracts. For instance, lead is a chemical that is absorbed at higher rates within children. Bearer (1995) explains, “it is estimated that an adult will absorb 10% of ingested lead, whereas a 1- to 2-year old child will absorb 50% of ingested lead” (p. 9). Once inside the body, children retain 32 percent of lead compared to the five percent retention rate of adults (Liu, Goyer, & Waalkes, 2008). Along the same line of thinking, children also metabolize substances at different rates than adults. In some cases, this ability may serve as a protective feature for children, but in most cases this ability leads them to experience greater exposure and accumulation of harmful chemicals (Bearer, 1995; Mott et al., 1997). Children also have a greater degree of biological vulnerability because they excrete chemicals differently than adults. The ability to purge chemicals is much less developed for children than it is for adults. For instance, Bearer (1995) reveals that infant kidneys do not have the capacity to filter out chemicals at the same rate as adults until the child is between 12 and 16 months old. This significant disadvantage also
means that toxic chemicals have a greater chance of staying in children for longer periods than in adults, thereby causing greater damage while in the body.

Independent Variables

Environmental toxins are ubiquitous chemicals that have the capacity to profoundly affect the physiology of all life on the planet. Since antiquity, plants and animals have used these compounds for self-defense; however, modern humans have advanced the science considerably and created artificial toxins called toxicants. No longer are these substances used solely for hunting and warfare, but rather for the production of goods ranging from pharmaceutical drugs to synthetic compounds like plastic. Of the estimated 70,000 to 75,000 chemicals which have been produced by industry (Mott, 1996), only a minority (＜20%) have been studied for their effects on human physiology (Johnson, 1995). This fact is especially troubling because these chemicals may have adverse effects on numerous biological processes, causing acute health conditions like bronchitis in the best-case scenarios and chronic conditions like lung cancer in the worst-case scenarios.

Neurological Toxicants

Neurotoxicants are chemicals that adversely impact the nervous system. Acute or chronic exposure to these chemicals can cause a variety of psychiatric symptoms because they have the capacity to bypass or degrade the blood-brain barrier (Gallo, 2008). Once past this critical barrier, these toxicants can directly interact with the brain and central nervous system through disruption of normal neuron functioning. Primarily, these chemicals affect neurons one of two ways: they can damage neuron structure or they can affect neurotransmission (Table 1). Neurological toxicants that change neural structure
usually destroy one or more parts of the neuron, and toxicants that affect neurotransmission change the way the neurons communicate with each other. The physical manifestation of these effects is as varied as the number of compounds released into the environment and largely depends on the specific classification of the toxicant.

<table>
<thead>
<tr>
<th>Target</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Neuron Structure</strong></td>
<td></td>
</tr>
<tr>
<td>Neuronopathy</td>
<td>Degeneration of an entire nerve cell including the dendrites and the axon</td>
</tr>
<tr>
<td>Axonopathy</td>
<td>Damage to the axon which causes the axon and myelin surrounding it to degenerate, while the cell body remains intact</td>
</tr>
<tr>
<td>Myelinopathy</td>
<td>Deterioration of the myelin sheath that results in impairment or loss of transmission of the nerve impulse</td>
</tr>
<tr>
<td><strong>Neurotransmission</strong></td>
<td></td>
</tr>
<tr>
<td>Blocking Agents</td>
<td>Bind the chemical to the presynaptic terminal, eliminating the chance that other neurotransmitters can affect the terminal</td>
</tr>
<tr>
<td>Depolarizing Agents</td>
<td>Eliminate the charge which alters the permeability of the cell towards sodium and potassium ions</td>
</tr>
<tr>
<td>Stimulant Agents</td>
<td>Increase the excitability (sensitivity) of neurons</td>
</tr>
<tr>
<td>Depressant Agents</td>
<td>Decrease the excitability (sensitivity) of neurons</td>
</tr>
<tr>
<td>Anticholinesterase Agents</td>
<td>Inhibit the enzyme that is responsible for breaking down acetylcholine, which results in increased and prolonged stimulation of the postsynaptic membrane</td>
</tr>
</tbody>
</table>

Table 1. Human health effects of exposure to neurotoxicants¹

¹ Definitions adapted from Kent, 1998 (p. 133-137)
Neurological toxicants can be classified into two broad categories, both of which may cause significant harm. Metals (e.g., lead and mercury) and solvents (e.g., benzene and trichloroethylene) have well-known physical and psychiatric signs and symptoms for anyone who comes into contact with these substances (i.e., through absorption, ingestion, or inhalation). Metals are one broad category of neurological toxicants released into the environment. Generally, metals are defined as solid materials that exhibit physical properties like electrical and thermal conductivity. Metals have been used extensively throughout human history, and it is estimated that metalworking began at least 7,000 years ago when humanity realized it could extract metal from ore (Liu, Goyer, & Waalkes, 2008). Historically, only a limited number of metals were used in industry, but advances in technology in the late 19th and early 20th century led to the widespread use of metals in extraction, manufacturing, and many others industries. Typically, employees in industries that directly work with metal (e.g., miners and steelworkers) are the most likely to come in contact with the compounds, but many industries dispose of metals or metal byproducts in the air and water, making everyone susceptible to the consequences.

Ohio industries release many types of metals, but three of those metals are dangerous to children. As previously mentioned, the metal that has the most profound effect on children’s health is lead because it is harmful even in small quantities. Inorganic lead, lead that does not contain carbon, is commonly used for products like paints and dyes, while organic lead can be used for products like water pipes and ammunition (Liu, Goyer, & Waalkes, 2008). Exposure pathways for lead may occur through using every day products containing lead, occupational exposure (e.g., metal smelting) or recreational exposure (e.g., planting flowers in contaminated soil). As far back as 400 B.C.,
Hippocrates recognized the toxicity of lead in miners, and the effects he noticed are similar to the effects seen today (Cain, 1988). In addition to decreasing intelligence and slowing psychomotor development, moderate to severe lead poisoning causes myelinopathy, which manifests itself through psychiatric symptoms affecting mood (e.g., depression), behavior (e.g., impulsivity), cognition (e.g., lack of attention), perception (e.g., hallucinations), and mental retardation or developmental delays in children (Brown, 2002; Kent, 1998).

Mercury is another well-researched metal with well-documented human toxicity. In use since at least 1500 B.C., this metal is employed for activities like mining and the manufacture of industrial chemicals (Liu, Goyer, & Waalkes, 2008). Seventy percent of mercury emissions are anthropogenic in nature, and atmospheric mercury frequently originates from heavy industry like power plants and metal smelters (Trasande, Landrigan, & Schechter, 2005). Once released into the air, mercury particles have the potential to combine with other chemicals to become organic methyl mercury and then bioaccumulate in the food chain, which is the most likely exposure route to be through food consumption (Kent, 1998). Methyl mercury is so prevalent in this state that the Ohio EPA recommends that everyone limit fish intake to one time per week in the cases of some species (e.g., Yellow Perch) and one time per month in the cases of other species (e.g., Largemouth Bass; Ohio EPA, 2010). Exposure to mercury in utero is especially dangerous because the blood-brain barrier is not developed, and “levels in the fetal brain [can rise] about 5–7 times that in maternal blood” (Clarkson, 2002, p. 12). Mercury poisoning was recognized in the 18th and 19th century when the substance was used to make felt hats; this practice caused numerous psychiatric symptoms, resulting in the
popular expression “mad as a hatter” (Kent, 1998). Exposure to excess levels of mercury can lead to neuronopathy, a condition which has psychiatric implications for mood (e.g., irritability), behavior (e.g., crying), cognition (e.g., memory loss), and perception (e.g., paranoia; Brown, 2002).

Manganese is another metal emitted by Ohio industries that may pose a serious health concern. This metal has been used since prehistoric times in the pigmenting of paint among other activities (Liu, Goyer, & Waalkes, 2008). Emissions related to manganese are from mining operations, metal smelting operations, and manufacturing operations for products containing manganese (e.g., steel alloys and batteries; U.S. EPA, 2009a). For the last 15 years, manganese has been used for a gasoline additive when lead was phased out. The additive, methylcyclopentadienyl manganese tricarbonyl (MMT), has raised serious concern among some health officials because of its unknown health effects and ability to leach into aquifers (Brown, 2002). Manganese is the only metal discussed here that is required for the human body to function. Manganese was designed to bypass the blood-brain barrier with little to no effort. In small doses, manganese is beneficial to the human body because it is necessary for many cellular functions like the production of essential enzymes (Liu, Goyer, & Waalkes, 2008). However, the benefits of this chemical can be negated when people are over-exposed to it, causing manganese poisoning (a.k.a., manganism). Exposure to excess levels of mercury can have psychiatric implications for mood (e.g., irritability and/or mild euphoria), behavior (e.g., uncontrollable laughing and/or crying), cognition (e.g., poor concentration), and perception (e.g., hallucinations; Brown, 2002).
Solvents, some of which are neurotoxicants, are another broad category of compounds commonly found in the environment (Reichardt, 1988). Solvents are classified as liquid organic chemicals because of their lipophilicity (i.e., the ability to dissolve in fats) and volatility. Alcohols (e.g., ethanol) represent a class of solvents that have been widely used throughout human history, but most solvents have been developed since the industrial revolution when petroleum use became common. These substances have the unique ability to, “dissolve, dilute or disperse materials that are insoluble in water” and consequently are used in products like paints, inks, aerosol sprays, dyes, and fuels (Bruckner, Anand, & Warren, 2008, p. 981). The low boiling point and speed with which solvents evaporate make inhalation the primary route to exposure, although solvents may also be absorbed into the body through ingestion. Widely used solvents like 1, 1, 1-trichloroethane are produced in the hundreds of millions of tons each year for use in cleaning products, which makes it highly likely that most people are exposed to similar solvents through occupational manufacture or in-home use (U.S. EPA, 2009a).

Several classes of solvents are more likely than others to impact mental health. Aromatic hydrocarbon, named as such because some of the compounds have a sweet aroma, represent one type of solvent that is of particular importance to children’s health.\(^2\) Benzene is the simplest aromatic hydrocarbons because it is made up of six carbon atoms. It is also one of the most recognized aromatic hydrocarbons, owing in part to its high production volume (ranked 22\(^{nd}\) in 2007), and its numerous uses include adhesives, dyes, ink, resins, gasoline, artificial leather, soaps, rubber, and waxes (Brown 2002, U.S.

\(^2\) None of the 17 polyhydrocarbons listed by the ATSDR are recognized or suspected neurotoxicants; therefore, they will not be discussed here.
Benzene enters the environment chiefly through industrial emissions, but it can also originate from natural sources like volcanoes and forest fires. Once in the atmosphere, benzene can precipitate to the Earth’s surface and contaminate soil and water. Exposure to benzene or its derivatives typically occurs through breathing in the chemical, whether it is smoke from a factory or gas particles from a gasoline station. Human behavior also plays an important part in benzene exposure because cigarettes contain high concentrations of benzene. While it is not known whether children are more susceptible than adults, benzene does have important health implications (ATSDR, 2007). In addition to affecting most other systems in the human body, exposure may cause neurological symptoms ranging from memory loss and nervousness to seizures and coma (Brown, 2002).

Chlorinated hydrocarbons are another general category of solvents that can negatively affect mental health. These chemicals are often the building blocks of more complex chemicals and are made up of chlorine, hydrogen and carbon atoms like their name suggests (U.S. EPA, 2009c). Trichloroethylene is a commonly used chlorinated hydrocarbon that industries employ for degreasing metal parts, removing paint and spots, and creating adhesives (ATSDR, 2003). This compound does not naturally occur in the environment, but people may come into contact with it through using products containing the chemical, working in an occupation where the chemical is used, or breathing contaminated air and drinking contaminated water. Children are not known to suffer greater effects from this chemical than adults, but their bodies are still more likely to absorb it than adults (ATSDR, 2009). Trichloroethylene is best known for its ability to cause cancer and immune disorders, but it also has the ability to impact neurological
health (Bruckner, Anand, & Warren, 2008). The most common psychiatric symptoms for persons exposed to this chemical impact mood (e.g., mood changes), behavior (e.g., mania), cognition (e.g., poor concentration), and perception (e.g., delusions; Brown, 2002).

**Neurotoxicants in Ohio**

Longitudinal data from the Environmental Protection Agency’s (EPAs) Toxic Release Inventory (TRI) database showed that Ohio citizens have been at great risk for exposure to neurotoxicants (U.S. EPA, 2009a; see Appendix A for complete list of neurotoxicants). From 2001 to 2008, industries released an average 41.8 million pounds of neurological toxicants into Ohio’s atmosphere, ranking Ohio among the top-5 dischargers of neurotoxicants in the nation every year. The toxic burden to Ohio’s atmosphere is even more dramatic once one multiplies the toxicity of the chemical by the pounds of each chemical released, thereby giving a more accurate estimation of the hazard associated with the chemicals. The weighted average of neurotoxic chemicals over eight years balloons to 24.2 billion pounds released into Ohio’s atmosphere. Although the numbers are extraordinarily high, it is important to note that neurotoxicant release in Ohio has actually decreased over time (Table 2).
Recent data suggests that emissions fell below the average amount released in previous years. In 2008, 32.7 million pounds of neurotoxicants were released into Ohio’s atmosphere, representing a weighted average of 19.3 billion pounds. These data indicate a 33.45 percent and 30.99 percent decrease respectively in the release of all airborne toxicants in Ohio. Federal EPA officials have explained that their own research has shown that facility closures and reduced production have been the primary reasons for the decrease in emissions in Ohio and the nation at large over this period (T. Antisdel, personal communication, February 22, 2010). Another reason for the decrease may stem from government programs aimed at reducing emissions. Recently, Ohio EPA officials began a campaign to encourage some of the top TRI reporters to voluntarily reduce their emissions through pollution reduction and prevention strategies, which would have influenced the emissions levels after 2007 (Ohio EPA, 2009).

An analysis by zip code revealed that most of the top-five zip codes experienced decreases in neurotoxicant release from 2001 to 2008 (Table 3). The most notable exception to the rule was the 44004 zip code in Ashtabula; that city experienced a 47.52 percent increase in emissions over time, most likely due to new industry. The 45802 zip

<table>
<thead>
<tr>
<th>Ohio Data</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
<th>2005</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Toxicants (Million lbs)</td>
<td>49.2</td>
<td>47.2</td>
<td>44.0</td>
<td>42.7</td>
<td>41.6</td>
<td>38.3</td>
<td>38.4</td>
<td>32.7</td>
</tr>
<tr>
<td>Percent change</td>
<td>N/A</td>
<td>-4.1</td>
<td>-6.6</td>
<td>-3.0</td>
<td>-2.6</td>
<td>-7.8</td>
<td>0.3</td>
<td>-14.9</td>
</tr>
<tr>
<td>Toxicity x Pounds (Billion lbs)</td>
<td>27.9</td>
<td>26.4</td>
<td>28.3</td>
<td>27.0</td>
<td>23.0</td>
<td>21.8</td>
<td>19.9</td>
<td>19.3</td>
</tr>
<tr>
<td>Percent change</td>
<td>N/A</td>
<td>-5.5</td>
<td>7.4</td>
<td>-4.6</td>
<td>-14.7</td>
<td>-5.5</td>
<td>-8.8</td>
<td>-2.9</td>
</tr>
</tbody>
</table>

Table 2. Ohio neurotoxicants
code in Lima also experienced an increase in emissions, but this increase was only 9.82 percent and could easily have resulted from the growth of existing industry. Three of the other five zip codes stayed the same over time. Defiance (i.e., 43512) and Croton (i.e., 43013) were replaced by Marysville (i.e., 43041) and Stratton (i.e., 43961) for the fourth and fifth positions in 2008. Defiance and Croton must have had a substantial decrease in neurotoxicant emissions, going from an unweighted average of 1.8 million pounds to less than 750 thousand pounds. Industry closure is the most likely reason for the decrease in emissions.

<table>
<thead>
<tr>
<th>Rank</th>
<th>Zip Code</th>
<th>City</th>
<th>Total Air Emissions 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44004</td>
<td>Ashtabula</td>
<td>8,289,259.70</td>
</tr>
<tr>
<td>2</td>
<td>45802</td>
<td>Lima</td>
<td>2,605,182.00</td>
</tr>
<tr>
<td>3</td>
<td>45750</td>
<td>Marietta</td>
<td>1,671,398.55</td>
</tr>
<tr>
<td>4</td>
<td>43041</td>
<td>Marysville</td>
<td>944,935.00</td>
</tr>
<tr>
<td>5</td>
<td>43961</td>
<td>Stratton</td>
<td>765,935.28</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rank</th>
<th>Zip Code</th>
<th>City</th>
<th>Total Air Emissions 2001</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44004</td>
<td>Ashtabula</td>
<td>5,618,888.07</td>
</tr>
<tr>
<td>2</td>
<td>45750</td>
<td>Marietta</td>
<td>2,722,659.00</td>
</tr>
<tr>
<td>3</td>
<td>45802</td>
<td>Lima</td>
<td>2,372,205.00</td>
</tr>
<tr>
<td>4</td>
<td>43512</td>
<td>Defiance</td>
<td>1,975,966.70</td>
</tr>
<tr>
<td>5</td>
<td>43013</td>
<td>Croton</td>
<td>1,652,154.00</td>
</tr>
</tbody>
</table>

Table 3. Zip codes with highest unweighted emissions

The zip codes most impacted by neurotoxicants change when the TRI data are weighted by toxicity (Table 4). Marietta (i.e., 45750) replaces Ashtabula (i.e., 44004) as the most toxic city in Ohio, emitting a whopping 18.1 billion weighted pounds of
neurotoxicants. Despite the 51.86 percent reduction in 2008, Marietta still emits more than 8.7 billion weighted pounds of airborne toxicants in 2008. Ashtabula remains near the top of the list, placing second in neurotoxicant release in 2001 and third in 2008. Just as the unweighted data for this city indicate an increase in 2008 emissions, so too does data associated with weighted neurotoxicants; emissions rise from 884.2 million to pounds in 2001 to 1.2 billion pounds of weighted toxicants in 2008 (an increase of 37.19%).

<table>
<thead>
<tr>
<th>Rank</th>
<th>Zip Code</th>
<th>City</th>
<th>Total Air Emissions 2008</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45750</td>
<td>Marietta</td>
<td>8,700,960,256.00</td>
</tr>
<tr>
<td>2</td>
<td>45640</td>
<td>Jackson</td>
<td>1,264,610,261.00</td>
</tr>
<tr>
<td>3</td>
<td>44004</td>
<td>Ashtabula</td>
<td>1,213,028,867.00</td>
</tr>
<tr>
<td>4</td>
<td>45662</td>
<td>New Boston</td>
<td>1,010,653,200.00</td>
</tr>
<tr>
<td>5</td>
<td>44483</td>
<td>Warren</td>
<td>431,019,530.00</td>
</tr>
</tbody>
</table>

Table 4. Zip codes with highest weighted emissions

The other three cities that rank highly in 2001 are replaced in 2008, which indicates a major change in the geographic location of industry. For example, Brook Park (i.e., 44142) ranks third in overall weighted emissions in 2001, but does not even show up on the list in 2008. In fact the average emissions for the top four polluters (excluding the outlier Marietta) raises from 616.0 million weighted pounds to 979.8 million
weighted pounds in 2008, representing a 59.05 percent growth. The change in the release of dangerous airborne chemicals most likely relates to new industries coming into Ohio and to an expansion of existing services. Finally, it is important to note that only two of the cities that show up in the top-five producers in unweighted chemicals also show up in the top-five for weighted chemicals. The importance of this fact cannot be understated because the facilities that emit the highest concentration of unweighted chemicals may not have the most dangerous emissions. If one ranks every zip code in Ohio for 2008, then Jackson (i.e., 45640), which ranks second in weighted emissions, ranks 50th in unweighted emissions. The same can be said for two other cities; New Boston (i.e., 45662) ranks 112th in unweighted emissions and Warren (i.e., 44483) ranks 66th in unweighted emissions.

A variety of industries contribute to neurotoxicant emissions in Ohio. To classify these industries, the North American Industry Classification System (NAICS) can be used to group industries into relatively homogeneous categories. While the NAICS code has 20 general categories for industries in the United States, only 11 of these categories contain industries that emit neurotoxicants. Additionally, some of these categories represent an extremely small portion of toxicant emissions and were combined into an “Other” category for this analysis.
Figure 1 illustrates the airborne release of neurotoxicants by industry. Chemical and allied products industries are the most frequent type of industry in Ohio, and these industries produce chemicals used by the consumer (e.g., bleach) and chemicals used by businesses for product development (e.g., methanol). Data from 2008 show this industry released the highest concentrations of airborne neurotoxicants (>14.5 million pounds). The manufacturing industries produce machinery for factories as well as car parts and auto bodies. The data reveal that this industry released the second highest concentration of airborne neurotoxicants (11.5 - 14.4 million pounds). The utilities industry (e.g., gas and electric) is the third largest producer of neurotoxicants, representing 11 percent of releases (3.7 - 14.3 million pounds). Metal refiners and smelters include businesses that process primary (i.e., new material) and secondary (i.e., recycled material) metals, and 2008 data illustrate these industries are the fourth highest producer of airborne
neurotoxicants (2.6 - 3.6 million pounds). Finally, the “other” category represents a large group of industries that contribute to less than one percent (≤ 2.5 million pounds) of overall neurotoxicant emissions.

Much like the analysis of airborne emissions among cities, the unweighted emissions hide which industries are the most toxic. Figure 2 illustrates the industries responsible for the most hazardous neurotoxicants. While metal refiners and smelters are ranked fourth in chemical releases, chemicals released by these industries are far more hazardous than any other industry.

![Figure 2. Weighted airborne neurotoxicants by Ohio industry](chart)

In fact, 73 percent of emissions come from this industry if the chemical toxicity is taken into account, which accounts for more than 13.9 billion weighted pounds of neurotoxicants. Another profound difference concerns chemical and allied products. This industry has the largest proportion of facilities emitting toxicants (45%), but these
toxicants are nowhere near as dangerous as compared to metal refiners and smelters. Once chemical toxicity is taken into account these facilities only represent 10 percent of the weighted emissions. The only other noticeable difference comes from the manufacturing sector. Whereas manufacturing is ranked second in unweighted chemicals, it comes in third once toxicity is taken into account, representing nine percent of all airborne emissions.

Dependent Variables

*Mental Health Outcomes*

Traditional mental health treatment involves therapeutic or behavioral interventions geared toward helping consumers obtain successful mental health outcomes. This noble goal has led to several important questions: “How does one define mental health outcomes?” and “How does one measure these outcomes?” Practitioners, policy makers, and researchers have struggled to define these outcomes because different outcomes are expected depending on the characteristics of the consumer (e.g., age and diagnosis). For example, children and adults are expected to have dissimilar mental health outcomes because of the vastly different developmental stages. Hoagwood, Jensen, Petti, and Burns (1996) explain, “developmental differences between adults and children suggest that assessment of outcomes for children must include attention to the biological and psychological capacities of the developing child across multiple domains” (p. 1058). Adding to the complexity of outcomes measurement is a lack of uniformity in the way outcomes are quantified. Academic researchers frequently measure conceptually different outcome-related domains; some researchers measure child functioning as an outcome.
(e.g., Canino, Costello, & Angold, 1999) while others measure symptomology (e.g., Achenbach, 1991) or social competence (e.g., Harter & Chao, 1992).

To help guide the field toward standardized outcome assessment, Hoagwood, Jensen, Petti, and Burns (1996) propose a theoretical paradigm for children receiving behavioral health services. The SFCES model focuses on five domains that capture children’s mental health outcomes in the context of the larger environment, each of which is clearly operationalized and can be targeted for treatment: symptoms and diagnosis; functioning; consumer perspectives; environment; and systems. The symptoms and diagnosis domain concerns common psychiatric and behavioral symptoms (e.g., distractibility and sleep difficulties) that may lead to a clinical diagnosis. Assessment of this domain includes the examination of the number, type, duration, and frequency of presenting symptoms. The functioning domain relates to the ability of the children to adapt to the demands in the larger environment (e.g., school performance, peer and family interactions). This domain is evaluated on a continuum in which good adaptation means that children could handle themselves well in circumstances that require normal adaptation while poor adaptation means that the children have an impaired ability to meet competing environmental demands. The third domain, consumer perspectives, refers to the experience of the child and family involved in treatment. Clinicians that evaluate this domain assess the overall satisfaction with treatment as well as other family characteristics like caregiver burden, hopefulness, and quality of life. The environmental domain is the fourth domain, and it focuses on changes in children’s immediate environments (e.g., family and school) due to the intervention. Evaluation of this domain examines positive or negative changes that occur during or following the intervention.
(e.g., family stability and classroom attendance). The final domain, systems, examines service outcomes (e.g., change in service utilization, change in restrictiveness of services) and organizational outcomes (e.g., integration and coordination). The goal of these outcome measurements is to evaluate whether treatment of children has impacted the system at large through impacting agency-level, community-level, and state-level structures.

*Psychiatric Symptomology and Diagnosis*

Empirical research focusing on the relationship between neurotoxicant exposure and psychiatric symptomology falls into three categories: 1) research that investigates the relationship between discrete events and psychiatric outcome (e.g., chemical exposure due to train derailment), 2) research that explores the relationship between mental health diagnosis and specific chemicals, and 3) in etiological studies of mental health diagnoses. Generally, research that focuses on discrete events explores the health effects of man-made disasters on people residing in near-by communities (e.g., Bhopal, Chernobyl, Hiroshima). Oftentimes, this research follows these communities for decades to examine the long-term consequences of the disaster on physical and mental status (e.g., Bromet & Havenaar, 2007); however, this type of research generally is limited by its inability to explain whether the event itself or the chemicals themselves actually caused the psychiatric and somatic symptoms. The other major category of research scrutinizes the effects of specific chemicals on mental health diagnosis (e.g., autism). Lead and mercury are two of the best-known chemicals because their properties have profound short-term and long-term consequences for those exposed to those elements (Brown, 2002).
together, these types of research offer a theoretical guide that can inform the relationship between mental health and exposure to neurotoxicants in the environment.

One example of a discrete event that affected psychiatric outcomes occurred with the 1984 Union Carbide disaster in Bhopal, India. Numerous empirical studies investigated the physical and psychiatric outcomes of chemical exposure after 27 tons of methyl isocyanate gas were accidentally released from the Union Carbide plant when the safety systems failed to contain a chemical reaction (Dhara & Dhara, 2002). All told, 40 square kilometers were contaminated with the poisonous gas, resulting in a death toll estimated at 15,000 to 20,000 people. The survivors of the disaster were beset with numerous health problems including a variety of psychiatric symptoms. Empirical research into the effects of the gas’ toxicity found that exposed people suffered increased posttraumatic stress disorder, pathological grief reaction (e.g., depression), emotional reactions to physical problems (e.g., anxiety), and exacerbation of previous psychiatric problems. Additionally, other research studies found that exposed persons had higher rates of mental illness and more psychomotor (e.g., motor and sensory) and neurological (e.g., numbness and tingling) problems. Dhara and Dhara (2002) concluded that the research established many severe effects of methyl isocyanate gas poisoning; however, the authors cautioned that most research studies reviewed were limited by unclear methodology, misclassification bias, and selection bias. Clearly, this research shows an association between toxicant release and psychiatric symptoms, but one cannot separate the trauma of the event from the physiological effects of the chemicals.

More recently, Greve, Bianchini, Stickle, Love, Doane, & Thompson (2007) conducted a case-control study as part of a lawsuit against a company held responsible for
a hazardous material spill in Eunice, Louisiana. In May 2000, a train derailment caused 17 tank cars to lose their hazardous material, resulting in the evacuation of 2,300 people due to a cloud of toxic chemicals that could be seen 18 miles away. Researchers randomly selected plaintiffs from the community affected by the toxicant release and control group members from a demographically similar community. All participants completed a clinical interview to discuss symptoms and problems associated with the release of hazardous materials, and they filled out standardized instruments that evaluated psychiatric, cognitive, social, behavioral functioning, and distress. Upon analysis of the data, researchers discovered children were psychologically impacted by the release of toxicants in the environment. Greve et al. (2007) explain results show, “that Exposed children experienced more psychological distress (particularly symptoms of anxiety) and more physical symptoms (e.g., headaches, respiratory and gastrointestinal problems as well as general concern over their physical functioning than Controls” (p. 318). While there could be some bias in children’s reports about symptoms due to the legal context of the research and young children’s inability to articulate their experience, the authors conclude that the presence of a control group helped eliminate much of the bias.

Research has also been carried out to explore the relationship between mental health diagnosis and specific chemicals. Windham, Zhang, Geenier, Croen, Grether (2006) conducted a case-control study that explored the associations between autism spectrum disorders and exposure to hazardous air pollutants (HAPs). Researchers selected 284 cases from California’s Department of Development Services and the Kaiser Permanente Medical Program, and 657 control group members were randomly selected from California’s 1994 birth-infant death certificate file. Geographic birth residence of
cases and controls were compared to level of ambient HAPs at the census tract level.
HAPs were classified into four broad categories for testing: metals, aromatic solvents, chlorinated solvents, and other chemicals. Using logistic regression, the authors found that diagnosis and symptoms of autism had the highest adjusted odds ratios for metals and chlorinated solvents. Further analysis by individual chemicals showed that two chemicals stood out from the others; the presence of autism diagnosis and symptoms increased the adjusted odds ratios for vinyl chloride by 1.75 ($\text{CI}_{95}=1.25–2.43$) and for mercury by 1.92 ($\text{CI}_{95}=1.36–2.71$). According to Windham et al. (2006), the major limitations of the study included the estimation of chemical emission, the inability to account for mobility during pregnancy, and the difficulty disentangling the effects of highly correlated toxicants. In conclusion, the authors stated their results suggested a relationship exists between ambient level of HAPs and autism, and they call upon other scientific investigators to verify their findings.

Palmer, Blanchard, Stein, Mendell, & Miller (2006) illustrated the relationship among mercury release, special education rates, and autism diagnosis in a cross-sectional study in Texas. The authors compared autism diagnoses found in school records to the industry emission of mercury at the county level. Using poisson regression modeling to account for county-level clustering effects, the researchers discovered a relationship between mercury release and autism. Palmer et al. (2006) explained, “on average, for each 1,000 lb. of environmentally released mercury, there was a 43% increase in the rate of special educations services and a 61% increase in the rate of autism” (p. 203). Admittedly, the authors stated there were several limitations to the study, including the inability to examine data at the individual level, the use of cross-sectional (i.e., not
longitudinal) data, and the potential bias in school administrative data related to autism prevalence. Despite these limitations, the authors said results from this study warranted further examination into the associations among mercury release, autism diagnosis and special education, potentially leading to legislation that would decrease the amount of mercury released by industry.

Bouchard, Laforest, Vandelac, Bellinger, and Mergler (2007) examined AD-HD symptoms in children exposed to manganese from tap water in Quebec Canada. A particular area of Quebec was chosen that had naturally occurring high levels of manganese and minute levels of other heavy metals (e.g., lead and mercury). Forty-two children were enrolled in the study; some received water from a natural well notoriously high in manganese and others received water from a well low in manganese. The water was sampled in each participants home and hair samples were taken. While most parents (91%) bought bottled water because of the poor taste of well water, they still used well water for cooking (96%) and other activities (e.g., mixing fruit juice). Children were evaluated by parents and teachers with the Revised Conner’s Scale for symptoms of AD-HD. Results indicated that manganese levels found in children’s hair were significantly higher in homes that had water from the well with high levels of manganese. Findings also indicated that children with elevated manganese levels in their hair (>3 µg/g) scored significantly higher on the Revised Conner’s Scale for AD-HD oppositional behaviors, hyperactivity and the overall AD-HD Index, even when age and gender were controlled for in regression modeling. Bouchard et al. (2007) suggest that the strengths of the study were the homogeneous sample and the trace levels of other elements in the water. The authors also acknowledged that their results are limited because of the small sample size
and possible self-selection bias toward parents who believed their children had behavioral problems.

Karagas et al. (2012) reviewed the literature about adverse health effects of low-level methylmercury exposure. Of particular interest to this study, the authors reviewed research about methylmercury exposure and ADHD. Several cross-sectional and case-control studies came to different conclusions about whether mercury had an impact on health, with some finding a relationship between exposure and ADHD (Boucher et al., 2010; Torrente, Colomina, & Domingo, 2005) and others not finding a relationship between exposure and ADHD (Ha et al., 2009; Surkan et al., 2009). In fact, it was much more common for the authors to find studies were methylmercury negatively impacted domains like memory and visual-motor functions rather than the behavioral domain. Karagas et al. (2012) point out that a significant limitation of these studies is that they do not take fish consumption into account, which is the primary method of exposure for methylmercury.

Other researchers discussed neurotoxicants in etiological studies of mental illness. Froehlich, Anixt, Loe, Chirdktiatgumchai, Kuan, and Gilman (2011) reviewed recent literature on the etiology of AD-HD. Environmental risk factors were mentioned as contributing factors to the formation of AD-HD in some cases (e.g., through exposure to high concentrations of lead, manganese, and mercury). Every study that evaluated the relationships between lead and AD-HD showed there was an increased risk of the disorder as exposure to lead increased (Cho et al., 2010; Nicolescu et al., 2010; Nigg, Nikolas, Mark Knottnerus, Cavanagh, Friderici, 2009). Likewise, exposure to manganese increased the odds of developing AD-HD (Farias et al., 2010). In fact, the only element
that was not found to have a relationship to AD-HD was mercury, even controlling for confounders like lead exposure (Nicolescu et al., 2010). Froehlich et al. (2010) said that each of these studies was limited in some way, but findings were typical of previous literature reviews.

Landrigan (2010) discussed the etiology of autism in a study that emphasized the environmental contributions to the disorder. Autism is known to have a strong familial genetic component, but genetic factors alone contribute less than 10 percent to the cases. The author pointed out that the developing brain was incredibly susceptible to environmental insults, both in utero and once the child was born, and he suggested that epigenetics played a large role in the development of the disorder. The most prominent chemicals found to have relationships with autism included lead, methylmercury, polychlorinated biphenyls, arsenic, manganese, organophosphate insecticides, DDT, and ethyl alcohol. Landrigan (2010) explained these chemicals represented a small portion of the chemicals with known toxicology; over 200 chemicals were recognized neurotoxicants, but an additional 1,000 have proven to be neurotoxic in animal models, some of which he speculated may contribute to the onset of autism. Landrigan (2010) concluded by calling for a three-fold research agenda that would better help to understand the development of autism, and he was hopeful that the ongoing National Children’s Health Study would better explain its etiology.

Control Variables

Given the complex etiology of child and adolescent mental illness, it is necessary to control for potentially confounding and intervening variables when examining the spatial relationship between mental health outcomes and exposure to neurotoxicants.
Special care also must be taken because those risk factors that influence the development of mental illness may not be the same as those risk factors that influence child admissions. To make the matter more complicated, it is important to realize that researchers have difficulty disentangling the effects of one risk factor from the other because they may either be highly related (i.e., exhibiting multicollinearity) or they may statistically interact with one another, creating a complex relationship (Raine, 2005). This study included control variables that are risk factors associated with the development of mental illness as well as risk factors associated with child admissions.

a. Risk factors associated with the development of mental illness

According to the Surgeon General’s report on mental health, risk factors for youth mental illness include both biological and psychological stressors (U.S. Department of Health and Human Services; USDHHS, 1999). Biological factors related to mental illness may include genetic predisposition, gender and race. Early onset schizophrenia is an example of one disorder that has a very high likelihood of genetic transmission. The environment can also influence biological risk factors. Children exposed in utero to alcohol, drugs (licit and/or illicit), and tobacco are at increased risk of mental illness. Abused children and children who suffer traumatic brain injury are also more likely to develop mental illness. Psychological risk factors that may influence a child’s mental illness are just as diverse. Children who experience a negative home environment through poor child-parent relationships (e.g., inconsistent discipline, attachment issues), parental discord, and sibling rivalry are at increased risk for mental illness. Parental history of psychopathology and criminality also influence the risk of children’s mental illness.

Environmental factors like crime, large family size, overcrowding and economic hardship
may also contribute to the development of mental illness. No matter whether the stressor is biological or psychological, these risk factors may work together or separately to produce the mental illness (USDHHS, 1999).

Several journal articles considered individual risk factors for mental illness. Fryers, Melzer, and Jenkins (2002) conducted a systematic review of research comparing social inequalities and mental disorders in developed countries. Specifically, the authors examined large population-based studies (> 3,000 people) that focused on participants with broad social class variation in relation to well-defined and validated measures of social inequality. Social inequality was defined by factors that included education, employment, income/material circumstances, and social status. Despite covering over twenty years of academic research, the authors only found nine empirical studies that met their criteria for inclusion into the systematic review. These studies were limited on many accounts, and the dissimilar operational definitions of social inequality, different methodologies, and diverse instruments precluded meta-analysis of the findings. Nonetheless, there were some important results that informed the relationship between social inequality and mental illness. As Fryers, Melzer, and Jenkins (2002) explain, “eight of these nine well-conducted studies…showed consistent links between the common mental disorders and one or other marker of social disadvantage, with none showing inverse trends” (p. 236). Social status was the only factor that did not show a consistent association; whereas, lower income (6/6 studies), unemployment (6/7 studies), and education level (4/5 studies) were all reported to significantly increase the odds ratios of mental disorders.
Muntaner, Eaton, Miech, and O’Campo (2004) illustrated the complex relationships among poverty-related variables, demographic factors, and mental illness in their literature review on the relationship between socioeconomic position and major mental disorders. Socioeconomic position (SEP) was operationally defined by examining factors related to social class and social status (i.e., deprivation, disadvantage, income inequality, poverty). After reviewing previous literature reviews, meta-analyses, and recent research published between 1998 and 2002, the authors used a life-course approach to explore a multitude of factors related to SEP like gender and mental health diagnosis. Results indicated that a majority of studies found an inverse relationship between SEP and disorders like schizophrenia and depression. While the magnitude of this association differed among studies, the empirical research usually concluded there was a higher risk of mental illness with lower levels of SEP. While research on schizophrenia does not address whether schizophrenia causes SEP of vice-versa (i.e., the selection-causation issue; Dohrenwend et al., 1992), cross-sectional and longitudinal research on depression illustrated a causal pathway from SEP to depression. That is, people with a low SEP were at greater risk for becoming clinically depressed based on their economic conditions. Other findings dealt with the risk factors associated with women and minorities. For example, the authors indicated that women of low SEP were particularly vulnerable to depression. Muntaner et al. (2004) explained, “compared with men, women are at higher risk of depression and depression persistence, in part because of their lower socioeconomic standing and higher exposure to stressors” (p. 58). Research also suggested minorities are vulnerable to mental illness. Ruling out fetal stress and immigration, the authors also concluded that there was a relationship between ethnicity,
schizophrenia, and SEP; these studies found an interaction between genetic vulnerability and ethnic groups of low SEP.

Mental illness has also been found to be related to crime and community violence. Lynch (2003) demonstrated the relationship between community violence and mental health in a literature review on research studies published between 1987 and 2002. Higher levels of community violence were predictive of a wide variety of ill effects on child mental health. Children exposed to community violence were found to have an increased likelihood of developing posttraumatic stress disorder (PTSD). It did not even matter whether the children were indirectly exposed (through hearing about the event) or directly exposed (through being a victim); all forms of exposure to neighborhood violence consistently led to PTSD symptoms. Community violence also has been shown to contribute to externalizing and internalizing problems in youth. Externalizing problems (e.g., antisocial behavior and aggression) have been documented in youth exposed to community violence even when prior levels of youth violence are controlled. Internalizing problems (e.g., anxiety and depression) also have increased with exposure to community violence in most studies; however, the relationship appeared to be context dependent. Younger children were most susceptible to the effects of community violence, not early-adolescents and older children. Another context-specific factor occurred with the way youth experienced violence. The relationship between depression and directly experiencing violence in the community was high, and the relationship between depression and witnessing community violence was much lower. Community violence even significantly impacted peer relations. Youth exposed to violence were much more
likely to report problematic peer relationships, interpersonal problems, and engagement in bullying than similar peers exposed to fewer incidents of community violence.

b. Risk factors associated with child admissions

Child admissions to mental health facilities have many correlates. Demographic variables like age, gender, and race have all been found to be predictors of child admissions. Farmer, Stangl, Burns, Costello, and Angold (1999) found that males were more likely to be admitted than females. Merikangas et al. (2010) and Merikangas et al. (2011) tested a more complex model with a nationally representative sample and found that males were more likely to be admitted if they had substance abuse or ADHD-related disorders, while females were more likely to be admitted if they mood or anxiety disorders. Younger children are more likely to be admitted than older children, although no study used the same age groupings. Farmer et al. (1999) studied children who were 9, 11, or 13 years of age at baseline and found that younger children were more likely to be admitted. Zahner and Daskalakis (1997) considered a younger population and found that 9-11 year olds had 1.56 the odds of being admitted compared to 6-8 year old children. Cohen and Hesselbart (1993) divided their studies of children into two groups and found that children less than 17 years of age were more likely to be admitted. Children who were minority race have also been found to be less likely to be admitted in one study. Zahner and Daskalakis (1997) found that Blacks were less likely to be admitted to mental health services than Whites. Merikangas et al. (2011) also found several statistical interactions between specific disorders and demographic variables: rural Whites were more likely to be admitted for mood disorders than Blacks; White males were more likely to be admitted for anxiety disorders than Black males; Hispanic youth were more likely
to be admitted as their number of diagnoses increased compared to youth of other ethnicities; and children of divorced parents were more likely to be admitted for substance abuse than children of married parents.

Parental education level was likely to contribute to admissions among children. Findings indicated child admission rates were higher for children with substance abuse disorders if their parents did not complete high school or college (Merikangas et al. 2010; 2011). Likewise, John, Offord, Boyle, and Racine (1995) found higher admission rates for children with parents who had less than an eighth grade education; children had 3.87 times the odds of using services compared to children with parents who had greater than a high school education. Not all studies agreed with these findings. Farmer et al. (1999) analyzed data from the Great Smoky Mountains Study and found that admissions to specialty mental health services (as opposed to school-based mental health services or both services combined) were greater among children with parents who had at least a Bachelor’s education.

Mixed results have been found with respect to the relationship between child admissions and economic status. Farmer et al. (1999) found that children living below poverty and children with Medicaid were more likely to be admitted to a mental health facility. This study’s findings do not agree with Cohen and Hesselbart (1993), who divided their sample into two groups and found that children in families making more than $50,000 had higher admission rates. Zahner and Daskalakis (1997) did not look at wages per say, but they examined social class and did not find any differences between admission rates for youth of different social classes.
Other variables that predict child admissions have been inconsistently studied in the literature. Researchers have found chronic mental illness, geographic type (urban vs. rural), parental history of psychopathology, and poor school performance predict child admissions to mental health treatment (Cohen & Hesselbart, 1993; Farmer et al., 1999; John et al., 1995). Sourander et al. (2004) conducted a prospective survey that followed boys from age 8 to age 18. The authors found that the factors related to future use of mental health services included, “teacher evaluation of need of referral or assessment, low school performance, and high level of symptoms in teacher evaluations predicted service use 10 years later” (Sourander et al., 2004, p. 1254). One factor especially, symptoms in teacher evaluations at age 8, predicted 40 percent of child admissions at age 18.

Research Justification

Despite the increased interest in the relationship between pollution and health, researchers have neither investigated the relationship between exposure and admissions for children and adolescents nor have they investigated the relationship between exposure to neurotoxicants and mental health assessment scores. As previously stated, empirical research is very limited in its scope of inquiry; researchers have only examined the relationship between isolated neurological toxicants and mental health after man-made disasters (e.g., Rehner, Kolbo, Trump, Smith & Reid, 2000), in select communities of interest (e.g., Bullard, 2000), and in etiological studies of mental health diagnoses (Froehlich et al., 2011; Landrigan, 2010). This knowledge gap is especially surprising because Ohio has been consistently among the top-five emitters of airborne
neurotoxicants in the United States (U.S. EPA, 2009a). Exposed youth in Ohio may suffer acute or chronic effects from this exposure, but no one really knows.

The study makes a unique contribution to the scholarly literature on several accounts. This research adds basic knowledge to the field about the spatial relationships between neurotoxicant exposure and mental health admissions. Currently, there is a scholarly debate about the relationship between admissions for specific diagnoses (e.g., autism) and magnitude of pollution emissions (e.g., Windham, Zhang, Gunier, Croen, & Grether, 2006). It is possible that information from this research could inform that debate through helping policy makers, academicians, and lay people to understand whether to be concerned about current levels of neurotoxicant exposure. Findings from this research also contribute knowledge about the spatial relationship between neurotoxicant exposure and mental health outcomes. While there is knowledge about specific neurological toxicants (e.g., lead), there is not any scholarly information about the general impact of low levels of known or suspected toxicants in the every-day environment. Knowledge about this association may enlighten Ohio’s citizens, informing them about actions they should take in regards to the pervasive environmental neurotoxicants.

*Research Objectives*

The specific aims of this research study address two important areas of investigation. Based on the literature, child admissions may be influenced by neurotoxicant exposure. While it is possible that admissions for many diagnostic categories are related to exposure, only the diagnostic categories implicated in the literature are examined for this analysis with one exception. It is possible that neurotoxicant exposure represents such a large threat to children’s health and wellbeing.
that child admissions for all diagnostic categories would be impacted; therefore, an objective specific to this line of inquiry is tested. Child assessment scores at intake are also explored in this study to determine whether neurotoxicant exposure has an impact at intake. This line of inquiry is also important because it may demonstrate that exposure and the onset of mental illness are related. In particular, domains important to mental health based on the literature are compared with exposure. Each of these research objectives are examined in a spatial context because there is considerable variability in child admissions, assessment scores, and exposure throughout Ohio. Spatial models test whether the intensity of variables are related to one another throughout the state (e.g., intensity of child admissions vs. intensity of exposure). The following is a list of specific research objectives:

1. The spatial relationship between neurotoxicant exposure and child admissions for mental illness
   a. At patient intake, determine if there is a spatial relationship between categories of neurotoxicant exposures (i.e., solvents & metals) and child admissions for all diagnostic categories
   b. At patient intake, determine if there is a spatial relationship between categories of neurotoxicant exposures (i.e., solvents & metals) and admissions for specific disorders known or suspected to be implicated in disease etiology, including:
      i. Attention deficit-hyperactivity disorder
      ii. Pervasive developmental disorder
   c. At patient intake, determine if there is there a spatial relationship between specific neurotoxicant exposures (i.e., lead, mercury and manganese) and...
admissions for specific disorders known or suspected to be implicated in disease etiology, including:

i. Attention deficit-hyperactivity disorder

ii. Pervasive developmental disorder

2. The spatial relationship between neurotoxicant exposure and mental health assessment scores

   a. At patient intake, determine if there is a spatial relationship between neurotoxicant exposure and the mental health outcome for hopefulness

   b. At patient intake, determine if there is a spatial relationship between neurotoxicant exposure and the mental health outcome for functioning

   c. At patient intake, determine if there is a spatial relationship between neurotoxicant exposure and the mental health outcome for problem severity
Chapter 3: Methodology

Chapter 3 reviews the methodology and procedures for this research study. This chapter begins with a discussion of the participants, sampling frame, sampling technique, and unit of analysis. Following this discussion, is an introduction to the specific procedures for data collection. Next, there is an in-depth look at the independent, dependent, and control variables, along with operational definitions for each of the variables used in the analyses. Data preparation is also examined, and this section provides specific guidelines on selecting data, cleaning data, geocoding, and missing data imputation. Finally, there is a review of each of the data analysis techniques.

Participants

Admission rates and average outcomes scores were aggregated to the census tract level for children and adolescents from the ages of 5 to 17. All of these children obtained treatment for mental illness from Ohio behavioral health services organizations during state fiscal years 2007 and 2008. Data from participants were considered for inclusion in this study as long as they had a primary or secondary diagnosis of mental illness found in the Diagnostic and Statistical Manual of Mental Disorders (DSM; APA, 2000), which indicated they were experiencing clinically significant distress on a frequent basis. Some diagnoses were excluded from the present analysis because the illnesses were not likely due to neurotoxicant exposure, but rather to factors like illicit substance use or child abuse. Children and adolescents were excluded if they had a primary or secondary
diagnosis of organic psychotic conditions (290.xx – 294.xx); substance abuse, dependence, or intoxication (303.xx – 305.xx); psychological factors associated with a general medical condition (316.xx); intellectual disabilities excluding pervasive developmental disorders (317.xx – 319.xx), any form of injury or poisoning (800.xx – 999.xx); symptoms due to bereavement or absence of a family member (V61.07, V61.08, V62.82); and abuse (v61.12, v61.13, v61.22, v61.13).

Sampling Frame and Sample Size

The number of children and adolescents who receive publicly funded mental health services varies year-to-year based on the number of children and adolescents requesting services. ODMH data revealed that approximately 100,092 youth were served in state fiscal year 2007 (ODMH, 2007) and 103,451 youth were served in state fiscal year 2008 (ODMH, 2008). It is important to note that these numbers overestimate the population of children and adolescents with Ohio Scales data because not all agencies submitted these data for the youth served by the behavioral health organizations. Data submission rates by provider organizations indicate that well over two-thirds of agencies have been submitting data since state fiscal year 2005. ODMH sent their dataset in calendar year (not the fiscal year format), so the sampling frame of children and adolescents includes 92,349 admissions records from 55,519 unique youth from calendar year 2007 and 68,302 admissions records from 45,453 unique youth from calendar year 2008.

Sampling Technique

Until state fiscal year 2010, the Ohio Department of Mental Health mandated all behavioral health organizations in Ohio that submit claims for Medicaid reimbursement
to offer Ohio Scales forms to their clientele. Children and adolescents, their parents, and case managers filled out the Ohio Scales at the intake appointment and every 90-days thereafter for the first year of treatment. Forms were filled out once a year after the first year of treatment. Consumers were not required to fill out these forms in whole or in part; therefore, responses to questionnaires included missing data. To complicate matters further, some behavioral health organizations required to offer these questionnaires did not do so consistently, meaning that agencies may have submitted between zero and 100 percent of Ohio Scales data. Addresses for consumers without any data were imputed and aggregated to the census tract level. The imputation process helped lessen some concerns with missing data, which could have potentially created false spatial patterns due to the non-random nature of the missing data.

Unit of Analysis

The unit of analysis for this study was the census tract, and all consumer-level data were aggregated into Ohio’s 2,934 census tracts as captured by the Census 2000 shapefile (U.S. Census Bureau, 2001). Census tracts are small geographic entities that generally follow the boundaries of states and counties. Typically, census tracts are units of between 2,500 and 8,000 people that have very similar characteristics. The U.S. Census Bureau (1994) explained, “when first established, census tracts were to be as homogeneous as possible with respect to population characteristics, economic status, and living conditions” (p. 10-1). Census tracts were chosen for this study instead of some other geographic entity for several reasons. Underlying the basic research question is the notion that exposure varies by factors like proximity to industry, lifestyle, and daily activity patterns. With that in mind, a reasonable geographic entity had to be chosen in
which children would spend most of their time. Census blocks and block groups were deemed too small to represent the area of daily activity, and counties were deemed too large of an area to investigate meaningful spatial relationships. Census tracts had the best chance of representing a larger area that has a chance of containing the child’s school along with most of the other areas the child is likely to go during the typical day. Census tracts were also chosen because they represent a typical unit of analysis in geographic research for which many governmental and non-governmental organizations create estimates of population parameters (Longley, Goodchild, Maguire & Rhind, 2005; U.S. Census Bureau, 2012). These geographical units are small enough to detect meaningful differences among phenomena of interest while maintaining some level of privacy for the participant (Dolinoy & Miranda, 2004).

Procedures

Data Collection

ODMH databases were obtained through a special request to the ODMH Office of Research and Evaluation [ORE]. In 2010, ODMH revised its data release policies to make internal and external requests go through their legal and executive departments. ODMH requested that the dissertation committee and the OSU IRB approve this research project before the data request would be reviewed by their agency. The IRB approval process was unique because the requested electronic medical records represented fully identified data as defined in HIPPA (UC Davis Health System, 2010); therefore, it required a HIPPA waiver. Once these steps had been met and their legal and executive teams reviewed the data request, ODMH staff provided the cross-sectional data on September 29, 2010. Unique client identifiers were transformed into random numbers by
ODMH staff before they sent the database. All consumer data were stored on a password protected computer in encrypted files to which only the researcher had access.

*Questionnaires*

At patient intake, it was possible for there to be three assessment-related questionnaires; therefore, the ODMH data request included all recorded variables from the Ohio Scales Youth questionnaire, the Ohio Scales Parent questionnaire, and the Ohio Scales Worker questionnaire. In this study, all children and adolescents, parents, and case managers submitting Ohio Scales were included in the analyses. The content in these questionnaires was very similar; each of the three questionnaires had the same items on problem severity and functioning. Only the parent and child versions of the questionnaire had the four items on hopefulness. When multiple data points were present for a child (e.g., youth and parent records) scores were averaged to get the best overall picture of the youth’s clinical condition.

*Independent Variables*

a. Quantifying exposure

The dissertation proposal stated that AERMOD would be used with 2007 and 2008 EPA data for all dispersion modeling. AERMOD was chosen in particular because it is considered the state-of-the-art modeling platform designed by the American Meteorological Society/Environmental Protection Agency Regulatory Model Improvement Committee (U.S. EPA, 2008). Unfortunately, AERMOD turned out to be inappropriate for several reasons. Upon in-depth examination of the program, AERMOD could only calculate a surface for one chemical at a time from one or more facilities, which was a problem since 200 chemicals are known and suspected neurotoxicants.
AERMOD was also not really designed to estimate dispersion patterns over large geographic areas like states, so separate models would have to be made for much smaller geographic units (i.e., conservatively estimated to be five). That means that over 1,000 air dispersion models would need to be run, which made the time requirements impractical for this study.

A geostatistical interpolation technique called kriging was also considered to estimate the quantities of neurotoxicants around the state; however this technique was also inappropriate for this study. Kriging pollutants would be most accurate if a dense network of air monitors were placed around Ohio to determine the average concentration of Neurotoxicant A through Neurotoxicant Z; however, no such network exists. Researchers like Liao et al. (2006) have used air monitors for particulate matter pollution to estimate exposure and related health effects, but those compounds are not evaluated in this study. Data from the EPA’s Toxic Release Inventory provide estimated and actual releases from industries around Ohio (e.g., fugitive and point source air emissions), which means that the prediction surface would be an estimate of hypothetical air releases from hypothetical sources and not an estimate of total exposure of Person A at Point A. Ideally, kriging would calculate that type of additive model for the purposes of this study, but it actually interprets point data as the real value at that location, so it forces neighboring values to be close to it, which may or may not be accurate. As an example, think of two industries that release lead into the atmosphere. Industry A might release one million pounds of lead and Industry B located across the street might release 25,000 pounds of lead. Kriging algorithms would try to force local values somewhere in between
one million pounds and 25,000 pounds even though total exposure would be an additive model and not a model based on averages.

b. National-scale Air Toxics Assessment

Fortunately, there was an acceptable solution to the problem of quantifying exposure. The National-scale Air Toxics Assessment (NATA) provides cumulative risk estimates for chronic exposure to airborne toxicants (U.S. EPA, 2009b). NATA originated from the Cumulative Exposure Project, and has created estimates every three years since 1996. IFC International (2011) explains the goals of NATA are to, “identify those air toxics of greatest potential concern with regard to their contribution to population risk” and “guide efforts to reduce toxic air pollution and to provide information that can be used to further the already significant emissions reductions achieved in the United States since 1990” (p. 4). The most recent dataset included risk estimates from 2005, and they became available in early 2011. Datasets are produced at various geographic levels for the United States, with risk estimates being more accurate as the population increases per geographic unit (e.g., county risk estimates are more reliable and accurate than census tract risk estimates).

The conceptual model of NATA displays the numerous factors taken into account when establishing a risk estimate (Appendix B). First, NATA identifies all outdoor emission sources. IFC International (2011) explains these sources may be stationary point sources (e.g., emissions from factories), non-point sources (e.g., fugitive emissions from dry cleaners and gasoline stations), on- and off-road mobile sources (e.g., cars and trains) and extrinsic background sources (e.g., natural sources that persist in the environment). Chemicals that are identified as hazardous air pollutants by the Clean Air Act along with
diesel particulate matter are compiled from various national databases like the Toxic Release Inventory and the National Emissions Inventory as well as state and local databases. Emissions data gathered at the local and state levels are considered more accurate than the national level data, and these data are given preferential treatment when available during the modeling process. Currently, NATA only considers an air-based pathway when building exposure models. While this pathway limits the exposure route to inhalation, NATA thoroughly evaluates the complex interactions:

… including the locations and nature of emissions, the emission release conditions, local meteorology, locations of receptor populations, and the specific behaviors and physiology of individuals in those populations. … (which includes) account[ing] for an individual’s movements among microenvironments such as residences, offices, schools, exterior work sites, and automobiles … (IFC International, 2011, pg. 12-13)

Emissions data from all sources undergo dispersion modeling in the next phase of the NATA risk assessment process. The Human Exposure Model-3 is used in conjunction with AERMOD to determine the dispersion of chemicals within 50 miles of their release. Facility data (e.g., stack location, stack height, gas velocity) are combined with meteorological data to determine the fate of the chemical at various geographic levels of resolution. The Community Multiscale Model (CMAQ) is used during this process to model unique air toxicants resulting from secondary formation (e.g., formation of formaldehyde through chemical transformation of volatile organic compounds). As mentioned previously, very little data on secondary transformation are incorporated into
NATA because of the lack of research into secondary formation. None of the neurotoxicants considered on in this study are modeled through secondary formation.

Modeling inhalation exposures is another important step in determining the cumulative risk estimates. This procedure refines the risk estimate by examining mobility patterns among individuals within microenvironments (e.g., travelling from home to school or work). For 2005 data, NATA derives estimates by creating exposure ratios based on the 1999 Hazardous Air Pollutant Exposure Model version 5 (HAPEM5). While this is not ideal because of the potential for exposure estimates to differ greatly, the U.S. EPA has not received funding to conduct a proper exposure assessment since 1999. The HAPEM5 data, off which NATA’s current estimates are based, are derived from several sources. Ambient air concentration data are averaged over a one-year period at the census tract level for all sources (e.g., on- and off-road emissions). Population demographic data are also input into the model to determine the exposure concentration to 10 unique groups (e.g., 5-11 males vs. 5-11 females).³ Activity pattern data, which includes commuting pattern data, are also calculated to understand the “frequency and duration of exposure” which are used to, “place a hypothetical individual who commutes to work either in the home tract or the work tract at each 3-hour time step, and in a specific microenvironment” (IFC International, 2011, p. 42). Finally, microenvironmental data are created that take into account penetration of emissions into microenvironments, the proximity of the ambient air concentrations compared to the outdoor concentrations, and an additive factor that accounts for other emission sources (IFC International, 2011).

³ Cohort level data are not publically available.
NATA collects chemical toxicity information and characterizes the specific effects of air toxicants in the next phase of emissions modeling. Using data from many sources, including but not limited to the Integrated Risk Information System, Agency for Toxic Substances and Diseases Registry, and California’s Office of Environmental Health Hazard Assessment. NATA thoroughly investigates the literature to develop a dose-response assessment for each chemical. This assessment helps NATA assign toxicity values that indicate the level of adverse effects present at certain doses of the chemical. All dose-response relationships are modeled for chronic exposure over a long period of time for non-cancerous and cancerous substances. Non-cancerous chemicals are modeled as hazard quotients, which relate to the “ratio of the potential exposure to the substance and the level at which no adverse effects are expected” (IFC International, 2011, p. A3-A4). Hazard quotients less than one are not likely to be associated with adverse effects, and hazard quotients equal to or greater than one may be associated with adverse effects.

NATA estimates have several limitations. NATA risk estimates did not reflect every chemical emitted from every source in the environment. Many chemicals were simply not tracked in EPA databases, or were not released in the quantity necessary to report to the EPA. For instance, regulation 42 U.S.C. 11023 promulgated that industries must report data to the EPA’s Toxic Release Inventory only if they use more than 10,000 pounds of the toxic chemical per year or manufacture/process more than 25,000 pounds of the toxic chemical per year (U.S. EPA, 2009d). Other chemicals were tracked, but data on airborne emissions may have been estimated and not based on direct readings of chemical presence. Another limitation stemmed from the lack of information for certain
parts of the NATA model. NATA estimated dose response values for each chemical, but 39 of the chemicals did not have research that reliably states any dose-response relationship. Data on chemical reactivity both with atmospheric conditions and with other chemicals was also very limited and only incorporated for a handful of chemicals. NATA estimates were also limited because they did not account for all pathways of exposure. Data were not incorporated that would relate to dermal or ingestion pathways, nor did they take bioaccumulation in the food chain into account. The reader is referred to the report from IFC International (2011) for more nuanced limitations of the dataset.

c. Operationally defining neurotoxicants

U.S. EPA staff with NATA compiled a list of 23 neurological toxicants for which neurologic-specific risk estimates were available out of 179 hazardous air pollutants (12.85%) surveyed in 2005. While this list was not as comprehensive as the list of neurological toxicants from the Environmental Defense Fund (2005b), cancer risk estimates for NATA chemicals suspected to be neurotoxicants cannot be combined with neurologic-specific risk estimates because of the different methodologies in their calculation. This study used NATA’s hazard quotients, which were the sum of the hazard indexes for each chemical developed from point sources, non-point sources, on- and off-road sources as well as extrinsic background sources. Neurotoxic chemicals on the list were subdivided into the two basic categories (Table 5), and some research questions used class-specific hazard indices. Upon request, staff at the National Emissions Inventory provided a helpful list of solvents to help determine the proper classification of chemicals.
### Table 5. NATA Neurotoxicants and their classification

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,1,1-Trichloroethane</td>
<td>Solvent</td>
</tr>
<tr>
<td>2,4-Dinitrotoluene</td>
<td>Solvent</td>
</tr>
<tr>
<td>Acrylamide</td>
<td>Solvent</td>
</tr>
<tr>
<td>Allyl chloride</td>
<td>Solvent</td>
</tr>
<tr>
<td>Benzidine</td>
<td>Solvent</td>
</tr>
<tr>
<td>Carbon disulfide</td>
<td>Solvent</td>
</tr>
<tr>
<td>Cresol cresylic acid (mixed isomers)</td>
<td>Solvent</td>
</tr>
<tr>
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<td>Solvent</td>
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<tr>
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<tr>
<td>Tetrachloroethylene (perchloroethylene)</td>
<td>Solvent</td>
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<tr>
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<td>Solvent</td>
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### Dependent Variables

a. Measures of mental health diagnosis

The Diagnostic and Statistical Manual of Mental Disorders, 4th edition-text revision identifies psychiatric disorders common to the United States (APA, 2000). This manual was used to develop general categories for psychiatric disorders among youth and the general population including: attention deficit and disruptive behavior disorders; adjustment disorders; pervasive developmental disorders; disorders diagnosed in infancy,
childhood, and adolescence; mood disorders; anxiety disorders; schizophrenia and other psychotic disorders; somatoform disorders; factitious disorders; dissociative disorders; eating disorders; and sleep disorders.

b. Measures of mental health outcomes at assessment

Ohio Youth Problem and Functioning Scales (Ohio Scales). The Ohio Scales (Appendix C) were multidimensional measures designed to evaluate the outcomes of youth (ages 5-18) who received treatment for serious emotional disorders (Ogles, Melendez, Davis & Lunnen, 2000; 2001). The Ohio Scales short form (48-items) had three parallel instruments that can be completed by the youth (ages 12-18), the youth’s parent or primary care-giver, and the youth’s direct-care provider. The Ohio Scales were constructed to measure four primary domains associated with mental health outcomes: 1) symptom distress and diagnosis, 2) functioning, 3) hopefulness, and 4) satisfaction with and inclusion in treatment services at behavioral health organizations. Operational definitions for each domain follow:

- **Symptom distress and diagnosis** (a.k.a., problem severity) – the summated scale of items 1 through 20 on the questionnaire based on a six-point summated rating scale given a numerical value ranging from *Not at all* (0) to *All of the time* (5).

- **Functioning** – the summated scale of items 29 through 48 on the questionnaire based on a six-point summated rating scale given a numerical value ranging from *Extreme troubles* (0) to *Doing very well* (5).

- **Consumer perspectives** – 1) hopefulness: the summated scale of items 21 through 24 on the questionnaire based on a six point summated rating scale given a numerical value ranging from *Very hopeful* (1) to *Not hopeful* (6). 2) service
satisfaction: the summated scale of items 25 through 28 on the questionnaire based on a six point summated rating scale given a numerical value ranging from Most satisfied (1) to Least satisfied (6) or Not at all (1) to A great deal (6).\(^4\)

- **Environment** – the final score on the Restrictiveness of Living Environments Scale (ROLES) on the worker form of the Ohio Scale. Agency workers indicate which environment the youth has lived in and use a statistical formula to evaluate the average restrictiveness of the youth’s living environment for the past 90 days (Hawkins, Almeida, Fabry, & Reitz, 1992).\(^5\)

- **System (service outcomes)** – the youth’s total service utilization cost of the prior 90-days.\(^6\)

The Ohio Scales have shown excellent psychometric properties (Ogles, Melendez, Davis & Lunnen, 2000; 2001). In clinical and community samples, the Ohio Scales short form has shown good internal consistency on the problem severity scale ($\alpha = .86$ to .93), the functioning scale ($\alpha = .91$ to .93), the hopefulness scale ($\alpha = .65$ to .84), and the service satisfaction scale ($\alpha = .72$ to .82). Test-retest reliability was adequate for the hopefulness ($\alpha = .74$ to .79) and the service satisfaction scales ($\alpha = .67$), but it has neither been estimated for the problem severity nor functioning scales. While researchers (Ogles, Melendez, Davis & Lunnen, 2000; 2001) did not examine the Ohio Scales short form for validity, they analyzed the Ohio Scales long form (72 items) for validity. That analysis is applicable to this discussion because of the high correlation between both form’s problem

\(^4\) The service satisfaction score will not be used, but was worth mentioning to fully understand the Outcomes tool.
\(^5\) The ROLES score will not be used, but was worth mentioning to fully understand the Outcomes tool.
\(^6\) The system variable will not be applicable because only new consumers will be examined at patient intake.
severity scales \((r = .80)\) and functioning scales \((r = .91)\). Results indicated the Ohio Scales long form had good convergent validity, as displayed through the weak to moderate correlations between the Ohio Scales problem severity scale and the Child and Adolescent Functioning Assessment Scale \((r = .59)\); the Ohio Scales functioning scale and the Vanderbilt Functioning Instrument \((r = .54)\); and the Ohio Scales functioning scale and the Children’s Global Assessment Scale \((r = .31)\).

Results from several independent research studies also indicated the Ohio Scales had good reliability and validity. In one study (TDMHMR, 2004), the Ohio Scales psychometric properties were compared to the other widely accepted instruments that measure problem severity and functioning, the Child Behavioral Checklist (CBCL) and Strengths and Difficulties Questionnaire (SDQ). The Ohio Scales displayed excellent internal consistency \((\alpha > .90)\). Testing for convergent validity also yielded promising results. Findings indicated the Ohio problem severity scale score correlated significantly with the CBCL problem scale \((r = .62 \text{ to } .64)\) and with the SDQ problems scale \((r = .37 \text{ to } .63)\). Results also showed significant correlations between the Ohio functioning scale score and the CBCL functioning scale \((r = -.52 \text{ to } -.54)\)\(^7\) and with the SDQ functioning scale \((r = -.28 \text{ to } -.47; \text{TDMHMR, 2004})\). Other researchers have expanded on these findings, demonstrating the Ohio Scales have convergent validity, showing it could discriminate between diagnostic categories (Turchik, Karpenko, & Ogles, 2007).

\(^7\) Normally, positive correlations are expected when testing for convergent validity, but many of the forms were scored incorrectly by the clinicians and left “as is” by the researchers.
Control Variables

The inclusion of control variables was an important part of this study. As previously mentioned, behavioral healthcare organizations were only required to submit Ohio Scales data when services are billed to Medicaid, which meant that most consumers in this study were served by Ohio’s Medicaid system. To qualify for Medicaid benefits in Ohio through Healthy Start and Healthy Families, the income eligibility guidelines specify that families must make 90 percent of the federal poverty level or children (up to age 19) must make 200 percent of the federal poverty level (ODJFS, 2010). The federal government also allows Medicaid benefits for children that are disabled (e.g., blind or deaf) and for children with a deceased or disabled parent (SSA, 2010; SSA, 2004). To help control for any spurious relationships caused by poverty or other known risk factors for mental health, contextual variables (e.g., average family size and child poverty), and demographic variables (e.g., gender and race) were controlled for in regression modeling (USDHHS, 1999).

a. Contextual variables

Control variables were incorporated from the American Community Survey (ACS). This survey provides a supplement to the decennial census for interim estimates of population characteristics (U.S. Census Bureau, 2009). Since 2005, approximately 250,000 surveys have been mailed out per month for a total of three million per year. The sampling frame comes from the same master address file developed by the U.S. Census Bureau for the decennial census. Housing units and group quarters facilities (e.g., nursing homes and prisons) are sampled through a multistage random sampling process. The
census bureau follows-up with non-respondents through computer-assisted telephone interviewing (approximately 85 thousand per month) and computer-assisted personal interviewing (approximately 40 thousand per month).

The ACS dataset used for this analysis was the 2009 five-year estimate dataset that covers the years 2005-2009. This dataset was preferred over the one-year or three-year estimate datasets because it provided the most reliable estimates that were based on the largest sample size. While this dataset does not precisely match the timeframe of the study, datasets based on five-year estimates are the only ones that contain census tract information (U.S. Census Bureau, 2013). Several of the numerous ACS population characteristics were considered. The most salient domains that related to child mental health outcomes were 1) average family size, 2) child poverty, 3) female-headed households, 4) high school education or greater, 5) less than high school education, 6) risk of lead paint exposure, 7) median income, 8) minority race, and 9) unemployment rate. Operational definitions for each domain follow:

- **Average family size** - the average size of family households, including adults and children, per census tract.
- **Child poverty** - the sum of number of children 5 years, 6-11 years, 12-14 years, 15 years, and 16-17 years living below the poverty level divided by the total child population, 5-17 years per census tract.
- **Female-headed households** - the number of female householders with no husband present in a family household per census tract divided by the total number of households in the tract.

- **High school education or greater** - the sum of the number of adults 18-24 years who completed high school or equivalent, have some college or have a bachelor's degree plus the sum of the number of adults 25 and over who have a high school education or higher divided by the sum of the population 18-24 years and the population 25 and over per census tract.

- **Less than high school education** - the sum of the number of adults 18-24 years and the population 25 who have not completed high school or equivalent divided by the sum of the total population 18-24 years and the total population 25 and over per census tract.

- **Risk of lead paint exposure** - the sum of house built before 1939 or earlier, from 1940 to 1949, from 1950 to 1959, from 1960 to 1969, and from 1970 to 1979 divided by the total number of houses built on or after 1980 per census tract.

- **Median income** - the 2009 inflation adjusted median earnings (past 12 months) for adults 25 years and over per census tract.

- **Minority race** - the summed total of all minority races divided by the total population per census tract.

- **Unemployment** - the percent of unemployed adults 16 years and over per census tract.
The inclusion of ACS data was important from the standpoint of understanding the neighborhood influences on mental health outcomes at assessment; however, it also posed a problem. Census tracts were created to have relatively homogeneous group characteristics, so many of the variables were expected to be significantly correlated with one another. Principal components analysis was performed to collapse the data into one or more factors to develop an index of poverty. This statistic produced an estimate of total variance among the variables in one component. Variables were considered for inclusion to the model if they 1) loaded above 0.25, and 2) had component loadings within a relatively narrow range. Factors were only retained if components loaded on one factor. Factor score coefficients were saved with the regression method, which were standardized to have a, “mean of 0 and a variance equal to the squared multiple correlation between the estimated factor scores and the true factor values” (SPSS Inc., 2010). Components were developed from the census tracts retained for the analysis and not off of census tracts from the entire state.

Messer et al. (2006) proposed a standardized neighborhood deprivation index that combined eight census variables into one scale. Many of these variables were very similar to the variables related to the prediction of child mental health and child admissions (e.g., poverty and parental education level). Additional variables suggested by Messer et al. (2006) were added to the model to reveal whether these variables may also have predicted child mental health. Operational definitions these additional domains follow:
Crowding - the sum of owner and renter occupied units with greater than one person per room divided by the total number of owner and renter occupied units per census tract.

Households on public assistance - the number of household on public assistance divided by the total number of households per census tract.

Males in management occupations – the number of males in management, professional, and related occupations for the civilian population age 16 and over divided by the total number of all people in other occupations per census tract.

Females in management occupations – the number of females in management, professional, and related occupations for the civilian population age 16 and over divided by the total number of all people in other occupations per census tract.

b. Demographic variables

A variety of variables were requested as part of the proposal, some of which were maintained by ODMH while others were not. Variables like child’s age, gender, and race were submitted by most clinical staff. The foster care variable was also maintained by ODMH, but these children were often linked with addresses of child welfare agencies, so this variable was not used for the analysis. Unfortunately, other variables requested like family size, family income, family employment, and service utilization cost were unavailable because they were not tracked for children at the state level. Operational definitions for each domain follow:

- **Age > 11** - the number of admissions for children aged 12 and older divided by the number of total admissions per census tract.
• **Age < 12** - the number of admissions for children aged 11 and younger divided by the number of total admissions per census tract.

• **Male gender** - the number of male admissions divided by the number of total admissions per census tract.

• **Female gender** - the number of female admissions divided by the number of total admissions per census tract.

• **White race** - the number of non-Hispanic White admissions divided by the number of total admissions per census tract.

• **Minority race** - the number of Black and other racial minority admissions divided by the number of total admissions per census tract.

*Data Preparation*

a. Selecting data files

ODMH provided the Ohio Scales data on September 29, 2010. Unfortunately, ODMH did not have the time to pull out the highly specific cases required for this study, so they provided a larger than expected database. The electronic medical records consisted of 642,425 records in three databases; 234,877 records were from parent forms; 141,998 records were from youth forms; and 265,550 records were from case manager forms. These records included intake and follow-up records for 127,384 unique children and youth over many years.

Culling out the necessary data required many different steps, some of which were performed alone and others of which required outside help. Many cases would have to be excluded because there were many additional treatment records that were follow-up records. A list of rules was developed in order to select the specific set of records
required. A specialist created a query in Structured Query Language (SQL) on a database with dummy values to make sure the code ran appropriately (Appendix D). The SQL code that parsed the records had to take into consideration many important steps. The variable “Code2” was the primary record identifier, and explained which case records were duplicated (i.e., had multiple dates of service). The variables that specified date of admission, “Admissdt,” and age at assessment, “Ageatassessment,” were also crucial because they helped SQL identify which records to keep and delete. The syntax broke down into five phases:

Phase 1:
SQL pulled the records out that had only one “Code2” and stored them in a separate table. The program then deleted them from the starting table and focused on the remaining records.

Phase 2:
SQL identified records that had the same “Code2” value, but had different ADMISSDT values and stored them in a separate table. The program then deleted them from the starting table and focused on the remaining records.

Phase 3:
SQL identified all items that had duplicates for the variable “Code2,” “Admissdt,” “Hope,” “Func,” “Satis,” “Pserver” and selected their minimum age at assessment with the variable “Ageatassessment.” The program then stored values in a separate table. Finally, the program deleted records from the starting table and focused on the remaining records.
Phase 4:

SQL aggregated the records that had the same “Code2” and “Admissdt” to remove any duplicated information - if it existed.

Phase 5:

SQL joined all of the phase tables together for final output.

After running the SQL code, the Ohio Scales data consisted of 385,128 records representing 127,203 unique children in three databases; 144,380 records were from parent forms; 88,296 records were from youth forms; and 152,452 records were from case manager forms. More records still had to be deleted because they fell outside the range of the study. The Ohio Scales dataset contained 257,809 records after deleting records for years that fell outside of the study (116,314) and records for children that fell outside the age range of the study (11,005).

Other records were excluded for various reasons. Children were taken out who had a primary or secondary diagnosis of substance abuse or dependence because it was unclear how neurotoxicants would affect their mental illness. Likewise, children whose mental illness could be the result of a general medical condition were excluded. Altogether, 14,610 admissions records were excluded for children with any of the following DSM diagnoses: 290.xx – 294.xx; 303.xx – 305.xx; 316.xx – 319.xx; 800.xx – 999.xx; V61.07; V61.08; V61.21; and V62.82. The resulting database contained 243,199 records.

b. Ohio Scales data cleaning

A preliminary examination of the files indicated there was occasional data entry error. Case managers sometimes designated that a client was male and other times
designated the client was female. The same issues occurred for race; sometimes a client was designated as White and other times as a racial minority. This problem was especially pronounced within the minority population where they were more likely to be in multiple racial categories. To resolve this problem, client gender and racial data were aggregated into one database. Client designations were rounded to reflect the most often scored category, and a client that ended up in the middle was randomly placed in a category. For example, a client who was listed as gender 1.5 (i.e., once designated as a male and once as a female), would be randomly assigned to either male or female.

c. Address data cleaning

ODMH provided an address database with 127,203 unique address (i.e., one for each client), and these addresses were corrected independently of the Ohio Scales dataset just in case they would ever be needed in the future. These addresses had neither been standardized nor geocoded, so significant cleaning was needed to prepare the addresses for geocoding. ARCMAP 10.0 has a tool that automatically standardizes addresses; however, this tool has a few well-known quirks, so every address had to be evaluated individually. The following steps were taken to clean the addresses before and after the ARCMAP tool had been used:

- Deleted all apartment information (e.g., #3; Apt. A; Up; UPPR; Lower; LWR; DN; Down; Front; FRT; FNT; FT; Rear; RR). These designation could be found anywhere in the string variable and were causing errors during address standardization.
• Removed all inappropriate ½ designations because the address program thought that the 2 in ½ was the primary number in the address and not the earlier 1/2/3/4/5 numbers of the address.

• Changed designations like CR, C R, C.R., and C. R. to “County Road.”

• Changed designations like SR, S R, S.R., S. R., ST RT, ST RTE, STE. RT., ST. RT. to “STATE RTE” or “OHIO.”

• Changed designation WY to “WAY.”

• Added spaces between numbers and letters where the program might become confused.

• Corrected spellings for street names and cities.

• Corrected zip codes when possible.

After standardizing the address, some of them had to be deleted for various reasons. For example, several text fields that came with the address database indicated something special about the child. The largest group of children was removed because their address was a PO Box, and there was no guarantee that they lived in the same census tract as the post office. Another group of children were in foster care, and their address corresponded to the County Department of Job and Family Services, Child Support Enforcement Agency, or Public Service Children’s Agency. To make sure all of the children were identified who did not have this attribute listed in a text field, any address that corresponded to organizations listed in the Ohio Department of Job and Family Services agency directory were taken out (ODJFS, 2011). All children labeled “homeless” were not included in the analysis because it was unclear whether they
actually lived at the address. Other children were taken out because they had no address. Altogether, these steps resulted in the loss of 6,815 address records.

d. Geocoding

Several geocoding services were used to get the maximum number of addresses geocoded after the address data were cleaned. Most geocoding was accomplished with the former University of Southern California WebGIS portal (110,154 addresses; Goldberg, 2011), but ArcMap 10.0 (7,561 addresses) and Google’s geocoding service (577 addresses; Google, 2011) were also used to geocode addresses. In total, 118,292 addresses were able to be geocoded (92.99%) at the street address level. Only a minority of addresses (2,096) were unable to be geocoded at any level through these services.

e. Database combination

The Ohio Scales and address databases were combined once they were each scrubbed, yielding 124,972 records for calendar year 2007 and 97,810 records for calendar year 2008. These databases were aggregated, so that there was only one record per unique admission instead of between one and three records if the children, parents, and case managers filled out separate forms. This process generated 67,905 unique admissions records for calendar year 2007 and 51,560 unique admissions records for calendar year 2008.

Variation in admissions from year to year is expected, but the 24.07 percent drop from 2007 to 2008 was something altogether different. A change in the political landscape occurred after the 2006 governor’s election. Some constituents groups (e.g., mental health boards and agencies) encouraged the governor to disband the Ohio Scales data warehouse due to the perceived burden of filling out forms. While no changes to the
political landscape occurred in 2007, constituent groups received word that the governor was going to work with the ODMH director to significantly change the system in 2008; some agencies in fact stopped submitting Ohio Scales data to ODMH. The proposal mentioned analyzing 2007 and 2008 data either separately or in a pooled analysis, but an exploratory spatial analysis revealed there was non-random dropout in some communities. Only 2007 Ohio Scales data were analyzed for this study because of potential bias in the spatial analysis. That does not mean 2008 data cannot be used for future spatial analysis, just that one can only use data from communities that consistently submitted data from 2007 to 2008.

f. Missing data imputation

Missing data theory and techniques have evolved substantially since the late 80's when multiple imputation (MI) was first developed (Little & Rubin, 1987). Whereas, early techniques focused on ignoring missing data (i.e., complete case analysis through listwise deletion), analyzing the covariance matrix (i.e., pairwise deletion) or mean substitution, current techniques like MI focus on adding a random error term to make the imputed values more reasonable (Graham, Cumsille, & Shevock, 2012). Multiple imputations are also preferred over regression-based single imputation because the parameter estimates may be incorrect. UCLA SCG (2012) comments on this problem, A limitation of single imputation is that it treats imputed values as though they were observed, which is not the case; imputations are only estimates. As a result, analyses of a single imputation will tend to overstate our confidence in the parameter estimates; the standard errors are too small. Multiple imputation addresses this problem by introducing an additional
form of error based on variation in the parameter estimates across the
imputations, so-called between imputation error.

Missing data theorists emphasize that MI is not trying to figure out how Participant A
would have answered, but to restore lost variability to the dataset. Graham, Cumsille, and
Shevock (2012) explain, “we impute in order to preserve important characteristics of the
whole dataset. That is, we impute to get better estimates of population parameters (e.g.,
variances, covariances, means, regression coefficients, etc.) with as much statistical
power as possible.” Current MI techniques focus on creating parameter estimates that are
as close to the population parameter as possible (i.e., unbiased) while making the
variability around the estimate as small as possible (i.e., efficient).

Prior to imputation, researchers should consider the cause of missingness or the
mechanisms of missingness in the data before proceeding with imputation. The goal of
this evaluation is to make sure that researchers understand whether the missing values are
due to a random or non-random process. Graham (2012) discusses the types of missing
data; he explains, “missing data generally fall into three categories: Missing Completely
at Random (MCAR), Missing At Random (MAR), and Missing Not At Random
(MNAR)” (p. 9). The evaluation of mechanisms of missingness examines the
relationships among the variables of interest to determining which process exists. Should
missing data definitely be MNAR instead of MCAR, then multiple imputation becomes a
substantively different process. Missing data that are MCAR could be completely random
like the roll of dice, or essentially random if a variable that is not captured by the
researcher explains the missingness, but is uncorrelated with other variables in the study.
Data that are MAR means that missingness is a function of one or more variables, and/or
there is a linear or non-linear correlation among two or more of the variables that explain pattern of missingness. No matter the reason, researchers have to include the variables responsible for this pattern in the imputation model, lest they create estimation bias (Graham, 2012). Missing data that are MNAR means that one or more variables that are responsible for and correlated with the missingness have not been measured and are not included in the model. Graham (2012) makes a point that researchers can never fully understand whether missing is MAR or MNAR unless follow-up is conducted with non-respondents; however, he also suggests that it may not make a practical or statistical difference because missing data theorists have demonstrated MNAR may only have a negligible impact on results.

Based on extensive testing, SAS 9.3 is one of the most recommended programs for multiple imputation (Graham, 2012). Fortunately, there are plenty examples of SAS syntax for MI, and syntax used for this analysis was largely based on the framework of others (Graham, 2012; SAS, 2012; UCLA SCG, 2012). Multiple imputation can be a complicated procedure, but SAS 9.3 accomplishes the procedure in less than 10 steps. First researchers must decide what variables to include in the model. Typically, researchers include any variables that are present in the model, including dependent and independent variables. Additionally, researchers also include auxiliary variables when possible; these variables may not be part of the model, but may be highly correlated with variables containing missing information and thus give a better estimate of missing values during MI (Graham, 2012). For this analysis, subscales that were tested in unique regression models (e.g., associated with specific diagnoses) were be included in the imputation model because of potential problems with multicollinearity. Variables that
expressed little correlation (< 0.1) with variables missing data were also not included in the imputation model for fear of adding noise to the imputed data (i.e., over or underinflating coefficients).

After writing the syntax that brings in the file and formats the variables, the next step is to examine the patterns of missing variables. This process involves running a basic procedure, PROC MI sans subcommands, and examining the output. UCLA SCG (2012) explains that missing data patterns can either be monotone or arbitrary, where monotone patterns have an ordered missing pattern and arbitrary patterns look random. Each of these missing data patterns have unique algorithms for MI, so researchers need to make sure to use the most appropriate formulae. Results from this basic command also help the researcher understand how SAS is modeling the estimates. For example, the output would show that SAS runs the expectation maximization algorithm with the Markov chain Monte Carlo (MCMC) algorithm to obtain the initial parameter estimates by default. The “prior” command would show that SAS selects a “Jeffrey’s prior” by default, which is recommended unless the model has a difficult time converging and the researcher wants to build in a ridge regression technique, or the researcher uses a covariance matrix from other data to model parameter estimates (Graham, 2012). The next step is to evaluate some of these default commands to make sure the model is as elegant as possible. To this end, the researcher tests whether PROC MI works when an expectation maximization subcommand is added and there are zero imputations. This step would show how many iterations it takes before model convergence and determine whether the fit function changes monotonically over iterations. Monotonic change should indicate a gradual decrease in the -2 Log Likelihood fit function between iterations or something is wrong.
with the model. The evaluation of the default MCMC subcommand is the next step. Here, researchers should make sure SAS is able to impute one dataset without any iteration. Once the defaults are known to work, then it is time to begin imputation. Graham (2012) recommends starting with 200 burn-in iterations (i.e., results are thrown out) to allow parameter estimates to become random, and he recommends the convergence criterion be set at 1E-5, so that results from this program would be similar to other MI programs. The iterations command is set to the number of iterations it took the dataset to converge in test runs. The number of imputed datasets you create depends on the percentage of missing data. While earlier research imputed as few as five datasets, recent research suggests that as many as 100 imputations may be needed depending on the percentage of missing data per variable (Graham, Olchowski, & Gilreath, 2007). Finally, researchers examine the MCMC diagnostic plots to make sure that the solutions are acceptable. Through this examination, researchers can evaluate the “plot of the autocorrelations of parameters over iterations of MCMC,” “the plot of parameter estimates themselves over all iterations of MCMC,” and the worst linear function to determine whether the number of burn in iterations is correct (Graham, 2012, p. 170).

Prior to imputation, some of Graham's (2012) techniques were used to evaluate missingness. The overall percent of missing data were examined for each variable as well as the overall patterns of missingness among all the variables. Missing data patterns were also examined for key demographic variables like gender and race. The only form of missingness present in this study was item non-response. Some clinicians, parents, and children may have left one or more items blank for a variety of reasons. As mentioned previously, clinicians were required to offer the Ohio Scales to parents and children, but
these individuals may have refused to fill out the forms. Responses for every questionnaire may be missing in cases like this example. These cases may have all of the responses missing. Fortunately, this situation was highly unlikely and these groups were much more likely to have skipped a question they did not understand or refused to answer a question that they deemed too personal.

It is important to note that imputation for this study did not fully satisfy all of the conditions of proper multiple imputation. Multiple imputation creates multiple datasets, so it is difficult to analyze results in some cases. In SAS, there is a procedure to pool the regression statistics for any number of datasets; however, this procedure is not compatible with spatial regression. Conversations with several people familiar with the R software package suggested pooled spatial regression estimates can be developed with R syntax, but there are still limitations. To the best of my knowledge, no equations exist to pool unique spatial statistics like Moran's I, tests for heteroskedasticity, tests for multicollinearity, and geographic weighted regression.

A single imputation built off of a multiple imputation (MI) algorithm was used to handle missing data before analyses. The same steps mentioned above were followed, but only the first imputed dataset was used for the analyses. Data were imputed with the MCMC algorithm (single chain) that uses a Jeffrey’s prior. Initial estimates for the MCMC were derived by an expectation maximization algorithm in posterior mode, and the convergence criterion was set at 1E-5. The number of burn-in iterations was increased from 200 to 300 because only the first imputed dataset was used and this may make the database more reliable. Selected results from the imputed dataset were compared to
results from an un-imputed dataset because the ideal procedures for multiple imputations were not being followed.

*Data Analysis*

Research objectives 1a through 2c were analyzed with each of the techniques presented in the following sections, which are briefly explained here. The spatial context was of critical importance to these questions because the research objectives wanted to determine whether the variables displayed complex relationships that were part of a spatial process. This concept was best captured by Tobler’s (1970) first law of geography, “everything is related to everything else, but near things are more related than distant things” (p. 236). More traditional non-spatial statistics would ignore spatial processes and could be biased toward rejecting the null hypothesis, underestimating variance in the data, and presenting smaller confidence intervals (Ward & Skrede-Gleditsch, 2008). To better understand the hypothesized spatial relationships, ArcMap 10.0, 10.1, and GeoDa 1.3.15 was used with data aggregated to the census tract level. SPSS 19.0 was also used to examine frequencies, perform correlation analyses, and perform principal components analysis.

a. Exploratory data analysis

Exploratory data analysis was performed to understand the linear and non-linear patterns in the data. Descriptive statistics and graphs (e.g., histograms) helped determine whether the independent and dependent variables exhibited skewness and/or kurtosis. In addition, unimputed and imputed datasets were compared with a paired samples t-test to determine whether imputed variables differ after imputation. Finally, independent and
dependent variables were also correlated with Pearson’s r and point-biserial correlations to discover the strength and direction of the relationships among the variables.

b. Exploratory spatial data analysis

Exploratory spatial data analyses were used to visualize spatial distributions, identify spatial outliers, and discover clusters or hot spots. Histograms, box plots, scatter plots, qq plots, and choropleth maps helped to understand the complex spatial arrangements within the data. For example, a scatterplot matrix tested for linearity among the variables; this graphic helped to determine whether the amount of neurotoxicant exposure had a linear effect on mental health assessment scores. When there was a non-linear relationship, then variables were transformed (e.g., through using a squared term).

c. Spatial autocorrelation

Each of the dependent variables was investigated for spatial autocorrelation with a variety of statistics. In contrast to traditional statistics that do not emphasize the spatial context, spatial autocorrelation in geostatistics is desirable because a researcher can test for clustering of observations. Global measures indicated when there was spatial autocorrelation across the state, and local measures indicated where there were different patterns of clustering within the regions of the state.

The global Moran’s I is a widely accepted test to evaluate spatial autocorrelation (Moran, 1950). Values for Moran’s I fall between -1 and +1. A correlation of -1 would indicate that observations are perfectly dispersed and a correlation of +1 would indicate that observations are perfectly clustered. A Moran’s I value of zero would indicate there is no spatial relationship among observations. After calculating the Moran’s I, a random permutation test was performed to test the stability of the results. This test calculates a
reference distributions based on the Moran’s I statistic and compares that distribution to
the original Moran’s I statistic. Typically, one performs this test multiple times and with
different numbers of permutations (e.g., 499 – 999) to discover whether the observed
spatial autocorrelation is extreme across any number of possible spatial arrangements. A
significant result means that it is unlikely a higher Moran’s I value would be found. The
p-value given in these results is considered a “pseudo p-value” because the result
increases or decreases, depending on the number of permutations. This statistic also
produces a plot with four quadrants. Observations in the upper right and lower left
quadrants (labeled high-high or low-low respectively) represent spatial clusters and
indicate positive local spatial autocorrelation. In contrast, observations in the upper-left
and lower right quadrants (labeled low-high or high-low respectively) represent spatial
outliers and indicate negative local spatial autocorrelation (Anselin, 2005).

The local indicators of spatial association (LISA) are widely accepted to evaluate
local spatial autocorrelation. This statistic produces two maps of the region of interest;
one map displays the type of clustering (e.g., high-high or low-low), and the other map
shows the significance level of the clustering. Much like the Moran’s I, results from this
analysis are based on a permutation test and pseudo p-value. A higher degree of
significance (e.g., 0.05 vs. 0.001) means that any clustering is likely to be stable over
time (and not caused by spurious data). The LISA permutation test is also performed
multiple times at differing numbers of permutations to evaluate the stability of the results.

d. Empirical Bayes Smoothing

Rate instability (a.k.a., the small numbers problem) commonly occurs when
researchers are studying geographic units with small populations. The problem arises
because the denominator for these geographic units is often so small that rates may be extreme or may fluctuate wildly from year to year. One way to deal with rate instability is to simply ignore the data. The National Cancer Institute uses this method when it calculates cancer rates for areas and it masks data with less than six events (BioMedware, 2012). Another way to deal with the small numbers problem is to use a smoothing algorithm. Rate smoothing is commonly used in geographic studies to take into account regional instability for spatial units (e.g., census tracts). Smoothing algorithms can focus on the global or local level, but their purpose is one in the same: an increase in the precision of risk estimates.

This study took a hybrid approach to the small numbers problem. Census tracts with less than five admissions were already removed from analyses to protect client confidentiality. This removal also served to take out the most likely cases to generate unstable weights. In addition, an empirical Bayes smoothing (EBS) algorithm based on poisson gamma rates was used with all admissions data because preliminary analysis indicated rate instability between 2007 and 2008. EBS transforms the rates so they are more similar to a global trend. ASU (2013) explained the technique, “is based on a raw rate for each areal unit that is averaged with a separately computed reference estimate based on the whole study region, such as the overall population mean.”

e. Spatial regression modeling

Spatial regression analysis determined when there was an important underlying spatial process among the independent and dependent variables. For each research question, model building involved finding the most effective combination of explanatory variables while controlling for any of the common problems for regression models (e.g.,
multicollinearity). Model building used several different regression techniques to develop the best explanatory model; it began with ordinary least squares (OLS) regression to establish a parsimonious global model. Geographically weighted regression was used to develop local explanatory models when necessary. Both sets of models were compared to determine the most parsimonious model.

An ordinary least squares (OLS) regression model examined the global processes in the models. Classic OLS investigates whether one or more independent variables predict a dependent variable. OLS coefficients are most reliable when the model meets all of the regression assumptions (Berry, 1993); however, several of these are difficult to meet in the analysis of spatial data (Bailey & Gatrell, 1995). In particular, OLS assumes that one is modeling a stationary process with fixed effects. Stationarity, the independence of statistical properties, suggests that first order effects (i.e., global trends) are of primary import while second order effects (i.e., local trends) should not exist (Bailey & Gatrell, 1995). Said another way, the random error in classic OLS is assumed to have a mean of zero, and random error terms are homoskedastic (i.e., uncorrelated with a constant variance). In reality, these assumptions are usually violated because some observations may cluster or drift together, which may represent an underlying spatial process. For instance, an OLS model may illustrate mental health admissions are the same in Appalachian and metropolitan regions (i.e., spatial homogeneity); whereas, a spatial regression model with nonstationarity may indicate mental health admissions are different in Appalachian and metropolitan regions (i.e., spatial heterogeneity). Despite these problems, OLS ensures that the model is properly specified before using other regression techniques.
Geographic weighted regression (GWR) was used to examine the intensity of spatial clustering in this study. This technique was preferred for spatial modeling because it developed a unique regression equation for each of the census tract, which was related to neighboring census tracts. By allowing local regression equations to vary, GWR was able to show where each model predicted well versus where it did not predict well. This ability was very important for research questions associated with this study because Ohio has many diverse regions, and the intensity of child admissions and mental health assessment scores were expected to vary significantly throughout the state.

GWR examines local processes within the models. Unlike OLS, GWR assumes nonstationarity of the data and explores local trends that occur across the study area, which is an excellent way to discover regional differences. This regression technique is different than standard OLS techniques because it creates a unique equation and r-squared coefficient for every spatial unit of interest. One can map these coefficients to create a surface to see the relationship among the independent and dependent variables. Areas indicating weak prediction may warrant the inclusion of other variables into the model; whereas, areas indicating strong prediction may indicate remediation is possible with modification of one or more independent variables.

Model building is an iterative process accomplished in several steps for both types of regression (Rosenshein & Scott, 2010). First, variable coefficients are investigated to determine if they have the expected positive or negative sign. For example, problem severity should be positively related to degree of child poverty based on research; the more problem’s experienced on a regular basis, the greater the likelihood of being poor.
Should one or more coefficients have the wrong sign, then the problem is thoroughly investigated by checking the integrity of the data.

The second step to evaluate the regression models is to make sure there is no redundancy in the explanatory variables. Multicollinearity, the ability of two or more variables to tell the same story, is a typical problem of research that includes highly related variables. In this study, some of the independent variables expressed a high correlation because they were developed to be related to each other (e.g., hopefulness and problem severity). Other variables were related to one another because they were either conceptually related or trended together (e.g., ACS child poverty and minority variables). Variables with variance inflation factors over 7.5 were considered for either deletion from the model or as contenders for the neighborhood index. After all, it did not make sense to keep variables that have redundancy in their explanatory power of the dependent variable.

Next, the statistical significance of the overall model as well as each variable within the regression model is investigated. In classic OLS, the Joint F-Statistic is used to determine overall model significance. However, if the Koenker Bruesch-Pagan statistic shows nonstationarity in the model, then the Joint Wald Statistic is more appropriate to interpret the overall model fit. P-values associated with each individual variable are interpreted to determine statistical significance as long as the Koenker Bruesch-Pagan statistic is not significant; otherwise the robust probabilities are interpreted.

The evaluation of each model’s performance is the next step in the process. The percentage of variance explained by a model is evaluated with the adjusted r-squared
value. For some models, it is likely the results may be low for this statistic. Some degree of model misspecification was expected in this study. As previously stated, the complex nature of child admissions and mental health outcomes at assessment made it highly likely that some predictor variables were left out because there was no way to easily capture them (e.g., crime). Aside from the adjusted r-squared statistic, each model was compared with the Akaike info criterion-corrected (AICc), which was a goodness of fit statistic that puts a penalty for using too many parameters in a model.

That step is followed by an evaluation of the regression model’s residuals. Classic OLS assumes that the residuals, the difference between the observed response and the predicted response, are normally distributed. If this assumption turns out to be invalid, then the model may be misspecified and inefficiently estimate the p-vaes of coefficients. Statistical significance of the Jarque-Bera statistic would indicate the model’s residuals are not normally distributed. This statistic may be improved through adding missing explanatory variables, removal of outliers, or the transformation of variables. GWR residuals were displayed in a simple histogram and compared to a normal distribution when this statistic was significant.

Finally, the GWR residuals resulting from the regression analysis undergo testing for clustering. All residuals are mapped to see if there are any visible clusters. Ideally, the residuals should look like a random pattern on the map; there should be no pattern to under- and over-predictions. A Moran’s I statistic also is used on the residuals to provide statistical verification of the presence or absence of spatial autocorrelation. The random permutation test for Moran’s I is also performed to discover whether observed spatial
autocorrelation is extreme across 999 possible spatial arrangements.

In geospatial statistics, the classic OLS model is compared with other models that may present a more accurate picture of the data. Earlier, it was mentioned that the several key assumptions in OLS were likely to be violated because of spatial dependence in the data. When, the Koenker Bruesch-Pagan statistic is statistically significant (i.e., the stationarity assumption is violated), then the model is a good candidate for GWR because relationships among variables differ across census tracts. OLS and GWR models are compared using the statistical tests mentioned when evaluating model performance, including the adjusted r-squared, and AICc.
Chapter 4: Results

Chapter 4 presents the results from the analyses mentioned in the previous chapter. This chapter begins with a discussion of the data aggregation and the resulting sample of census tracts. Next, results from the imputation are presented followed by sections on the transformation of key variables and results from the principal components analysis. Each of the dependent variables was analyzed for spatial autocorrelation, and these results are presented with tables for global spatial autocorrelation and maps for local spatial autocorrelation. Finally, the regression analyses are presented in the order of the research objectives.

Data Aggregation

As previously mentioned, all data were received at the client level in three databases: youth surveys, parent surveys, and case manager surveys. Each database was combined into one comprehensive dataset. Data were sorted by unique client identifier, age at assessment, primary diagnosis, and diagnostic category. Data were aggregated so there was one unique admission per client episode, and data were aggregated a second time down to the census tract level. The intermediary aggregation step allowed unique statistics to be calculated for admission level data (e.g., rate of admissions).

Sample

The final sample consisted of 2,608 census tracts out of a possible 2,941 census tracts (88.68%), which included census tracts that could be counted multiple times
because they were split into multiple parts. The chief reason census tracts were removed dealt with the HIPPA criteria; these areas contained less than five admissions during 2007. Omitted census tracts were located primarily in urban areas except for the western side of the state where there were several large groups of rural tracts omitted. Figure 3 is colored green to show the areas that were included in the analysis, and Figure 4 displays in red the census tracts that were removed from the analysis.

Figure 3. Census tracts included in all analyses
Census tracts within this dataset had between one and 15 neighboring census tracts from which the spatial weights were determined (Figure 5). The majority of census tracts had between five and six neighboring census tracts (22.4%), but census tracts with six to seven neighbors was almost as common (20.4%). The removal of census tracts created three neighborless tracts or islands (i.e., 390490020, 390852032, and 391130102), so data were hand entered for these tracts to give them relationships with the closest tracts for the queen contiguity spatial weights file. Reciprocal relationships were not entered for the census tract neighbors because it would dilute their existing spatial weight scheme and possibly lead to misestimation of local relationships.
Data at the client admission level were evaluated for missingness. Missing data range from zero to almost 30 percent, depending on the variable. The dependent variable hopefulness had the greatest amount of missing information (28.71%), followed by the independent variable race (10.29%). The other dependent variables, functioning and problem severity, had very little missing information (4.36% and 3.63% respectively), and the independent variable gender also had very little missing information (1.15%). The hopefulness variable likely has a higher degree of information because it is only on two of the three Ohio Scale surveys, giving it a higher probability of appearing missing after the first aggregation.

Graham (2012) recommends that any variable highly correlated with variables missing information be included during the imputation. All of the variables were tested,
but very few made it into the final model because they did not meet the criteria of having at least a 0.1 correlation with at least one other variable. Table 6 display the correlations among variables included in the imputation process. The Pearson’s correlations conducted are also applicable to the nominal variables (e.g., gender and race) because a point-biserial correlation is a unique case of the Pearson product moment correlation. The highest correlations are among the dependent variables; this strong relationship was expected because they were developed to relate to one another. Functioning exhibits a moderately negative statistically significant correlation with problem severity \((r = -0.671, p < 0.01)\) and hopefulness \((r = -0.571, p < 0.01)\). Problem severity and hopefulness exhibit a moderately positive statistically significant correlation \((r = 0.544, p < 0.01)\). Race exhibits a moderate statistically significant correlation with neurotoxicant solvents \((r_{pb} = 0.415, p < 0.01)\). Age exhibits a statistically significant weak correlation with gender \((r_{pb} = 0.126, p < 0.01)\) and functioning \((r_{pb} = 0.106, p < 0.01)\).

\[
\begin{array}{cccccc}
\text{Age} & \text{Gender} & \text{Race} & \text{Hopefulness} & \text{Functioning} & \text{Problem Severity} & \text{Neuro. Solvents} \\
1 & 0.126** & -0.01** & 0.028** & 0.106** & -0.098** & -0.002 \\
1 & -0.02** & 0.036** & 0.079** & 0.002 & -0.009* & \\
1 & -0.06** & -0.041** & 0.006 & 0.415** & \\
1 & -0.571** & 0.544** & -0.049** & \\
1 & -0.671** & -0.044** & \\
1 & 0.026** & \\
\end{array}
\]

Table 6. Pairwise correlations

\(^{8}\) *p < 0.05; **p < 0.01
Appendix E shows an analysis of the missing data patterns. Altogether, there are 28 patterns of missing and non-missing data in the dataset that appeared arbitrary and not monotone. The most frequent pattern found in the dataset is complete cases across all variables (63.10%). The second most frequent pattern is complete cases across all variables with the exception of the hopefulness variable (22.41%), and the third most frequent pattern is complete data across all variables with the exception of the race variable (6.78%). All other missing data patterns account for a relatively small percentage of the patterns of missingness (i.e., between 2.77% and 0.00%). It is difficult to tell whether the groups means presented in the table are significantly different without coding each of the 66,712 cases individually, but group means look very similar for the first three patterns which represents a majority of the data (92.29%).

Data were imputed with the MCMC algorithm (single chain) that used a Jeffrey’s prior with 300 burn-in iterations. Initial estimates for the MCMC were derived by an expectation maximization algorithm in posterior mode, and the convergence criterion set at 1E-5. Preliminary tests showed that the expectation maximization algorithm ran with no imputations, and that the MCMC converged in one iteration. Several test runs of the final algorithm indicated that the model converged in 12 iterations, so 12 was chosen as the number of iterations between imputations in a chain. Monotonic change indicated a gradual decrease in the -2 Log Likelihood fit function between iterations (Table 7). Most of the variables stabilized within five iterations, but hopefulness stabilized around eight iterations. Once the data were imputed, no pathological information was indicated by the worst linear function graphs, autocorrelation plots, or time-series plots.
Table 7. Expectation maximization iteration history

<table>
<thead>
<tr>
<th>Iteration</th>
<th>-2 Log Likelihood</th>
<th>Gender</th>
<th>Race</th>
<th>Hopefulness</th>
<th>Functioning</th>
<th>Problem Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>613882</td>
<td>1.42342</td>
<td>1.40410</td>
<td>11.3105</td>
<td>47.0555</td>
<td>25.8523</td>
</tr>
<tr>
<td>1</td>
<td>541936</td>
<td>1.42342</td>
<td>1.40410</td>
<td>11.3105</td>
<td>47.0555</td>
<td>25.8523</td>
</tr>
<tr>
<td>2</td>
<td>538578</td>
<td>1.42347</td>
<td>1.40475</td>
<td>11.3314</td>
<td>47.0515</td>
<td>25.8578</td>
</tr>
<tr>
<td>3</td>
<td>538250</td>
<td>1.42348</td>
<td>1.40490</td>
<td>11.3423</td>
<td>47.0512</td>
<td>25.8587</td>
</tr>
<tr>
<td>4</td>
<td>538220</td>
<td>1.42348</td>
<td>1.40493</td>
<td>11.3467</td>
<td>47.0512</td>
<td>25.8590</td>
</tr>
<tr>
<td>5</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40493</td>
<td>11.3482</td>
<td>47.0512</td>
<td>25.8590</td>
</tr>
<tr>
<td>6</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3488</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
<tr>
<td>7</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3489</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
<tr>
<td>8</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3490</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
<tr>
<td>9</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3490</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
<tr>
<td>10</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3490</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
<tr>
<td>11</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3490</td>
<td>47.0511</td>
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</tr>
<tr>
<td>12</td>
<td>538217</td>
<td>1.42348</td>
<td>1.40494</td>
<td>11.3490</td>
<td>47.0511</td>
<td>25.8591</td>
</tr>
</tbody>
</table>

Missing values were also present in American Community Survey dataset. In one instance, census tract data had null values for all contextual variables. While this situation is possible, it is highly unlikely to be correct because five children that made it into the final sample were living in that tract; whereas, ACS data indicated that no one was living in the tract. Instead of using imputation to fill in missing values, it was more appropriate to substitute the mean values from neighboring census tracts. Data for census tract 390998033 was averaged for all contextual variables from census tracts 390998029, 390998031, 390998032, and 390998124.

The un-imputed dataset only contained four census tracts with missing information on the child’s race variable by the second aggregation. Race data for these four census tracts was replaced by average race data from neighboring census tracts. In three instances racial profiles from neighboring census tracts was unavailable because no
children were admitted. Census tract data for 391093001 was replaced with the average of 391093151, 391093153, 391093901, 39109365101; data for 39113050401 was replaced with the average of 39113050501 and 39113050303; data for 39113050302 was replaced with the average of 39113050101, 39113050303, and 39113050301; and data for 391499715 was replaced with the average of 391499714, 391499717, and 390110406. Variable means from the unimputed and imputed datasets were compared with a paired-samples t-test. After aggregation to the census tract level, results showed some differences among variables (Table 8). The three dependent variables were not statistically different between the unimputed and imputed datasets. Means were different for demographic variables. The percentage of males ($t_{2,603} = -7.903$, $p < 0.0001$) was significantly higher as was the percentage of females ($t_{2,603} = -7.197$, $p < 0.0001$). The percentage of Whites ($t_{2,603} = -26.932$, $p < 0.0001$) was significantly higher as was the percentage of minorities ($t_{2,603} = -28.712$, $p < 0.0001$). Interestingly, the means for all imputed variables were slightly higher than means for the unimputed data, with the mean increasing the most for the percentage of White in a census tract. The Pearson’s product moment correlation was excellent between the unimputed and imputed variables, typically ranging from 0.97 to 0.99. Hopefulness exhibited the lowest correlation of the group, but it was still very strong overall ($r = 0.88$, $p < 0.0001$).
<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean Difference</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>t-value</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hopefulness</td>
<td>-0.009</td>
<td>0.647</td>
<td>0.013</td>
<td>-0.721</td>
<td>2603</td>
<td>0.471</td>
</tr>
<tr>
<td>Functioning</td>
<td>-0.014</td>
<td>0.726</td>
<td>0.014</td>
<td>-1.018</td>
<td>2603</td>
<td>0.309</td>
</tr>
<tr>
<td>Problem Severity</td>
<td>-0.001</td>
<td>0.749</td>
<td>0.015</td>
<td>-0.059</td>
<td>2603</td>
<td>0.953</td>
</tr>
<tr>
<td>Male %</td>
<td>-0.170</td>
<td>1.096</td>
<td>0.021</td>
<td>-7.903</td>
<td>2603</td>
<td>0.000*</td>
</tr>
<tr>
<td>Female %</td>
<td>-0.124</td>
<td>0.882</td>
<td>0.017</td>
<td>-7.197</td>
<td>2603</td>
<td>0.000*</td>
</tr>
<tr>
<td>White %</td>
<td>-1.495</td>
<td>2.833</td>
<td>0.056</td>
<td>-26.932</td>
<td>2603</td>
<td>0.000*</td>
</tr>
<tr>
<td>Minority %</td>
<td>-1.127</td>
<td>2.003</td>
<td>0.039</td>
<td>-28.712</td>
<td>2603</td>
<td>0.000*</td>
</tr>
</tbody>
</table>

Table 8. Differences among unimputed and imputed variables.

**Variable Transformation**

Residuals from preliminary regression modeling were not normally distributed in a few cases, so a minority of variables was transformed. All variables related to hazard quotients/indices (e.g., hazard quotient for total neurotoxicant risk) were transformed with a natural logarithm, and the variable associated with risk of lead paint exposure was transformed with the arcsine technique. Normal score transformation was considered in place of other transformations, but was not used because of concerns with over fitting models. Jarque-Bera scores improved after transformation, although they typically remained statistically significant.

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\( p < 0.0001 \)
Principal Components Analysis

As expected, the proposed independent variables were correlated at statistically significant levels. Principal components analysis was conducted to combine related variables into one or more factors. A preliminary model was run that contained all of the ACS data proposed in the study along with some unique variables found to be important in a neighborhood poverty index (Messer et al., 2006). Early results showed promise with the following variables: race, unemployment, female-headed family households with one or more children, average household size for female-headed households, average household size for all family households, crowded housing, households on public assistance, percent child poverty, greater than high school education, less than high school education, males in managerial positions, females in managerial positions, income less than $35,000, median income for adults over 16, median income for males over 16, and median income for females over 16. The preliminary model was simplified for parsimony and to retain the variables that expressed the highest degree of correlation (Appendix F). The final model yielded seven variables that accounted for 66.14 percent of the variance on one factor. While this number sounds high, one must remember that there is a 33.86 percent loss of information when substituting these seven variables with one variable. Both Bartlett’s test for sphericity ($\chi^2 (21) = 11,861.93, p < 0.0001$) and the KMO test (0.89) confirmed that there was sampling adequacy for correlation matrices. Communalities for each of the variables ranged from 0.588 to 0.800, which indicated that each variable explained a high proportion of the variance within the factor structure (Table 9).
Variables that were significant in the PCA were slightly different than the standardized neighborhood deprivation index (SNDI). The PCA retained four of the eight variables from the SNDI, but two of the variables did not turn out to be related to the construct: males in a management and professional occupations and crowded housing. One variable, the percent of minorities per census tract, was even added that was not included in the SNDI. It is possible that those two variables were not included in the model because two of the SNDI variables were modified for this analysis (e.g., the percent of household poverty became the percent of child poverty), or because Messer et al. (2006) were looking at much smaller geographies in metropolitan and suburban settings. Either way the variables retained for the factor best represented the concepts of people at-risk for poverty and poor economic outcomes, and will hereafter be referred to as the neighborhood economic risk index (NERI).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Extraction Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Income less than $35,000</td>
<td>0.800</td>
</tr>
<tr>
<td>Child poverty</td>
<td>0.713</td>
</tr>
<tr>
<td>Female-headed family households with one or more children under 18</td>
<td>0.703</td>
</tr>
<tr>
<td>Unemployment</td>
<td>0.631</td>
</tr>
<tr>
<td>Households on public assistance</td>
<td>0.604</td>
</tr>
<tr>
<td>Less than high school education</td>
<td>0.590</td>
</tr>
<tr>
<td>Percent minority</td>
<td>0.588</td>
</tr>
</tbody>
</table>

Table 9. Communalities for the NERI
Spatial Autocorrelation

a. Global autocorrelation

Dependent variables associated with the research questions were examined for spatial autocorrelation. Global Moran’s I revealed that Ohio Scale measures did not exhibit spatial autocorrelation. Hopefulness, functioning and problem severity all displayed random patterns, and their Moran’s I values were not statistically significant (Figure 6). The random permutation tests for all three variables were significant across 999 possible spatial arrangements, indicating that it was unlikely a higher Moran’s I value would be found (Table 10).

Figure 6. Comparing Moran’s I plots for hopefulness, functioning and problem severity scores

Global Moran’s I revealed that some admissions variables exhibited spatial autocorrelation. Overall child admissions and admissions for AD-HD were positively correlated; they displayed moderate clustering across the state. In contrast, admissions for PDD did not display significant clustering across the state (Figure 7). The random
permutation tests for all three variables were significant across 999 possible spatial arrangements, indicating that it was unlikely a higher Moran’s I value would be found (Table 10).

![Figure 7. Comparing Moran’s I plots for general admissions, AD-HD admissions, and PDD admissions](image)

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Dependent Variable</th>
<th>Moran’s I</th>
<th>R²</th>
<th>E[I]</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>z-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hopefulness</strong></td>
<td></td>
<td>0.071</td>
<td>0.018</td>
<td>-0.0004</td>
<td>0.0123</td>
<td>5.762</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>Functioning</strong></td>
<td></td>
<td>0.106</td>
<td>0.037</td>
<td>-0.0004</td>
<td>0.0125</td>
<td>8.472</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>Problem Severity</strong></td>
<td></td>
<td>0.095</td>
<td>0.031</td>
<td>-0.0004</td>
<td>0.0121</td>
<td>7.933</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>Child Admissions</strong></td>
<td></td>
<td>0.469</td>
<td>0.416</td>
<td>-0.0004</td>
<td>0.0125</td>
<td>37.650</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>AD-HD Admissions</strong></td>
<td></td>
<td>0.365</td>
<td>0.290</td>
<td>-0.0004</td>
<td>0.0125</td>
<td>29.594</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>PDD Admissions</strong></td>
<td></td>
<td>0.021</td>
<td>0.002</td>
<td>-0.0004</td>
<td>0.0124</td>
<td>1.782</td>
<td>0.001</td>
<td></td>
</tr>
</tbody>
</table>

Table 10. Global spatial autocorrelation results

---

10 999 permutations
b. Local autocorrelation

Local indicators for spatial autocorrelation (LISA) showed some local patterns. Hopefulness had 359 statistically significant local clusters (Figure 8). Most of these clusters were significant at the 0.05 level (266, 74.09%), and others were significant at the 0.01 level (82, 22.84%), or the 0.001 level (11, 3.06%). Census tracts significant at the 0.05 level gathered in several regions. Ashtabula County and Montgomery County had many clusters where children were less hopeful at intake (High-High), and Cuyahoga County had many clusters where children appeared more hopeful at intake (Low-Low). Census tracts statistically significant at other levels did not appear to cluster in any particular region.

Figure 8. LISA results for hopefulness
Functioning had 459 statistically significant local clusters (Figure 9). As with hopefulness, most of these clusters were significant at the 0.05 level (323, 70.37%), and others were significant at the 0.01 level (119, 25.93%), or the 0.001 level (17, 3.70%). Census tracts significant at the 0.05 level gathered in many regions. Jefferson, Medina, Monroe, eastern Preble, Stark, Summit, and Wayne had many clusters where children had better functioning at intake (High-High). Hamilton and Scioto counties had several clusters where children had worse functioning at intake (Low-Low). Lucas and Tuscarawas counties had clusters where children had better functioning at intake (High-High), and these values were significant at the 0.01 level. There was also minor clustering in Hamilton, Montgomery and Scioto counties for children who displayed worse functioning at intake (Low-Low), which was significant at the 0.01 level. Census tracts statistically significant at the 0.001 level did not appear to cluster in any particular region.
Problem severity had 383 statistically significant local clusters (Figure 10). Most of these clusters were significant at the 0.05 level (267, 69.71%), and others were significant at the 0.01 level (92, 24.02%), or the 0.001 level (24, 6.27%). Census tracts significant at the 0.05 level gathered in many regions. Hamilton, Lawrence, Montgomery, and Scioto had several clusters where children had worse problem severity at intake (High-High). There was also one cluster on the border of Ashtabula and Trumbull counties were children also had worse problem severity at intake. Geauga, Holmes, Lorain, Lucas, Mercer, and Wayne counties had several clusters where children had lower scores on problem severity at intake (Low-Low). Census tracts significant at the 0.01 level gathered in several regions. Montgomery and Scioto counties had clusters where children had worse problem severity at intake (High-High). There was also
clustering in Allen, Geauga, Lucas, and Preble counties for children who displayed less problem severity at intake. Census tracts statistically significant at the 0.001 level did not appear to cluster in any particular region.

![Figure 10. LISA results for problem severity](image)

Child admissions had 911 statistically significant local clusters (Figure 11). Many of these clusters were significant at the 0.05 level (481, 52.80%), and others were significant at the 0.01 level (307, 33.70%), or the 0.001 level (123, 13.50%). Census tracts significant at the 0.05 and 0.01 levels typically occurred both in rural areas and in major metropolitan cities. The majority of clusters were dispersed throughout the state and indicated a lower than expected level of admissions (Low-Low). A higher than
expected level of admissions occurred in counties with major metropolitan cities (i.e., Cuyahoga, Franklin, Hamilton, and Mahoning) as well as Appalachian counties (i.e., Athens and Jefferson) and one rural county (i.e., Clark). Even at the 0.001 level, there were still counties that displayed clustering; Butler, Darke, Delaware, Licking, Union, and Warren counties had lower than expected levels of child admissions, while Summit county had a higher than expected level of admissions.

Figure 11. LISA results for all child admissions

Admissions due to attention deficit-hyperactivity disorder (AD-HD) had 744 statistically significant local clusters (Figure 12). Many of these clusters were significant at the 0.05 level (447, 60.08%), and others were significant at the 0.01 level (226,
30.77%), or the 0.001 level (71, 9.54%). Census tracts significant at the 0.05 and 0.01 levels typically occurred both in rural areas and in major metropolitan cities. The majority of clusters were dispersed throughout the state and indicated a lower than expected level of AD-HD admissions (Low-Low). A higher than expected level of AD-HD admissions occurred in counties with major metropolitan cities (i.e., Cuyahoga, Franklin, Hamilton, Mahoning, and Montgomery) as well as one rural county (i.e., Marion). Even at the 0.001 level, there were still counties the displayed clustering; Medina, Mercer, Tuscarawas, Union and Warren counties had lower than expected levels of AD-HD admissions, while Clark, Hamilton and Summit counties had a higher than expected level of AD-HD admissions.

Figure 12. LISA results for admissions due to attention deficit-hyperactivity disorder
Admissions due to pervasive developmental disorder (PDD) had 427 statistically significant local clusters (Figure 13). Most of these clusters were significant at the 0.05 level (296, 69.32%), and others were significant at the 0.01 level (101, 23.65%), or the 0.001 level (30, 7.03%). Census tracts significant at the 0.05 and 0.01 levels typically occurred in rural and suburban areas. The majority of clusters were dispersed throughout the state and indicated a lower than expected level of PDD admissions (Low-Low). A higher than expected level of PDD admissions occurred in rural areas (i.e., Hancock, Muskingum, and Trumbull) as well as to the west of Youngstown. Even at the 0.001 level, there were still counties the displayed clustering. Clusters were present between the border of Holmes and Tuscarawas counties as well as the border of Delaware and Union counties. Butler and Warren counties also had clusters of lower than expected admissions. No counties exhibited a higher than expected level of PDD admissions.

Figure 13. LISA results for admissions due to pervasive developmental disorder
Preliminary Regression Analyses

Preliminary regression analyses were run on all research questions for several reasons. First, some variables of interest were “mirrors” of one another (e.g., % male admissions and % female admissions), so only the variables that had the strongest and most significant coefficients were incorporated. Second, it was important to discover whether quadratic terms or two-way interactions had any impact on the regression models. Upon examination, a few of these variables were statistically significant in some models and were incorporated in every initial model. For the sake of brevity the “initial model” for all of the research questions contained the following variables: hopefulness, functioning, problem severity, average family size, the percent of admissions greater than age 11, the percent of male admissions, the percent of minority admissions, the percent of crowding, the percent of risk of lead paint exposure (arcsine), NERI score, interaction between functioning and risk of lead paint exposure, interaction between average family size and risk of lead paint exposure, the squared functioning score, the squared percent of male admissions, and the squared NERI score. The following variables were also incorporated into the initial model depending on the research question and what performed best in preliminary tests: total hazard quotient (ln), solvent hazard quotient (ln), metal hazard quotient (ln), lead hazard index (ln), manganese hazard index (ln), and mercury hazard index (ln). Models that used a variable as a dependent variable (e.g., hope) did not also include that variable as an independent variable in the initial model.

Research Question 1a

Research question 1a asks whether there is a spatial relationship between neurotoxicant exposure and the mental health admissions at patient intake. OLS
regression models helped to develop the most parsimonious model before spatial modeling (Table 11). Model 1’s Koenker BP statistic indicated nonstationarity (55.52, 16, p < 0.0001), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (3,238.76, 16, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.06 – 246.31). The Jarque-Bera test indicated residuals were normally distributed (4.46, 2, p = 0.1070). Model 2 was similar to model 1, but it only included the variables that were found to be statistically significant: average family size, functioning, the percent of male admissions, the NERI score, the squared NERI score, and the interaction term for average family size and risk of lead paint exposure. This model showed nonstationarity (29.60, 6, p < 0.0001). Model 2 was also statistically significant and explained 51 percent of the variation in child admissions (2,705.07, 6, p < 0.0001). Unlike the previous model, the Jarque-Bera test indicated residuals were not normally distributed in model 2 (6.90, 2, p = 0.0317).

<table>
<thead>
<tr>
<th></th>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>206.47***</td>
<td>456.87***</td>
<td>845.25***</td>
<td>N/A</td>
</tr>
<tr>
<td>Joint Wald</td>
<td>3,238.76***</td>
<td>2,705.07***</td>
<td>2,454.57***</td>
<td>N/A</td>
</tr>
<tr>
<td>Adjusted R²</td>
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<td>0.51</td>
<td>0.49</td>
<td>0.46</td>
</tr>
<tr>
<td>AICc</td>
<td>3,456.24</td>
<td>3,702.70</td>
<td>3,800.42</td>
<td>3,364.22</td>
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<tr>
<td>Jarque-Bera Test</td>
<td>4.46</td>
<td>6.90**</td>
<td>8.46**</td>
<td>N/A</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>55.52***</td>
<td>29.60***</td>
<td>8.57**</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 11. Comparing child admission models

---

11 *Bandwidth set to 279 neighbors with an adaptive kernel; **p < 0.05; ***p < 0.0001
Model 2 underwent further refinement because the variables functioning and the percent of male admissions were no longer significant when other variables were taken out of Model 1. The interaction term for average family size and risk of lead paint exposure was also taken out because only one of the variables remained in the model. Model 3 contains average family size, the NERI score, and the squared NERI score. Model 3 showed nonstationarity like other models (8.57, 3, p = 0.0356). Model 3 was also statistically significant and explained 49 percent of the variation in child admissions (2,454.57, 3, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (8.46, 2, p = 0.0146).

Table 12 displays the OLS results from Model 3. Each of the variables in the model is statistically significant, and none of the variables shows multicollinearity per the VIF. One variable, the NERI score, show positive relationships with child admissions, while average family size and the squared NERI score show negative relationships with child admissions. The child admissions model is the one of three models where a nonlinear term made it into the final model. The squared term is negative, which indicates an inverted-U shape where the linear slope becomes less positive as x increases. In this regression equation, the squared NERI score is decreasing as child admissions increase; the larger the squared value of child admissions, the smaller the NERI score. This decrease is not substantial (only -0.14), but it is noticeable and the regression equation suffers significantly if the term is taken out. The largest coefficient was found between child admissions and the NERI score; for each one unit increase in the NERI score, the natural log of child admissions increases by 0.58, holding all other variables constant.
That is, children living in neighborhoods at greater risk of economic distress experience greater admission rates.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust* Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
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<td>-30.13</td>
<td>0.00**</td>
<td></td>
</tr>
<tr>
<td>Average family size</td>
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<td>0.03</td>
<td>-10.68</td>
<td>0.00**</td>
<td>1.05</td>
</tr>
<tr>
<td>NERI</td>
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<td>0.01</td>
<td>41.66</td>
<td>0.00**</td>
<td>2.00</td>
</tr>
<tr>
<td>NERI squared</td>
<td>-0.14</td>
<td>0.01</td>
<td>-16.84</td>
<td>0.00**</td>
<td>1.96</td>
</tr>
</tbody>
</table>

Table 12. Model 3 variables for child admissions

Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account. The AICc was lower than in both Model 2 and Model 3, but the adjusted $R^2$ decreased slightly from other models (Table 11). The Jarque-Bera test in Model 3 indicated that the residuals were not normally distributed, but Figure 14 shows the standardized residuals appear to be unimodal and approximate a normal distribution with mesokurtic peakedness (3.12) and skewness that is close to zero (-0.10).

Figure 14. Distribution of standardized residuals for child admissions

---

12 *Robust values displayed because of significant Koenker BP statistic; **p < 0.0001
Global Moran’s I revealed the final child admissions model exhibited positive spatial autocorrelation in GWR residuals ($I = 0.13; z = 10.05, p < 0.0001$). Figure 15 displays the standardized residuals across the state. Child admissions closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. As one would expect from a normal distribution, most of the residuals are located near the center of the distribution and taper off as the standard deviation increases. Clustering appeared most prevalent in census tracts whose residuals fall into the middle of the distribution (-1.5 to 1.5 standard deviations).

Figure 15. GWR standardized residuals for child admissions
Model 4 predicts child admissions very well in some areas. The percent of the local variance accounted for by Model 4 ranges from 6.38 percent to 73.02 percent, and Figure 16 highlights the variance explained per census tract. Model 4 best predicts child admissions (>65%) in central Ohio and the Akron region. The model also predicts well (between 53% and 64%) in urban areas (e.g., Canton, Cincinnati, and Dayton) and suburban areas (e.g., Lorain and Warren counties). The model predicts the poorest (between 6% and 37%) in some urban areas (e.g., Cuyahoga, Lucas, and Mahoning counties) and most rural and Appalachian areas (e.g., northwestern and southeastern counties).

Figure 16. GWR $R^2$ for child admissions\textsuperscript{13}

\textsuperscript{13} Natural breaks used to generate intervals
Research Question 1bi

Research question 1bi asks whether there is a spatial relationship between neurotoxicant exposure and attention deficit-hyperactivity disorder (AD-HD) admissions at patient intake. OLS regression models helped to develop the most parsimonious model before spatial modeling (Table 13). Model 1’s Koenker BP statistic indicated nonstationarity (33.25, 17, p = 0.0105), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (1,802.88, 17, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.19 – 246.43). The Jarque-Bera test indicated residuals were not normally distributed (71.49, 2, p < 0.0001). Model 2 was similar to model 1, but it only included the variables that were found to be statistically significant: average family size, functioning, the percent of admissions greater than age 11, the percent of male admissions, the NERI score, and the squared NERI score. This model showed nonstationarity (14.86, 6, p = 0.0214). Model 2 was also statistically significant and explained 36 percent of the variation in AD-HD admissions (1,475.28, 6, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (63.90, 2, p < 0.0001).
<table>
<thead>
<tr>
<th></th>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>106.42***</td>
<td>248.15***</td>
<td>290.55***</td>
<td>N/A</td>
</tr>
<tr>
<td>Joint Wald</td>
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<td>1,475.28***</td>
<td>1,431.00***</td>
<td>N/A</td>
</tr>
<tr>
<td>Adjusted R²</td>
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<td>0.36</td>
<td>0.35</td>
<td>0.40</td>
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<tr>
<td>AICc</td>
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<td>5,379.71</td>
<td>5,245.32</td>
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<tr>
<td>Jarque-Bera Test</td>
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<td>63.90***</td>
<td>65.02***</td>
<td>N/A</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>33.25**</td>
<td>14.86**</td>
<td>16.54**</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 13. Comparing AD-HD models

Unfortunately, Model 2 would not converge in GWR because of local multicollinearity.

After checking all possible combinations of Model 2’s variables with exploratory regression, the best model that would converge was Model 3. This model included all the variables from Model 2 except for functioning. Like the other models, it showed nonstationarity (16.54, 5, p = 0.0054). Model 3 was also statistically significant and explained 35 percent of the variation in AD-HD admissions (1,431.00, 5, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (65.02, 2, p < 0.0001).

Table 14 displays the OLS results from Model 3. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. Two variables, the percent of male admissions and the NERI score, show positive relationships with AD-HD admissions, while average family size, the percent of admissions greater than age 11, and the squared NERI score show negative relationships with AD-HD admissions. The largest coefficient was found between AD-HD admissions

---

14 *Bandwidth set to 833 neighbors with an adaptive kernel; **p < 0.05; ***p < 0.0001
15 Evaluated through testing different models and examining the multicollinearity condition number
and the NERI score; for each one unit increase in the NERI score, the natural log of admissions for children with AD-HD increases by 0.59, holding all other variables constant. That is, children living in neighborhoods at greater risk of economic distress experience greater admissions for AD-HD.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust* Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-4.57</td>
<td>0.13</td>
<td>-35.25</td>
<td>0.00**</td>
<td></td>
</tr>
<tr>
<td>Average family size</td>
<td>-0.26</td>
<td>0.05</td>
<td>-5.97</td>
<td>0.00**</td>
<td>1.05</td>
</tr>
<tr>
<td>% Admissions &gt; age 11</td>
<td>-0.46</td>
<td>0.08</td>
<td>-5.53</td>
<td>0.00**</td>
<td>1.01</td>
</tr>
<tr>
<td>% Male Admissions</td>
<td>0.53</td>
<td>0.08</td>
<td>6.29</td>
<td>0.00**</td>
<td>1.01</td>
</tr>
<tr>
<td>NERI</td>
<td>0.59</td>
<td>0.02</td>
<td>31.51</td>
<td>0.00**</td>
<td>2.01</td>
</tr>
<tr>
<td>NERI squared</td>
<td>-0.14</td>
<td>0.01</td>
<td>-13.93</td>
<td>0.00**</td>
<td>1.96</td>
</tr>
</tbody>
</table>

Table 14. Model 3 variables for AD-HD admissions

Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account. The adjusted $R^2$ was higher and the AICc was lower than in both Model 2 and Model 3 (Table 13). The Jarque-Bera test in Model 3 indicated that the residuals were not normally distributed, but Figure 17 shows the standardized residuals appear to be unimodal and approximate a normal distribution with mesokurtic peakedness (2.94). There is a slight negative skew, but the skewness number is still close to zero (-0.38).

---

16 *Robust values displayed because of significant Koenker BP statistic; **p < 0.0001
Global Moran’s I revealed the final AD-HD admissions model exhibited positive spatial autocorrelation in GWR residuals ($I = 0.12; z = 8.97, p < 0.0001$). Figure 18 displays the standardized residuals across the state. AD-HD admissions closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. As one would expect from a normal distribution, most of the residuals are located near the center of the distribution and taper off as the standard deviation increases. Clustering appeared most prevalent for census tracts whose residuals fall into the middle of the distribution (-1.5 to 1.5 standard deviations).
Model 4 predicts child admissions well in some areas. The percent of the local variance accounted for by Model 4 ranges from 13.41 percent to 46.07 percent, and Figure 19 highlights the variance explained per census tract. Model 4 best predicts AD-HD admissions (>43%) in urban areas around Ohio (e.g., Akron, Cincinnati, Columbus, and Dayton) and in the suburban communities surrounding these urban areas (e.g., Lorain and Media counties). Interestingly, Model 4 does not predict well in Toledo, and Cuyahoga appears to be bifurcated, with higher $R^2$’s on the west side of the county. The model predicts the poorest (between 13% and 30%) in most rural and Appalachian areas (e.g., northwestern and southeastern counties).
Research Question 1bii

Research question 1bii asks whether there is a spatial relationship between neurotoxicant exposure and pervasive developmental disorder (PDD) admissions at patient intake. OLS regression models helped to develop the most parsimonious model before spatial modeling (Table 15). Model 1’s Koenker BP statistic indicated nonstationarity (80.59, 17, p < 0.0001), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (164.03, 17, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.19 – 246.23). The Jarque-

17 Natural breaks used to generate intervals
Bera test indicated residuals were not normally distributed (2,040.67, 2, p < 0.0001). Model 2 was similar to model 1, but it only included the variables that were found to be statistically significant: average family size, functioning, problem severity, the NERI score, and the squared NERI score. This model showed nonstationarity (22.57, 5, p = 0.0004). Model 2 was also statistically significant and explained three percent of the variation in PDD admissions (111.73, 5, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (2,122.39, 2, p < 0.0001).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>7.20***</td>
<td>18.33***</td>
<td>26.53***</td>
<td>N/A</td>
</tr>
<tr>
<td>Joint Wald</td>
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<td>111.73***</td>
<td>96.10***</td>
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</tr>
<tr>
<td>Adjusted R²</td>
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<td>0.03</td>
<td>0.05</td>
</tr>
<tr>
<td>AICc</td>
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<td>5,358.21</td>
<td>5,041.45</td>
<td>5,017.11</td>
</tr>
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<td>Jarque-Bera test</td>
<td>2,040.67***</td>
<td>2,122.39***</td>
<td>2,159.25***</td>
<td>N/A</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>80.59***</td>
<td>22.57**</td>
<td>11.64**</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 15. Comparing PDD models

Unfortunately, Model 2 would not converge in GWR because of local multicollinearity. After checking all possible combinations of Model 2’s variables with exploratory regression, the best model that would converge was Model 3. This model included all the variables from Model 2 except for functioning and problem severity. Like the other models, it showed nonstationarity (11.64, 3, p = 0.0087). Model 3 was also statistically significant and explained five percent of the variation in PDD admissions.
(96.10, 3, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (2,159.25, 2, p < 0.0001).

Table 16 displays the OLS results from Model 3. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. One variable, the NERI score, shows a positive relationship with PDD admissions, while average family size and the squared NERI score show negative relationships with PDD admissions. The largest coefficient was found between PDD admissions and average family size; for each one unit increase in the average family size, the natural log of admissions for children with PDD decreases by 0.21, holding all other variables constant. That is, children with larger families experience fewer admissions for PDD.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-7.83</td>
<td>0.09</td>
<td>-90.42</td>
<td>0.00**</td>
<td>---</td>
</tr>
<tr>
<td>Average family size</td>
<td>-0.21</td>
<td>0.04</td>
<td>-5.89</td>
<td>0.00**</td>
<td>1.05</td>
</tr>
<tr>
<td>NERI</td>
<td>0.10</td>
<td>0.02</td>
<td>5.78</td>
<td>0.00**</td>
<td>2.01</td>
</tr>
<tr>
<td>NERI squared</td>
<td>-0.04</td>
<td>0.01</td>
<td>-4.27</td>
<td>0.00**</td>
<td>1.96</td>
</tr>
</tbody>
</table>

Table 16. Model 3 variables for PDD admissions

Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account. The adjusted $R^2$ was higher and the AICc was lower than in both Model 2 and Model 3 (Table 15). The Jarque-Bera test in Model 3 indicated that the residuals were not normally distributed. In fact, these

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19 Robust values displayed because of significant Koenker BP statistic; **p < 0.0001
numbers are huge compared to Jarque-Bera results in other research questions. Figure 20 explains the likely cause of these huge numbers; the standardized residuals appear to be bimodal (or exhibiting positive skew depending on how one looks at it). The distribution on the left appears normal with little skew and mesokurtic peakedness, while the distribution on the right has a right skew and platykurtic peakedness.

Figure 20. Distribution of standardized residuals for PDD admissions

Global Moran’s I revealed the final PDD admissions model exhibited no spatial autocorrelation in GWR residuals (I = 0.01; z = 0.67, p = 0.4999). Figure 21 displays the standardized residuals across the state. PDD admissions closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. Even though the Moran’s I did not find statistically significant clustering, there appears to be some minor clustering for residuals for where PDD admissions fell below the sample mean (between -1.5 to -0.5 standard deviations) and also above the sample mean (≥1.5 standard deviations).
Figure 21. GWR standardized residuals for PDD admissions

Model 4 predicts PDD admissions rather poorly in all areas. The percent of the local variance accounted for by Model 4 ranges from zero to 0.90 percent, and Figure 22 highlights the variance explained per census tract. Model 4 has the highest $R^2$'s for PDD admissions ($>0.90\%$) in the southwestern region as well as in Lorain and Medina counties. The model predicts the poorest (between 0.00% and 0.30%) in some urban areas (e.g., Cuyahoga and Lucas counties), suburban areas (e.g., Geauga and Lake counties), and most Appalachian areas.


Research Question 1ci

Research question 1ci asks whether there is a spatial relationship between lead, mercury, or manganese neurotoxicant exposures and AD-HD admissions at patient intake. OLS regression models helped to develop the most parsimonious model before spatial modeling (Table 17). Model 1’s Koenker BP statistic indicated nonstationarity (33.93, 18, p = 0.0128), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (1,811.88, 18, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.06 – 246.42). The Jarque-Bera test indicated residuals were not normally distributed (71.54, 2, p < 0.0001). Model 2 was similar to

---

20 Natural breaks used to generate intervals
model 1, but it only included the variables that were found to be statistically significant: average family size, functioning, the percent of admissions greater than age 11, the percent of male admissions, manganese hazard index (ln), the NERI score, and the squared NERI score. This model showed nonstationarity (16.02, 7, p = 0.0250). Model 2 was also statistically significant and explained 36 percent of the variation in AD-HD admissions (1,487.30, 7, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (63.11, 2, p < 0.0001).

<table>
<thead>
<tr>
<th></th>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>100.89***</td>
<td>213.60***</td>
<td>62.71***</td>
<td>N/A</td>
</tr>
<tr>
<td>Joint Wald</td>
<td>1,811.88***</td>
<td>1,487.30***</td>
<td>225.74***</td>
<td>N/A</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.41</td>
<td>0.36</td>
<td>0.09</td>
<td>0.15</td>
</tr>
<tr>
<td>AICc</td>
<td>5,176.76</td>
<td>5,355.82</td>
<td>6,294.66</td>
<td>4,082.18</td>
</tr>
<tr>
<td>Jarque-Bera test</td>
<td>71.54***</td>
<td>63.11***</td>
<td>34.11***</td>
<td>N/A</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>33.93**</td>
<td>16.02**</td>
<td>36.26***</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 17. Comparing AD-HD models, manganese

Unfortunately, Model 2 would not converge in GWR because of local multicollinearity. After checking all possible combinations of Model 2’s variables with exploratory regression, the best model that would converge with a neurotoxicant variable was Model 3. This model included average family size, the percent of admissions greater than age 11, the percent of male admissions, and manganese hazard quotient (ln). Like the other models, it showed nonstationarity (36.26, 4, p < 0.0001). Model 3 was also

---

21 Bandwidth set to 1,000 neighbors with an adaptive kernel; **p < 0.05, ***p < 0.0001
statistically significant and explained 36 percent of the variation in child problem severity scores (225.74, 4, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (34.11, 2, p < 0.0001).

Table 18 displays the OLS results from Model 3. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. Two variables, the percent of male admissions and the manganese hazard quotient (ln) show positive relationships with AD-HD admissions, while average family size and the percent of admissions greater than age 11 show negative relationships with AD-HD admissions. The largest coefficient was found between AD-HD admissions and the percent of admissions greater than age 11; for each one unit increase in the admissions for children 12 and over, the natural log of AD-HD admissions decreases by 0.56, holding all other variables constant. That is, AD-HD admissions decrease as the proportion of children 12 and over admitted increase.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-3.71</td>
<td>0.16</td>
<td>-23.74</td>
<td>0.00***</td>
<td>-----</td>
</tr>
<tr>
<td>Average family size</td>
<td>-0.50</td>
<td>0.05</td>
<td>-10.09</td>
<td>0.00***</td>
<td>1.02</td>
</tr>
<tr>
<td>% Admissions &gt; age 11</td>
<td>-0.56</td>
<td>0.10</td>
<td>-5.69</td>
<td>0.00***</td>
<td>1.01</td>
</tr>
<tr>
<td>% Male admissions</td>
<td>0.64</td>
<td>0.10</td>
<td>6.41</td>
<td>0.00***</td>
<td>1.01</td>
</tr>
<tr>
<td>Manganese HI</td>
<td>0.11</td>
<td>0.02</td>
<td>5.47</td>
<td>0.00***</td>
<td>1.03</td>
</tr>
</tbody>
</table>

Table 18. Model 3 variables for AD-HD admissions, manganese

---

22 Robust values displayed because of significant Koenker BP statistic; **p < 0.001; ***p < 0.0001
Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account; however, there is a significant caveat. While the AICc was lower than all other models, the $R^2$ was significantly lower than Model 2 (Table 17), which indicates this model is not ideal. The Jarque-Bera test in Model 4 indicated that the residuals were not normally distributed, but Figure 23 shows the standardized residuals to be unimodal and approximate a normal distribution with mesokurtic peakedness (2.85) and skewness that is close to zero (-0.25).

Figure 23. Distribution of standardized residuals for AD-HD admissions, manganese

Global Moran’s I revealed the final AD-HD admissions model exhibited positive spatial autocorrelation in GWR residuals ($I = 0.32; z = 24.70, p < 0.0001$). Figure 24 displays the standardized residuals across the state. AD-HD admissions closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. As one would expect from a normal distribution, most of the residuals are located near the center of the distribution and taper off as the standard deviation increases. Clustering appeared most prevalent in
census tracts whose residuals fall into the middle of the distribution (-1.5 to 1.5 standard deviations).

Figure 24. GWR standardized residuals for AD-HD admissions, manganese

Model 4 predicts AD-HD admissions rather poorly in all areas. The percent of the local variance accounted for by Model 4 ranges from 4.76 percent to 19.07 percent, and Figure 25 highlights the variance explained per census tract. Model 4 best predicts AD-HD admissions (≥17%) in southwestern Ohio; however, there is a caveat here too. Once more, this model is not ideal because the R² results are likely unstable in 808 census tracts grouped together in a giant C-shape across the entire western edge of Ohio because the local multicollinearity condition numbers are above 30 (range 31.00 – 34.62).
model predicts the poorest (between 5% and 10.29%) in many rural and Appalachian areas (e.g., eastern and southeastern counties).

![Figure 25. GWR R² for AD-HD admissions, manganese](image)

**Research Question 1cii**

Research question 1cii asks whether there is a spatial relationship between lead, mercury, or manganese neurotoxicant exposures and PDD admissions at patient intake. The first OLS regression model indicated that neither lead, mercury nor manganese were found to be statistically significantly related to child admissions. Model 1’s Koenker BP statistic indicated nonstationarity (79.89, 18, p < 0.0001), so the Joint-Wald test was used.

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23 Natural breaks used to generate intervals
to judge the significance of the model. Model 1 was statistically significant (166.30, 18, \( p < 0.0001 \)), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.05 – 246.42). The Jarque-Bera test indicated residuals were not normally distributed (2,046.68, 2, \( p < 0.0001 \)). Model 1 came to the exact same conclusion as the previous PDD model in question 1b2, so no further results will be presented for this model; neither lead, mercury, nor manganese hazard indices were statistically significant predictors in PDD admissions.

**Research Question 2a**

Research question 2a asks whether there is a spatial relationship between neurotoxicant exposure and the mental health outcome for hopefulness at patient intake. OLS regression models helped to develop the most parsimonious model before spatial modeling (Table 19). Model 1’s Koenker BP statistic indicated nonstationarity (148.13, 15, \( p < 0.0001 \)), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (1,570.29, 15, \( p < 0.0001 \)), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.22 – 245.95). The Jarque-Bera test indicated residuals were not normally distributed (82.74, 2, \( p < 0.0001 \)). Model 2 was similar to model 1, but it only included the variables that were found to be statistically significant: problem severity, the percent of admissions greater than age 11, the NERI score, percent of minority admissions, and total hazard quotient (ln). This model showed nonstationarity (67.27, 5, \( p < 0.0001 \)). Model 2 was also statistically significant and explained 37 percent of the variation in child hopefulness scores (1,064.47, 5, \( p < 0.0001 \)). The Jarque-Bera test indicated residuals were not normally distributed (83.90, 2, \( p < 0.0001 \)).
<table>
<thead>
<tr>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>143.77**</td>
<td>308.24**</td>
<td>N/A</td>
</tr>
<tr>
<td>Joint Wald</td>
<td>1,570.29**</td>
<td>1,064.47**</td>
<td>N/A</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>0.45</td>
<td>0.37</td>
<td>0.37</td>
</tr>
<tr>
<td>AICc</td>
<td>6,582.37</td>
<td>6,927.84</td>
<td>6,946.34</td>
</tr>
<tr>
<td>Jarque-Bera test</td>
<td>82.74**</td>
<td>83.90**</td>
<td>81.85**</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>148.13**</td>
<td>67.27**</td>
<td>64.82**</td>
</tr>
</tbody>
</table>

Table 19. Comparing hopefulness models\(^2\)

Unfortunately, Model 2 would not converge in GWR because of local multicollinearity. After checking all possible combinations of Model 2’s variables with exploratory regression, the best model that would converge was Model 3. This model included all the variables from Model 2 except for total hazard quotient (ln). Like the other models, it showed nonstationarity (64.82, 4, p < 0.0001). Model 3 was also statistically significant and explained 37 percent of the variation in child hopefulness scores (1,025.53, 4, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (81.85, 2, p < 0.0001).

Table 20 displays the OLS results from Model 3. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. Two variables, problem severity and the percent of admissions greater than age 11, show positive relationships with hopefulness, while the NERI score and the percent of minority admissions show negative relationships with hopefulness. The largest coefficient was found between hopefulness and the percent of admissions greater than

---

\(^2\) Bandwidth set to 375 neighbors with an adaptive kernel; \(*\)*p < 0.0001
age 11; for each one unit increase in the admissions for children 12 and over, hopefulness increases by 0.90, holding all other variables constant. That is, hopefulness scores increase (i.e., a child becomes less hopeful) as the proportion of children 12 and over admitted increase.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>7.11</td>
<td>0.15</td>
<td>45.98</td>
<td>0.00**</td>
<td>---</td>
</tr>
<tr>
<td>Problem Severity</td>
<td>0.15</td>
<td>0.01</td>
<td>30.25</td>
<td>0.00**</td>
<td>1.03</td>
</tr>
<tr>
<td>% Admissions &gt; age 11</td>
<td>0.90</td>
<td>0.12</td>
<td>7.35</td>
<td>0.00**</td>
<td>1.01</td>
</tr>
<tr>
<td>% Minority Admissions</td>
<td>-0.45</td>
<td>0.08</td>
<td>-6.05</td>
<td>0.00**</td>
<td>1.60</td>
</tr>
<tr>
<td>NERI</td>
<td>-0.12</td>
<td>0.02</td>
<td>-5.01</td>
<td>0.00**</td>
<td>1.62</td>
</tr>
</tbody>
</table>

Table 20. Model 3 variables for hopefulness

Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account. The adjusted $R^2$ was higher and the AICc was lower than in both Model 2 and Model 3 (Table 19). The Jarque-Bera test in Model 3 indicated that the residuals were not normally distributed, but Figure 26 shows the standardized residuals to be unimodal and approximate a normal distribution with mesokurtic peakedness (3.88) and skewness that is close to zero (0.07).

---

Robust values displayed because of significant Koenker BP statistic; **p < 0.0001
Global Moran’s I revealed the final hopefulness model exhibited no spatial autocorrelation in GWR residuals (I = 0.01; z = 0.43, p = 0.6666). Figure 27 displays the standardized residuals across the state. Hopefulness scores closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. As one would expect from a normal distribution, most of the residuals are located near the center of the distribution and taper off as the standard deviation increases. Clustering appeared most prevalent in census tracts whose residuals fall into the middle of the distribution (-1.5 to 1.5 standard deviations).
Figure 27. GWR standardized residuals for hopefulness

Model 4 predicts hopefulness scores well in some areas. The percent of the local variance accounted for by Model 4 ranges from 20.02 percent to 55.96 percent, and Figure 28 highlights the variance explained per census tract. Model 4 best predicts hopefulness scores (≥48%) in rural areas (e.g., Allen, Hancock, and Putnam counties) and suburban areas (e.g., Lake and Geauga counties). The model also predicts well (between 40% and 47%) in urban areas (e.g., Akron Cincinnati, and Dayton) and rural areas in northwestern Ohio. The model predicts the poorest (between 20% and 32%) in some urban areas (e.g., central Ohio), suburban areas (e.g., Lorain county), and most Appalachian areas (e.g., eastern and southeastern counties).
Research Question 2b

Research question 2b asks whether there is a spatial relationship between neurotoxicant exposure and the mental health outcome for functioning at patient intake. OLS regression models helped to develop the most parsimonious model before spatial modeling (Table 21). Model 1’s Koenker BP statistic indicated nonstationarity (126.18, 14, p < 0.0001), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (3,262.79, 14, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.06 – 58.92). The Jarque-Bera test indicated residuals were not normally distributed (179.79, 2, p < 0.0001). Model 2 was similar to

\(^{26}\) Natural breaks used to generate intervals
model 1, but it only included the variables that were found to be statistically significant: hopefulness, problem severity, the percent of minority admissions, the percent of admissions greater than age 11, and the solvent hazard quotient (ln). This model showed stationarity (2.84, 5, \( p = 0.7239 \)), so the Joint F-statistic was used to judge the significance of the model. Model 2 was also statistically significant and explained 58 percent of the variation in child functioning scores (\( F_{5,2602}=708.76, p < 0.0001 \)). The Jarque-Bera test indicated residuals were not normally distributed (209.03, 2, \( p < 0.0001 \)).

<table>
<thead>
<tr>
<th></th>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>266.07*</td>
<td>708.76*</td>
</tr>
<tr>
<td>Joint Wald</td>
<td>3,262.79*</td>
<td>3,000.02*</td>
</tr>
<tr>
<td>Adjusted ( R^2 )</td>
<td>0.59</td>
<td>0.58</td>
</tr>
<tr>
<td>AICc</td>
<td>12,320.39</td>
<td>12,383.35</td>
</tr>
<tr>
<td>Jarque-Bera test</td>
<td>179.79*</td>
<td>209.03*</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>126.18*</td>
<td>2.84</td>
</tr>
</tbody>
</table>

Table 21. Comparing functioning models\(^{27}\)

Model 2 was the first model to show stationarity, which indicated that it was not appropriate for geographically weighted regression. The model underwent further refinement because the Jarque-Bera test was significant, but a stable model did not emerge because no combinations of the remaining variable through exploratory regression had a Jarque-Bera statistic that was not significant. Table 22 displays the OLS

\(^{27}\) *\( p < 0.0001 \)
results from Model 2. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. One variable, the percent of admissions greater than age 11, shows a positive relationship with functioning, while hopefulness, problem severity, the percent of minority admissions, and the solvent hazard quotient (ln) show negative relationships with functioning. The largest coefficient was found between functioning and the percent of minority admissions; for each one unit increase in the admissions for minorities, functioning decreases 2.08, holding all other variables constant. That is, functioning scores decrease (i.e., a child becomes less functional) as the proportion of minorities admitted increase.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Std. Error</th>
<th>t-test</th>
<th>Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>69.36</td>
<td>0.89</td>
<td>77.77</td>
<td>0.00*</td>
<td></td>
</tr>
<tr>
<td>Hopefulness</td>
<td>-1.08</td>
<td>0.06</td>
<td>-19.48</td>
<td>0.00*</td>
<td>1.58</td>
</tr>
<tr>
<td>Problem Severity</td>
<td>-0.46</td>
<td>0.01</td>
<td>-31.96</td>
<td>0.00*</td>
<td>1.56</td>
</tr>
<tr>
<td>% Admissions &gt; age 11</td>
<td>1.30</td>
<td>0.30</td>
<td>4.26</td>
<td>0.00*</td>
<td>1.03</td>
</tr>
<tr>
<td>% Minority Admissions</td>
<td>-2.08</td>
<td>0.21</td>
<td>-9.88</td>
<td>0.00*</td>
<td>1.76</td>
</tr>
<tr>
<td>Solvents HQ</td>
<td>-0.52</td>
<td>0.18</td>
<td>-2.96</td>
<td>0.00*</td>
<td>1.76</td>
</tr>
</tbody>
</table>

Table 22. Model 2 variables for functioning

Research Question 2c

Research question 2c asks whether there is a spatial relationship between neurotoxicant exposure and the mental health outcome for problem severity at patient intake. OLS regression models helped to develop the most parsimonious model before

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28 *p < 0.0001
spatial modeling (Table 23). Model 1’s Koenker BP statistic indicated nonstationarity (203.84, 15, p < 0.0001), so the Joint-Wald test was used to judge the significance of the model. Model 1 was statistically significant (2,592.16, 15, p < 0.0001), but the variance accounted for by these factors was artificially inflated by the high multicollinearity among some of the variables (VIF range: 1.23 – 246.31). The Jarque-Bera test indicated residuals were not normally distributed (85.39, 2, p < 0.0001). Model 2 was similar to model 1, but it only included the variables that were found to be statistically significant: hope, average family size, the percent of admissions greater than age 11, the NERI score, the squared NERI score, the percent of minority admissions, and total hazard quotient (ln). This model showed nonstationarity (62.53, 7, p < 0.0001). Model 2 was also statistically significant and explained 37 percent of the variation in child problem severity scores (1,209.41, 7, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (149.54, 2, p < 0.0001).

<table>
<thead>
<tr>
<th>Model 1 OLS</th>
<th>Model 2 OLS</th>
<th>Model 3 OLS</th>
<th>Model 4 OLS</th>
<th>Model 5 GWR*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint F</td>
<td>218.59**</td>
<td>219.17**</td>
<td>255.50**</td>
<td>372.21**</td>
</tr>
<tr>
<td>Joint Wald</td>
<td>2,592.16**</td>
<td>1,209.41**</td>
<td>1,207.84**</td>
<td>1,165.24**</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>0.56</td>
<td>0.37</td>
<td>0.37</td>
<td>0.36</td>
</tr>
<tr>
<td>AICc</td>
<td>13,027.71</td>
<td>13,934.25</td>
<td>13,933.31</td>
<td>13,958.06</td>
</tr>
<tr>
<td>Jarque-Bera test</td>
<td>85.39**</td>
<td>149.54**</td>
<td>61.91**</td>
<td>177.22**</td>
</tr>
<tr>
<td>Koenker (BP) test</td>
<td>203.84**</td>
<td>62.53**</td>
<td>56.31**</td>
<td>50.96**</td>
</tr>
</tbody>
</table>

Table 23. Comparing problem severity models

*Bandwidth set to 850 neighbors with an adaptive kernel; **p < 0.0001
Model 2 underwent further refinement because the variable average family size was no longer significant when other variables were taken out of Model 1. Model 3 contains hopefulness, the percent of admissions greater than age 11, NERI score, the squared NERI score, the percent of minority admissions, and total hazard quotient (ln). Model 3 showed nonstationarity like other models (56.31, 6, p < 0.0001). Model 3 was also statistically significant and explained 37 percent of the variation in child problem severity scores (1,207.84, 6, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (61.91, 2, p < 0.0001).

Unfortunately, Model 3 would not converge in GWR because of local multicollinearity. After checking all possible combinations of Model 3’s variables with exploratory regression, the best model that would converge was Model 4. This model included all the variables from Model 3 except for the percent of minority admissions and total hazard quotient (ln). Like the other models, it showed nonstationarity (50.96, 4, p < 0.0001). Model 4 was also statistically significant and explained 36 percent of the variation in child problem severity scores (1,165.24, 4, p < 0.0001). The Jarque-Bera test indicated residuals were not normally distributed (177.22, 2, p < 0.0001).

Table 24 displays the OLS results from Model 4. Each of the variables in the model is statistically significant, and none of the variables show multicollinearity per the VIF. Two variables, hopefulness and the NERI score, show positive relationships with problem severity, while the percent of admissions greater than age 11 and the squared NERI score show negative relationships with problem severity. The largest coefficient was found between problem severity and the percent of admissions greater than age 11;
for each one unit increase in the admissions for children 12 and over, problem severity decreases by 3.36, holding all other variables constant. That is, problem severity scores decrease (i.e., a child reports fewer problems) as the proportion of children 12 and over admitted increase.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Robust Std. Error</th>
<th>Robust t-test</th>
<th>Robust Probability</th>
<th>VIF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>2.33</td>
<td>0.80</td>
<td>2.91</td>
<td>0.00**</td>
<td>----</td>
</tr>
<tr>
<td>Hopefulness</td>
<td>2.23</td>
<td>0.07</td>
<td>33.04</td>
<td>0.00***</td>
<td>1.02</td>
</tr>
<tr>
<td>% Admissions &gt; age 11</td>
<td>-3.36</td>
<td>0.48</td>
<td>-6.93</td>
<td>0.00***</td>
<td>1.01</td>
</tr>
<tr>
<td>NERI</td>
<td>1.09</td>
<td>0.10</td>
<td>10.85</td>
<td>0.00***</td>
<td>1.91</td>
</tr>
<tr>
<td>NERI squared</td>
<td>-0.22</td>
<td>0.05</td>
<td>-4.07</td>
<td>0.00***</td>
<td>1.90</td>
</tr>
</tbody>
</table>

Table 24. Model 4 variables for problem severity

Results from the GWR indicated that the model performed better than the OLS models once local relationships were taken into account. The adjusted $R^2$ was higher and the AICc was lower than all other models (Table 23). The Jarque-Bera test in Model 4 indicated that the residuals were not normally distributed, but Figure 29 shows the standardized residuals to be unimodal and approximate a normal distribution with mesokurtic peakedness (4.13) and skewness that is close to zero (0.26).

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30 Robust values displayed because of significant Koenker BP statistic; **p < 0.001; ***p < 0.0001
Global Moran’s I revealed the final problem severity model exhibited positive spatial autocorrelation in GWR residuals ($I = 0.04; z = 3.48, p = 0.0005$). Figure 30 displays the standardized residuals across the state. Problem severity scores closest to the sample mean are colored in yellow, while those below the sample mean are in shades of blue and those above the sample mean are in shades of red. As one would expect from a normal distribution, most of the residuals are located near the center of the distribution and taper off as the standard deviation increases. Clustering appeared most prevalent in census tracts whose residuals fall into the middle of the distribution (-1.5 to 1.5 standard deviations).
Model 5 predicts problem severity scores well in some areas. The percent of the local variance accounted for by Model 5 ranges from 22.71 percent to 48.43 percent, and Figure 31 highlights the variance explained per census tract. Model 5 best predicts problem severity scores ($\geq 47\%$) in northwestern and southwestern Ohio. The model predicts the poorest (between 23% and 29%) in some urban areas (e.g., Cuyahoga and Franklin counties) and most Appalachian areas (e.g., eastern and southeastern counties). The $R^2$ results may be unstable in 57 census tracts grouped together in Columbiana, Carroll, Huron, and Jefferson counties because the local multicollinearity condition numbers are slightly above 30 (range 31.00 – 32.02).
Figure 31. GWR $R^2$ for problem severity\textsuperscript{31}

\textsuperscript{31} Natural breaks used to generate intervals
Chapter 5: Discussion

Chapter 5 reviews the findings presented in the previous chapter. The results are presented in order of the research objectives and related to the literature presented in Chapter 2. Implications for the research results are discussed in regards to the governing macro and micro theories, policy, and future researchers. The major limitations are presented along with a brief discussion of how they impacted the study. Finally, the major conclusions from the research study are summarized.

Research Question 1a

Research question 1a investigated the spatial relationship between neurotoxicant exposure and the mental health admissions at patient intake, and results indicated no statistically significant association was found. The total hazard quotient (ln) for neurotoxicants was not statistically significant in the very first OLS model. Even when this variable was stratified into general categories of metal and solvent chemical subtypes, the hazard quotients were still not statistically significant in the first OLS model (data not shown).

Research suggests that neurotoxicants have an impact on mental health based on discrete events (Bromet & Havenaar, 2007; Dhara & Dhara, 2002; Greve et al., 2007), specific chemicals (Bouchard et al., 2007; Brown, 2002; Karagas et al., 2012; Palmer et al., 2006; Windham et al., 2006), and etiological studies (Froehlich et al., 2011; Landrigan, 2010). Moreover, one would expect an association between exposure and
health effects to be apparent because Ohio has consistently ranked in the top-5 emitters of neurotoxicants for over a decade (U.S. EPA., 2009a). While this study did not entirely confirm these findings, it is important to think about how this study differs from the aforementioned studies. This study is unique because it attempted to relate total hazard quotients (ln) for 23 chemicals to child admissions, which has not been heretofore investigated, making this research different compared to other studies. The research questions examined aggregate data at the census tract level and not individual level data, which may show a fundamentally different relationship between neurotoxicants and child admissions. Ideally, there would be similar studies at the census tract level, but the absence of these studies means some ecologic fallacy will be present when comparing findings from the population and individual levels. Also, some of the previously mentioned research used direct measures of exposure (e.g., blood draws) that would yield a more accurate understanding of the exposure instead of the indirect measure of exposure (i.e., hazard quotients/indices) utilized in this study. Finally, most of the studies examined short term effects of exposure; they did not examine exposure that occurred several years before the outcome measure as in this study. These hazard quotients may be very similar to the hazard quotients that existed in 2007, but there is no way to make that determination.

Even though the intent of this research question was to investigate neurotoxicants, a stable model for child admissions emerged from the data. The third OLS model indicated that average family size, NERI score, and the squared NERI score were all excellent predictors of child admissions. The model indicated the NERI score increased as census tract admissions increased, while average family size, and the squared NERI
score decreased as child admissions increased. The GWR model confirmed that there was spatial heterogeneity in the data and that the model predicted heterogeneity better in some areas than others. Overall, the model predicted admissions well in central Ohio and it predicted admissions poorly in most rural and Appalachian areas.

Findings for the child admissions model are moderately supported by previous research. The NERI scale is comprised of several variables that have been found to relate to admissions. Components of the NERI like child poverty and parental education have both been found to be important predictors in previous research (John, Offord, Boyle & Racine, 1995; Farmer et al., 1999; Fryers, Melzer, Jenkins, 2002; Muntaner et al., 2004; USDHHS, 1999). The significance of the NERI is not surprising because over 90 percent of the children submitting claims to ODMH are on Medicaid; in fact, it would have been surprising if it was not significant. The squared component of the NERI score may explain why some studies have reported conflicting results about parental education and wealth. For example, research indicated child admissions were more likely when the parents lived in poverty and also more likely when parents made over $50,000 (Cohen & Hesselbart, 1993; Farmer et al., 1999). Assuming both these findings are valid, they would best be graphed by a nonlinear relationship. In this study, the equation for child admissions has a nonlinear component that indicates the NERI score and average family size have a large role in child admissions, but only up to a point. In fact, when X increases beyond a certain point, this association becomes weaker. Had the Medicaid and non-Medicaid been studied together, it is possible that a similar type of nonlinear relationship would have been found. However in the context of this study, extreme
poverty associated with a high NERI score may lead to parental disorganization and decrease the likelihood of taking the child to be admitted.

Findings from this research did not support studies that found age, gender, and race were predictors of child admissions (Farmer et al., 1999; Zahner & Daskalakis, 1997). These findings may not support other studies because the unit of analysis was the proportion of each group in a census tract and not the individual. None of the unique interaction effects between race and admission were found (Merikangas et al., 2011), although this result may have been an artifact due to fewer racial categories in this study. Study results also contradict previous research findings that found larger average family size is related to child admissions because it indicates just the opposite (USDHHS, 1999); larger family sizes make it less likely for children to be admitted.

*Research Question 1bi*

Research question 1bi investigated the spatial relationship between neurotoxicant exposure and attention deficit-hyperactivity disorder (AD-HD) admissions at patient intake, and results indicated no statistically significant association was found. The total hazard quotient (In) for neurotoxicants was not statistically significant in the very first OLS model. Even when this variable was stratified into general categories of metal and solvent chemical subtypes, the hazard quotients were still not statistically significant in the first OLS model (data not shown).

Research suggests that neurotoxicants have an impact on the etiology of AD-HD (Bouchard et al., 2007; Brown, 2002; Karagas et al., 2012; Froehlich et al., 2011). This research question faces the same limitation as the previous research question. That is, this study examined aggregate data at the census tract level and not individual level data,
relied on indirect measures of exposure to neurotoxicants, and measured exposure that occurred several years before the outcome measure.

Even though the intent of this research question was to investigate neurotoxicants, a stable model for AD-HD admissions emerged from the data. The third OLS model indicated that average family size, the percent of admissions greater than age 11, the percent of male admissions, NERI score, and the squared NERI score were good predictors for the AD-HD admissions. The model indicated the NERI score and the percent of male admissions increased as census tract AD-HD admissions increased, while average family size, the percent of admissions greater than age 11, and the squared NERI score decreased as AD-HD admissions increased. The GWR model confirmed that there was spatial heterogeneity in the data and that the model predicted heterogeneity better in some areas than others. Overall, the model predicted admissions best in central, northern, and southwestern Ohio, and it predicted admissions poorly in most rural and Appalachian areas.

Findings from the AD-HD admissions model are strongly supported by previous research. The NERI scale comprised several variables that have been found to relate to admissions. Components of the NERI like child poverty and parental education have both been found to be important predictors in previous research (John, Offord, Boyle & Racine, 1995; Farmer et al., 1999; Fryers, Melzer, Jenkins, 2002; Muntaner et al., 2004; USDHHS, 1999). Once more, the significance of the NERI is not surprising because over 90 percent of the children submitting claims to ODMH are on Medicaid, and the squared component of the NERI score may explain why some studies have found conflicting findings in regards to admissions in relationship to parental education and wealth (Cohen
& Hesselbart, 1993; Farmer et al., 1999). Unlike the previous research question, this study supports research that indicated gender was an important predictor of admissions. Merikangas et al. (2010) and Merikangas et al. (2011) found that males were more likely to be admitted if they had AD-HD, which is exactly what was found in this study. Present findings also support the notion that younger children are more likely to be admitted, which agree with previous research that indicate younger children were at increased odds for being admitted (Farmer et al., 1999; Zahner & Daskalakis, 1997). Finally, these results may support research that shows there is a latency effect between manganese exposure and onset of health effects. Brown (2002) explained there is one to nine month latency before the onset of illness. While the interval between 2005 NATA data and 2007 ODMH data is greater than nine months, it is possible that parents took time to recognize their child’s illness and take them in for treatment.

Findings from this present study do not support studies which found race was a predictor of child admissions (Zahner & Daskalakis, 1997). Again, this finding may not support other studies because the unit of analysis was the proportion of each group in a census tract and not the individual. None of the unique interaction effects between race and admission were found (Merikangas et al., 2011), although this result may have been an artifact related to fewer racial categories in this study. Study results also contradict previous research findings that found larger average family size is related to child admissions because it indicates just the opposite (USDHHS, 1999); larger family sizes make it less likely for children to be admitted.
Research Question 1bii

Research question 1bii investigated the spatial relationship between neurotoxicant exposure and pervasive developmental disorder (PDD) admissions at patient intake, and results indicated no statistically significant association was found. The total hazard quotient (ln) for neurotoxicants was not statistically significant in the very first OLS model. Even when this variable was stratified into general categories of metal and solvent chemical subtypes, the hazard quotients were still not statistically significant in the first OLS model (data not shown).

Research has not been conducted specifically on PDD admissions, but it has been conducted on components of PDD (e.g., autism), which suggests that neurotoxicants have an impact on the etiology of at least some of the diagnoses within PDD (Brown, 2002; Landrigan, 2010; Palmer et al., 2006; Windham et al., 2006). This research question faces the same limitation as the previous research question. That is, the present study examined aggregate data at the census tract level and not individual level data, relied on indirect measures of exposure to neurotoxicants, and measured exposure that occurred several years before the outcome measure.

In contrast to other admissions models, the final model explained very little of the variation in PDD admissions. The third OLS model indicated that average family size, NERI score, and the squared NERI score were predictors for PDD admissions. The model indicated the NERI score increased as census tract admissions increased, while average family size and the squared NERI score decreased as PDD admissions increased. The GWR model confirmed that there was spatial heterogeneity in the data; however, the overall $R^2$ value indicated this model was still trivial at best because it only accounted for
five percent of the variation in PDD admissions. Overall, the model predicted PDD admissions poorly in all areas of the state.

Findings for the PDD admissions model are partially supported by previous research. Like other admissions variables, components of the NERI like child poverty and parental education have both been found to be important predictors in previous research (John, Offord, Boyle & Racine, 1995; Farmer et al., 1999), and the squared component of the NERI score may explain why some studies have found conflicting findings in regards to admissions in relationship to parental education and wealth (Cohen & Hesselbart, 1993; Farmer et al., 1999). Despite being part of the model, these variables contributed very little to the overall model, which means that more research will be needed to more fully understand predictors of PDD admissions.

Findings from this research do not support studies which found age, gender, and race were predictors of child admissions (Farmer et al., 1999; Zahner & Daskalakis, 1997). These findings may not support other studies because the unit of analysis was the proportion of each group in a census tract and not the individual. None of the unique interaction effects between race and admission were found (Merikangas et al., 2011), although this result may have been an artifact related to fewer racial categories in this study.

Research Question 1ci

Research question 1ci investigated the spatial relationship between lead, mercury, or manganese neurotoxicant exposures and AD-HD admissions at patient intake. Results indicated that only the manganese hazard index (ln) was related to AD-HD admissions. In
fact, the hazard indices (ln) for lead and mercury were not statistically significant in the very first OLS model.

This research question had the only model in which a specific neurotoxicant was found to impact admissions. The second OLS model explained 36 percent of the variation in admissions; however, this model would not converge in GWR due to local multicollinearity. The second model was refined through variable deletion to see whether the manganese hazard index (ln) would remain included in the model. In fact, the manganese hazard index (ln) stayed part of a majority of models tested in exploratory regression, but most of these models also would not converge in GWR. The third OLS model contained the manganese index (ln) quotient along with a majority of the other variables; however, it only explained nine percent of the variation in AD-HD admissions. Variables like average family size, the percent of admissions greater than age 11, the percent of male admissions, NERI score, and the squared NERI score, and the manganese hazard index (ln) remained in the model, but they were weak predictors of AD-HD admissions. The model indicated the manganese hazard index (ln) and the percent of male admissions increased as census tract AD-HD admissions increased, while average family size and the percent of admissions greater than age 11 decreased as AD-HD admissions increased. The sign of the manganese hazard index (ln) was positive in some of the exploratory regression models and negative in others, so this model must be interpreted with caution. For example, in the second OLS model the sign was negative, indicating that the manganese hazard index (ln) decreased as admissions increased; whereas, there was an opposite trend in model three. The GWR model confirmed that there was spatial heterogeneity in the data and that the model predicted heterogeneity.
better in some areas than others. Overall, the model predicted admissions fairly well in southwestern Ohio, and it predicted admissions poorly in most other areas. This model must also be interpreted with caution because the local multicollinearity coefficients were higher than normal in almost one-third of western Ohio’s census tracts.

Findings for the AD-HD admissions model with manganese are supported by previous research. Model two contained similar variables as those in research question 1bi, so the same conclusions hold true. That is, components of the NERI like child poverty and parental education were important predictors in previous research (John, Offord, Boyle & Racine, 1995; Farmer et al., 1999; Fryers, Melzer, Jenkins, 2002; Muntaner et al., 2004; USDHHS, 1999), and demographic variables like age and gender were important predictors in previous research (Farmer et al., 1999; Zahner & Daskalakis, 1997; Merikangas et al., 2010; Merikangas et al., 2011). Model three takes away the NERI at the expense of the percentage of variance explained, but it allows the manganese hazard index (ln) to remain part of the model and converge in GWR. Results from this finding are supported in the literature (Brown, 2002; Farias et al., 2010; Landrigan, 2010), where researchers have found increases in AD-HD admissions with manganese exposure. This research question faces the same limitation as the previous research question. That is, this study examined aggregate data at the census tract level and not individual level data, relied on indirect measures of exposure to neurotoxicants, and measured exposure that occurred several years before the outcome measure. Other conclusions from this model are the same as the conclusions reached in research question 1bi, and will not be restated here.
Research Question 1cii

Research question 1cii investigated the spatial relationship between lead, mercury, or manganese neurotoxicant exposures and PDD admissions at patient intake. Results indicated none of these chemicals were individually related to PDD admissions. In fact, the hazard indices for each of the neurotoxicants were not statistically significant in the very first OLS model. Findings for the PDD admissions model are not supported by previous research. Other researchers have found that specific neurotoxicants like lead, manganese, and mercury are related to the development of autism (Brown, 2002; Landrigan, 2010; Palmer et al., 2006). This research question faces the same limitation as the previous research question. That is, this study examined aggregate data at the census tract level and not individual level data, relied on indirect measures of exposure to neurotoxicants, and measured exposure that occurred several years before the outcome measure. Other conclusions from this model are the same as the conclusions reached in research question 1bii and will not be restated here.

Research Question 2a

Research question 2a investigated the spatial relationship between the total hazard quotient for neurotoxicants (ln) and child hopefulness on the Ohio Scales instrument at client intake. Results indicated total hazard quotient for neurotoxicants (ln) was related to child hopefulness. These results still held true even if the variable was stratified into general categories of metal and solvent chemical subtypes (data not shown).

Previous research suggests that child functioning and problem severity are related to hopefulness (Hoagwood et al., 1996). The present study was the first to test whether other variables were also related to child hopefulness at the census tract level. OLS model
two found that problem severity and the percent of admissions greater than age 11 had positive relationships with hopefulness. As children in a census tract become less hopeful, they experience more problem severity and experience more admissions for children older than 11.\textsuperscript{32} Hopefulness had negative relationships with the percent of minority admissions, NERI score, and the total hazard quotient (ln). As children in a census tract became more hopeful, they are less likely to be a minority, less likely to have a high NERI score, and less likely to live in an area with a high total hazard quotient (ln).

Unfortunately, this model would not converge in GWR, so the model was refined with exploratory regression. After removing the total hazard quotient (ln), the model would converge with only a loss of one percent of the variation explained in child hopefulness. The GWR model confirmed that there was spatial heterogeneity in the data and that the model predicted heterogeneity better in some areas than others. Overall, the model predicted hopefulness scores well in some urban areas (e.g., Akron, Cincinnati, and Dayton) and rural areas in northwestern Ohio. The model predicted the poorest in other urban areas (e.g., central Ohio), suburban areas (e.g., Lorain county), and most Appalachian areas (e.g., eastern and southeastern counties).

Findings for the hopefulness model are moderately supported by previous research. Hopefulness was associated with problem severity in the expected direction (Hoagwood et al., 1996). Hopefulness also was associated with functioning in the expected direction; however, this association was not statistically significant as expected (Hoagwood et al., 1996). This finding may be related to the unit of analysis; this study examined census tracts, while previous research has investigated research questions at the

\textsuperscript{32} Again, an increase in the hopefulness score actually means the child becomes less hopeful.
individual level. Many of the predictors that were found to be related to child admissions were also related to hopefulness scores, which should expand the literature for measuring mental health outcomes at assessment.

Research Question 2b

Research question 2b investigated the spatial relationship between the hazard quotients for metals (ln) and solvents (ln) and child functioning on the Ohio Scales instrument at client intake. Results indicated the solvent hazard quotient (ln) was related to child functioning. These results did not hold true if the variable was combined into a total hazard quotient variable (data not shown).

Previous research suggests that child hopefulness and problem severity are related to functioning (Hoagwood et al., 1996). The present study was the first to test whether other variables were also related to child functioning at the census tract level. OLS model two found that the percent of admissions for children older than 11 had a positive relationship with functioning. As the children in a census tract experience greater functioning, the percent of children admitted older than 11 also increase.33 Functioning had negative relationships with hopefulness, problem severity, the percent of minority admissions, and the solvent hazard quotient (ln). As children in a census tract experienced poorer levels of functioning, they were likely to be less hopeful, more likely to have higher levels of problem severity, more likely to be of minority status, and more likely to have higher solvent hazard quotients (ln). This model turned out not to be appropriate for GWR because the Koenker BP statistic indicated stationarity. Even though this model explained a high percentage of the variance, it must be interpreted with

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33 Again, an increase in the functioning score means the child has better functioning.
caution because the model’s residuals were not normally distributed. Further study is needed and will hopefully result in a model with normally distributed residuals.

Findings for the functioning model are highly supported by previous research. Functioning was associated with hopefulness and problem severity in the expected directions (Hoagwood et al., 1996). Many of the predictors that were found to be related to child admissions were also related to functioning scores, which should expand the literature for measuring mental health outcomes at assessment.

*Research Question 2c*

Research question 2c investigated the spatial relationship between the total hazard quotient (ln) and child problem severity on the Ohio Scales instrument at client intake. Results indicated total hazard quotient (ln) was related to child problem severity. These results did not hold true if the variable was stratified into general categories of metal and solvent chemical subtypes (data not shown).

Research suggests that child hopefulness and functioning are related to problem severity (Hoagwood et al., 1996). The present study was the first to test whether other variables were also related to child problem severity at the census tract level. OLS model two found that hopefulness, the percent of minority admissions, NERI score, and the total hazard quotient (ln) had positive relationships with problem severity. As the children in a census tract experienced more problems, those children exhibited less hopefulness, had a greater likelihood for minority admissions, displayed a higher NERI score, and lived in an area with a higher total hazard quotient (ln).[^34] Problem severity had negative relationships with the percent of admissions for children older than 11 and the squared

[^34]: Again, an increase in the problem severity score means the child has more problems.
NERI score. As the children in a census tract experienced more problems, those children were less likely to have admissions for children older than 11.

Unfortunately, this model would not converge in GWR, so the model was refined with exploratory regression. After removing the total hazard quotient (ln) and the percent of minority admissions, the model would converge with only a loss of one the percent of the variation explained in the child problem severity. The GWR model confirmed that there was spatial heterogeneity in the data and that the model predicted heterogeneity better in some areas than others. Overall, the model predicted problem severity scores well in northwestern and southwestern Ohio. The model predicted the poorest in some urban areas (e.g., Cuyahoga and Franklin counties) and most Appalachian areas (e.g., eastern and southeastern counties).

Findings for the problem severity model were moderately supported by previous research. Problem severity was associated with hopefulness in the expected direction (Hoagwood et al., 1996). Problem severity also was associated with functioning in the expected direction; however, this association was not statistically significant as expected (Hoagwood et al., 1996). This finding may be related to the unit of analysis; this study focused on the census tract level, while previous research has investigated research questions at the individual level. Many of the predictors that turned out related to child admissions were also related to problem severity scores, which should expand the literature for measuring mental health outcomes at assessment.

Implications for Theory

Macro and micro theories guided this research effort. Deep ecology helped to understand humanity’s interconnectedness with the environment and presented a
worldview that suggested harm to one part of the system touches all parts of the system (Naess, 1973; Van Wormer, Besthorn, & Keefe, 2007). The micro theory about children’s increased vulnerability also illuminated the relationship between children and environmental toxicants. Children’s unique characteristics like differential intake of food and water, rapid growth and development, and their distinctive means of absorption, metabolism, and excretion of substances contribute to the greater exposure they have to chemicals (Bearer, 1995; Galvez, Forman, & Landrigan, 2005; Landrigan & Garg, 2002; Mott et al., 1997; NRC, 1993).

Results from this study indicated that the neurotoxicant hazard quotients were statistically significant in some OLS models; however, these models would not converge due to local multicollinearity. For example, hopefulness and problems severity models both included the total hazard quotient (ln) in model two, but the variable was taken out in successive models, allowing the GWR model to converge. The solvent hazard quotient (ln) stayed in the final model for functioning, and the manganese hazard index (ln) stayed in the final model for AD-HD admissions. However, even these models were less than ideal because the OLS residuals were non-normally distributed in one case and the variance explained decreased significantly in the other case. Regardless, results should be interpreted with caution until further research is done to refine and stabilize these models.

Findings provide tentative support for the macro and micro theories governing this research study. A child’s functioning at assessment may indeed be impacted by solvent neurotoxicants, just as AD-HD admissions may really be influenced by manganese emissions. Should these relationships be validated in future research efforts, then the theories can be refined to include these relationships. Deep ecology could be
expanded to show that while all organisms are interconnected, some bear a disproportionate share of the toxic burden. The micro theory of children’s increased vulnerability also would have additional evidence, and it could be refined to show that children’s functioning levels are impacted and the incidence of mental illness increases with greater neurotoxicant emissions.

Implications for Policy

Despite their tentative nature, the findings lend themselves to policy implications. Results from this study may challenge federal and state EPA officials to critically examine existing regulation and consider mandating new reporting requirements for industries that release the toxicants. At present, databases that contribute information to NATA like the TRI are not required to measure all toxicants (U.S. EPA, 2009d; Dolinoy & Miranda, 2004). This knowledge gap limits Ohio citizen’s awareness about their total exposure to neurotoxicants; thereby also limiting the facts that may otherwise spur citizens to action. New reporting requirements could compel organizations to be transparent about pollution release and give citizens the knowledge to understand whether the chemicals are harmfully impacting their health and wellness.

Should the relationship between manganese and AD-HD admissions continue to be significant in other studies, then policy makers may want to consider screening children for manganese poisoning as a regular part of the intake process. People with iron deficiency are at greatest risk for manganese poisoning because of the chemical relationship between iron and manganese (Liu, Goyer, & Waalkes, 2008), so there is a medical precedent for a vulnerable population. Manganism can be treated by medical
physicians, but its effects may still last a lifetime and require ongoing psychiatric treatment (Brown, 2002).

**Implications for Future Research**

This study has several important implications for future researchers. The findings suggest neurotoxicants may play a role in child AD-HD admissions and child functioning scores. Future researchers could focus on smaller geographic areas (e.g., counties or ADAMHS boards) instead of a state, which may take care of issues involving local multicollinearity and GWR model convergence; however, the tradeoff would mean that any resulting models are only applicable to that geographic unit. The difficulty seen in GWR model convergence likely means that different parts of the state have different models, and the models represented in this research only apply to the state level. It is quite possible that neurotoxicants play a role in some communities more so than others. Researchers should be careful when they select communities potentially impacted by neurotoxicants and base their decisions on theory and prior research so they are not cherry picking their results. For example, one could look at the communities with the highest r-squared values for AD-HD and manganese and “prove” that a relationship exists between these variables. Given results from this study, researchers should acknowledge the modifiable areal unit problem and be especially careful to point out that there could be different models for different communities.

Researchers could also use different statistical tests like spatial dependence modeling and hierarchical linear modeling (HLM) to investigate the variables with other techniques. Spatial dependence modeling is another form of spatial regression, and it tests whether the outcomes at different locations are dependent upon the location. Spatial
dependence may enter a regression model through a dependent lagged term or through assuming errors are correlated. In spatially lagged models, the dependent variable is lagged and related to a group of explanatory variables, with the goal of discovering whether the outcomes in a geographic unit are influenced by the outcomes of neighboring units; whereas, spatial error models treat error as a problem and attempt to remove it (Ward, & Skrede-Gleditsch, 2008). Future researchers could examine child admissions and mental health assessment scores in the context of spatial dependence to discover whether the outcomes of the model depend on location. Should these models be significant, than they would further develop the literature on factors that influence mental health admissions and assessment scores in a spatial context.

HLM would be an interesting technique to use with this data because of its multilevel nature. In one model proposed in Appendix G, child assessment scores at client intake are seen as nested within clinical evaluator (i.e., case manager, parent, and youth), then nested within census tract, and nested again within county. NATA hazard quotients are primary predictors at the census tract level, but other predictors could be added as well. Independent variables at the county level could include NATA hazard quotients and any funds provided to residents from county commissioners or levies. The third tier variable, county, is then crossed with agency and Alcohol, Drug Addiction, and Mental Health Services (ADAMHS) Board because a client may not receive services from agencies in their county or board area. Independent variables like an agency’s budget, the number of full time employees, and the number of evidence based practices could all be predictors of children’s assessment scores at the agency level. At the ADAMHS board level, independent variables like budget, levy dollars, and leadership
style could also be important predictors. This would be a pertinent model to test, but there is only one problem: the mathematics behind it do not exist. Even one of the most advanced HLM software platforms, HLM7, can only handle spatial dependence for a limited number of models. Conversations with a program developer also indicated the random coefficients were not working within the two-level model, so it will be some time before this model could be tested.

More information needs to be collected about the recovery from mental illness for people affected by neurotoxicant pollution. It is quite possible that the treatment and recovery process would be different for people not affected by neurotoxicant pollution. On one hand it could be easier if those affected could be detoxified with medication and any psychiatric impacts were short-term in nature. On the other hand, it could be much more difficult if the chemicals produced lasting psychiatric conditions. Long-term exposure could in fact make the reoccurrence of mental illness much more likely, but only additional research would either prove or disprove this conjecture.

Researchers could conduct similar research on children who are not on Medicaid or who are on both private and public insurance. Results from this study are based on children who are typically on Medicaid (>90%). The models that predict admissions and assessment scores may be the same as the ones found in this study, or they could be very different. The index that was a combination of poverty factors, NERI, might still be an important predictor once children with other payer types are included, but there is no way of knowing without further research.

Finally, state and federal governments should encourage more basic research on chemicals emitted by industry. Most of the chemicals on the Environmental Defense
Fund list that cause harm to the nervous system are “suspected” neurotoxicants and not “recognized” neurotoxicants. Scientists in academia and industries may be motivated by the findings to strengthen the knowledge about the degree of chemical toxicity as well as explore the toxicity of new chemicals coming into the market. Other important areas for research would involve understanding the short and long-term implications for exposure to neurotoxicants.

**Limitations**

As with every study, this research had several important limitations. NATA risk estimates reflect an indirect measure of exposure and cannot specify the amount of a chemical in a child’s brain or nervous system. Moreover the IFC International (2011) has cautioned these indirect estimates are most precise at larger geographic levels (e.g., the county level), which makes census tract estimates less stable than estimates for larger areas. Despite these limitations, NATA hazard quotients still remain the best risk estimates at the census tract level. Aside from the decades of research that have gone into the refinement of the NATA model, it is unquestionably the most comprehensive exposure model because it calculates risk from various sources down to various endpoints.

Another important source of neurotoxicant exposure that cannot be modeled was cigarette smoking. Children and adolescents completing the Ohio Scales may smoke themselves, but these children would only have been excluded if they qualified for a diagnosis of nicotine abuse or dependence. Even if they did not personally smoke, parents or friends of the youth may have smoked, leading them to be exposed to second-hand smoke. The presence of either of these conditions may have led to a biased
estimation of the relationship between neurotoxicant hazard quotients/indices and mental health admissions and/or assessment scores because this factor was not controlled in the analyses.

An unanticipated limitation was a problem with the MACSIS address records. MACSIS address records were retrieved on September 29, 2010, but they were (theoretically) updated on an ongoing basis. Unfortunately, ODMH did not preserve old addresses in their data warehouse, so these addresses might not have reflected where the child lived during the 2007 calendar year. Fortunately, ODMH staff were able to find one variable that preserved the county of admission for each admission record. Very few admissions records indicated the children moved out of the county. Of the 14,738 admissions records that indicated any move, most children only moved to one (41.5%), two (32.1%) or three (26.1%) other counties, and fewer still moved to between four and seven counties (0.33%). With this ODMH variable, the time of the move could be identified, and admissions records were kept if they corresponded to the county of residence (6,278), while other addresses were deleted (8,460). After the MACSIS and address databases were joined, the removal of bad addresses resulted in the deletion of 170 admissions records in 2007. This relatively small number meant that some of those moves corresponded to admissions from inapplicable years, inapplicable child ages, etc. This particular issue meant MACSIS addresses were most correct at the county level and that some measurement error would have been be part of the analyses. Ideally, I would have received these addresses earlier, but ODMH wanted the official approval from the proposal committee, the OSU IRB, their legal staff, and their executive team before they
would give me the data. While regrettable, their request was logical since I was requesting fully identified data from a protected population.

Finally, the generalizability of the findings was another limitation of the study. Like all geographic research, this study was subject to the modifiable areal unit problem. The results may have been different if the Ohio Scales assessment scores and admissions data were aggregated into different spatial units. For example, total hazard quotients (In) for neurotoxicants may have been related to child admissions at the zip code level or the county, but not at the census tract level. The generalizability of the findings was also limited to interpretation at the census tract level and not the individual level, lest one commit the ecologic fallacy. For example, the conclusions that lead and mercury were not associated with an increase in AD-HD admissions only held true for the census tract level. It was unclear whether those same findings would hold true if individual records were analyzed, but this study cannot make a judgment either way. Finally, the generalizability was limited because participants were child and adolescent consumers who are typically on Medicaid. The findings will only apply to that group and further research will be warranted to see whether findings are applicable to other groups.

Conclusion

This ecologic study investigated spatial relationships between neurotoxicants and mental illness. Theories related to deep ecology and children’s increased vulnerability informed the research questions and suggested that children could be negatively impacted by neurotoxicants. Two basic questions guided the study: 1) How do neurotoxicant hazard quotients/indices impact child admissions, and 2) How do neurotoxicant hazard quotients/indices impact mental health assessment scores? Data from the Ohio
Department of Mental Health and the National-scale Air Toxics Assessment were united and modeled with several geographic information systems programs to answer a series of concrete research questions that led the investigation.

Models for the first research question typically explained a large percentage of the variance in child admissions; however, only one of these models indicated the importance of neurotoxicants. Overall child admissions were best explained by average family size, the NERI score, and the squared NERI score. AD-HD admissions were best explained by average family size, the percent of admissions for children older than age 11, the percent of minority admissions, the NERI score, and the squared NERI score. Variables that explained PDD admissions were average family size, the NERI score, and the squared NERI score; however, this model explained very little of the variation in admissions. Once individual compounds were taken into account, AD-HD admissions were best explained by average family size, the percent of admissions for children older than age 11, the percent of minority admissions, the NERI score, the squared NERI score, and the manganese hazard index (ln). Unfortunately, this model would not converge in GWR due to local multicollinearity without excluding the NERI score and the squared NERI score. The PDD admissions model was no different when individual hazard indices for metal compounds were taken into account.

Models for the second research question typically explained a moderate percentage of the variance in mental health assessment scores; however, only one of the GWR models suggested indicated the importance of neurotoxicants. Child hopefulness scores were best explained by problem severity, the percent of admissions greater than age 11, the NERI score, the percent of minority admissions, and total hazard quotient.
(ln). Unfortunately, the total hazard quotient (ln) had to be removed for the model to converge in GWR. Child functioning scores were best explained by hopefulness, problem severity, the percent of minority admissions, the percent of admissions greater than age 11, and the solvent hazard quotient (ln). Child problem severity scores were best explained by hopefulness, average family size, the percent of admissions greater than age 11, the NERI score, the squared NERI score, the percent of minority admissions, and total hazard quotient (ln). Unfortunately, variables related to average family size, the percent of minority admissions, and the total hazard quotient (ln) had to be taken out for the model to converge in GWR.

Findings from this study suggest neurotoxicants may play a role in child AD-HD admissions and child functioning scores. More research needs to be conducted to validate these findings because of several problems with some OLS and GWR models. Future researchers could focus on smaller geographic areas instead of the state, which may take care of issues involving local multicollinearity and GWR model convergence. Researchers could also use different statistical tests like spatial dependence modeling and HLM to investigate the variables with other techniques. Finally, researchers could conduct similar research on children who are not on Medicaid, and they could look at direct exposure to neurotoxicants.

Where these results take future researchers is only limited by their imagination. This study did not find some of the suspected relationships with neurotoxicants, yet it still found interesting results for models dealing with child admissions and outcomes at assessment. Hopefully, researchers can use this study to refine their own thinking about admissions and outcomes paradigms. Perhaps they will find a relationship between these
domains and neurotoxicants or perhaps not. Whether a set of research questions is validated is not as important as the search for the truth.
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Appendix A: 200 Neurotoxicants Compiled by the Environmental Defense Fund
1,1-dichloro-1-fluoroethane
1,1-dimethyl hydrazine
1,1,1-trichloroethane
1,1,1,2-tetrachloroethane
1,1,2-trichloroethane
1,1,2,2-tetrachloroethane
1,2-dibromo-3-chloropropane
1,2-dibromoethane
1,2-dichlorobenzene
1,2-dichloroethane
1,2-dichloroethylene
1,2-dichloropropene
1,2,3-trichloropropane
1,2,4-trichlorobenzene
1,2,4-trimethylbenzene
1,3-butadiene
1,3-dichloropropylene
1,4-dichloro-2-butene
1,4-dichlorobenzene
1,4-dioxane
2-ethoxyethanol
2-mercaptobenzothiazole
2-methoxyethanol
2-methylpyridine
2-nitropropane
2,4-d
2,4-d 2-ethylhexyl ester
2,4-d butoxyethyl ester
2,4-dinitrotoluene
2,6-dinitrotoluene
3-iodo-2-propynyl butylcarbamate
4-nitrophenol
4,4'-isopropylidenediphenol
4,4'-methylenebis(2-chloroaniline)
Acetaldehyde
Acetonitrile
Acrolein
Acrylamide
Acrylonitrile
Allyl alcohol
Allyl chloride
Aluminum (fume or dust)
Aluminum oxide (fibrous forms)
Ammonia
Aniline
Antimony
Arsenic
Arsenic compounds
Atrazine
Barium
Benzal chloride
Benzene
Benzidine
Benzoic trichloride
Benzyl chloride
Bifenthrin
Biphenyl
Bis(2-chloroethyl) ether
Bromine
Bromochlorodifluoromethane
Bromomethane
Bromotrifluoromethane
Cadmium
Carbaryl
Carbon disulfide
Carbon tetrachloride
Carbonyl sulfide
Catechol
Certain glycol ethers
Chlordane
Chlorine
Chlorobenzene
Chlorodifluoromethane
Chloroethane
Chloroform
Chloromethane
Chloropicrin
Chloroprene
Chlorpyrifos methyl
Cobalt
Creosote
Cresol (mixed isomers)
Cumene
Cyanide compounds
Cyclohexane
Cyclohexanol
Cyfluthrin
Diazinon
Dibutyl phthalate
Dichlorodifluoromethane
Dichloromethane
Dichlorotetrafluoroethane (cfc-114)
Dicyclopentadiene
Diethanolamine
Dimethyl phthalate
Dimethyl sulfate
Dimethylamine
Diphenylamine
Epichlorohydrin
Ethyl acrylate
Ethylbenzene
Ethylene
Ethylene glycol
Ethylene oxide
Ethyleneimine
Ethylidene dichloride
Formaldehyde
Formic acid
Freon 113
Heptachlor
Hexachloro-1,3-butadiene
Hexachlorobenzene
Hexachlorocyclopentadiene
Hexachloroethane
Hexachlorophene
Hydrazine
Hydrogen cyanide
Hydrogen fluoride
Hydroquinone
Lead
Lead compounds
Lindane
Lithium carbonate
M-cresol
M-xylene
Malathion
Malononitrile
Manganese
Manganese compounds
Mercury
Mercury compounds
Methacrylonitrile
Methanol
Methoxychlor
Methyl acrylate
Methyl iodide
Methyl isobutyl ketone
Methyl methacrylate
Methyl tert-butyl ether
Methylene bromide
Molybdenum trioxide
N-butyl alcohol
N-hexane
N-methyl-2-pyrrolidone
N-methylolacrylamide
N-nitroso-n-ethylurea
N,n-dimethylformamide
Naphthalene
Nickel
Nicotine and salts
Nitrobenzene
O-cresol
O-toluidine
O-xylene
Ozone
P-cresol
P-phenylenediamine
P-xylene
Paraldehyde
Pentachlorobenzene
Pentachloroethane
Pentachlorophenol
Permethrin
Phenol
Phenothrin
Phosphorus (yellow or white)
Phthalic anhydride
Polychlorinated biphenyls
Propane sultone
Propargyl alcohol
Propionaldehyde
Propoxur
Propylene oxide
Pyridine
Quinoline
Quinone
Safrole
Sec-butyl alcohol
Selenium
Selenium compounds
Sodium dimethyldithiocarbamate
Sodium nitrite
Strychnine and salts
Styrene
Tert-butyl alcohol
Tetrachloroethylene
Thallium compounds
Thiram
Toluene
Toluene diisocyanate (mixed isomers)
Toluene-2,4-diisocyanate
Toxaphene
Trichloroethylene
Trichlorofluoromethane
Triethylamine
Tris(2,3-dibromopropyl) phosphate
Vinyl acetate
Vinyl chloride
Vinylidene chloride
Xylene (mixed isomers)
Appendix B: Conceptual Model for NATA
Blue boxes indicate elements included in the 2005 NATA; clear boxes indicate elements that could be included in future assessments. In the “Sources” included here, “Major stationary” includes both major and area sources as defined for regulatory purposes in the Clean Air Act. “Non-point” refers to smaller (and sometimes less discrete) sources that are typically estimated on a top-down basis (e.g., by county). Additional explanation of source types included in NATA is presented in Section 2. DPM refers to diesel particulate matter. PBTs refers to chemicals that are persistent, bioaccumulative, and toxic. HQ and HI refer to hazard quotient and hazard index, respectively.

Figure 32. Conceptual model for NATA
Appendix C: Ohio Youth Problem and Functioning Scales
**Ohio Mental Health Consumer Outcomes System**  
**Ohio Youth Problem, Functioning, and Satisfaction Scales**  
Youth Rating – Short Form (Ages 12-18)

<table>
<thead>
<tr>
<th>Instructions: Please rate the degree to which you have experienced the following problems in the past 30 days.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Score</strong></td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>1. Arguing with others</td>
</tr>
<tr>
<td>2. Getting into fights</td>
</tr>
<tr>
<td>3. Yelling, swearing, or screaming at others</td>
</tr>
<tr>
<td>4. Fits of anger</td>
</tr>
<tr>
<td>5. Refusing to do things teachers or parents ask</td>
</tr>
<tr>
<td>6. Causing trouble for no reason</td>
</tr>
<tr>
<td>7. Using drugs or alcohol</td>
</tr>
<tr>
<td>8. Breaking rules or breaking the law (out past curfew, stealing)</td>
</tr>
<tr>
<td>9. Skipping school or classes</td>
</tr>
<tr>
<td>10. Lying</td>
</tr>
<tr>
<td>11. Can’t seem to sit still, having too much energy</td>
</tr>
<tr>
<td>12. Hurting self (cutting or scratching self, taking pills)</td>
</tr>
<tr>
<td>13. Talking or thinking about death</td>
</tr>
<tr>
<td>14. Feeling worthless or useless</td>
</tr>
<tr>
<td>15. Feeling lonely and having no friends</td>
</tr>
<tr>
<td>16. Feeling anxious or fearful</td>
</tr>
<tr>
<td>17. Worrying that something bad is going to happen</td>
</tr>
<tr>
<td>18. Feeling sad or depressed</td>
</tr>
<tr>
<td>19. Nightmares</td>
</tr>
<tr>
<td>20. Eating problems</td>
</tr>
</tbody>
</table>

(Add ratings together) Total ______
### Instructions:

Please circle your response to each question.

1. Overall, how satisfied are you with your life right now?
   1. Extremely satisfied
   2. Moderately satisfied
   3. Somewhat satisfied
   4. Somewhat dissatisfied
   5. Moderately dissatisfied
   6. Extremely dissatisfied

2. How energetic and healthy do you feel right now?
   1. Extremely healthy
   2. Moderately healthy
   3. Somewhat healthy
   4. Somewhat unhealthy
   5. Moderately unhealthy
   6. Extremely unhealthy

3. How much stress or pressure is in your life right now?
   1. Very little stress
   2. Some stress
   3. Quite a bit of stress
   4. A moderate amount of stress
   5. A great deal of stress
   6. Unbearable amounts of stress

4. How optimistic are you about the future?
   1. The future looks very bright
   2. The future looks somewhat bright
   3. The future looks OK
   4. The future looks both good and bad
   5. The future looks bad
   6. The future looks very bad

---

**Total:**

---

### Instructions:

Below are some ways your problems might get in the way of your ability to do everyday activities. Read each item and circle the number that best describes your current situation.

<table>
<thead>
<tr>
<th>Item</th>
<th>Extreme Trouble</th>
<th>Quite a Few Troubles</th>
<th>Some Trouble</th>
<th>OK</th>
<th>Doing Very Well</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Getting along with friends</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. Getting along with family</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. Dating or developing relationships with boyfriends or girlfriends</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. Getting along with adults outside the family (teachers, principal)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. Keeping neat and clean, looking good</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. Caring for health needs and keeping good health habits (tak...</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. Controlling emotions and staying out of trouble</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. Being motivated and finishing projects</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. Participating in hobbies (baseball cards, coins, stamps, art)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. Participating in recreational activities (sports, swimming, bike riding)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. Completing household chores (cleaning room, other chores)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. Attending school and getting passing grades in school</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. Learning skills that will be useful for future jobs</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. Feeling good about self</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. Thinking clearly and making good decisions</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. Concentrating, paying attention, and completing tasks</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. Earning money and learning how to use money wisely</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. Doing things without supervision or restrictions</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. Accepting responsibility for actions</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. Ability to express feelings</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

(Add ratings together) **Total:**

---

---
Appendix D: SQL Code to Parse Outcomes Database
CREATE TABLE `massatti_original` (  `CODE2` varchar(50) default NULL,  `AGEATASSESSMENT` varchar(50) default NULL,  `GENDER` varchar(50) default NULL,  `RACEW` varchar(50) default NULL,  `RACEN` varchar(50) default NULL,  `RACEB` varchar(50) default NULL,  `RACEH` varchar(50) default NULL,  `RACEA` varchar(50) default NULL,  `RACEO` varchar(50) default NULL,  `RACEU` varchar(50) default NULL,  `ADMISSDT` varchar(50) default NULL,  `AGEINTAKE` varchar(50) default NULL,  `YEARINTAKE` varchar(50) default NULL,  `DIAGTYPE` varchar(50) default NULL,  `PDIAG` varchar(50) default NULL,  `SDIAG` varchar(50) default NULL,  `HOPE` varchar(50) default NULL,  `FUNC` varchar(50) default NULL,  `SATIS` varchar(50) default NULL,  `PSERVER` varchar(50) default NULL,  `PRIMARYLAST` varchar(50) default NULL,  `AUTO_ID` int(11) NOT NULL auto_increment,  PRIMARY KEY (`AUTO_ID`) ) ENGINE=MyISAM AUTO_INCREMENT=141283 DEFAULT CHARSET=latin1;

CREATE TABLE `massatti` (  `CODE2` varchar(50) default NULL,  `AGEATASSESSMENT` decimal(50,30) default NULL,  `GENDER` varchar(50) default NULL,  `RACEW` varchar(50) default NULL,  `RACEN` varchar(50) default NULL,  `RACEB` varchar(50) default NULL,  `RACEH` varchar(50) default NULL,  `RACEA` varchar(50) default NULL,  `RACEO` varchar(50) default NULL,  `RACEU` varchar(50) default NULL,  `ADMISSDT` varchar(50) default NULL,  `AGEINTAKE` varchar(50) default NULL,  `YEARINTAKE` varchar(50) default NULL,
`DIAGTYPE` varchar(50) default NULL,
`PDIAG` varchar(50) default NULL,
`SDIAG` varchar(50) default NULL,
`HOPE` varchar(50) default NULL,
`FUNC` varchar(50) default NULL,
`SATIS` varchar(50) default NULL,
`PSERVER` varchar(50) default NULL,
`PRIMARYLAST` varchar(50) default NULL,
`AUTO_ID` int(11) NOT NULL auto_increment,
PRIMARY KEY (`AUTO_ID`) ) ENGINE=MyISAM AUTO_INCREMENT=141283 DEFAULT CHARSET=latin1;

CREATE TABLE `massatti_p1` (
`CODE2` varchar(50) default NULL,
`AGEATASSESSMENT` decimal(50,30) default NULL,
`GENDER` varchar(50) default NULL,
`RACEW` varchar(50) default NULL,
`RACEN` varchar(50) default NULL,
`RACEB` varchar(50) default NULL,
`RACEH` varchar(50) default NULL,
`RACEA` varchar(50) default NULL,
`RACEO` varchar(50) default NULL,
`RACEU` varchar(50) default NULL,
`ADMISSDT` varchar(50) default NULL,
`AGEINTAKE` varchar(50) default NULL,
`YEARINTAKE` varchar(50) default NULL,
`DIAGTYPE` varchar(50) default NULL,
`PDIAG` varchar(50) default NULL,
`SDIAG` varchar(50) default NULL,
`HOPE` varchar(50) default NULL,
`FUNC` varchar(50) default NULL,
`SATIS` varchar(50) default NULL,
`PSERVER` varchar(50) default NULL,
`PRIMARYLAST` varchar(50) default NULL,
`AUTO_ID` int(11) NOT NULL auto_increment,
PRIMARY KEY (`AUTO_ID`) ) ENGINE=MyISAM AUTO_INCREMENT=141283 DEFAULT CHARSET=latin1;

CREATE TABLE `massatti_p2` (
`CODE2` varchar(50) default NULL,
`AGEATASSESSMENT` decimal(50,30) default NULL,
`GENDER` varchar(50) default NULL,
`RACEW` varchar(50) default NULL,
`RACEN` varchar(50) default NULL,
`RACEB` varchar(50) default NULL,
CREATE TABLE `massatti_p3` (  `CODE2` varchar(50) default NULL,  `AGEATASSESSMENT` decimal(50,30) default NULL,  `GENDER` varchar(50) default NULL,  `RACEW` varchar(50) default NULL,  `RACEN` varchar(50) default NULL,  `RACEB` varchar(50) default NULL,  `RACEH` varchar(50) default NULL,  `RACEA` varchar(50) default NULL,  `RACEO` varchar(50) default NULL,  `RACEU` varchar(50) default NULL,  `ADMISSDT` varchar(50) default NULL,  `AGEINTAKE` varchar(50) default NULL,  `YEARINTAKE` varchar(50) default NULL,  `DIAGTYPE` varchar(50) default NULL,  `PDIAG` varchar(50) default NULL,  `SDIAG` varchar(50) default NULL,  `HOPE` varchar(50) default NULL,  `FUNC` varchar(50) default NULL,  `SATIS` varchar(50) default NULL,  `PSERVER` varchar(50) default NULL,  `PRIMARYLAST` varchar(50) default NULL,  `AUTO_ID` int(11) NOT NULL auto_increment,  PRIMARY KEY (`AUTO_ID`) ) ENGINE=MyISAM AUTO_INCREMENT=141283 DEFAULT CHARSET=latin1;
create view massatti_p3_view as
SELECT * FROM `massatti` group by CODE2, ADMISSDT;

CREATE TABLE tmp_CODE2 (CODE2 VARCHAR(50));

CREATE VIEW massatti_final AS
SELECT 'CODE2','AGEATASSESSMENT','GENDER','RACEW','RACEN','RACEB','RACEH','RACEA','RACEO','RACEU','ADMISSDT','AGEINTAKE','YEARINTAKE','DIAGTYPE','PDIAG','SDIAG','HOPE','FUNC','SATIS','PSERVER','PRIMARYLAST','PFORM','SPECFOST','FOSTER','AUTO_ID'
UNION
SELECT *
FROM massatti_p1
UNION
SELECT *
FROM massatti_p2
UNION
SELECT *
FROM massatti_p3;

create index c2 on massatti(CODE2);
create index c2 on massatti_p1(CODE2);
create index c2 on massatti_p2(CODE2);
create index c2 on massatti_p3(CODE2);
Appendix E: Missing Data Patterns
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Table 25. Missing data patterns
Appendix F: Correlations among ACS Variables
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<th>Unemployment</th>
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Table 26. Correlations among ACS variables used in principal components analysis\(^{35}\)

\(^{35}\) *p < 0.0001
Appendix G: Hierarchical Linear Model
Figure 33. Hierarchical linear model