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Lead Exposure and Crime

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LEAD EXPOSURE AND CRIME

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ABSTRACT

While lead exposure during childhood has been linked to criminal activity later in life, prior research has failed to develop a theoretical foundation explaining why lead and crime rates are positively related at the aggregate level. Utilizing tract-level data, I examine the relationship among elevated blood lead level rates, levels of concentrated disadvantage, and crime rates. Through a biosocial approach, I explore the lead-crime relationship using a measure of concentrated disadvantage to account for the variations across tracts. The results of this study suggest that the effect of lead on crime is more predominant in areas with higher levels of concentrated disadvantage. I conclude with a discussion of the implications this study has for public policy and future research.

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CHAPTER 1

Introduction

Unfortunately for the study of crime, Caesar Lombroso left an unintentional legacy that kept biological sources of crime out of criminology for decades. His crude methodologies led to conclusions with extremely limited applicability (i.e., the death penalty), the belief in biological determinism, and misguided eugenic practices (Raine, 2013). Criminology has, therefore, remained a relatively exclusive social science since Lombroso's time. Even as the biological sciences continue to make great strides in understanding human behavior, many criminologists still fight the integration of biology into criminology. This is largely due to a fear that the discovery of biological correlates of crime will once again lead to eugenic practices or even push the social ideological cores of criminology to the wayside (Wright & Cullen, 2012). However, now it is understood that biological influences predispose many individuals to act in certain ways, and the social environment interacts with these predispositions for better or worse (Brennan & Raine, 1997).

Human beings consist of genes, hormones, brains, and an evolutionary history (Walsh & Beaver, 2009). The exclusion of such biological influences in the understanding of human behavior, particularly criminal behavior, is a disservice to scholarly advancement. Biosocial criminology does not seek to pit nature against nurture; instead it seeks to understand the interaction between the two (Walsh & Beaver, 2009). Therefore, biosocial approaches can expand criminology as a science, making it even

more interdisciplinary by including biological influences (Wright & Boisvert, 2009). Furthermore, biosocial criminology provides new opportunities for research, including primary data collection methodologies aside from surveys. Biosocial criminology also allows for innovative and effective prevention ideas based in each stage of human development (Wright & Boisvert, 2009).

The chemical toxin lead serves as an example of a biosocial hazard. Exposure affects the biological and neuropsychological development of an individual. However, exposure to lead varies based on social context. Furthermore, an individual's ability to cope with neurological deficits is dependent on social supports available to that individual. The study of lead and its effect on the brain and central nervous system extends beyond the field of biology into the realms of neurology, neuropsychology, and physiology. This paper will only use the term "biosocial" in an attempt to garner further support for biological research in criminology before expanding to other individual fields that criminologists may be reluctant to explore. While there are specific distinctions between biology and fields such as neurology, "neurocriminology" and "biosocial criminology" are used relatively interchangeably (for an example, see Raine, 2013). This paper seeks to supplement the growing literature supporting the interaction between biological and social influences through a thorough investigation of the chemical lead as a criminogenic risk factor.

Theoretical Framework

Lead at the Individual Level

Exposure. Although the mean blood lead level (BLL) for U.S. children from 2007 to 2010 was 1.3 micrograms per deciliter (μg/dL), 2.6% of children had BLLs

above or equal to 5 μg/dL (Centers for Disease Control and Prevention [CDC], 2013). The Centers for Disease Control and Prevention (CDC) considers BLLs above or equal to 5 μg/dL to be elevated blood lead levels (EBLLs) based on the 97.5th percentile distribution of children's BLLs. The CDC requires case management (e.g., nutrition guidance) once BLLs reach 10 μg/dL. Chelation therapy, which is treatment to breakdown heavy metals in the body, is recommended at 45 μg/dL (CDC, 2012). At 70 μg/dL, clinical symptoms, such as seizures, comas, and even death, may occur (Jones et al., 2009).

The health dangers associated with lead exposure gain national attention through media coverage of scandals like children's toy recalls. For example, in 2007 Mattel recalled 967,000 toys spanning 83 products due to the amount of lead-based paint covering the toys (Story, 2007). However, individuals can be exposed to this "multimedia pollutant" from numerous sources in their everyday environments (Bellinger, 2008). While toy recalls garner short-lived uproars about lead hazards, lead-based paint is a well-known danger. The primary sources of lead exposure are lead-based paint and the dust resulting from its deterioration (Levin et al, 2008). Renovation in homes with lead-based paint increases the risk for EBLLs, with the greatest risk coming from hand sanding surfaces in preparation for painting (Reissman, Matte, Gurnitz, Kaufmann, & Leighton, 2002). Spanier, Wilson, Ho, Horning and Lanphear (2013) found that the BLLs of children living in houses undergoing interior renovation were 12% higher than the BLLs of children living in homes that were not undergoing interior renovation. Although lead paint is the primary source of lead exposure, over 30% of children with EBLLs were not exposed to lead paint hazards (Levin et al., 2008).

Emissions from industrial sites have become the most predominant cause of lead in the air since the cessation of the use of leaded gasoline (Levin et al., 2008). Areas with smelting and manufacturing plants have the greatest environmental risk. Lead particles are able to bind strongly to soil once they are released into the air. Therefore, locations with high amounts of traffic cause lead to be stirred up in the air more often than rural and quiet locations, which creates a greater risk for urban areas (Levin et al., 2008).

Lead can also be found in soldered cans and the printer ink on labels of food packaging, particularly when packaged in Mexico (Levin et al., 2008). Crystal and ceramic dishware contribute to lead in the diet, which can be transferred to babies through breast milk from a mother. Formula-fed babies can also be exposed through contaminated water. Metal piping provides an opportunity for lead to seep into water. Even "lead-free" plumbing fixtures are legally permitted to be 8% lead (Levin et al., 2008).

Polyvinyl chloride (PVC) is a lead hazard after extended exposure to sunlight (Levin et al., 2008). The dust that forms on vinyl miniblinds is, therefore, likely dangerous. Lead dust from PVC and lead paint accumulates on floors, creating a problem for children who crawl and are frequently on the ground. The Environmental Protection Agency (EPA) regulates lead dust on floors, maintaining that it is dangerous once it accumulates to 40 micrograms per square foot ($\mu g/ft^2$) (Environmental Protection Agency [EPA], 2001). However, Dixon et al. (2009) found that 4.6% of children have EBLLs when floor lead dust is equal to 12 $\mu g/ft^2$. Lead, in one form or another, is present in many locations that facilitate human exposure.

Developmental Effects. Once it enters the body, lead causes problems in behavioral and cognitive development by disrupting various brain mechanisms (Goyer, 1996). Calcium is an element that aids proteins and presynaptic cells in healthy brain functioning. Lead particles are able to mimic calcium, allowing lead to attach to brain structures and inhibit healthy functioning (Needleman, 2004). Lead is able to interfere with the central nervous system's ability to relay information throughout the brain through processes such as synaptic firing by attaching to these various structures. Lead can also have deleterious health effects outside of the brain, affecting other bodily functions such as the kidneys and blood pressure (Needleman, 2004).

EBLLs have been associated with decreased gray and white matter in the brain (Brubaker et al., 2009; Cecil et al., 2008). Gray matter contains the brain's neuronal bodies. Cecil et al. (2008) found that childhood BLLs are associated with decreased adult gray matter volume in the prefrontal cortex. This is important because the prefrontal cortex is responsible for managing attention, behavior regulation, and assessing new conflicts and tasks. Further, this region of the brain is responsible for comprehending and making decisions that are reward- or emotionally-based (Cecil et al., 2008).

Brubaker et al. (2009) found that childhood BLLs were also associated with decreases in adult white matter volume. White matter contains the axons that connect neurons and are responsible for communication between cells. Brubaker et al. (2009) discovered that axonal integrity was weakened and myelination was changed in children with EBLLs. This finding suggests that lead creates problems in cells' abilities to communicate, disrupting cognitive functioning and creating difficulty in individuals to easily regulate their behavior.

Evidence regarding prenatal lead exposure also supports the findings of reduced brain matter. Dietrich et al. (1987) found that prenatal lead exposure was associated with neurobehavioral deficits in three-month-old infants. Prenatal lead exposure, especially during the third trimester, is also associated with lower childhood IQs (Schnaas et al., 2006). The third trimester is particularly important because during this developmental phase secondary and tertiary sulci form (Stiles & Jernigan, 2010). The sulci are the folds of the brain and interference in their development can lead to an overall reduction in brain matter volume. Because the blood-brain barrier is less developed, the brain is more susceptible to lead exposure in the womb (Goyer, 1996). Capillaries composed of endothelial cells form the blood-brain barrier, which protects the brain and central nervous system from neurotoxins in the blood (i.e. lead) (Abbott, Patabendige, Dolman, Yusof, & Begley, 2010).

Postnatal exposure to lead is also associated with decreased intelligence, decreased academic achievement, and increased behavioral difficulties (Baghurst et al., 1992; Bellinger, Stiles, & Needleman, 1992; Calderón et al., 2001; Canfield et al., 2003; Lanphear et al., 2005; Needleman et al., 1979). Although the CDC requires case management for children with lead levels exceeding 10 μg/dL (CDC, 2012), the majority of studies involving lead have found that lead levels can be dangerous below 10 μg/dL. For instance, Needleman et al. (1979) found that dentine lead levels (i.e., lead levels in teeth) were associated with attention problems and decreased auditory and verbal processing, and children with lower dentine lead levels were most susceptible. Calderón et al. (2001) found that attention deficits in elementary school children were just as likely in children with lower levels of lead exposure. Lanphear et al. (2005) found that there

was no threshold of lead exposure to see a lower IQ in individuals, and that lowered IQ can be present at 7.5 μg/d. Canfield and colleagues (2003) support this idea by finding that IQ decreases are more significant when associated with 1-10 μg/dL, as opposed to 11-20 μg/dL.

Peak BLLs, which occur around 24 months of age, were significantly associated with a decrease in intelligence and academic achievement at age 10 (Bellinger, Stiles, & Needleman, 1992). The researchers suggest that lead levels peak at 24 months because children are now mobile but close to the ground, which puts them at a high risk of exposure to any lead dust accumulated on the ground. This is also the age when children put toys and other objects in their mouths, further increasing their risk of ingesting lead dust. Peak exposure, therefore, occurs during the toddler phase because this kind of tactile behavior typically stops with age (Reismann et al., 2012).

Current research on children supports the idea that lead is most deleterious at a younger age. By age six, children's brains are developed to 90% of their adult size (Stiles & Jernigan, 2010). Development of oligodendrocytes and myelination occurs in early childhood (Stiles & Jernigan, 2010). Because lead particles still have a chance to damage the growth of oligodendrocytes and myelin in early childhood, the brain is more susceptible at this time. Oligodendrocytes facilitate the growth of myelin, which assists with neuronal transmission. Any problems in their development could decrease neurons' abilities to communicate with one another. This would, presumably, create deficits in learning, leading to lower intelligence levels, as well as problems with attention, due to an inability to regulate actions and listen actively.

Interestingly, the dangers of toxin exposure in relation to intelligence and attention seem specific to lead, meaning that lower levels of lead are more dangerous than lower levels of other neurotoxins. An older study by Thatcher, Lester, McAlaster, and Horst (1982) found that lead did decrease intelligence, but they suggest cadmium may have the same effects and exposure hazards. Therefore, they suggest that lead is not the only dangerous toxin. While this certainly seems likely, Kim et al. (2013) found that lead exposure increases a child's odds of having attention deficit hyperactivity disorder (ADHD). Mercury and cadmium exposure, however, did not have significant effects. Surely, any environmental toxin is dangerous at certain levels, but the low levels at which lead is able to have an effect make it particularly dangerous to development.

Delinquency. Individual effects of lead are problematic, but the outcome of such effects can be detrimental to society as a whole. Multiple studies have found a positive association between lead concentrations in the body and criminal behavior, which is a societal issue in addition to the individual's concern. Needleman, Riess, Tobin, Biesecker, and Greenhouse (1996) found that boys aged 7 and 11 who had higher bone lead levels were more likely to self-report antisocial and delinquent behaviors. Parents and teachers were also more likely to report antisocial and delinquent behaviors for these boys. Needleman, McFarland, Ness, Fienberg, and Tobin (2002) found that adjudicated delinquents were more likely to have higher bone lead concentrations. Dietrich, Ris, Succop, Berger, and Bornschein (2001) conducted a birth cohort longitudinal study that found a significant and positive relationship between reported antisocial and delinquent behaviors at 15 to 17 years of age for those individuals with pre- and postnatal EBLLs. Prenatal BLLs have also been positively associated with total arrest rates later in life,

while postnatal BLLs have been positively associated with higher violent arrest rates (Wright et al., 2008).

While studies have found a positive association between lead and criminal activity, the relationship is likely indirect, acting by creating challenges in the social environment for individuals with biological impairments that have been exposed to lead (Needleman et al., 2002). Lead has a positive association with a decrease in prefrontal cortex gray matter (Cecil et al., 2008). Raine (2002) explains that dysfunction in the prefrontal cortex makes an individual less able to regulate his or her emotions and aggression, predisposing him or her to violence. Behavioral histories of people with damage to the prefrontal cortex reveal that those suffering this damage before 16 months of age have more antisocial behaviors in their past when compared to those with adult damage (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999). Individuals with early damage to the prefrontal cortex also showed a lack of remorse for their behavior and decreased moral reasoning abilities.

Moffitt's (1993) life-course-persistent offender provides the example for how the environment and individual interact to create antisocial outcomes. Neurological deficits start this offender's trajectory. Due to a lower ability to regulate behavior and lower cognitive functioning, individuals with neurological deficits are predisposed for antisocial behavior. Children that have behavioral problems illicit negative responses from parents and teachers. These negative responses encourage antisocial behaviors, leading to a life of crime (Moffitt, 1993). The neuropsychological deficits positively associated with lead levels described in the previous section increase an individual's risk for negative

interactions with his social environment, perpetuating a dangerous cycle of risk factors for delinquency (Raine, 2002).

Moffitt (1993) posits that neuropsychological deficits in verbal and executive functioning are positively associated with antisocial behavior. These deficits manifest themselves through reading and problem-solving difficulties, ADHD, expressive speech and writing problems, and poor test-taking skills. As children continue to have problems at school and home, their teachers and parents react differently (Moffitt, 1993). Once children begin exhibiting troubles in the classroom, they are at risk for future problems. Children may lose connections with teachers, creating a lack of motivation for success (Eccles et al., 1993). The family environment can work the same way (Eccles et al., 1993). Children with behavioral problems, or even difficulties in school, may create frustrations for parents, making negative interactions more frequent at home. This sequence of provocations and adverse reactions can easily produce an individual ripe for life-course-persistent antisocial behavior and offending (Moffitt, 1993).

Lead at the Macro Level

Exposure. The health hazards of lead exposure, while real to everyone, disproportionally affect those individuals living in areas concentrated disadvantage (Elreedy et al., 1999; Levin et al., 2008; Mahaffey, Annest, Roberts, & Murphy, 1982; McLoyd, 1998). Low socioeconomic status (SES), for example, is a consistent predictor of EBLLs. Mahaffey et al. (1982) demonstrate through an investigation of the National Health and Nutrition Examination Survey (NHANES) that EBLLs are more common in children from families whose annual income is less than 6,000 dollars. More recently, an examination of the Third NHANES found that individuals in the lowest tercile of a

poverty index had significantly higher BLLs than individuals in higher terciles (Lanphear, Dietrich, Auinger, & Cox, 2000). Elreedy and colleagues (1999) provide similar findings in their study of tibia lead concentrations in men. Their study suggests that tibia lead concentrations are significantly and positively associated with individual and geographic measures of SES. Those individuals with low incomes living in impoverished areas had significantly higher tibia lead levels than their low-income counterparts living in nonimpoverished areas, suggesting that geographic measures of SES interact with the individual measures of SES for increased tibia lead levels (Elreedy et al., 1999).

Children living in urban areas, especially inner city locations, have an increased risk for EBLLs (Levin et al, 2008; McLoyd, 1998). Living in a house built prior to the ban on lead paint in 1978, for instance, is positively associated with EBLLs (Levin et al., 2008). Older houses are more likely to be in the middle of the city because cities tend to develop outwards. Families with lower incomes are less able to afford newer houses and, therefore, low SES individuals are more likely to live in older homes in decaying urban centers.

Wilson (1987) coined the term *concentration effects* to describe experiences of inner-city, low-income families. Individuals who are poor, live in single-parent households, unemployed, and often black, live in the inner-city where these effects are able to build on each other, creating a degree of social isolation from more advantaged individuals. In addition to the previously established concentration effects, lead discriminately affects these individuals living in areas of concentrated disadvantage. Because there is a race gap in concentrated disadvantage, it is not surprising to find a race

gap in the literature studying the negative effects of lead. Estimates from early NHANES studies when the CDC defined EBLLs as 10 μg/dL or higher suggested that 12.2% of African-American children had EBLLs, while only 2% of Caucasian children had EBLLs (Mahaffey et al., 1982). However, prevalence estimates made from NHANES after the CDC lowered the level of EBLLs to 5 μg/dL dramatically increased. Levin et al. (2008) estimate that 48.6% of African-American children are now considered to have EBLLs, while 18.7% of Caucasian children have EBLLs.

McLoyd (1998) suggests that discriminatory housing practices have pushed many African-American individuals into impoverished, urban communities. Wilson's (1987) argument adds to this by demonstrating that poor white individuals are less likely to live in poor communities than poor black individuals. The differences in living situations likely contributes to the race gap in EBLLs that had been documented in a number of lead studies (Bernard & McGeehin, 2003; Brody et al., 1997; Canfield et al., 2003; Levin et al., 2008; Needleman et al., 2002).

Individuals living in urban areas are at an increased risk of lead exposure due not only to older housing, but also to air and soil pollution resulting from heavy traffic or industrial emissions (Levin et al., 2008). Annest et al. (1983) found that an average 37% drop in EBLLs between 1976 and 1980 was likely due to a decrease in the use of leaded gasoline. If leaded gasoline did cause EBLLs, those in city centers would be at the highest risk of exposure due to traffic pollution. While industrial emissions are more probable in urban areas, parents with low-paying factory jobs may also transfer lead dust from occupational exposure to their children, further increasing a low SES child's risk for exposure to lead (Levin et al., 2008).

Finally, individuals with a low SES are also more susceptible to the effects of lead because of the nutritional value of foods typically consumed by impoverished individuals. Consuming fewer calories from fat and adding calcium, iron, and other vitamins and nutrients to the diet can help the body fight the effects of lead exposure (Mahaffey, 1990). Unfortunately, research has demonstrated that low SES individuals are more likely to consume less nutritional foods because they are cheaper (Appelhans et al., 2012). A review of studies on "food deserts", or areas that are marked by less access to affordable and healthy food, found that low-income areas have fewer food retailers when compared to more affluent areas (Beaulac, Kristjansson, & Cummins, 2009). Individuals in low-income areas, therefore, have a longer drive to access nutritious foods, but this extra time may not be a luxury afforded to a single parent working multiple jobs. Beaulac and colleagues (2009) found in their review that largely African-American areas also had fewer supermarkets and chain stores, contributing to the race gap literature. The literature on food and nutrition demonstrate that while low SES individuals are more likely to be exposed to lead, their bodies are also less able to combat its effects due to a lack in nutritional health.

Crime. The sociological correlates of lead exposure, including concentration effects, described in the previous section are nearly parallel to those within the concept of *concentrated disadvantage*. This term is used to portray the situation of neighborhoods that face multiple hardships, including higher levels of poverty and unemployment, which leads to lower levels of collective efficacy and informal social controls (Sampson, Raudenbush, & Earls, 1997). These aggregate-level measures include the percentage of individuals living below the poverty line, receiving public assistance, who are

unemployed, living in female-headed households, who are children, and who are a minority, specifically African-American. While these individual measures present their own challenges separately, when taken together, they create situations where individuals are more likely to perceive higher levels of violence and have a greater risk of violent victimization (Sampson et al., 1997). Furthermore, concentrated disadvantage is also positively associated with greater rates of intimate partner violence against women (Benson & Fox, 2004).

The theoretical similarities amongst concentrated disadvantage and the risk factors for lead exposure suggest that lead exposure may partially mediate the association between concentrated disadvantage and crime at the aggregate level. The current research linking lead and crime at the macro level does so by examining crime trends and attributing the changes to BLL trends. For instance, Nevin (2007) uses best-fit lags and trend regression to find a positive association between multinational BLL trends and violent crime trends. Nevin (2007) examined the association between preschool BLL rate and crime rate trends in subsequent years, using peak-offending information as the point of reference (e.g. property crime peaks at 15 to 20 years of age, so he used crime data 15 to 20 years after the BLL measures). The internationality of Nevin's (2007) findings suggests that lead may, indeed, play a key role in crime.

Similarly, Reyes (2007) links violent crime increases and decreases in the United States with the consumption and ban of leaded gasoline, respectively. Her results support the connection between concentrated disadvantage, lead, and crime, especially if one considers the arguments of Levin et al. (2008). They suggest that residual effects of

leaded gasoline would disproportionately affect those individuals residing in urban centers, where the air and soil are more polluted from heavy traffic (Levin et al., 2008).

While the studies of Nevin (2007) and Reyes (2007) make convincing arguments for a relationship between lead and crime at the macro level using national and international trends, research on lead and crime has yet to set a foundation in criminological theory. This paper will take a biosocial approach to examine the relationship of EBLL rates and concentrated disadvantage and crime, while controlling for other structural characteristics. This paper seeks to fill the gap between lead and crime by accounting for concentrated disadvantage, a known crime correlate (Sampson et al., 1997). With this in mind, the following research questions were developed: (1) Is there a significant relationship between rates of EBLLs and crime? and (2) How do EBLL rates relate to the correlation between concentrated disadvantage and crime rates?

To answer these questions, five hypotheses are tested in this study:

H_1 Concentrated disadvantage is significantly related to EBLL rates.

H_2 Concentrated disadvantage is significantly related to crime rates.

H_3 EBLL rates are significantly related to crime rates.

H_4 EBLL rates partially mediate the relationship between concentrated disadvantage and crime rates.

H_5 Concentrated disadvantage moderates the relationship between EBLL rates and crime rates.

Lead, a seemingly biological danger, is a social problem. It is more likely to affect those individuals in socially disadvantaged areas who are least likely to be able to overcome its effects. The limited access to affordable healthcare, good nutrition, and

social capital allow crime to continue to happen disproportionately in impoverished communities. The chemical is discriminatory in the sense that exposure to it is more likely for those individuals living in areas of concentrated disadvantage. Exposure then leads to neurological deficits, which are difficult to overcome in individuals that have little social support in school, at home, and in the neighborhood. This, in turn, puts individuals at an increased risk for a life-time of offending (Moffitt, 1993). The current study aims to study this relationship at the aggregate level by analyzing rates of EBLLs, concentrated disadvantage and crime.

CHAPTER 2

METHODS

Data

Census tracts are the unit of analysis in this study. The data used come from a variety of sources including the National Neighborhood Crime Study (NNCS), the U.S. Census, and the Massachusetts Childhood Lead Poisoning Prevention Program (CLPPP). The 2000 Decennial Census served as the temporal point of interest because not all necessary data for the 2010 Census are currently available. BLLs data are from 2001 in order to establish temporal ordering between structural indicators provided by census data and the outcomes of interest. The crime data provided by the NNCS are crime rate averages for the years 1999 to 2001. Police departments provided the crime data at the incident or tract level in order to compile the NNCS dataset.

While Massachusetts boasts the best childhood lead poisoning screening program, reporting from laboratories varies widely across the state (Massachusetts Department of Public Health [MDPH], 2009). Crime data were not available at the tract level for the whole of Massachusetts, so the study was restricted to Boston and Worcester, Massachusetts. However, these are two of the most populous cities in the state with 589,141 and 172,648 residents, respectively (U.S. Census Bureau, 2000). Figure 2.1 displays their locations within Massachusetts. Of the possible 190 census tracts in the two cities, 143 (75%) had complete data from all three datasets.

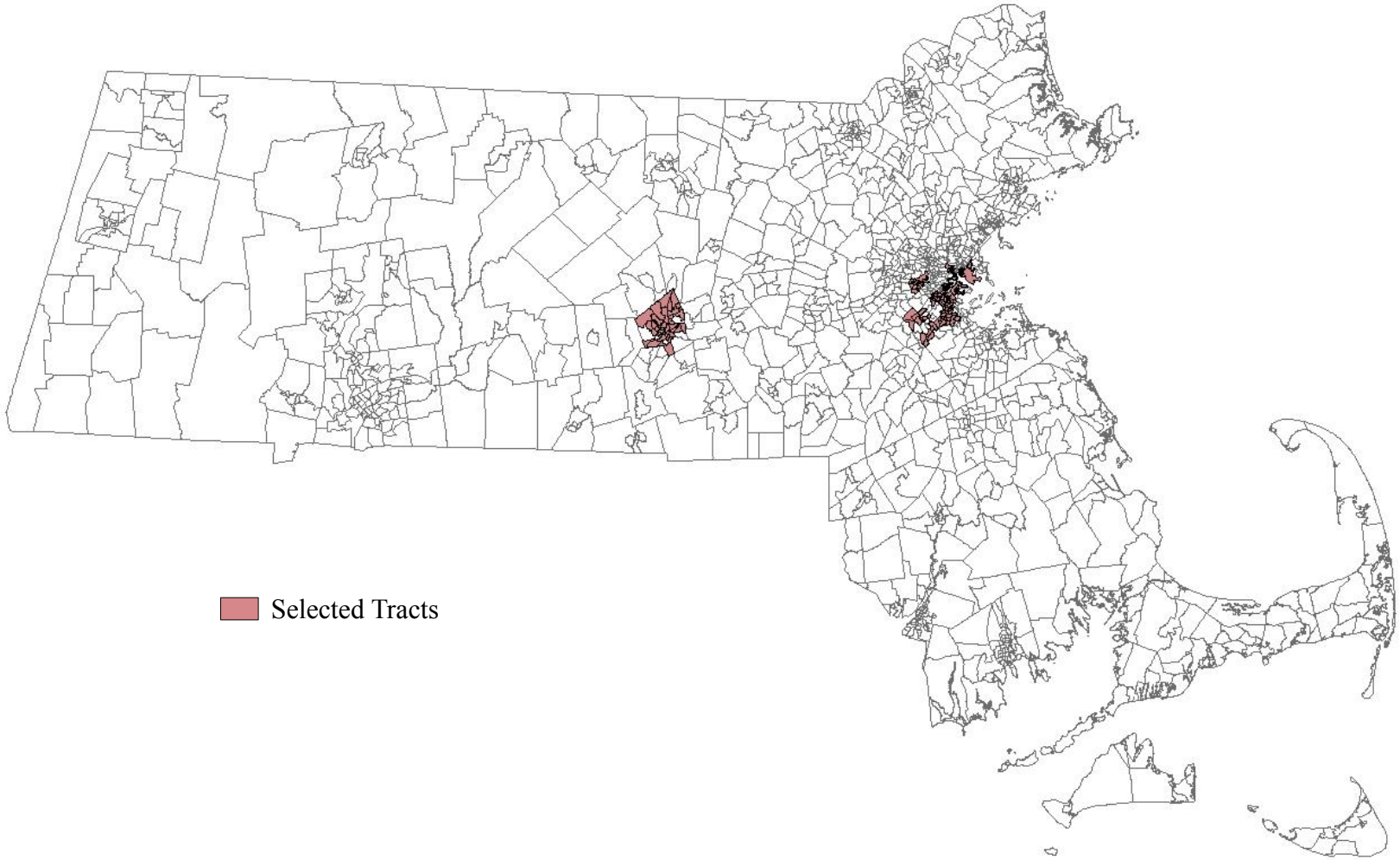

Figure 2.1 Tracts selected for analysis.

Variables

Crime rates. The crime rates for Boston and Worcester census tracts are the primary outcome variables in this study. Crime data from the NNCS, obtained through the Inter-university Consortium for Political and Social Research, included three-year averages of crime rates, measuring crime from 1999 to 2001 (Peterson & Krivo, 2000). Three-year averages were used to account for the potential variation for crime rates in a single tract from year to year. The rates were calculated per 1,000 persons and were computed for *murder*, *rape*, *robbery*, *aggravated assault*, *burglary*, *larceny*, and *motor vehicle theft*. *Violent crime rates* were created from the murder, rape, robbery, and aggravated assault variables as a summated scale. *Property crime rates* were created from the burglary, larceny, and motor vehicle theft variables as a summated scale. *Total crime rates* were calculated by summing the violent and property crime rates. Because the majority of these variables, with the exception of rape and motor vehicle theft, were highly skewed, kurtotic, or both, the rates were logged[1]. The natural log crime rate variables were used for analysis.

Blood lead levels. The data on BLLs used for this study come from data collected as required by the Massachusetts Lead Poisoning and Prevention Act (MDPH, 2009). The Code of Massachusetts Regulation requires that all children in Massachusetts be screened for lead poisoning between nine and 12 months of age and again at 2 and 3 years of age (Lead Poisoning Prevention and Control, 2002). If a child has not been screened before

[1] Total crime rate pre-transformation: skewness = 3.859, kurtosis = 23.094.
Total crime rate post-transformation: skewness = .211, kurtosis = 3.665.
Violent crime rate pre-transformation: skewness = 1.733, kurtosis = 8.961.
Violent crime rate post-transformation: skewness = -.608, kurtosis = 2.797.
Property crime rate pre-transformation: skewness = 4.279, kurtosis = 25.631.
Property crime rate post-transformation: skewness = .654, kurtosis = 4.531.

entering kindergarten, they must provide documentation of having done so prior to starting kindergarten. Physicians and laboratories submit test results, even if a child has low BLLs, to the Massachusetts Department of Public Health (MDPH). Individuals with the CLPPP maintain the data and make aggregated BLL information publically available (MDPH, 2009).

The BLL database includes counts for children between the ages of 0 and 71 months of age who were tested in 2001 for lead poisoning aggregated to the census tract level. While there were 41,008 children between the ages of 0 and 71 months in Boston and Worcester, only 15,041 children (37%) were tested in 2001. However, this screening rate is not necessarily a problem because a large percentage of children may have been tested prior to or after 2001 in order to comply with Massachusetts' regulation. The 37% tested in 2001 does not actually represent a true response rate, which would be nearly impossible to measure. BLL tests may be venous or capillary tests; however, one positive (i.e. 10 μg/dL or above) through a capillary test requires a verification test, either venous or capillary, to be considered a positive case. If a child was tested more than once in 2001, the highest reading is included in the dataset, although a venous test always takes precedence. Even if a child had multiple tests done in 2001, he or she only represents one count in the data.

CLPPP provided the dataset used in this study. The dataset includes counts for children in the following ranges of BLLs: 0-9 μg/dL (n=14,404), 10-24 μg/dL (n=607), and 25 or more μg/dL (n=30). Because the dataset does not allow for separation of the 0-9 μg/dL category, 10 μg/dL or higher are considered to be EBLLs in this study, rather than the CDC-recommended 5 μg/dL value. Therefore, this study conservatively

measures EBLLs. Percentages for each range (i.e. 0-9 μg/dL) were created by dividing the number of children per range by the total number of children screened in that tract and then multiplied by 100. This was done to help account for the variation of screening counts between tracts. A general measure of EBLLs was created for analysis by combining the 10-24 μg/dL and 25 or more μg/dL percentages for each tract (n=637). While only 4% of the total children screened had EBLLs, the percentage of EBLLs for each tract varies widely, ranging from 0% to 22%.

Because this variation may still be due to screening differences between tracts, a categorical measure of *EBLLs* was created using 4 percentiles (coded 0 = 0 to 25th percentile, 1 = 26th to 50th percentile, 2 = 51st to 75th percentile, 3 = 76th to 100th percentile). This categorical measure allows for the examination of groups of tracts, rather than individual tracts, which decreases the likelihood of one tract being too influential due to high EBLLs in an area with low screening rates. The four categories allow for comparisons based on percentiles rather than differences that may be arbitrary.

Structural characteristics. A measure of *concentrated disadvantage* was created to gage the social context of the census tracts. Consistent with previous research, concentrated disadvantage was measured using six variables from the 2000 U.S. Census: poverty, unemployment, receipt of public assistance, racial composition (i.e. percent African-American), female-headed households, and density of children (Sampson et al., 1997; Sampson, Sharkey, & Raudenbush, 2008; U.S. Census Bureau, 2000). Each of these variables is a percentage created by dividing the subset of the population characterizing each variable by the total population. The Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.73 for the census variables, suggesting that further

tests of unidimensionality were appropriate (Kaiser, 1970). A principal factor analysis (PFA) between the six structural variables revealed that one factor representing the concept of concentrated disadvantage emerged ($\lambda = 4.07$; factor loadings $\geq .66$). Cronbach's alpha was 0.76, which demonstrates good internal consistency between the variables (Cronbach, 1951). Concentrated disadvantage was then created as a weighted factor scale derived from the PFA (See Table 2.1 for factor scores).

Several other structural characteristics are also used as control variables in all multivariate models. A weighted-factor scale of *residential instability* was constructed from the percentage of the population who lived in a different residence in 1995 and the percentage of renter-occupied housing (KMO = .50; $\lambda = 1.36$; factor loadings $\geq .60$). *Immigrant concentration* at the tract level was created using the percentage of the population that is Hispanic and the percentage of the population that is foreign born using a weighted-factor scale (KMO = .50; $\lambda = 1.42$; factor loadings $\geq .65$). In order to create a measure of *population density*, the total population of the tract was divided by the land area, which was measured in meters. This variable was used to account for the variation of populations between tracts.

Table 2.1 Factor Loadings for Concentrated Disadvantage Scale Items

Female Householder	.98
Households on Public Assistance	.85
Under Age 18	.83
Individuals Below Poverty	.69
Unemployed	.68
Black	.66

Note. Loadings derived from a principal factor analysis.

Finally, a measure of *pre-79 housing* was created to control for the dangers of lead-based paint that is found in houses built before federal regulations regarding this

practice were enacted. This measure was created using the percentage of houses in each tract built prior to 1979. The Lead-Based Paint Poisoning Prevention Act was passed in 1971 and banned the use of lead-based paint in federally-funded housing (Department of Housing and Urban Development [HUD]). In 1978, the Act was amended to include all housing. Because the Census only provides information at certain increments (e.g. 1959, 1969, and 1979), the year 1979 was chosen as the housing cutoff in order to include all housing that may pose a potential danger by being built before any ban on lead-based paint was enacted. The Code of Massachusetts Regulation considers children living in houses built prior to 1978 to be at a high risk for lead poisoning, which further supports the current operationalization of the pre-79 housing variable (Lead Poisoning Prevention and Control, 2002).

Analytic Strategy

To test the present study's hypotheses, the analysis progressed in a series of stages. First, a set of analyses is used to examine whether the relationship between concentrated disadvantage and crime rates is partially confounded by EBLL rates in the census tracts. A series of ordered logistic regression and ordinary least squares (OLS) regression equations are estimated to examine this relationship. Ordered logistic regression is used to determine the relationship between concentrated disadvantage and EBLL rates, controlling for structural characteristics of residential instability, immigrant concentration, population density, and pre-79 housing. This model attempts to determine whether a significant relationship exists between the variables of interest and also satisfies a necessary condition for detecting mediation in later analyses (i.e., the

independent variable—concentrated disadvantage—is associated with the proposed mediator—EBLL) (Baron & Kenny, 1986).

OLS is utilized in the remaining models of this study given the continuous nature of the crime dependent variables. The first set of these analyses involves the estimation of three OLS models to determine the extent to which the relationship between concentrated disadvantage and crime rates is confounded by EBLLs. In doing so, Model 1 regresses the logged total crime rate on concentrated disadvantage and the statistical controls. The independent effect of EBLLs on logged total crime rate, net of statistical controls, is then estimated in Model 2. The third model (Model 3) examines the simultaneous (i.e., additive) effect of concentrated disadvantage and EBLLs, net of controls, on the logged total crime rate. Model 3 allows for the examination of whether EBLLs partially mediate the relationship between concentrated disadvantage and crime rates. The Clogg, Petkova, and Haritou (1995) *z*-test will be used to determine whether any reduction in the concentrated disadvantage effect is statistically significant (see, also, Paternoster, Brame, Mazerolle, & Piquero, 1998).

The final stage of the analysis seeks to determine if the relationship between EBLLs and crime rates is conditioned by the level of concentrated disadvantage in a given tract. This model regresses logged total crime on the interaction between concentrated disadvantage and EBLLs and the statistical controls. Based on prior literature, EBLLs are expected to have a greater effect on crime rates in tracts where concentrated disadvantage is more extreme. The *margins* command available in STATA 13 will be used to examine this potential conditional relationship.

CHAPTER 3

RESULTS

Table 3.1 presents the descriptive statistics for the variables used in this study. Figure 3.1 displays the distribution of crime rates, concentrated disadvantage level, and EBLL rates across tracts. There were no issues of multicollinearity in any of the models (i.e., all variance-inflation factors were below 2.0, Tabachnick & Fidell, 2007).

Table 3.1 Descriptive Statistics for Variables of Interest (n = 143)

Variables	Mean	SD	Min	Max
Total Crime Rate[a]	3.904	.638	2.375	6.000
Violent Crime Rate[a]	2.182	.968	-.540	4.286
Property Crime Rate[a]	3.670	.605	2.297	5.826
Concentrated Disadvantage	5.260[b]	1.022	-1.371	4.117
Elevated Blood Lead Levels	1.503	1.106	0	3
Residential Instability	-4.020[b]	.728	-1.866	1.848
Immigrant Concentration	-2.840[b]	.769	-1.166	3.097
Population Density	.008	.006	.000	.034
Pre-79 Housing	89.824	7.833	66.300	99.200

[a] Values presented are for the logged variable.
[b] Coefficient multiplied by 10^{-16}.

Elevated Blood Lead Levels

Table 3.2 presents the results of the ordered logistic regression model that examined the relationship between concentrated disadvantage and EBLL rates. As expected in H_1, concentrated disadvantage was positively associated with higher EBLL rates (b = .565, p < .05). For every one-unit increase in concentrated disadvantage, there is an expected 0.565 increase in the log odds of a tract being in higher levels of EBLL rates when all other variables are held constant. Generally, tracts with higher levels of

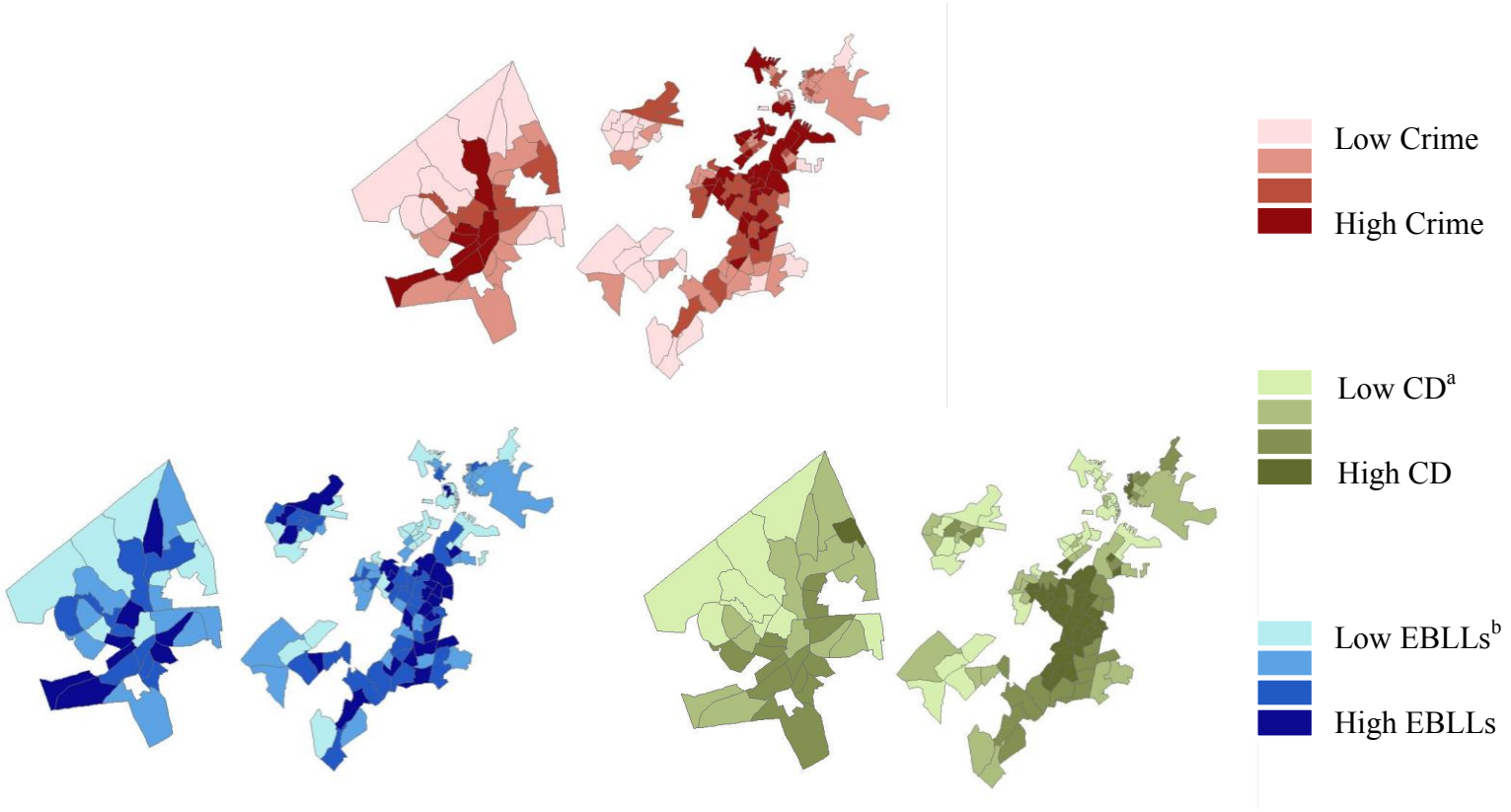

Figure 3.1 Graphic representation of variables.
[a] CD = concentrated disadvantage.
[b] EBLLs = elevated blood lead levels.

concentrated disadvantage have an increased chance of being in a higher level of EBLL rates.

Table 3.2 Ordered Logistic Regression Model for Elevated Blood Lead Levels

Variables	*b*	SE	e^b	*z*-ratio
Concentrated Disadvantage	.565	.232	1.759	2.44*
Residential Instability	-.233	.286	.792	-0.81
Immigrant Concentration	-.005	.217	.995	-0.02
Population Density	-28.826	42.367	3.027[a]	-0.68
Pre-79 Housing	.084	.021	1.088	3.98**
χ^2	30.01**			
df	5			
Pseudo R^2	.076			

Notes. e^b = exponentiated *b*. *$p < .05$, **$p < .01$ (two-tailed test). SE represents the robust standard error for the unstandardized coefficient. The Brant (1990) test suggested that each of the regression coefficients estimated in model were similar across categories of EBLL rates. Values for cut points were excluded from the table.
[a] Coefficient multiplied by 10^{-13}.

The ordered logistic regression model in Table 3.2 demonstrates that the percentage of houses built prior to 1979 was also positively associated with EBLL rates. One-unit increase in pre-79 housing predicts a .084 increase in the log odds of a tract being in a higher level of EBLL rates. Tracts with a higher percentage of houses built before 1979 had a higher chance of being in a higher level of EBLL rates.

Crime Rates

Total crime rates. Table 3.3 presents the OLS regression results that examined the association among concentrated disadvantage, EBLL rates, and total crime rates. Separate models tested the independent effects of concentrated disadvantage, EBLL rates, and the interactive effect of concentrated disadvantage and EBLL rates on total crime rates (Tables 3.4 and 3.5 report similar OLS regression model results, but the outcome variables are violent crime rates and property crime rates, respectively). Model 1 examined the direct effect of concentrated disadvantage on total crime rates.

Concentrated disadvantage and the control variables accounted for 35.8% of the variance in total crime rates. The results of this model supported H_2 that concentrated disadvantage is significantly and positively related to crime rates (b = .195, p < .01), so as the level of concentrated disadvantage increases, total crime rates increase.

In Model 1 of Table 3.3, residential instability was also significantly and positively related to total crime rates (b = .326, p < .01). As expected, tracts with higher levels of residential instability had higher total crime rates. Contrary to expectations, however, pre-79 housing was significantly and negatively associated with total crime rates in Model 1 (b = -.032, p < .01). This finding suggests that as the percentage of houses built before 1979 in a tract increases, crime rates decrease.

Model 2 in Table 3.3 examines the direct effect of EBLL rates on total crime rates. EBLL rates, net of controls, had a significant effect on total crime rates (b = .129, p < .01), supporting H_3. As expected, total crime rates were greater with higher EBLL rates. In Model 2, residential stability remained statistically significant. While EBLL rates were positively associated with total crime rates, pre-79 housing remained negatively associated with total crime rates (b = -.037, p < .01). This suggests that the pre-79 housing variable may not actually be an appropriate measure of potential lead exposure or that there is an unknown mechanism operating through the percentage of houses built pre-1979 on crime rates. In this model, immigrant concentration was significantly and positively associated with total crime rates (b = 161, p <.01), which suggests that as immigrant concentration in a tract increases, so, too, do total crime rates.

This study also hypothesized that EBLL rates would partially mediate the effect of concentrated disadvantage on crime (H_4). In Model 3 in Table 3.3, the inclusion of

EBLL rates into the equation slightly reduced the magnitude of concentrated disadvantage's effect on total crime rates but did reduce the effect to nonsignificance. Rather, concentrated disadvantage appears to partially mediate the relationship between EBLL rates and total crime rates (compare Models 2 and 3). Concentrated disadvantage decreased the magnitude of EBLL rates' effect on total crime rates and reduced its significance level from $p = .001$ to $p = .025$. Because the Clogg et al. (1995) *z*-test was not statistically significant (i.e., the drop in magnitude was not statistically significant), caution must be used when interpreting this result.

The theoretical framework of this paper also suggests that an interactive effect between concentrated disadvantage and EBLL rates on crime is likely to occur. If this logic holds, total crime rates in tracts with higher levels of concentrated disadvantage should be more influenced by EBLL rates than they are in areas of lower concentrated disadvantage. Therefore, Model 4 in Table 3.3 tested H_5 regarding this interaction. EBLL rates were mean-centered for interpretability of the interaction. The model was significant [$F(7, 135) = 17.72$, $p < .01$] and increased the explanatory power of the equation (Model 3 $R^2 = .380$; Model 4 $R^2 = .414$). The interaction term was significantly and positively associated with total crime rates ($b = .110$, $p < .01$). This finding suggests that concentrated disadvantage interacts with EBLL rates in affecting total crime rates in the census tracts.

Figure 3.2 presents a graphical depiction of this interaction. The solid line represents the minimum level of EBLL rates (-1.500), while the dotted line represents the maximum level of EBLL rates (1.497). The graph shows that the effect of concentrated disadvantage on total crime rates differs based on level of EBLL rates. With all else held

Table 3.3 OLS Regression Models for Total Crime Rates

Variables	Total Crime Rates											
	Model 1			Model 2			Model 3			Model 4		
	b (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*
Concentrated Disadvantage	.195 (.053)	3.71**	.313	---	---	---	.171 (.049)	3.52**	.274	.206 (.036)	5.70**	.331
Elevated Blood Lead Levels	---	---	---	.129 (.039)	3.27**	.224	.095 (.042)	2.27*	.164	.091 (.038)	2.38*	.157
Residential Instability	.326 (.086)	3.81**	.372	.327 (.087)	3.76**	.373	.338 (.085)	3.98**	.386	.354 (.082)	4.31**	.404
Immigrant Concentration	.057 (.048)	1.18	.068	.161 (.055)	2.96**	.194	.058 (.047)	1.24	.070	.052 (.043)	1.21	.063
Population Density	-4.811 (7.683)	-0.63	-.045	-5.374 (7.654)	-0.70	-.051	-3.323 (7.885)	-0.42	-.031	-6.083 (7.561)	-0.80	-.057
Pre-79 Housing	-.032 (.007)	-4.52**	-.393	-.037 (.007)	-5.56**	-.449	-.036 (.007)	-5.36**	-.445	-.034 (.006)	-5.28**	-.414
Concentrated Disadvantage X Elevated Blood Lead Levels	---	---		---	---		---	---		.110 (.032)	3.43**	.196
Constant	6.814 (.670)	10.17**		7.033 (.637)	11.04**		7.044 (.654)	10.78**		6.945 (.610)	11.38**	
F-test	11.87**			17.18**			17.11**			17.72**		
R^2	.358			.324			.380			.414		

Notes. **p* < .05, ***p* < .01 (two-tailed test). SE represents the robust standard error for the unstandardized coefficient.

constant, the effect of EBLL rates on total crime rates is greater in areas with higher levels of concentrated disadvantage.

Violent and property crime rates. To test the robustness of the above findings, OLS regression models were also conducted for violent crime rates (Table 3.4) and property crime rates (Table 3.5). Similar results to the total crime rate models were obtained in these equations. Concentrated disadvantage and EBLL rates were significantly and positively associated with both violent and property crime rates. The interaction term was significantly and positively associated with violent crime rates (b = .176, p < .01) and property crime rates (b = .094, p < .01).

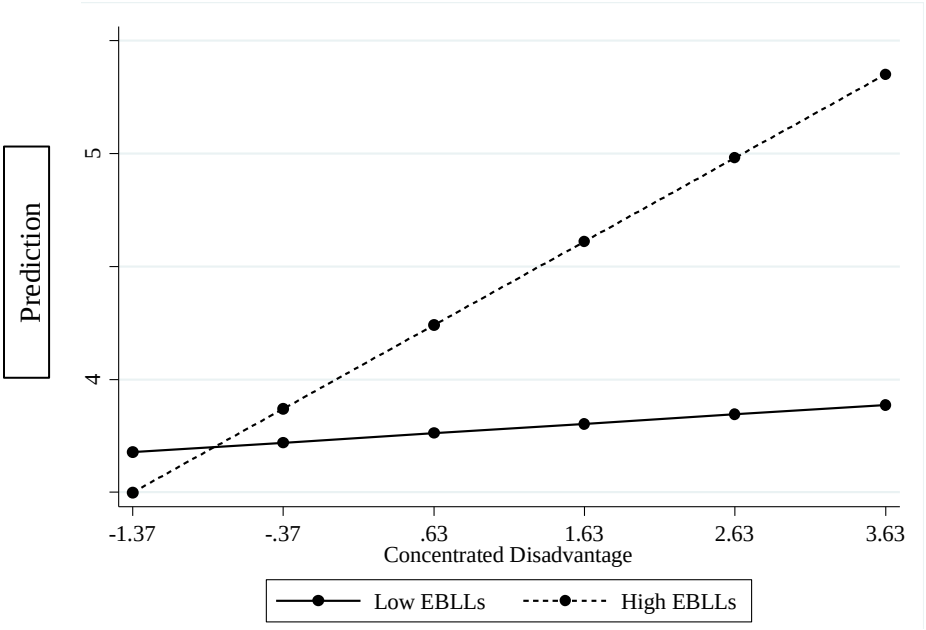

Figure 3.2 Interactive effect of concentrated disadvantage and EBLL rates on total crime rates.

Figure 3.3 displays this interaction. Similar to the total crime model, the effect of EBLL rates on violent crime is greater in areas of higher concentrated disadvantage. This interaction is also visible in the property crime model depicted in Figure 3.4. These

consistent interaction results suggest that EBLL rates are more detrimental to those areas with higher levels of concentrated disadvantage.

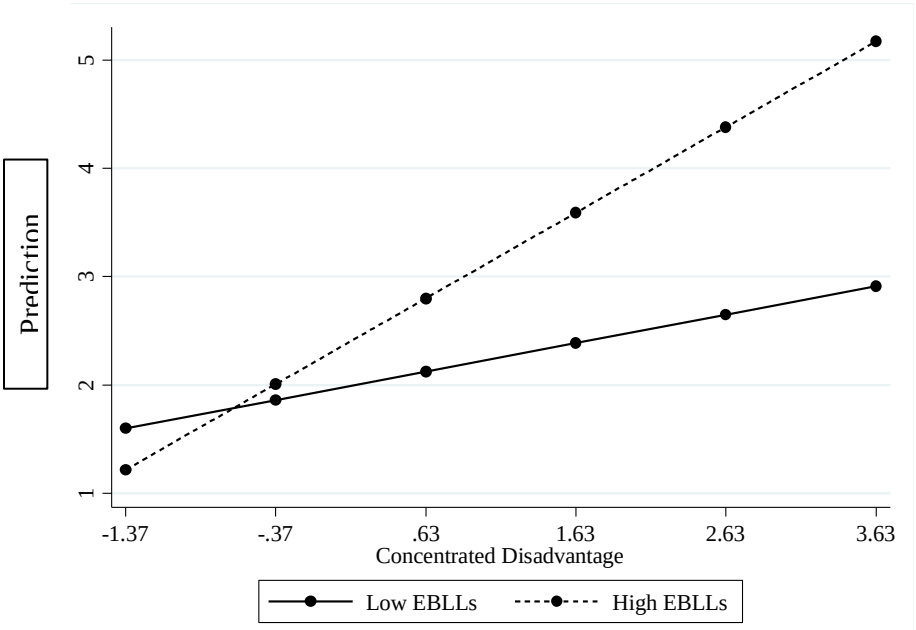

Figure 3.3 Interactive effect of concentrated disadvantage and EBLL rates on violent crime rates.

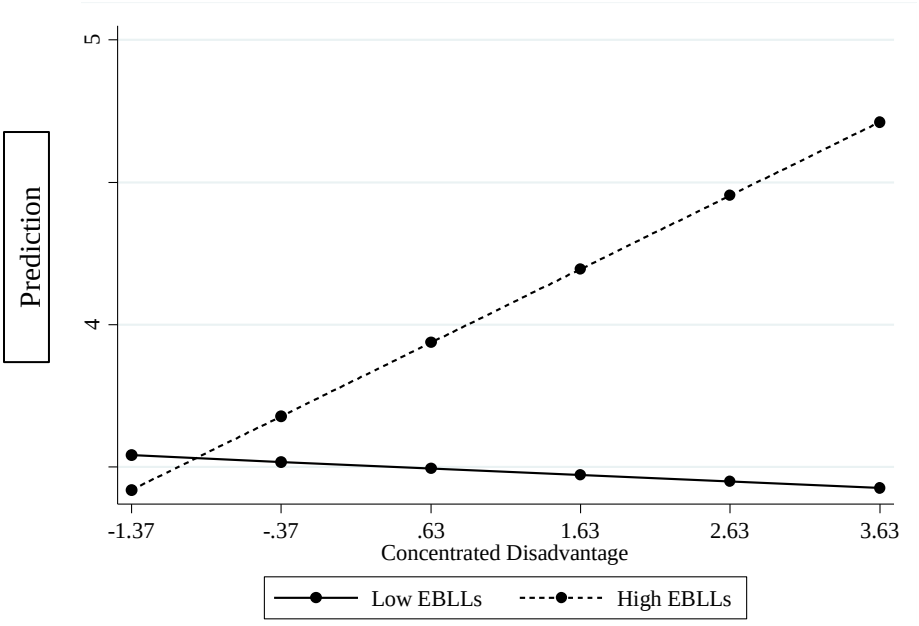

Figure 3.4 Interactive effect of concentrated disadvantage and EBLL rates on property crime rates.

Table 3.4 OLS Regression Models for Violent Crime Rates

Variables	Violent Crime Rates											
	Model 1			Model 2			Model 3			Model 4		
	b (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*
Concentrated Disadvantage	.502 (.090)	5.57**	.530	---	---	---	.471 (.089)	5.36**	.497	.528 (.056)	9.42**	.557
Elevated Blood Lead Levels	---	---	---	.216 (.063)	3.45**	.247	.121 (.066)	1.84	.138	.115 (.055)	2.08*	.131
Residential Instability	.262 (.118)	2.23**	.197	.247 (.125)	1.98*	.186	.278 (.115)	2.43*	.209	.303 (.109)	2.79**	.228
Immigrant Concentration	.205 (.070)	2.92*	.163	.492 (.095)	5.16**	.391	.207 (.070)	2.97**	.164	.198 (.065)	3.04**	.157
Population Density	9.603 (11.043)	0.87	.060	5.853 (10.948)	0.53	.036	11.505 (11.235)	1.02	.071	7.059 (10.565)	0.67	.044
Pre-79 Housing	-.028 (.008)	-3.43**	-.224	-.034 (.009)	-3.98**	-.274	-.033 (.008)	-4.10**	-.268	-.029 (.008)	-3.77**	-.235
Concentrated Disadvantage X Elevated Blood Lead Levels	---	---		---	---		---	---		.176 (.053)	3.31**	.208
Constant	4.596 (.765)	6.01**		4.859 (.792)	6.14**		4.890 (.757)	6.46**		4.684 (.729)	6.42**	
F-test	18.20**			16.26**			23.39**			27.04**		
R^2	.479			.310			.495			.533		

Notes. **p* < .05, ***p* < .01 (two-tailed test). SE represents the robust standard error for the unstandardized coefficient.

Table 3.5 OLS Regression Models for Property Crime Rates

Variables	Violent Crime Rates											
	Model 1			Model 2			Model 3			Model 4		
	b (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*	*b* (SE)	*t*-ratio	*β*
Concentrated Disadvantage	.111 (.045)	2.45*	.188	---	---	---	.087 (.041)	2.14*	.148	.118 (.035)	3.40**	.199
Elevated Blood Lead Levels	---	---	---	.109 (.037)	2.92**	.200	.091 (.039)	2.35*	.167	.088 (.037)	2.37*	.161
Residential Instability	. 347 (.082)	4.23**	.417	.353 (.083)	4.26**	.425	.359 (.082)	4.37**	.432	.372 (.080)	4.65**	.448
Immigrant Concentration	.036 (.048)	0.75	.046	.090 (.050)	1.80	.114	.037 (.047)	0.80	.047	.032 (.044)	0.74	.041
Population Density	-8.819 (7.401)	-1.19	-.088	-8.431 (7.340)	-1.15	-.084	-7.381 (7.549)	-0.98	-.073	-9.746 (7.379)	-1.32	-.097
Pre-79 Housing	-.032 (.007)	-4.43**	-.413	-.036 (.007)	-5.39**	-.468	-.036 (.007)	-5.23**	-.466	-.034 (.007)	-5.15**	-.438
Concentrated Disadvantage X Elevated Blood Lead Levels	---	---		---	---		---	---		.094 (.030)	3.18**	.177
Constant	6.600 (.679)	9.72**		6.817 (.649)	10.50**		6.822 (.664)	10.28**		6.753 (.624)	10.82**	
F-test	9.65**			14.15**			12.83**			12.92**		
R^2	.316			.323			.339			.367		

Notes. $*p < .05$, $**p < .01$ (two-tailed test). SE represents the robust standard error for the unstandardized coefficient.

CHAPTER 4

DISCUSSION

Previous literature has established a positive association between elevated lead levels and criminal activity at the individual level; people with higher levels of lead during childhood are more likely to partake in delinquent acts later in life than those with normal levels of lead during childhood (Dietrich et al., 2001; Needleman et al., 2002; Needleman et al., 1996; Wright et al., 2008). This relationship to delinquency is likely due to the neuropsychological deficits that lead causes and how society responds to children with the behavioral and learning problems created by these deficits (Moffitt, 1993; Raine, 2002).

Unfortunately, the children at an increased risk for lead exposure are those already at risk for delinquency. Children living in poor, urban, and black neighborhoods facing concentrated disadvantage are those most likely to engage in criminal activity as adolescents and adults (Sampson et al., 1997). Children living in areas of higher concentrated disadvantage are more likely to be exposed to lead, adding a biological risk for criminal activity to the numerous social challenges they already encounter (Elreedy et al., 1999; Levin et al., 2008; Mahaffey et al., 1982; McLoyd, 1998). While lead and crime trend studies suggest that there is indeed a relationship between the two at the aggregate level, researchers have yet to integrate this finding into a theoretical perspective (Nevin, 2007; Reyes, 2007). This paper has attempted fill this gap in the

literature by explaining the potential interaction between this individual-level risk and the social factors that perpetuate the risk. Toward that end, the findings of this study warrant further discussion.

First, the current study found an effect of EBLL rates on crime rates. While lead is usually an individual-level factor, these data suggest that there is a contextual effect of lead exposure. Because this study suggests that the environmental toxin not only affects an individual but also can affect a community, it calls further attention to the dangers of lead.

Second, the data in this study support the hypothesis that concentrated disadvantage – an established social correlate of crime – and EBLLs – an established biological correlate of crime – are independently and significantly related to crime rates. Both of these variables had independent effects on crime rates even when controlling for the other. This suggests that both social and biological factors at the aggregate level are important in the study of crime and neither should be discounted or ignored. Third, the data in this study support the idea that crime may result from the interaction of nature, the biological, and nurture, the social. The results suggest that concentrated disadvantage and EBLLs interact to affect crime outcomes. This interaction significantly predicted total, violent, and property crime. These findings clearly demonstrate the interaction between concentrated disadvantage and EBLL rates is important, as opposed to one over the other.

Finally, the interaction demonstrated that the effect of EBLL rates on crime was stronger in areas of higher concentrated disadvantage, as opposed to those with lower levels of concentrated disadvantage. This could be because individuals in the more disadvantaged areas are more likely to be exposed to lead or because they are less likely

to be able to combat its effects; however, it is more probable that a combination of these two reasons creates the biggest problem for those in areas of higher concentrated disadvantage.

This study's findings, while intriguing, must be considered preliminary. These results were based on the data from 143 census tracts in Massachusetts. Although the cities from which the tracts were drawn are large and diverse, results may vary in different locations, especially those in which housing is newer than it may be in one of the oldest parts of the United States. Therefore, future research should attempt to make this study more generalizable by examining tracts in different areas of the country. Future studies should also attempt to control for the risk of lead exposure; however, future researchers will need to find determine a more effective measure than that of the percentage of housing built prior to 1979 used in this study.

Lead data is another limitation of the current study and one that contributed to a small sample size. While Massachusetts appears to have the best screening and reporting program in the U.S., it varies widely across the state (MDPH, 2009). Based on a preliminary search for data at the beginning of this project, it was discovered that lead data are unavailable for a portion of states and is largely unreliable in those in which it is available. The data used in this study are considered the best available because they come from the state with that boasts the best screening and reporting program (MDPH, 2009). Massachusetts also provided the most accessible and thorough data, according to the preliminary search. In order to make this study more valid, data were only used from the areas in Massachusetts where the data were complete. Future research in this area should

aim to improve on the current findings by examining states that pay more attention to the hazards of lead and, consequently, collect better data.

Although this study suggests that these hazards discriminately impacts those facing concentrated disadvantage, the prevention of lead as a criminogenic risk factor is not hopeless. While the current study suggests that lead and concentrated disadvantage interact, policy can more easily impact lead than it can change social forces creating concentrated disadvantage. This paper, therefore, will conclude with a discussion of the policy and prevention implications that can be garnered from the biosocial understanding of lead.

Policies banning lead have made great strides in reducing EBLLs. Annest et al. (1983) demonstrated that the ban on leaded gasoline from the Clean Air Act of 1970 was correlated with the 37% drop in EBLLs from 1976 to 1980. Further, Binns, Campbell, and Brown (2007) attribute the dramatic decrease in BLLs greater than 10 μg/dL (88.2% in 1976-1980, 1.6% in 1999-2002) to the bans on lead-based paint. While these general policies may benefit everyone, individuals living in dilapidated city centers are still at a greater risk for lead exposure. Future initiatives like lead abatement support for poor families in older homes may have similar results that aid those living in areas of higher concentrated disadvantage (Reissman et al., 2002).

Developmental prevention programs may also be effective in combating the negative effects caused by lead. Routine prenatal screening could help identify those children in danger of neurological deficits caused by lead early in the developmental process (Gardella, 2001). Once these at-risk families are identified, practitioners can help teach good parenting skills that could serve as a protective factor against lead.

Furthermore, teachers in disadvantaged neighborhoods could be better trained to deal with those children who may have attention problems due to lead exposure. Through identification and intervention from positive social supports, children at risk for criminal activity from the biological effects of lead exposure can be led down a more positive lifetime trajectory. Finally, nutrition programs can be implemented in areas of higher concentrated disadvantage. Helping to introduce more calcium and other lead-fighting nutrition to children that may not be able to afford nutrient-rich diets may also help reduce the effects of lead (Mahaffey, 1990). Future research, however, is needed to ensure that such programs can offset the harmful effects of lead.

The current study has shown the importance of using both biological and social correlates of crime in the study of criminal behavior at the aggregate level. While these two areas of research often reject each other, this study has demonstrated that future research should seek to know how they interact to more fully understand the correlates of crime rates. Furthermore, if policy makers want to be as effective as possible in the reduction of crime, they must understand the entire story and not just a portion of it told by either social or biological factors.

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