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# Association of Childhood Blood Lead Levels with Firearm Violence Perpetration and Victimization

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ASSOCIATION OF CHILDHOOD BLOOD LEAD LEVELS WITH FIREARM VIOLENCE  
PERPETRATION AND VICTIMIZATION

by  
Lindsay R. Emer

A Dissertation Submitted in  
Partial Fulfillment of the  
Requirements for the Degree of

Doctor of Philosophy  
in Public Health

at  
The University of Wisconsin-Milwaukee  
December 2017

## ABSTRACT

### ASSOCIATION OF CHILDHOOD BLOOD LEAD LEVELS WITH FIREARM VIOLENCE PERPETRATION AND VICTIMIZATION

by

Lindsay R. Emer

The University of Wisconsin-Milwaukee, 2017  
Under the Supervision of Professor Dr. Lance S. Weinhardt

Over 10,000 Americans are killed in firearm homicides each year, and an additional 40,000 are injured in nonfatal shootings. There is a significant public health need to identify risk factors that can be modified to prevent firearm violence. Environmental lead exposure is a demonstrated neurotoxicant which causes behavior changes that are known to be criminogenic. More recent research has demonstrated that homicides and nonfatal shootings differ by the circumstances that lead to the shootings (i.e. gang, domestic violence, arguments) and aggregating them could lead to biased results. Although studies have found a relationship between childhood lead exposure and criminal behaviors, no studies have examined individual-level childhood lead exposure and firearm homicide and nonfatal shooting risk, including in the context of shooting type, and there is limited conceptual understanding of this relationship.

For this study, I performed a comprehensive review of the literature and developed a conceptual model illustrating the corresponding factors involved in childhood lead exposure and firearm violence to deepen our conceptual understanding of the relationship. Next, I conducted a retrospective cohort study using linked longitudinal data from the Milwaukee Health Department, Milwaukee Public Schools and Milwaukee Homicide Review Commission on 89,129 individuals born between June 1st, 1986 - December 31st, 2003 who had at least one

valid blood lead test result reported to the Milwaukee Health Department in the first 6 years of life and had stable childhood and adolescent residency to evaluate the association between childhood lead exposure and firearm violence victimization and perpetration. Victims or perpetrators of firearm violence were identified in the Milwaukee Homicide Review Commission homicide and nonfatal shooting database between January 1st, 2005 and December 31st, 2015. Last, I conducted a study on 1091 victims and 589 perpetrators of firearm violence identified in the Milwaukee Homicide Review Commission homicide and nonfatal shooting database who had at least one valid blood lead test result reported to the Milwaukee Health Department in the first 6 years of life to evaluate the association between childhood lead exposure and shooting type.

A conceptual model illustrating the relationship between childhood lead exposure and firearm violence is described with factors included at the Individual, Peer, Family, Multilevel, and Policy levels to guide future research, policy and practice. After adjustment for confounders, our results of the retrospective cohort study show that for every 1 $\mu$ g/dL increase in the mean or peak childhood lead level, the odds of an individual becoming a victim of firearm violence increases (Lead Mean: OR 1.04, 95% CI 1.03, 1.045; Lead Peak: OR 1.02, 95% CI 1.01, 1.03) and the odds of an individual becoming a perpetrator of firearm violence increases (OR 1.03, 95% CI 1.02, 1.04; Lead Peak: OR 1.02, 95% CI 1.01, 1.023). When childhood blood lead levels were categorized, a significant dose-response relationship was found. After adjustment for confounders, the results of our final study show that the relative risk of being a victim of an Argument/Fight-related shooting compared to an Other type of shooting is 37% higher for each 5  $\mu$ g/dL increase in the mean childhood lead level (RRR 1.37, 95% CI 1.02, 1.85). The relative risk of being a perpetrator of an Argument/Fight-related shooting compared

to a Retaliation-related shooting is 27% higher for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, after adjustment for confounding (RRR 1.27, 95% CI 1.03, 1.55). Our findings confirmed earlier clinical observations and recent research that have linked childhood lead exposure with violent crime and, more specifically, argument-related firearm violence, creating greater urgency for primary and secondary childhood lead exposure prevention and paving the way for strategies to reduce the occurrence of firearm violence.

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To everyone who has supported me along the way,  
especially my husband,  
and to the residents of Milwaukee.

## TABLE OF CONTENTS

Abstract	ii
List of Figures	ix
List of Tables	x
Acknowledgements	xi
CHAPTER	
1. Introduction	1
Background	2
Prevalence and Consequences of Firearm Violence	2
Defining Firearm Violence	5
Lead and Firearm Violence	7
Firearm Violence Prevention	8
Gaps in the Research	10
Specific Aims	17
Literature Review	20
Background on Lead	20
Sources of Lead Exposure	20
Lead Exposure in Children	22
Disparities in Lead Exposure	23
History of “Safe” Levels of Exposure	24
Lead in Wisconsin	25
Lead in Milwaukee	27
Lead and the Child Brain	29
Lead and Violence	30
2. Manuscript #1 - Childhood Lead Exposure and Firearm Violence: A Conceptual Model	35
3. Manuscript #2 - Association of Childhood Blood Lead Levels with Firearm Violence Perpetration and Victimization in Milwaukee	75
4. Manuscript #3 - Association of Childhood Blood Lead Levels with Firearm Violence Perpetration and Victimization by	106



	Shooting Type	
5.	Integrated Discussion, Conclusions and Recommendations	137
	Discussion	137
	Implications	143
	Limitations	146
	Recommendations/Conclusions	147
6.	References	153
7.	Appendix: Shooting Type Definitions	184
8.	Curriculum Vitae	186

## LIST OF FIGURES

### **Chapter 1**

Figure 1: Individual-Level Pathways of Lead Exposure to Delinquent  
and Criminal Behaviors 16

Figure 2: Community-Level Pathways of Lead Exposure to Higher Crime Rates 16

### **Chapter 2**

Figure 1: Individual-Level Pathways of Lead Exposure to Delinquent  
and Criminal Behaviors 41

Figure 2: Community-Level Pathways of Lead Exposure to Higher Crime Rates 41

Figure 3: Conceptual Model of Lead Exposure and Firearm Violence 71

### **Chapter 3**

Figure 1: Study Population Flow Diagram 83

Figure 2: Sample Datasets Timeline 85

## LIST OF TABLES

### **Chapter 1**

Table 1. Firearm Homicides, Nonfatal Shootings and Total Violent Crime in Milwaukee	11
---	----

### **Chapter 3**

Table 1: Study Variables	90
Table 2: Population Characteristics of Sample Population, Victims and Perpetrators	93
Table 3: Odds ratios for the association between childhood lead levels and firearm violence victimization and perpetration in Milwaukee	96

### **Chapter 4**

Table 1: Study Variables	120
Table 2: Population Characteristics of Victims of Firearm Violence in Milwaukee	125
Table 3: Population Characteristics of Perpetrators of Firearm Violence in Milwaukee	126
Table 4: Odds ratios and relative risk ratios for the association between childhood lead levels and shooting type in Milwaukee	127

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# ASSOCIATION OF CHILDHOOD BLOOD LEAD LEVELS WITH FIREARM VIOLENCE PERPETRATION AND VICTIMIZATION

## CHAPTER 1: INTRODUCTION

### INTRODUCTION

In August 2016, there were 24 homicides in Milwaukee, 20 of which were firearm homicides (Milwaukee Journal Sentinel, 2016). This was the deadliest month in Milwaukee history since July 1991, the month the victims of serial killer Jeffrey Dahmer were discovered (Luthern, 2016a). While Chicago received national media coverage for declaring August 2016 its deadliest month in nearly two decades, Milwaukee had a higher per capita rate of killings in August (4 per 100,000 people) than Chicago (3.3 per 100,000 people) (Luthern, 2016a). The majority of firearm violence does not attract national media attention, yet it remains a persistent, preventable, public health problem in the United States (U.S.). Identifying key risk and protective factors for firearm violence is paramount to developing effective prevention strategies.

For this study, I will examine childhood lead exposure as a key risk factor for firearm violence. In chapter one, I describe the problem and provide background information on firearm violence. Next, I introduce childhood lead exposure as a potential key risk factor for firearm violence and conduct a literature review on lead and childhood lead exposure. The chapter continues with the current gaps in the research and then ends with the specific aims of the study.

Chapter two through four are three separate manuscripts that align with the specific aims of this study. Chapter two reviews the relevant literature and proposes a conceptual framework for understanding the pathways between childhood lead exposure and firearm violence. I

examine the linkages between the lead literature and firearm violence literature and propose a conceptual framework illustrating these linkages. In chapter three, I evaluate the extent to which childhood lead exposure may contribute to firearm violence perpetration or victimization by estimating the association using logistic regression and controlling for confounding. In chapter four, I evaluate the extent to which childhood lead exposure may contribute to argument-related firearm violence perpetration or victimization compared to other types of firearm violence by estimating the relative risk using multinomial logistic regression methods and controlling for confounding. Finally, chapter five closes the dissertation with an integrated discussion that will include limitations of the research and recommendations for future research.

## **Background**

### **Prevalence and Consequences of Firearm Violence**

Nationwide, an average of over 200 people lose their lives and 1200 people are injured by firearm violence each week (Center for Disease Control and Prevention, 2015). More youth aged 10-24 years old die from homicide (80% of which is perpetrated with a firearm) than cancer, heart disease, birth defects, flu and pneumonia, respiratory disease, stroke and diabetes combined (David-Ferdon & Simon, 2014). Worldwide, the United States vastly outpaces its counterparts in firearm violence. If we examine firearm deaths among 24 countries with similar incomes and populations, 80% of all firearm deaths occur in the United States (American Public Health Association, 2015). Children are especially vulnerable to firearm violence in the U.S. compared to children in other developed nations. Of all children under 14 years old killed by firearms in this same group of 24 nations, 87 percent are U.S. children killed in the U.S. (American Public Health Association, 2015).

Overall, the rates of violent crime including murder/nonnegligent manslaughter have been on the decline since the early 1990s (Federal Bureau of Investigation, 2017). Despite this downward trend, some larger cities have experienced an uptick in certain violent crimes in recent years (Davey & Smith, 2015; Robertson, 2016). For example, 35 cities saw an increase in murders in the first 8 months of 2015 compared to the first 8 months of 2014 (Davey & Smith, 2015) and about two dozen cities experienced notable increases in murders in the first quarter of 2016 compared to the first quarter of 2015 (Major Cities Chiefs Association, 2016). At the same time, even with the recent spikes in some cities, the number of murders in these cities remains far below those of the late 1980s and early 1990s and many others have shown a decline or remained the same (Davey & Smith, 2015).

Locally, Milwaukee is experiencing increasing firearm violence. In 2015, there were 145 homicides in Milwaukee, the highest number recorded since 1993 (Milwaukee Homicide Review Commission, 2015a). Of the 145 homicides, 119 of them were firearm homicides (Milwaukee Homicide Review Commission, 2015a). That same year, there were 633 nonfatal shootings - the highest amount recorded since the Milwaukee Homicide Review Commission began tracking nonfatal shootings in 2006 (Milwaukee Homicide Review Commission, 2015a).

Fatal and nonfatal shootings disproportionately affect young, African American males living in socially and economically disadvantaged urban areas. Nationally, African American males between the ages of 15-29 years old are killed during firearm-related assaults at a rate nearly 10 times that of White males of the same age (15-19 years: 4.9 vs. 45/100,000; 20-24 years: 9.1 vs. 86.6/100,000; 25-29: 7.7 vs. 78.6/100,000) (Heron, 2015). Locally, these firearm-related assault disparities can be even greater. A 2015 mid-year report by the Milwaukee Homicide Review Commission found that in 2014, African American men ages 15-24 years old

were 24 times more likely to be involved in a nonfatal shooting than White males of the same age group (47 vs. 1171/100,000) (Milwaukee Homicide Review Commission, 2015b).

Estimates of the financial costs of firearm violence in the U.S. range from \$100 billion to more than \$450 billion per year (American Public Health Association, 2015; Cook & Ludwig, 2002; Miller, Cohen, & Wiersema, 1996). One estimate of the societal costs per shooting, including ambulance transport, healthcare (medical and mental health), insurance claims processing, criminal justice response, loss of employment and wages, and reduced quality of life, was \$5.1 million for each fatal shooting and \$433,000 for each nonfatal shooting that resulted in a hospital admission (American Public Health Association, 2015). And these estimates do not include intangible costs such as pain and suffering and living in fear of firearm violence (Cook & Ludwig, 2002). A more recent study examined the cost of homicides committed by 654 convicted and incarcerated murderers (DeLisi et al., 2010). They found that the cost of each homicide, including victim costs, criminal justice system costs, offender productivity costs, and the amount of money that citizens would be willing to pay to prevent crimes averaged more than \$17.25 million (DeLisi et al., 2010). Victims of nonfatal shootings may suffer from physical disabilities, short or long term physical impairments and mental health problems as a result of a nonfatal shooting injury (Fowler, Dahlberg, Haileyesus, & Annest, 2015). For example, studies have shown that nonfatal firearm injury is the leading cause of spinal cord injuries in the United States (Hemenway, 2006). Spinal cord injuries resulting from firearm violence are also more likely to result in paraplegia than other types of spinal cord injuries (McKinley, Johns, & Musgrove, 1999). In addition to the human toll for victims and their families, firearm violence can increase health care costs, decrease property values and disrupt social services in certain neighborhoods (Cook & Ludwig, 2002).



It is estimated that as many as 10 million Americans have experienced the murder of a family member or a close friend (American Public Health Association, 2015). A study by Finkelhor et al found that more than 25% of children witnessed an act of violence in their home, school or community in the past year (Finkelhor, 2009). Of these children, more than 5% had witnessed a shooting (Finkelhor, 2009). Children exposed to violence can experience short and/or long-term psychological effects and inhibited brain development, and are at increased risk of negative health outcomes (Garbarino, Bradshaw, & Vorrasi, 2002). These children are also at increased risk of future firearm violence perpetration and victimization. Studies show that one predictor of future firearm violence perpetration is previous firearm violence exposure (Weaver, Borkowski, & Whitman, 2008). Future perpetration risk was consistent regardless of whether children were injured by firearm violence, witnessed firearm violence or lived in neighborhoods with high levels of firearm violence (Garbarino et al., 2002; Weaver et al., 2008).

### **Defining Firearm Violence**

Aggression is defined as a behavior intended to harm someone who does not consent to be harmed (B. J. Bushman & Huesman, 2010; B. J. Bushman et al., 2016). Violence is defined as an extreme form of aggression where the intention of the behavior is serious physical harm or death (B. J. Bushman & Huesman, 2010; B. J. Bushman et al., 2016). Firearm violence therefore is violence that results in a fatal or nonfatal “firearm shot wound or penetrating injury from a weapon that uses a powder charge to fire a projectile” (Fowler et al., 2015, pg. 6).

According to the National Electronic Injury Surveillance System (NEISS), injury intent is defined as either assault/intentional, self-inflicted, unintentional (accidental) or unknown intent, and legal intervention (United States Consumer Product Safety Commission, 2016). The context

and circumstances surrounding firearm violence varies greatly by intent, therefore, this research is only examining fatal and nonfatal firearm violence where the intent is assault/intentional. Additionally, there are differences in the types of fatal and nonfatal firearm assaults. Mass rampage shootings, like the Newtown massacre, typically capture the most media attention, but are relatively rare events. The most prevalent types of fatal and nonfatal firearm assaults in the U.S. are street shootings (B. J. Bushman et al., 2016). Mass rampage shootings and street shootings are shootings defined by the location of the shooting and the characteristics of the shooter. Mass rampage shootings typically happen in a low-crime, small, rural town or suburb (92% of incidents in Newman et al study (Newman & Fox, 2005)). They are predominantly carried out by white males from middle class rural or suburban families (85% in Newman et al study (Newman & Fox, 2005) and 76% in Vossekui et al study (Vossekui, 2002)) who do not have a history of violent offending (63% were never in trouble in Vosselkui et al study (Vossekui, 2002)) and many commit suicide following the act (40% in Blair and Schweit study) (Blair & Schweit, 2014; B. J. Bushman et al., 2016). Conversely, street shootings occur in urban settings on the street or in homes and are carried out by a small number of mostly non-white offenders living in neighborhoods with high concentrations of poverty, crime and segregation who often have a history of violent offending (B. J. Bushman et al., 2016). The victims of street shootings share many of the same characteristics as the perpetrators. The majority of Milwaukee's shootings are street shootings. For example, in 2015, 88% of homicides and 84% of nonfatal shootings in Milwaukee occurred in a residence, the street or a vehicle and 82% of homicides and 83% of nonfatal shootings happened in Milwaukee's lowest SES zip codes (Milwaukee Homicide Review Commission, 2015a). Most homicide and nonfatal shooting suspects and victims were African American (suspects - homicide: 80%, nonfatal

shooting: 87%; victims - homicide: 84%, nonfatal shooting: 88%) males (suspects - homicide: 93%, nonfatal shooting: 90%; victims - homicide: 88%, nonfatal shooting: 88%) between the age of 18 - 29 years old (suspects - homicide: 70%, nonfatal shooting: 67%; victims - homicide: 57%, nonfatal shooting: 57%) with a criminal history (suspects - homicides: 99%, nonfatal shootings: 97%; victims - homicide: 83%, nonfatal shooting: 77%) (Milwaukee Homicide Review Commission, 2015a). This research will focus solely on street shootings.

### **Lead and Firearm Violence**

Environmental stressors are defined as “external physical, chemical, biological, social and economic factors that influence a person’s comfort, performance, health, safety and well-being” (Hwang, 2007, pg. 313). One environmental stressor that is a potential key risk factor for future violent crime is childhood lead exposure (Boutwell et al., 2016; Feigenbaum & Muller, 2016; D. M. Fergusson, Boden, & Horwood, 2008; Mielke & Zahran, 2012; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; Reyes, 2007; P. B. Stretesky & Lynch, 2001; J. P. Wright et al., 2008). Lead is an environmental toxicant that is particularly harmful to children and may cause irreversible, long term damage to a child’s brain. Varying degrees of lead exposure during childhood have shown to have deleterious effects on intelligence and cognition, (Baghurst et al., 1992; Banks, Ferretti, & Shucard, 1997; D. C. Bellinger, Stiles, & Needleman, 1992; Canfield et al., 2003; K. N. Dietrich, Berger, Succop, Hammond, & Bornschein, 1993; B. P. Lanphear et al., 2005) behavior, (K. N. Dietrich, Douglas, Succop, Berger, & Bornschein, 2001; H. L. Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996; P. B. Stretesky & Lynch, 2004; J. P. Wright et al., 2008) and brain structure (Cecil et al., 2008). Research suggests that the physical, mental and behavioral effects of childhood lead exposure may be conducive to future criminal behavior, including violent crime (Narag, Pizarro, & Gibbs, 2009). For example, lead exposure

is associated with cognitive impairments such as lower IQ, poor executive functioning, and attention-deficit/hyperactivity disorder (ADHD), all of which are criminogenic (Narag et al., 2009, pg. 963).

## **Firearm Violence Prevention**

There is a general consensus among researchers that firearm violence is the result of the complex interplay of biological, social and environmental risk and protective factors experienced at the individual, relationship, community and societal level over the life course (B. J. Bushman et al., 2016; David-Ferdon & Simon, 2014; Halliday-Boykins & Graham, 2001; Hong, Cho, Allen-Meares, & Espelage, 2011; Rubens & Shehadeh, 2014; Seal, Nguyen, & Beyer, 2014; World Health Organization (WHO), 2014). The prevailing approach to understanding the causes of adult violence and crime has focused heavily on social factors such as poverty, poor education, family instability, and delinquent peer groups (Carpenter & Nevin, 2010; Taylor, Forbes, Opeskin, Parr, & Lanphear, 2016). Most violence prevention interventions have involved targeting these factors (Carpenter & Nevin, 2010). In 2015, the CDC compiled a comprehensive list of the current evidence-based approaches to preventing youth violence. This list includes the following approaches: universal school-based, parenting skills and family relationship building, intensive family-focused, policy, environmental and structural, street outreach and community mobilization, early childhood home visitation and early childhood education (David-Ferdon & Simon, 2014). A review by Bushman et al also lays out several evidence-based youth violence prevention strategies. These strategies include developing self-control and social competence skills, strengthening effective parenting and family-based protective factors, reducing youth access to firearms, reducing alcohol and substance abuse in youth, and improving school climates (B. J. Bushman et al., 2016). While there has been some

success of current prevention programs, generally, success in preventing future violent behavior, especially at later ages, has been very limited (Carpenter & Nevin, 2010).

Present day firearm violence prevention interventions in Milwaukee tend to be reactive or focus on downstream strategies. For example, historically, firearm violence prevention efforts have focused on law enforcement strategies (Bieler, Kijakazi, LaVigne, Vinik, & Overton, 2016). Despite these efforts, high rates of firearm violence in communities of color persist (Bieler et al., 2016). Yet recent events suggest that there is the political will to begin taking a more public health approach to firearm violence prevention in Milwaukee (Bieler et al., 2016). There was a firearm violence summit in Milwaukee at the beginning of August 2016 that brought local leaders together to discuss potential firearm violence prevention strategies for the city (Luthern, 2016b). The discussion revolved around recommendations to come out of a recent study by the Joyce Foundation, Urban Institute and the Joint Center for Political and Economic Studies (Bieler et al., 2016; Luthern, 2016a; Luthern, 2016b). The recommendations from the report included reducing access to firearms for people at high risk of engaging in violence, improving relations between police and communities of color, investing in evidence-based social service interventions such as job training, youth programming and mental health and substance abuse counseling, and creating a comprehensive, city-wide violence prevention plan (Bieler et al., 2016; Luthern, 2016a; Luthern, 2016b).

Childhood lead exposure is a biologically plausible environmental predictor of firearm violence. Despite the evidence linking it to violent and criminal behavior, lead exposure prevention and abatement are not currently considered firearm violence prevention strategies. Determining the extent to which childhood lead exposure contributes to firearm violence offers us a route of prevention at the societal level. Based on recent work in

Milwaukee, the findings from this research would be supportive of the current efforts to apply a public health approach to firearm violence prevention.

### **Gaps in the Research**

While elevated childhood lead levels have been found to be associated with juvenile delinquency (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; H. L. Needleman, McFarland, Ness, Fienberg, & Tobin, 2002; Olympio, Gonçalves, Günther, & Bechara, 2009) and adult criminal behavior and violent offending, (D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004) researchers have yet to examine the relationship between childhood lead exposure and adult firearm violence, specifically. Pihl and Ervin found that adult male inmates convicted of violent crimes were more likely than adult male inmates convicted of property crimes to have elevated hair lead levels, but violent crime was aggregated to include murder, assault, armed robbery, and violent rape (Pihl & Ervin, 1990). Similarly, Wright et al reported a positive relationship between prenatal and childhood blood lead concentrations and the number of adult arrests (J. P. Wright et al., 2008). Once again, violent arrests were aggregated to include murder, rape, domestic violence, assault, robbery and possession of a firearm (J. P. Wright et al., 2008). Both Needleman et al and Dietrich et al found links between lead exposure and self-reported antisocial and delinquent behaviors (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; H. L. Needleman et al., 2002). While antisocial and delinquent behaviors are both risk factors for firearm violence, they are not firearm violence specifically. Finally, Stretesky and Lynch and Nevin found positive associations between aggregate lead levels and crime trends, including homicide and violent crime, but they did not examine firearm violence specifically (R. Nevin, 2000; P. B. Stretesky & Lynch, 2001; P. B. Stretesky & Lynch, 2004).

It is important to disaggregate violent crime data because violent crimes differ in many ways, such as by frequency, lethality and sanction. Violent crime varies in frequency by type of crime. By FBI Uniform Crime Reporting (UCR) standards, violent crime is defined as homicide, rape, aggravated assaults, and robberies. Nonfatal shootings are included under these definitions as either aggravated assaults or robberies. Firearm homicides and nonfatal shootings make up a small percentage of annual violent crime in Milwaukee (Table 1). On average, from 2009 - 2013, firearm homicides made up less than 1% of total violent crime in Milwaukee. During that same period, nonfatal shootings made up a little over 6% of total violent crime in Milwaukee.

**Table 1: Firearm Homicides, Nonfatal Shootings and Total Violent Crime in Milwaukee**

Year	FH	NFS	Total VC	FH/VC	NFS/VC	FH + NFS/VC
2009	53	439	7382	.7%	6%	6.7%
2010	66	402	6974	.9%	5.8%	6.7%
2011	60	473	6940	.9%	6.8%	7.7%
2012	75	500	7936	.9%	6.3%	7.2%
2013	81	530	8025	1%	6.6%	7.6%

FH = Firearm Homicide, NFS = Nonfatal Shooting, VC = Total Violent Crime

Firearm violence is the most lethal of all violent crime. The case fatality rate (the proportion of individuals injured or infected with a disease who die as a result of that injury or disease) of firearm-related assault in Milwaukee in 2015 was 15.8% (Milwaukee Homicide Review Commission, 2015a). In other words, 15.8% of individuals who were victims of a

firearm assault died because of that assault. In comparison, the case fatality rate for *Cryptosporidium* is .02% (Milwaukee Homicide Review Commission, 2015a). The Center for Disease Control and Prevention estimates that Milwaukee spent more than \$2 million dollars in response to the *Cryptosporidium* outbreak in 1993 (Corso et al., 2003).

Violent crimes also differ in ways such as severity, certainty and swiftness of arrest. Sanctions for certain violent crimes involve lengthy periods of incarceration, while others, on average, may result in shorter periods of confinement (Cherry & List, 2002). Increased sanctions may deter some violent crimes but not others. Violent crimes often characterized as crimes of passion, i.e. murder and rape, may not be deterred by the threat of increased sanctions (Cherry & List, 2002). Additionally, the probability of arrest differs regionally and across violent crime types. In 2015, Milwaukee's homicide clearance rate was approximately 60%, down from about 90% in 2008 (Milwaukee Police Department, 2015). The national average clearance rate for homicides has hovered around 55-60% for the past 10 years (Milwaukee Police Department, 2015). Rape is highly underreported and therefore clearance rates remain consistently low nationwide.

Aggregating violent crime data can produce biased results. A study by Cherry and List found significantly different parameter estimates when they examined the deterrence hypothesis with aggregated and disaggregated crime data (Cherry & List, 2002). The study examined three models: one model aggregated all types of crime into one crime index, one model aggregated crime into the two major FBI crime groups (violent and property) and one model disaggregated crime types entirely (murder, rape, robbery, assault, burglary, larceny and motor vehicle). The study found significant variability in the parameter estimates in each model. In some cases, the sign for the parameter estimate switched when the crime types were disaggregated (Cherry &



List, 2002). For example, this occurred when they examined the effect the size of the police force had on crime. The parameter estimate for the aggregated crime index was .413, but for the disaggregated model, the parameter estimates fluctuated between -.119 (assault) and -.395 (larceny) (Cherry & List, 2002).

When firearm violence has been disaggregated from violent crime, prior research has focused predominantly on examining either perpetration or victimization risk of firearm homicides. While homicide data tends to be more robust because of the resources spent on homicide investigations, homicides are relatively rare events (Papachristos, Wildeman, & Roberto, 2015). Nonfatal shootings are far more common than homicides. Even with the record number of homicides in 2015, there were over five times more nonfatal shootings than firearm homicides that year in Milwaukee (Milwaukee Homicide Review Commission, 2015a). Despite being far more common and having similar causes and consequences, nonfatal shootings remain significantly understudied (Lee, 2012; Papachristos et al., 2015).

In addition to focusing on homicides, prior research has historically treated firearm violence as a dichotomous outcome. More recent research has demonstrated that shootings differ by the circumstances that lead up to the shootings and the motives behind them (Flewelling & Williams, 1999; J. M. Pizarro, 2005; J. M. Pizarro, 2008). For example, a shooting could be during a drug transaction, a robbery or a dispute between intimate partners. Different shooting types may require different prevention strategies. Prior research has examined deviant homicides, (Decker, 1996) drug homicides, (Goldstein, 1985) family homicides, (Diem & Pizarro, 2010), gang homicides, (Decker & Curry, 2002; Papachristos, 2009) as well as other groupings (Robbery, Other felony, Acquaintance and Intimate partner homicides) (Parker, 1989). The results of this research suggest that homicides (and nonfatal shootings) are not

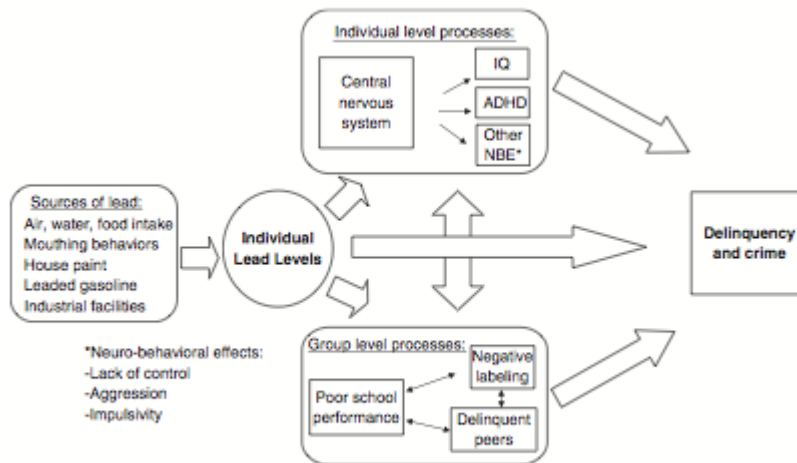
unitary offenses and support the utility of categorizing shootings into more specific and homogenous categories in future research (Flewelling & Williams, 1999; J. M. Pizarro, Zgoba, & Jennings, 2011).

One of the reasons why there is limited research on the public health risk factors of firearm violence as well as on nonfatal shootings and the different shooting types, is the lack of data available to support this research. This research requires large-scale, integrated data linking longitudinal public health and criminal justice data. In addition, it demands access to confidential police records and proper coding of those records. The Milwaukee Homicide Review Commission has been able to overcome both of these limitations. From 2005 - 2015, the Commission has had direct access to Milwaukee Police Department crime incident records. Staff have been consistently collecting and coding data on homicides since 2005 and nonfatal shootings since 2006. Additionally, the Commission developed an integrated data system called DataShare MKE. Large-scale data integration is complete for the following data sets: Milwaukee Police Department arrests and incidents, Milwaukee County Pretrial Services, Milwaukee Health Department (births, immunizations, blood lead, and communicable disease), Milwaukee County District Attorney's Office, Wisconsin State Courts, Milwaukee Public Schools, ATF eTrace, National Integrated Ballistic Information Network and ShotSpotter®. DataShare MKE gives us access to identifiable and linkable data from multiple domains (e.g., community, school, family, individual) and at multiple levels (i.e., community, individual), including information from across the life course (i.e., childhood, adolescence, early adulthood).

While recent research has begun examining the link between childhood lead exposure and adult delinquency and crime, there is limited literature describing the theory driving this

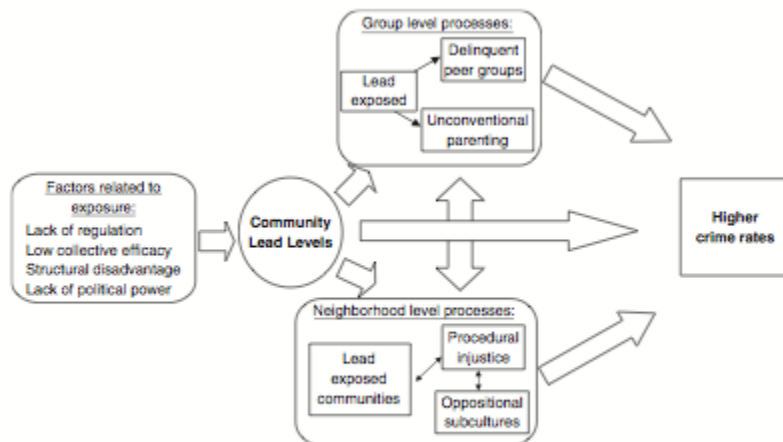
relationship. A literature review by Narag, Pizarro and Gibbs examining the implications of lead exposure on criminological theory appears to present the most comprehensive illustration of the individual and community-level pathways from childhood lead exposure to delinquency and crime (See Figures 1 and 2) (Narag et al., 2009). Narag et al posits that, at the individual level, the relationship between individual lead exposure and delinquency and crime are mediated by a combination of individual level processes such as IQ, ADHD, lack of self-control, aggression and impulsivity, and group level processes such as poor school performance, negative labeling and delinquent peers (Narag et al., 2009). At the community level, the relationship between community lead exposure and crime rates is mediated by a combination of group level processes such as delinquent peer groups and unconventional parenting, and neighborhood level processes such as procedural injustice and oppositional subcultures (Narag et al., 2009). While the conceptual model presented by Narag et al is a significant contribution to the literature, it has limitations when it is applied to specific crimes such as street shootings. The key risk factors for street shootings fall under the following categories: individual (neurobiological, personality, and alcohol and drug use), family, school, neighborhood, and access to firearms (B. J. Bushman et al., 2016). The conceptual framework presented by Narag et al captures most of the factors that fall into these categories, but it does not fully encompass all the key risk factors of street shootings. For example, the model includes personality traits such as aggressiveness, impulsivity and difficulties with self-control, all of which increase an individual's risk of street shooting perpetration, but the model does not include other key risk factors such as individual-level alcohol and other drug use, access to firearms, and neighborhood level violence.

**Figure 1**



**Figure 1: Individual-Level Pathways of Lead Exposure to Delinquent and Criminal Behaviors**

**Figure 2**



**Figure 2: Community-Level Pathways of Lead Exposure to Higher Crime Rates**

## Specific Aims

Over 10,000 Americans are killed in firearm homicides each year, and an additional 40,000 are injured in nonfatal shootings. There is a significant public health need to identify risk factors that can be modified by policies and interventions to prevent firearm homicides and nonfatal shootings. The prevailing approaches to understanding the causes of violence and crime have focused heavily on social factors such as poverty, poor education, family instability, and delinquent peer groups. While current prevention programs targeting these factors have yielded some positive results, generally success in preventing future violent behavior, especially at later ages, has been limited.

Environmental lead exposure is a demonstrated neurotoxicant which causes behavior changes that are known to be criminogenic, such as impulsivity and aggression. Although studies have found a relationship between individual-level childhood lead exposure and criminal behaviors as well as community-level childhood lead exposure and crime rates, no studies have examined individual-level childhood lead exposure and firearm homicide and nonfatal shooting risk, specifically. Childhood lead exposure is a biologically plausible environmental predictor of firearm violence and an important research area which has not been addressed.

Prior firearm violence research has focused on either perpetration or victimization risk of firearm homicides. Despite being far more common and having similar causes and consequences, nonfatal shootings remain significantly understudied. In addition, prior research has typically treated firearm violence as a dichotomous outcome. More recent research has demonstrated that homicides and nonfatal shootings differ by type (i.e. gang, domestic violence, arguments) and aggregating them could lead to biased results.

To overcome previous limitations and address current gaps in the research, I will examine

the relationship between individual level childhood lead exposure and firearm homicide and nonfatal shooting perpetration and victimization risk, including by shooting type. I hypothesize that higher levels of lead exposure during childhood will lead to higher odds of firearm violence victimization and perpetration, especially argument-related shootings, after controlling for potential confounding variables (sex, race, SES, and time). To address these hypotheses, I will first use a retrospective cohort study using linked longitudinal data from the Milwaukee Health Department, Milwaukee Public Schools and Milwaukee Homicide Review Commission on 89,129 individuals born between June 1st, 1986 - December 31st, 2003 who had at least one valid blood lead test result reported to the Milwaukee Health Department in the first 6 years of life, were living in the city of Milwaukee at the time of all of their blood lead tests, and were present in Milwaukee Public Schools records between July 1st, 2004 - July 18th, 2016. Firearm violence incidents were identified in the Milwaukee Homicide Review Commission homicide and nonfatal shooting database between January 1st, 2005 and December 31st, 2015 and then individuals were identified as victims or perpetrators linked to those incidents. Then I will conduct a second study using linked longitudinal data from the Milwaukee Health Department and Milwaukee Homicide Review Commission on 1091 victims and 589 perpetrators only who had at least one valid blood lead test result reported to the Milwaukee Health Department in the first 6 years of life. The shooting type was identified in the Milwaukee Homicide Review Commission homicide and nonfatal shooting database.

My specific aims are:

**Aim 1.** To deepen our conceptual understanding of the relationship between childhood lead exposure and firearm violence, I will review the relevant literature and discuss the corresponding factors involved with lead exposure and firearm violence and propose a conceptual model

illustrating this relationship.

**Aim 2.** To evaluate the extent to which childhood lead exposure may contribute to later firearm violence victimization and perpetration, I will estimate the association using traditional regression methods and control for confounding.

**Aim 3.** To evaluate the extent to which childhood lead exposure may contribute to later argument-related firearm violence perpetration and victimization compared to other types of firearm violence perpetration and victimization, I will estimate the relative risk using traditional regression methods and control for confounding.

The findings from this study will elucidate the extent to which elevated childhood lead exposure is a risk factor for firearm violence and, more specifically, argument-related firearm violence, paving the way for strategies to reduce the occurrence of firearm violence.

## **LITERATURE REVIEW**

### **Background on Lead**

Lead is a naturally-occurring metal found in the earth's crust (US Environmental Protection Agency, 2016). It is a soft, malleable and heavy metal that is bluish-white when freshly cut and tarnishes to a grayish color when exposed to air (Occupational Safety and Health Administration, 2016). Lead is found in small amounts in ore with other elements such as silver, zinc or copper (National Institute of Environmental Health Services, 2016). Despite naturally occurring in small amounts, there is an abundance of lead in the earth and it is easy to extract (National Institute of Environmental Health Services, 2016). Behind China and Australia, the U.S. is the third largest producer of lead in the world (Occupational Safety and Health Administration, 2016).

Lead has been mined for centuries for industrial use (Narag et al., 2009; Occupational Safety and Health Administration, 2016). Lead can be used on its own, mixed with another metal to form an alloy or as a chemical compound (Occupational Safety and Health Administration, 2016). Currently, the primary use of lead in the U.S. is for lead alloy rechargeable electric batteries in automobiles (Occupational Safety and Health Administration, 2016). Lead has been used in a variety of products including: paints, gasoline, ammunition, pipes, cable covering, building material, solder, radiation shielding, collapsible tubes, ceramic glazes, fishing weights, and cosmetics (Narag et al., 2009; Occupational Safety and Health Administration, 2016).

### **Sources of Lead Exposure**

Due to its many uses in industry over centuries, lead can be found in the air, soil, food, water, and in our homes. The source and concentration of lead varies regionally. Lead may



enter the environment from past or current uses. For example, soil may be contaminated because it is the site of a former smelting facility or current automobile emissions are settling to the ground.

Airborne lead comes from emissions of automobiles, airplanes, smelters, battery plants, and industrial facilities (Narag et al., 2009; Occupational Safety and Health Administration, 2016). The settling of airborne lead on to the ground can create lead-contaminated soil and surfaces. Chipping lead-based paint used on home exteriors also contributes to lead-contaminated soil. Once lead enters the soil it can remain there for hundreds of years (Datko-Williams, Wilkie, & Richmond-Bryant, 2014). A review by Datko-Williams, Wilkie, and Richmond-Bryant examining soil lead levels in the United States found no significant change in soil lead levels over the time period evaluated (Datko-Williams et al., 2014). In addition, it found that in all but one city, lead levels were higher in urban compared to suburban areas (Datko-Williams et al., 2014).

Food is another source of lead (Narag et al., 2009). Crops planted in lead-contaminated soil will take up lead as they grow (Narag et al., 2009). Crops grown in fields close to heavily traveled roads may be exposed to more lead from automobile emissions (Narag et al., 2009). In addition, the processing, use of preservatives and use of soldered cans can contaminate food with lead (Narag et al., 2009).

Lead is also found on surfaces inside older residential homes. Lead-based paint was widely used inside homes until its use was banned by the Consumer Product Safety Commission in 1977 (US Consumer Product Safety Commission, 1977). The chips or dust from lead-based paint can contaminate the interior of a home. The dust may be so fine that it is not visible to the

naked eye.

Finally, lead is found in water when drinking water is pumped through leaded pipes (National Institute of Environmental Health Services, 2016; US Environmental Protection Agency, 2016). Upwards of 10 million homes and buildings receive water through service lines that contain lead (National Institute of Environmental Health Services, 2016). Homes built before 1986 are more likely to have lead services lines (National Institute of Environmental Health Services, 2016). Utilities can treat water with an anticorrosive to prevent lead particles from detaching from the pipes and entering the water supply (National Institute of Environmental Health Services, 2016). Lead is more likely to leach off into the water supply if the pipes are disturbed, such as during nearby construction, or when the leaded pipes corrode, which was the case in Flint, Michigan in 2014. The drinking water in Flint became contaminated because the utility did not maintain corrosive controls.

### **Lead Exposure in Children**

The most common pathway to lead exposure varies by group. For the purposes of this paper, we will focus on lead exposure in children. Children are more likely to be exposed to lead than adults due to the nature of their daily activities (D. C. Bellinger, 2004). Children spend most of their day playing close to the ground and exploring their environment by touching objects with their hands and putting their hands in their mouths (D. C. Bellinger, 2004; K. N. Dietrich, 2010). This increases their exposure to contaminated soil, vegetation and dust (K. N. Dietrich, 2010). In addition, children are particularly vulnerable to higher doses of lead exposure compared to adults. Children eat more grams of food per kilogram of body weight, have higher water requirements, intake more air and consume more vegetables than adults (K. N.

Dietrich, 2010). Finally, children's bodies absorb more of the lead they are exposed to compared to adult bodies. Children absorb about 50% of the lead they ingest, while adults only absorb about 10-15% of ingested lead (K. N. Dietrich, 2010). This absorption can be even higher when children have nutritional deficiencies in iron or calcium (D. C. Bellinger, 2004; Bradman, Eskenazi, Sutton, Athanasoulis, & Goldman, 2001; R. O. Wright, Shannon, Wright, & Hu, 1999).

The most common types of lead exposure in the United States are lead-based paint chips and dust in older homes, contaminated soil, and contaminated drinking water. Lead exposure in children most often occurs when they ingest lead-based paint via paint chips, floor dust or soil most commonly found in housing built before 1978 (Nagaraja, 2013). Children of color living in structurally disadvantaged neighborhoods with deteriorating housing are disproportionately exposed to lead (B. P. Lanphear, Dietrich, & Berger, 2003; Narag et al., 2009). When patterns of exposure are examined by group, children of color are less likely to be screened, more likely to have higher lead levels and less likely to be treated when they are exposed (Hwang, 2007; Jones et al., 2009; P. B. Stretesky, 2003).

### **Disparities in Lead Exposure**

Environmental lead levels vary greatly geographically, but the variation is not random. A study by Hird and Reese found that African Americans and Hispanics were more likely than Caucasians to live in a U.S. county with elevated air pollution rates which included lead air concentrations (Hird & Reese, 1998). Using data from the U.S. Census Bureau and the U.S. Environmental Protection Agency, a study by Stretesky found that the counties with the largest proportions of African American youth under 16 years of age had 8% more lead in the air than

counties with no African American youth (P. B. Stretesky, 2003). Risk factors for lead exposure include living in lower income and rental housing, living near major highways and living near industrial zones or rural areas near smelter sites (R. Nevin, 2000). Minorities are more likely than non-minorities to reside in all of these high-risk areas and, due to barriers such economic constraints, minorities are less able to move away from these high lead risk neighborhoods (Narag et al., 2009; P. B. Stretesky & Lynch, 2004).

### **History of “Safe” Levels of Exposure**

In 1892, Australia was the first country to discover lead poisoning in children (H. Needleman, 2004). Like household exposure in the U.S., Australian children were predominantly exposed through consuming deteriorating lead-based paint in the household (H. Needleman, 2004). Australia banned the use of household lead-based paint in 1920 (H. Needleman, 2004).

Childhood lead poisoning was first recognized in the U.S. in 1914 (H. Needleman, 2004). Until a study published by Byers and Lord in 1943, the belief was that lead poisoning resulted in death or a full recovery (without lasting effects) (H. Needleman, 2004). The Byers and Lord study found that 19 of 20 survivors of acute lead poisoning suffered from significant long-term health deficits including behavioral issues and academic difficulties (Byers & Lord, 1943). It wasn't until the 1970s, that researchers began to recognize that even low-level lead exposure can cause deficits in IQ, attention and language (Burdé & Choate, 1972; Landrigan et al., 1975; H. Needleman, 2004; Perino & Ernhart, 1974).

Surmounting evidence from epidemiological studies has lead experts to gradually adjust the threshold defining lead levels as elevated over time. Since 1960, the actionable blood lead

levels have been set at the following levels: 60 µg/dL from 1960-1970, 40 µg/dL from 1970-1975, 30 µg/dL from 1975-1985, 25 from 1985-1991, 10 µg/dL 1991-2012, and 5µg/dL from 2012 to present (K. N. Dietrich, 2010). These are actionable levels, not “safe” levels, as there is no safe threshold for blood lead (Narag et al., 2009). In fact, recent research has found that the association between lead levels and adverse outcomes is similar across a range of blood lead levels (Goodlad, Marcus, & Fulton, 2013; Nigg et al., 2008; Nigg, Nikolas, Knottnerus, Cavanagh, & Friderici, 2010). Most notably, recent studies by Nigg and colleagues found significant associations between lead and ADHD symptoms in samples with below actionable lead levels (Goodlad et al., 2013; Nigg et al., 2008; Nigg et al., 2010).

### **Lead in Wisconsin**

Wisconsin has consistently reported high numbers of lead-poisoned children compared to other states. Wisconsin is among the top nine states nationwide in number of children with blood lead levels of 5 µg/dL or higher (Wisconsin Department of Health Services, 2014). When compared to other states in the Midwest in 2012 (Ohio, Indiana, Michigan and Minnesota), Wisconsin reported the second highest number (6,996) and proportion (7.13%) of lead-poisoned children (Wisconsin Department of Health Services, 2014).

Medicaid-enrolled children in Wisconsin are at three time greater risk of lead poisoning than children who are not enrolled in Medicaid (Wisconsin Department of Health Services, 2014). In 2014, 88% of the lead poisoned children were enrolled in Medicaid (Wisconsin Department of Health Services, 2014). While White children make up the greatest proportion of children tested for lead (44.2%), lead poisoning rates are highest for minority children (Wisconsin Department of Health Services, 2014). Black children made up only a quarter of

children tested in 2014, but represented nearly half of the children found to be lead-poisoned (Wisconsin Department of Health Services, 2014).

In 1998, the Department of Health Services released the Wisconsin Blood Lead Screening Recommendations (Wisconsin Department of Health Services, 2014). Developed by an advisory committee, the guidelines focused on screening children at greatest risk of lead exposure and poisoning (Wisconsin Department of Health Services, 2014). Milwaukee and Racine both have a high proportion of older housing stock which puts children living in those areas at higher risk for exposure. To account for this elevated risk, testing guidelines are different for those two cities than for the rest of the state. The guidelines recommend universal testing of all children living in the cities of Milwaukee and Racine three times before the age of 3 years (12 months, 18 months, and 24 months) (Wisconsin Department of Health Services, 2014). Children ages 3 to 5 years old should be tested if they: live in a house built before 1950, live in a house built before 1978 and recently renovated, have a sibling or playmate with lead poisoning, is enrolled in Medicaid, WIC or is uninsured or if they have no prior tests (Wisconsin Department of Health Services, 2014). Children enrolled in Medicaid are required to have their blood lead tested as part of their Early and Periodic Screening, Diagnostic and Treatment (EPSDT) services at 12 and 24 months of age (HCFA, 1999). Children between 24 and 72 months of age, enrolled in Medicaid and with no prior testing should also receive a blood lead test (HCFA, 1999).

Even with these guidelines, not all children are being tested for blood lead in accordance with these recommendations. In 2014, there were 455,524 children under the age of 6 years old in Wisconsin, but only 87,987 were tested for lead poisoning (19.3%) (Wisconsin Department of Health Services, 2014). Approximately 4.5% of those tested (3,922) had blood lead levels

greater than or equal to 5 µg/dL (Wisconsin Department of Health Services, 2014). The greatest number of children tested for lead poisoning in 2014 were one year old (Wisconsin Department of Health Services, 2014). A total of 38,096 of 75,717 eligible one year olds were tested (50.3%) (Wisconsin Department of Health Services, 2014). For most children, the test at one year old is the only time they are tested. The incidence of lead-poisoned children (first identification of lead poisoning) is highest around 2 years old and the prevalence (total burden of lead poisoning) is highest between 2 and 4 years old (Wisconsin Department of Health Services, 2014). In other words, less children are being tested at the ages when they are most likely to be identified as lead poisoned.

Despite the federal testing requirements, not all Wisconsin children on Medicaid are tested for lead poisoning or tested at the appropriate ages (Wisconsin Department of Health Services, 2014). Only 62% of 1-year-olds, 48% of 2-year-olds, and 16% of children aged 3-5 who were not previously tested were tested in 2014 (Wisconsin Department of Health Services, 2014). Overall, only 42% of Wisconsin children enrolled in Medicaid were tested at the recommended 1 and 2 years of age (Wisconsin Department of Health Services, 2014).

### **Lead in Milwaukee**

Results from a City of Milwaukee Community Health Assessment show that children under 6 years old and living in Milwaukee are at a higher risk for having elevated blood lead levels compared to children living in the rest of the state (City of Milwaukee Health Department, 2016). The percentage of children living in Milwaukee with blood lead levels above the level in which public health action is recommended by the CDC (5µg/dL) has fallen from 38% in 2003 to 10% in 2014, but remains more than twice as high as the statewide prevalence (4.5%) (City of

Milwaukee Health Department, 2016). The proportion of Milwaukee's children 12 to 35 months who were tested has fluctuated over time. In 1997, about 25% of children were tested for lead, in 2011 just over 70% were tested, and in 2014, the number declined to 60% (City of Milwaukee Health Department, 2016).

To help prevent lead exposure in children, the Milwaukee Health Department Childhood Lead Poisoning Prevention Program removes lead from high risk homes. Between 1997 and 2015, the Milwaukee Health Department Childhood Lead Poisoning Prevention Program removed lead from nearly 18,000 homes (City of Milwaukee Health Department, 2016). Unfortunately, funding constraints only allow for 400 remediations per year (City of Milwaukee Health Department, 2016). There are at least 17,000 housing units in Milwaukee where lead paint hazards remain (City of Milwaukee Health Department, 2016). If the funding for lead remediation remains at its current level, it will take Milwaukee 42 years to fully remediate all of these homes (City of Milwaukee Health Department, 2016).

Residents in Milwaukee can also be exposed to lead in their drinking water. In Milwaukee, drinking water is still delivered through lead lateral service pipes to 70,000 residential properties built before 1951 (Behm, 2016b). While Milwaukee uses corrosive controls to prevent lead from leaching into the water, work on and around the pipes, as well as the work involved in replacing them, can disturb the pipes and cause lead to break off into the water (Behm, 2016b). In September of 2016, Mayor Tom Barrett advised all residents living in one of these properties to use water filters (Behm, 2016a). Replacing all of the lead service lines would cost an estimated \$511 to \$756 million (Behm, 2016a).



## **Lead and the Child Brain**

There are five environmental toxicants that are widely recognized to be neuroteratogenic in humans (harmful to fetal brain development): arsenic and arsenical compounds, lead and lead compounds, methylmercury, toluene and PCBs (Grandjean & Landrigan, 2006). Out of all five environmental toxicants, pediatric neuropsychologists are most likely to encounter children with lead exposure.

The developing human brain is particularly vulnerable to injury caused by neurotoxicant exposure compared to fully developed brains of adults (K. N. Dietrich, 2010). Studies show that the developing brain in fetuses and children is one of the most sensitive human organs to damage (K. N. Dietrich, 2010). This vulnerability begins as early as conception (K. N. Dietrich, 2010). Lead crosses the placenta by diffusion and collects in the fetal organs (K. N. Dietrich, 2010). The blood-brain barrier in a fetus or young infant is immature and therefore more permeable to toxicants (K. N. Dietrich, 2010). In addition, a fetus does not possess the capability to metabolize and detoxify toxicants (K. N. Dietrich, 2010). There is no clear delineation between fetal and infant CNS maturation (K. N. Dietrich, 2010). “Processes such as cell proliferation and growth, and especially cortical cellular migration, myelination, dendritic arborization, synaptogenesis, and programmed apoptosis, continue for a considerable period after birth” (K. N. Dietrich, 2010, pg. 218) Since brains are not fully developed until 21 years after conception, they remain vulnerable into early adulthood (K. N. Dietrich, 2010). While it is known that the developing brain is more vulnerable to the impact of toxins, specific windows of vulnerability have not been well-established (B. P. Lanphear, 2015).

According to Silbergeld, lead affects the developing brain in two distinct

ways: neurodevelopmental toxicity and neuropharmacological toxicity (1992) (Silbergeld, 1992). First, it causes neurodevelopmental toxicity by “interfering with the hard wiring and differentiation of the central nervous system (CNS)” (Silbergeld, 1992, pg. 3202). This interference may cause permanent damage to the CNS (Silbergeld, 1992). Second, it causes neuropharmacological toxicity by interacting with calcium and zinc and interfering with “ionic mechanisms of neurotransmission” (Silbergeld, 1992, pg. 3202) These effects on the CNS as it develops result in cognitive and motor impairments in lead-exposed children (K. N. Dietrich, 2010).

### **Lead and Violence**

Elevated childhood lead levels have been found to be associated with juvenile delinquency, (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; Olympio et al., 2009) and juvenile and adult criminal behavior, including violent offending (Denno, 1990; D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; H. L. Needleman et al., 2002; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004) in both cross-sectional and longitudinal research. The lead-delinquency relationship was first reported early in the lead literature. In a study by Byers and Lord in 1943, they found that children with acute lead exposure exhibited violent and aggressive behavioral difficulties (Byers & Lord, 1943). Needleman et al conducted a retrospective cohort study examining the dentine lead levels of 301 male primary school students who participated in the Pittsburgh Youth Study (a study of the developmental predictors of delinquency) (H. L. Needleman et al., 1996). The study found significant associations between elevated dentine lead levels at 11 years old and delinquent, aggressive, internalizing and externalizing behaviors reported by both parents and teachers and self-reported delinquency, after adjusting for covariates (H. L. Needleman et al., 1996). In the

first prospective longitudinal birth cohort study of its kind, Dietrich et al studied the effects of lead on development in 195 urban, inner city children (K. N. Dietrich et al., 2001). Dietrich et al found that prenatal lead exposure was significantly associated with the frequency of delinquent and antisocial behaviors reported by the children's parents and both prenatal and postnatal (average childhood level and level measured at 6 years of age) lead exposure were significantly associated with self-reported frequency of delinquency and antisocial behaviors, after controlling for covariates (K. N. Dietrich et al., 2001).

A longitudinal study by Denno tracked 987 African-American youth born in Philadelphia from birth to age 22 years to try to identify early predictors of juvenile and adult criminal behavior (Denno, 1990). The study found that among males, lead intoxication at age 7 years was one of the strongest predictors of disciplinary problems from ages 13 to 14, juvenile delinquency and adult offenses for male participants (Denno, 1990). Needleman et al found similar results when they examined the relationship between lead and adjudicated delinquents. In a case control study of 194 adjudicated delinquent juveniles and 146 nondelinquent juveniles, the bone lead concentrations of the adjudicated delinquent juveniles were significantly higher than those of the nondelinquent juveniles after controlling for covariates (H. L. Needleman et al., 2002). Adjudicated delinquents were four times more likely than controls to have bone lead concentrations above 25ppm (H. L. Needleman et al., 2002). Fergusson and colleagues examined a 21-year longitudinal New Zealand birth cohort to determine if there was a link between dentine lead exposure measured between 6-9 years old and both officially recorded and self-reported criminal convictions between the ages of 14-21 years old (D. M. Fergusson et al., 2008). They found a significant dose-response relationship between childhood lead levels and rates of violent and property crime (officially recorded and self-reported), after controlling for confounding (D.

M. Fergusson et al., 2008). A study by Pihl and Ervin examined the differences in hair element levels, including lead, between 30 violent and 19 nonviolent males incarcerated in an institute for psychiatrically disturbed criminals (Pihl & Ervin, 1990). Chi-squared analysis revealed that violent offenders had significantly higher lead levels in their hair than the nonviolent offenders (Pihl & Ervin, 1990). These results have been replicated in other correlational studies examining violent criminal offenders as well (Schauss, 1981). More recently, Wright et al examined the relationship between early lead exposure (prenatal, average childhood, and at 6.5 years) and criminal arrests in young adulthood in 250 participants in the Cincinnati Lead Study birth cohort (J. P. Wright et al., 2008). They found that for every 5 $\mu$ g/dL increase in prenatal and 6 year old blood lead concentration, the adjusted total arrest rates were significantly greater (prenatal: RR=1.40, 95% CI 1.07-1.85 and 6.5 years: RR=1.27, 95% CI 1.03-1.57) (J. P. Wright et al., 2008). The adjusted arrest rates for violent crimes were also significantly greater for each 5  $\mu$ g/dL increase in average childhood lead (RR=1.30, 95% CI 1.03-1.64) and 6 year blood lead (RR=1.48, 95% CI 1.15-1.89) (J. P. Wright et al., 2008).

In addition to individual level studies, there is evidence of an association between lead and crime at the city, state, national and international levels. In a 2000 paper, Nevin found that leaded gasoline emissions from vehicle tailpipes explained 90 percent of the variation in violent crime in America (R. Nevin, 2000). Nevin found that leaded gasoline emissions nearly quadrupled from the 1940s through the early 70s and then dropped dramatically as lead was removed from gasoline (R. Nevin, 2000). Additionally, crime rates followed a similar pattern, only they were offset by about 20 years (R. Nevin, 2000). Crime rates began increasing from the 1960s through the 80s and then began dropping the early 1990s (R. Nevin, 2000). Nevin posited that children who were exposed to leaded gasoline emissions during the 1940s and 50s were

more likely to commit violent crimes in the 1960s-1980s (R. Nevin, 2000).

A study by Reyes in 2007 took Nevin's 2000 work a step further and examined the effects of lead vehicle emissions on crime at the state level. Reyes reported while leaded gasoline emissions declined in the 1970s and 80s as a result of the introduction of new technologies in vehicle exhaust systems and the implementation of the 1970 Clean Air Act, the degree in which they declined varied greatly by state (Reyes, 2007). Upon closer examination, Reyes found that the pace of the decline in the violent crime rate corresponded with pace of the decline in the consumption of leaded gasoline. More specifically, Reyes concluded that the reduction in childhood lead exposure resulted in a 56% decline in violent crime in the 1990s (Reyes, 2007). Lead vehicle emissions did not influence property crime rates.

Stretesky and Lynch examined the effect of air lead concentrations on crime at the county level, and focused solely on homicide rates (P. B. Stretesky & Lynch, 2001). They found that air lead concentration was the only indicator to be associated with homicides rates, after controlling for multiple confounding factors, including 9 other measures of air pollution (P. B. Stretesky & Lynch, 2001). From 1989-1991, the adjusted homicide rate was over four times higher in counties with the highest level air lead concentrations (.17mcg/m<sup>3</sup>) compared to those with the lowest concentrations (0mcg/m<sup>3</sup>) (P. B. Stretesky & Lynch, 2001).

Nevin followed up his national-level work with a 2007 study examining lead and international crime trends (R. Nevin, 2007). In this study, he examined the relationship between preschool blood lead and future crime trends over several decades in the US, Britain, Canada, France, Australia, Finland, Italy, West Germany, and New Zealand (R. Nevin, 2007). Nevin found a strong association between preschool blood lead and trends in property crimes (theft and

burglary) and violent crimes (murder, rape, robbery, and aggravated assault) at lags consistent with peak offending ages in all of the countries included in the study (R. Nevin, 2007).

A study by Mielke and Zahran examined the relationship between annual vehicle lead emissions and city-level aggravated assault rates. The relationship was tested in Chicago, Indianapolis, Minneapolis, San Diego, Atlanta and New Orleans (Mielke & Zahran, 2012). Mielke and Zahran found that, with all other factors held equal, a 1% increase in tonnages of lead air emissions released 22 years previous raised the current aggravated assault rate by 0.46% (Mielke & Zahran, 2012). Most recently, Boutwell and colleagues examined the relationship between aggregate-level blood lead levels of children less than 72 months old during an 11 year period (1996-2007) and crime in St. Louis, MO from 2010-2012 (Boutwell et al., 2016). Using spatial statistical models, they found that greater blood lead levels at the census-tract level were associated with increases in violent, non-violent and total crime (Boutwell et al., 2016).

Finally, a study by Feigenbaum and Muller in 2016 shifted away from air emissions and focused the effects of lead exposure via lead water service lines on city-level homicide rates between 1921 and 1936. The study used historical data on the water supply of U.S. cities in the late nineteenth century (Feigenbaum & Muller, 2016). They found that homicide rates were 24% higher in the cities that used lead service pipes compared to those that did not (Feigenbaum & Muller, 2016).

## **CHAPTER 2: MANUSCRIPT #1**

### **Childhood Lead Exposure and Firearm Violence: A Conceptual Model**

#### **Abstract**

Over 10,000 Americans are killed in firearm homicides each year, and an additional 60,000 are injured in nonfatal shootings. Identifying key risk and protective factors for firearm violence is paramount to developing effective prevention strategies. Environmental lead exposure is a demonstrated neurotoxicant which causes behavior changes that are known to be criminogenic, such as impulsivity and aggression. Despite research linking lead and crime, there is limited conceptual understanding of the relationship between childhood lead exposure and firearm violence. The purpose of this article is to provide a more comprehensive conceptual model of the corresponding factors involved in this relationship as well as the mechanisms by which childhood lead exposure leads to firearm violence. We conducted a comprehensive review of the childhood lead and firearm violence literature and used the social ecological model to guide the development of the conceptual model. A conceptual model illustrating the relationship between childhood lead exposure and firearm violence is described with factors included at the Individual (Race, Gender, IQ, ADHD, Aggression, Academic Outcomes, AODA, and Access to Firearms and Firearm Carrying), Peer (Deviant Peers), Family, Multilevel (SES and Exposure to Violence), and Policy (Laws and Regulations) levels. This conceptual model represents a starting point for childhood lead and firearm violence research and can be used to guide future research, policy and practice.

## **Introduction**

Despite experiencing a decline in violent crime since the early 1990s, firearm violence remains a significant public health problem in the United States (Federal Bureau of Investigation, 2017). An average of over 200 people killed and 1200 people are injured by firearm violence each week (Center for Disease Control and Prevention, 2015a). While mass rampage shootings such as the Newtown school shooting often garner the most media attention, street shootings are the most prevalent types of fatal and nonfatal firearm assaults in the U.S. (Bushman et al., 2016). Fatal and nonfatal street shootings disproportionately affect young, African American males living in socially and economically disadvantaged urban areas. This population has the highest victimization and perpetration rates of firearm violence (J. L. Lauritsen & Lab, 2007).

It is important to study firearm violence not only because of the loss of human life but also the multitude of short- and long-term costs to individuals, families and society. Estimates of the financial costs of firearm violence in the U.S. range from \$100 billion to more than \$450 billion per year (American Public Health Association, 2015; Cook & Ludwig, 2002; Miller, Cohen, & Wiersema, 1996). Victims of nonfatal shootings may suffer from physical disabilities, short or long term physical impairments and mental health problems, loss of employment and wages, and reduced quality of life (Fowler, Dahlberg, Haileyesus, & Annet, 2015). In addition to the human toll for victims and their families, firearm violence can increase health care costs, decrease property values and disrupt social services in neighborhoods (Cook & Ludwig, 2002).

Identifying key risk and protective factors for firearm violence is paramount to developing effective prevention strategies. One environmental stressor that is a potential key risk factor for future violent crime is childhood lead exposure (Boutwell et al., 2016; Feigenbaum & Muller, 2016; D. M. Fergusson, Boden, & Horwood, 2008; Mielke & Zahran,



2012; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; Reyes, 2007; P. B. Stretesky & Lynch, 2001; J. P. Wright et al., 2008). Lead is an environmental toxicant that is particularly harmful to children and may cause irreversible, long term damage to a child's brain. Varying degrees of lead exposure during childhood have shown to have deleterious effects on intelligence and cognition, (Baghurst et al., 1992; Banks, Ferretti, & Shucard, 1997; D. C. Bellinger, Stiles, & Needleman, 1992; Canfield et al., 2003; K. N. Dietrich, Berger, Succop, Hammond, & Bornschein, 1993; B. P. Lanphear et al., 2005) behavior, (K. N. Dietrich, Douglas, Succop, Berger, & Bornschein, 2001; H. L. Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996; P. B. Stretesky & Lynch, 2004; J. P. Wright et al., 2008) and brain structure (Cecil et al., 2008). Research suggests that the physical, mental and behavioral effects of childhood lead exposure may be conducive to future criminal behavior, including violent crime (Narag, Pizarro, & Gibbs, 2009).

Elevated childhood lead levels have been found to be associated with juvenile delinquency, (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; Olympio, Gonçalves, Günther, & Bechara, 2009) and juvenile and adult criminal behavior, including violent offending (Denno, 1990; D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; H. L. Needleman, McFarland, Ness, Fienberg, & Tobin, 2002; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004). This relationship was identified early in the lead literature and continues to be replicated in present day cross-sectional and longitudinal research. In a study by Byers and Lord in 1943, they found that children with acute lead exposure exhibited violent and aggressive behaviors (Byers & Lord, 1943). Almost 50 years later, a study by Pihl & Ervin found similar results in adults when they examined the differences in hair element levels, including lead, between 30 violent and 19 nonviolent males incarcerated in an institute for

psychiatrically disturbed criminals (Pihl & Ervin, 1990). Chi-squared analysis revealed that violent offenders had significantly higher lead levels in their hair than the nonviolent offenders (Pihl & Ervin, 1990). A study by Needleman et al produced similar results in juveniles. The case control study of 194 adjudicated delinquent juveniles and 146 nondelinquent juveniles found that the bone lead concentrations of the adjudicated delinquent juveniles were significantly higher than those of the nondelinquent juveniles after adjusting for covariates (H. L. Needleman et al., 2002). Adjudicated delinquents were four times more likely than controls to have bone lead concentrations above 25 ppm (H. L. Needleman et al., 2002).

Several longitudinal studies examining the early predictors of juvenile and adult criminal behavior have identified childhood lead exposure as a key predictor. Denno conducted a longitudinal study that tracked 987 African-American youth born in Philadelphia from birth to age 22 years old (Denno, 1990). The study found lead intoxication at age 7 years was one of the strongest predictors of disciplinary problems from ages 13 to 14, juvenile delinquency, and adult offenses for male participants (Denno, 1990). In a prospective longitudinal birth cohort study, Dietrich et al studied the effects of lead on development in 195 urban, inner city children (K. N. Dietrich et al., 2001). They found significant positive associations between prenatal lead exposure and the frequency of delinquent and antisocial behaviors reported by the children's parents and child as well as between postnatal (average childhood level and level measured at 6 years of age) lead exposure and self-reported frequency of delinquency and antisocial behaviors, after controlling for covariates (K. N. Dietrich et al., 2001). More recently, Wright et al examined the relationship between early lead exposure (prenatal, average childhood, and at 6.5 years) and criminal arrests in young adulthood in 250 participants of the Cincinnati Lead Study birth cohort (J. P. Wright et al., 2008). They found that for every 5 µg/dL increase in prenatal

and 6-year-old blood lead concentration, the adjusted total arrest rates were significantly greater (prenatal: RR=1.40, 95% CI 1.07-1.85 and 6.5 years: RR=1.27, 95% CI 1.03-1.57) (J. P. Wright et al., 2008). The adjusted arrest rates for violent crimes were also significantly greater for each 5 µg/dL increase in average childhood lead (RR=1.30, 95% CI 1.03-1.64) and 6-year-old blood lead (RR=1.48, 95% CI 1.15-1.89) (J. P. Wright et al., 2008). Finally, Fergusson and colleagues examined a 21-year longitudinal New Zealand birth cohort to determine if there was a link between dentine lead exposure measured between 6-9 years old and both officially recorded and self-reported criminal convictions between the ages of 14-21 years old (D. M. Fergusson et al., 2008). They found a significant dose-response relationship between childhood lead levels and rates of violent and property crime (officially recorded and self-reported), after controlling for confounding (D. M. Fergusson et al., 2008).

While prior research has found an association between childhood lead exposure and delinquency and criminal offending, there is limited conceptual understanding of the different factors involved in this relationship. A literature review by Narag, Pizarro and Gibbs examining the implications of lead exposure on criminological theory appears to present the most comprehensive description of the individual and community-level pathways from childhood lead exposure to delinquency and crime (Figures 1 and 2) (Narag et al., 2009). The conceptual model presented by Narag et al is a significant contribution to the literature, but it has limitations when applied to specific crimes, such as firearm violence. The key risk factors for firearm violence fall under the following categories: individual, peer, family, neighborhood, and policy level (Bushman et al., 2016). The conceptual framework presented by Narag et al captures most of the factors that fall into these categories, but it does not fully encompass all the risk factors of

firearm violence. For example, the model does not include key risk factors such as individual-level alcohol and other drug use, access to firearms, and neighborhood level violence.

To get a more all-encompassing understanding of the factors involved with both childhood lead exposure and firearm violence, the following paper reviews and integrates the literature on the predictors of childhood lead exposure, effects of childhood lead exposure, and predictors of firearm violence. Based on the results of the integrated review, we propose a conceptual model that illustrates the corresponding factors involved with childhood lead exposure and firearm violence at and across the individual, peer, family, and policy levels. The purpose of this article is to provide a more comprehensive conceptual model of the corresponding factors as well as the mechanisms by which childhood lead exposure leads to firearm violence to guide future research, policy and practice.

Figure 1

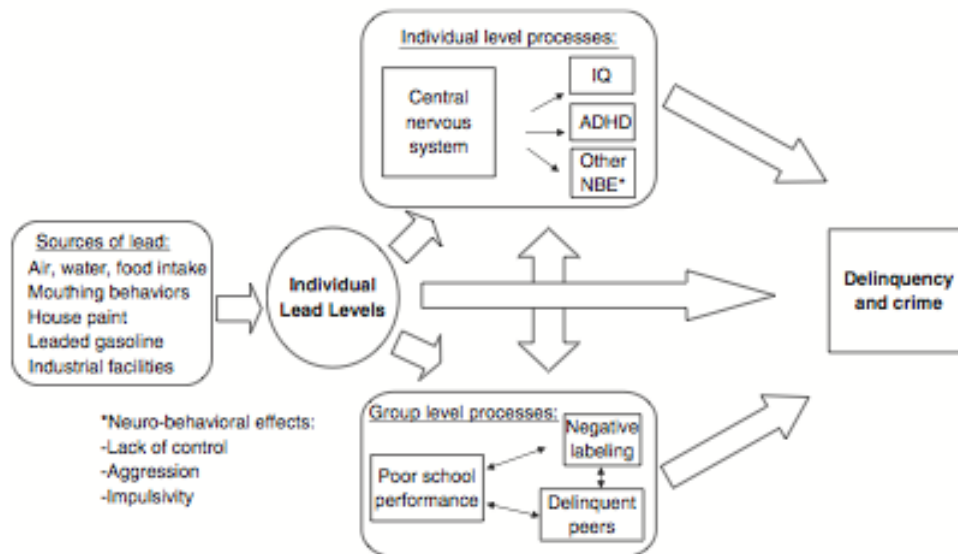


Figure 1: Individual-Level Pathways of Lead Exposure to Delinquent and Criminal Behaviors

Figure 2

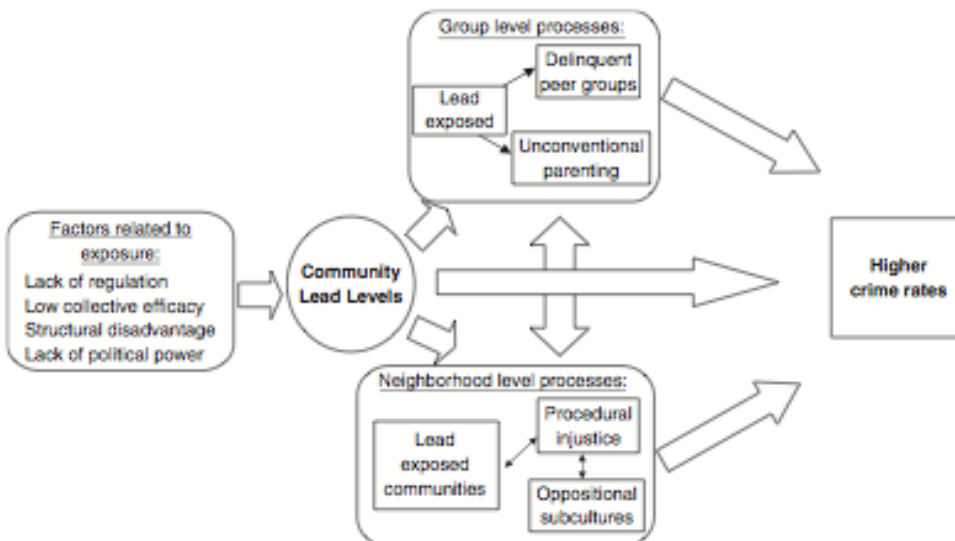


Figure 2: Community-Level Pathways of Lead Exposure to Higher Crime Rates

## **Methods**

### **Inclusion and Exclusion Criteria**

To be eligible for this review, studies needed to evaluate the predictors and effects of childhood lead exposure and the predictors of firearm violence perpetration and victimization. All forms of lead exposure measures were included (i.e. bone, dentine, blood, etc.). Articles on firearm violence needed to explicitly define violence as violence perpetrated with a firearm or a criminal arrest for committing an aggravated assault or homicide. To include seminal lead and firearm violence research, there was no date range restriction. All study designs, populations, and geographical areas were eligible for this review.

### **Search Strategies for Identification of Studies**

A conceptual model or framework begins with conducting a thorough review of the literature. The following strategies were used to perform a search for literature fitting the inclusion criteria. It should be noted that our search strategy was reviewed and approved by a University of Wisconsin-Milwaukee librarian (Linda Kopecky, Head of Research Services). A keyword search was conducted in 10 full text and abstract databases (Web of Science, Academic Search, PubMed, Cochrane Reviews, ProQuest Dissertations, PsychINFO, Criminal Justice Abstracts, NCJRS Abstracts, CINAHL, and Environment Complete). Keyword searches included (but were not limited to) a combination of the following: 'lead exposure,' 'lead poisoning,' 'lead poisoning in children,' 'child,' 'victim,' 'violence,' 'firearms,' 'guns,' 'youth violence,' 'juvenile delinquency,' and 'violent offending.' Next, we reviewed the references of the articles and reports found in my search for additional literature. During each step, we prioritized identifying review articles and highly cited articles. These articles assisted with

directing and narrowing the literature search as well as refining keyword searches. We scanned the titles, read the abstracts of the related literature, and saved the full-text versions of relevant articles. The relevant articles were sorted into three categories: predictors of childhood lead exposure, effects of childhood lead exposure, and predictors of firearm violence.

### **Developing the Conceptual Model**

A conceptual model or framework is “the system of concepts, assumptions, expectations, beliefs, and theories that support and inform your research” (Maxwell, 2005, pg. 39). It provides the foundation for a study by providing an overview of the literature, highlighting trends in the research and illustrating the linkages between key constructs (Rocco & Plakhotnik, 2009). In addition, it conceptualizes the study, guides the development of the hypotheses, research design and instrumentation, and supports the interpretation of the study’s findings (Rocco & Plakhotnik, 2009). Finally, it demonstrates how a study advances knowledge and identifies opportunities for future research (Rocco & Plakhotnik, 2009). Building a model assists researchers with “defining a concept, mapping the research terrain or conceptual scope, systematizing relations among concepts, and identifying gaps in the literature” (Rocco & Plakhotnik, 2009) pg. 128. Conceptual models are essential for integrated interdisciplinary research.

It has been established in the literature that firearm violence is shaped by factors at multiple levels (Bushman et al., 2016; David-Ferdon & Simon, 2014; Halliday-Boykins & Graham, 2001; Hong, Cho, Allen-Meares, & Espelage, 2011; Rubens & Shehadeh, 2014; Seal, Nguyen, & Beyer, 2014; World Health Organization (WHO), 2014). The social ecological model posits that individual behavior is influenced by factors at the individual, relationship,

community and societal levels (McLeroy, Bibeau, Steckler, & Glanz, 1988). Therefore, the social ecological model was used to guide the development of the conceptual model. Within each category (predictors of childhood lead exposure, effects of childhood lead exposure, and predictors of firearm violence), we organized the articles by individual, relationship, community and societal level factors. Through this process, we identified where there was overlap in the literature, determined the key variables for the model and defined the relationships between the variables. Finally, we performed an iterative process of reading and synthesizing the literature, building the conceptual model, and returning to the literature. The final model illustration was created to depict the multi-layered factors involved with childhood lead exposure and firearm violence. Arrows were used to represent the direction of the relationship between constructs.

## **Results**

### **Individual Level Factors**

#### *Race*

Environmental lead levels vary greatly geographically, but the variation is not random. Lead levels are higher in areas where non-Whites reside. A study by Hird and Reese found that African Americans and Hispanics were more likely than Caucasians to live in a U.S. county with elevated air pollution rates which included lead air concentrations (Hird & Reese, 1998). Using data from the U.S. Census Bureau and the U.S. Environmental Protection Agency, a study by Stretesky found that the counties with the largest proportions of African American youth under 16 years of age had 8% more lead in the air than counties with no African American youth (P. B. Stretesky, 2003). Risk factors for lead exposure include living in lower income and rental housing, living near major highways and living near industrial zones or rural areas near smelter



sites (R. Nevin, 2000). Minorities are more likely than non-minorities to reside in these high-risk areas and, due to barriers such economic constraints, minorities are less able to move away from these high lead risk neighborhoods (Narag et al., 2009; P. B. Stretesky & Lynch, 2004).

While there has been progress in reducing childhood lead exposure over time, racial disparities remain (Wheeler & Brown, 2013). African American, non-Hispanic children are disproportionately exposed to and have higher levels of lead compared to children of other races. A Morbidity and Mortality Weekly Report (MMWR) by Wheeler found that African American, non-Hispanic children had significantly higher geometric mean blood lead levels compared to Mexican American and non-Hispanic White children from 1999-2010(Wheeler & Brown, 2013). A systematic review of U.S. studies that reported childhood blood lead levels and the race/ethnicity of at least two groups found that African American children had consistently higher blood lead levels when compared to children of other races (White, Bonilha, & Ellis Jr, 2016). In the studies, African American children reported the highest mean lead levels and were more likely to have elevated blood lead ranges (White et al., 2016).

The majority of firearm violence victims and suspects of both fatal and nonfatal shootings are African American. Nationally, firearm violence is the leading cause of death for non-Hispanic African American men aged 15-34 years (David-Ferdon & Simon, 2014; Wintemute, 2015). African American males between the ages of 15-29 years old are killed during firearm-related assaults at a rate nearly 10 times that of White males of the same age (15-19 years: 4.9 vs. 45/100,000; 20-24 years: 9.1 vs. 86.6/100,000; 25-29: 7.7 vs. 78.6/100,000) (Heron, 2015). The homicide rate for non-Hispanic African American youth was 13.7 times higher than the rate for non-Hispanic White youth (28.8 vs. 2 per 100,000) and 4.1 times higher than the rate for Hispanic youth (28.8 vs. 7.1 per 100,000) in 2011(David-Ferdon & Simon,

2014). At ages 20-29 years, the firearm homicide rate for non-Hispanic African American males was 20 times higher than the rate for non-Hispanic White males and five times higher than Hispanic males in 2012 (Wintemute, 2015). The firearm homicide rate is higher for non-Hispanic African American females as well (Wintemute, 2015). African Americans are also more likely to commit firearm violence than non-Hispanic Whites and Hispanics (Federal Bureau of Investigation, 2015; J. L. Lauritsen, Heimer, & Lynch, 2009; Nielsen, Martinez Jr, & Rosenfeld, 2005).

### *Gender*

Despite being studied in multiple geographic regions, the role gender plays in modifying the relationship between lead and crime is not clear. While experiencing similar levels of lead exposure, some studies have found that males may experience worse health outcomes from lead exposure than girls (D. Bellinger, Leviton, & Sloman, 1990a; K. N. Dietrich, Krafft, Shukla, Bornschein, & Succop, 1987; Jedrychowski et al., 2009; Khanna, 2015; Ris, Dietrich, Succop, Berger, & Bornschein, 2004). One of the publications from the Cincinnati Lead Study posited that male brains may be more sensitive to toxic chemical exposures in utero after finding that males had greater neurobehavioral deficits in response to maternal blood lead than females at age 6 months (K. N. Dietrich et al., 1987). A later publication examined the neuropsychological effects (memory, learning/IQ, attention, visuoconstruction, and fine-motor) of lead exposure (Ris et al., 2004). When the interaction effect of gender on lead exposure was examined, the study found that males were at heightened risk for attention and visuoconstruction deficits (Ris et al., 2004). In addition, a study by Bellinger et al examined the effects of lead exposure on cognitive development at 24 months and 57 months (D. Bellinger, Leviton, & Sloman, 1990b). They found that girls did not experience the same level of deficits from lead exposure as boys (D.

Bellinger, Leviton, & Sloman, 1990b). A pilot study by Khanna examined the cognitive effects of lead exposure in 40 children (aged 3-6 years old) residing near a U.S. Environmental Protection Agency-designated lead Superfund site (Khanna, 2015). The study found an interaction effect between lead status and sex (Khanna, 2015). Girls with elevated lead levels scored as well as girls without elevated lead levels on cognitive measures (Khanna, 2015). Lead exposure had a much greater effect on males than females (Khanna, 2015). A study in Poland by Jedrychowski et al examined the relationship between low level prenatal lead exposure and cognitive deficits at 3 years old by gender (Jedrychowski et al., 2009). They found that prenatal lead exposure was inversely associated with cognitive function in boys but not girls at 36 months of age (Jedrychowski et al., 2009). In contrast, some studies have found girls to have worse health outcomes from lead exposure than boys. For example, as part of the Port Pirie study, Baghurst and colleagues examined the effects of lead exposure on children's intelligence (Baghurst et al., 1992). The study found that girls were more sensitive to the effects of lead than boys. The full-scale IQ decrement for girls was 7.8 points compared to 2.6 points for the boys (Baghurst et al., 1992).

Males bear the greatest burden of firearm-related mortality and nonfatal firearm injuries treated in the U.S. (Fowler et al., 2015). From 2010-2012, the annual rate of firearm violence death was 5 times higher for males than females (Fowler et al., 2015). During that same time period, the average annual rate of nonfatal firearm injury from firearm assaults for males was 9 times higher than the rate for females (27.9 vs. 3.2 per 100,000) (Fowler et al., 2015). The youth male homicide rate in 2011 was 6 times higher for males than females (12.3 vs. 2.1 per 100,000) and the nonfatal physical assault injury rate in 2012 was 1.6 times higher for males than females (1,141 vs 704 per 100,000) (David-Ferdon & Simon, 2014). Males also commit firearm violence

at higher rates than females (Federal Bureau of Investigation, 2015; J. L. Lauritsen et al., 2009). For example, an annual report on homicides and nonfatal shootings in Milwaukee, WI found that, in 2015, 93% of homicide suspects and slightly less than 90% of nonfatal shooting suspects were male (Milwaukee Homicide Review Commission, 2015). One year prior, 86% of homicide suspects and 92% of nonfatal shooting suspects were male (Milwaukee Homicide Review Commission, 2015). Similar trends have been identified at the national level.

### *Intelligence Quotient (IQ)*

Following the advent of lead screening, lead studies in the 1970s focused on children who were exposed to lead but not showing overt symptoms of acute lead poisoning (Banks et al., 1997). These studies predominantly compared cognitive and behavioral outcomes of children with highly elevated lead levels (i.e.  $> 40 \mu\text{g/dL}$  in de la Burde 1972) to control children with lower lead levels (Banks et al., 1997). Three of these studies found significant associations between lead and IQ (Burdé & Choate, 1972; Landrigan et al., 1975; Perino & Ernhart, 1974) and two did not (Kotok, 1972; Lansdown et al., 1974). These studies suffered from design flaws such as small sample sizes, delayed lead exposure measures (i.e. lead exposure measured at 6 years of age when peak exposure is around 2 years old), miscategorization of elevated lead exposure levels (i.e. “low” level exposure would later be considered “high” exposure in studies in the 1980s), and limited confounders included in the final analysis (Banks et al., 1997; H. Needleman, 2004).

A landmark study by Herbert Needleman et al in 1979 attempted to address the design issues of earlier studies. The study found a significant negative association between dentine (tooth) lead levels and child IQ, after controlling for multiple covariates (H. L. Needleman et al.,

1979). Children with higher dentine lead levels scored an average of 4.5 points lower on the full-scale IQ test (Wechsler Intelligence Scale for Children Revised) than children with lower lead levels (H. L. Needleman et al., 1979). In addition, the teachers of the over 2,000 children in the sample blindly rated each child's classroom behavior. The ratings showed a dose-response relationship between dentine lead level and behavioral problems and academic difficulties (H. L. Needleman et al., 1979).

After the mixed results of early studies, the negative relationship between childhood lead exposure and child IQ has since been well-documented in the scientific literature. Multiple cross-sectional and longitudinal studies have found that moderate to low levels of lead exposure experienced prenatally, postnatally or during early childhood is associated with lower IQ scores in children living the United States and abroad, after important confounding variables were held constant (Al-Saleh et al., 2001; Baghurst et al., 1992; D. C. Bellinger et al., 1992; Canfield et al., 2003; A. Chen, Dietrich, Ware, Radcliffe, & Rogan, 2005; A. Chen, Cai, Dietrich, Radcliffe, & Rogan, 2007; L. Chiodo, Jacobson, & Jacobson, 2004; de la Burd  & Choate, 1975; K. N. Dietrich et al., 1993; Dudek & Merez, 1997; Factor-Litvak, Wasserman, Kline, & Graziano, 1999; Kordas et al., 2006; Ris et al., 2004; Rummo, Routh, Rummo, & Brown, 1979; L. Schnaas, Rothenberg, & Flores, 2006; L. Schnaas et al., 2000; G. Wasserman et al., 2000; G. A. Wasserman et al., 2003; G. A. Wasserman et al., 1997). For example, a pooled analysis of seven prospective studies with 1,333 children from Boston, MA, Cincinnati, OH, Cleveland, OH, Rochester, NY, Mexico City, MX, Port Pirie, AU, and Kosovo, YU, found a significant negative relationship between childhood lead levels and IQ (B. P. Lanphear et al., 2005). The study found that an increase in blood lead concentration from 1 to 10  $\mu\text{g}/\text{dL}$  would result in a 6.2-point reduction in IQ score and an increase in blood lead concentration from 10 to 20  $\mu\text{g}/\text{dL}$  would

result in a 1.9-point reduction in IQ score (B. P. Lanphear et al., 2005). Several meta-analyses have replicated the dose-response relationship between childhood lead exposure and IQ (H. L. Needleman & Gatsonis, 1990; Pocock & Ashby, 1985; Schwartz, 1994). The meta-analysis by Schwartz concluded that a child's IQ decreases 0.25 per 1 µg/dL increase in blood lead (Schwartz, 1994).

The link between IQ and delinquency has been accepted by criminologists for many years (McGloin, Pratt, & Maahs, 2004). A notable study by Hirschi and Hindelang in 1977 found that the IQ of delinquents and nondelinquents differed by one standard deviation (Hirschi & Hindelang, 1977). While in the years since Hirschi and Hindelang's study there has been disagreement over the magnitude and nature of the role IQ has on delinquency compared to other risk factors, the relationship between IQ and delinquency has been demonstrated in the empirical literature nonetheless (Ellis & Walsh, 2003; Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt, Gabrielli, Mednick, & Schulsinger, 1981; Moffitt, 1990). A review by Farrington found that low intelligence is a major long-term predictor of male youth violence (Farrington, 1998a). Additionally, a longitudinal study in Pittsburgh followed three cohorts of over 1500 young men from late childhood into their 20s to understand the development of juvenile offending, mental health problems, and drug use in inner city males (Loeber et al., 2012). The study found a significant negative relationship between IQ and the probability of being arrested for violent offenses (Loeber et al., 2012). Narag et al posited that children with low IQ tend to also show characteristics of low self-control and impulsivity, both of which are associated with violent offending (Narag et al., 2009). Older and more recent studies have found that the IQ-delinquency relationship is mediated by poor school performance (McGloin et al., 2004; Narag et al., 2009).

In addition to analyzing low IQ as a risk factor, research has examined whether high IQ is a protective factor for future violent offending. A meta-analysis of 15 longitudinal studies examined the extent to which above average intelligence serves as an interactive and/or risk-based protective factor for offending and violence (Ttofi, Farrington, Piquero, & DeLisi, 2016). The study found that, overall, higher IQ predicted lower levels of offending in both high-risk and low-risk groups and that the effects were significantly different based on risk level (Ttofi et al., 2016). IQ was protective of future offending in high risk individuals, but it did not have a significant protective effect on offending for low risk individuals (Ttofi et al., 2016). This means that interventions focused on improving cognitive development and intelligence may be more effective at reducing offending in high risk individuals than low risk individuals.

#### *Attention-deficit/hyperactivity disorder (ADHD)*

While not quite as extensive as IQ, the positive significant association between childhood lead levels and attention deficit and hyperactivity symptoms and/or ADHD diagnosis for children in the U.S. and abroad (after controlling for covariates) is well established in the literature (D. Bellinger, Leviton, Allred, & Rabinowitz, 1994; Burns, Baghurst, Sawyer, McMichael, & Tong, 1999; A. Chen et al., 2007; L. M. Chiodo et al., 2007; L. Chiodo et al., 2004; D. M. Fergusson, Horwood, & Lynskey, 1993; Ha et al., 2009; H. L. Needleman et al., 1996; H. L. Needleman et al., 1979; Nicolescu et al., 2010; Nigg et al., 2008; Nigg, Nikolas, Knottnerus, Cavanagh, & Friderici, 2010; Ris et al., 2004; Rummo et al., 1979; Silva, Hughes, Williams, & Faed, 1988; Thomson et al., 1989; Wang et al., 2008). A meta-analysis of 33 studies examining the relationship between lead and attention and hyperactivity symptoms found that childhood lead level has a significant, small to moderate positive association with both symptoms ( $r=.16$ ,  $k=27$ ,  $p<.001$  for inattention and  $r=.13$ ,  $k=21$ ,  $p<.001$  for hyperactivity/impulsivity symptoms)

(Goodlad, Marcus, & Fulton, 2013). The magnitude of these effect sizes is equitable to the associations found in the meta analyses examining lead level and intelligence and lead level and conduct problems (Goodlad et al., 2013; Marcus, Fulton, & Clarke, 2010; H. L. Needleman & Gatsonis, 1990). Unlike many of the lead and IQ studies, lead levels were measured in preschool, primary and adolescent aged children. The effects of prenatal lead exposure on attention and hyperactivity were not included in this meta-analysis.

This positive association has been found even at low lead levels of lead exposure. For example, in two studies by Chiodo and colleagues, significant positive associations were found between concurrent lead levels of seven-year-old children and attention problems, hyperactivity and social behaviors (L. M. Chiodo et al., 2007; L. Chiodo et al., 2004). The lead levels for children enrolled in the study averaged 5 µg/dL, with 90% of child lead levels falling below 10 µg/dL (L. M. Chiodo et al., 2007; L. Chiodo et al., 2004). A study by Braun et al examined the relationship between child lead exposure and ADHD diagnosis in a representative U.S. dataset (Braun, Kahn, Froehlich, Auinger, & Lanphear, 2006). The study found that children with lead levels in the top 20th percentile were 4 more times likely to be diagnosed with ADHD than children with lead levels in the bottom 20th percentile (Braun et al., 2006). Children in the top 20th percentile had lead levels that ranged between 2.7 - 5.5 µg/dL (Braun et al., 2006).

Childhood ADHD is associated with future adult offending, including violent offending, as well as risk factors for adult offending including neuropsychological deficits, poor academic and cognitive skills, truancy, and aggression (Barkley, 2002; Biederman et al., 2006; Mannuzza, Klein, & Moulton, 2008; Pratt, Cullen, Blevins, Daigle, & Unnever, 2002; Retz & Rösler, 2009; Schilling, Walsh, & Yun, 2011). A longitudinal study of 411 London males found that attention deficit/hyperactivity was one of the most important childhood (8-11 years old) predictors of adult



offending (up to 32 years old) (Farrington, Loeber, & Van Kammen, 1990). Attention deficit/hyperactivity was predictive of whether the child also had conduct problems (Farrington et al., 1990). Farrington found in a later study that impulsivity, a prominent deficit for people with ADHD, was a major long-term predictor of male youth violence (Farrington et al., 1990). A study of 207 New York boys (ages 6-12 years) with ADHD, but free of conduct disorder, were followed up on at ages 18 and 25 years old, and then their lifetime criminal arrest histories were obtained when they were 38 years old. When compared to a similar group of boys who did not have ADHD as children, the ADHD group of boys were significantly more likely to be arrested and convicted of felonies and aggressive offenses, including murder, rape, robbery, assault, arson, extortion, and weapons offenses (Mannuzza et al., 2008). A population-based longitudinal study of 2713 Finnish boys examined the childhood predictors of adult criminality for males. Teacher reports of hyperactivity issues at age 8 years old independently predicted drug offenses, violent offenses, property offenses, and traffic offenses in early adulthood (Sourander et al., 2006). It also predicted that a boy would commit more than five offenses as an adult (Sourander et al., 2006). Finally, a meta-analysis by Pratt et al also confirmed that ADHD is a significant risk factors for young adulthood crime and delinquency (Pratt et al., 2002).

### *Aggression*

Multiple studies have found an association between childhood lead exposure and conduct problems or aggression within the past 20 years (Braun et al., 2008; A. Chen et al., 2007; L. M. Chiodo et al., 2007; L. Chiodo et al., 2004; K. N. Dietrich et al., 2001; Nigg et al., 2008). Teachers and parents are more likely to report misbehavior in school and at home for children with high lead levels. A meta-analysis conducted by Marcus et al in 2010 reviewed 19 studies that examined the association between lead exposure and conduct disorder, oppositional defiant

disorder, aggressive and violent behavior, and delinquent, antisocial and criminal behavior (Marcus et al., 2010). The study found a positive medium effect size between lead exposure and conduct problems (Marcus et al., 2010). The magnitude of the effect size is similar in size to the association between lead exposure and IQ (Marcus et al., 2010). Lead exposure in these studies was most often measured using the concurrent blood lead levels in young children to late adolescents (3.8 to 18.4 years). Unlike IQ, the effects of prenatal, postnatal or toddler lead exposure was not assessed. Like the attention and hyperactivity studies, there was no association between the average blood lead level of the participants and the effect size of the association. In other words, studies with higher average lead levels in its sample did not find higher effect sizes (Marcus et al., 2010).

Childhood aggression is one of the strongest predictors of future violent offending (American Psychological Association, 2013; Huesmann, Eron, & Dubow, 2002). According to a review by Farrington, male youth violent offenders often show aggression as a child (Farrington, 1998a). And the earlier and more frequent a child is aggressive, the higher their risk for adult violent offending (American Psychological Association, 2013). A longitudinal study by Loeber et al compared violent and nonviolent offenders as well as violent offenders who committed a homicide versus those who did not, to determine risk factors for violence and homicide in young males in Pittsburgh. The study identified delinquency before 10 years of age and physical aggression as predictors of violent offending (Loeber et al., 2005). In addition, 31 of the 33 homicide offenders had a history of violence before committing the homicide (Loeber et al., 2005). Two additional longitudinal studies yielded similar results. A study by Dubow et al found that higher aggression at ages 8 and 19 years was a risk factor for adult violent offending (Dubow, Huesmann, Boxer, & Smith, 2016). The same population-based longitudinal study of

2713 Finnish boys that examined ADHD also found that parental reports of conduct problems at age 8 years old independently predicted violent, property, traffic, and drunk driving offenses and committing more than five offenses as a young adult (Sourander et al., 2006). The same study found that self-reports of bullying at age 8 years old independently predicted violent offenses in young adulthood (Sourander et al., 2006). Finally, a study by Resnick et al used a representative sample of Add Health data to study predictors of youth violence perpetration. The authors found that a history of violence as a child is a predictor of youth violence perpetration (Resnick, Ireland, & Borowsky, 2004).

### *Academic Outcomes*

Numerous studies show that lead is associated with school performance, including reading, math and science test scores, learning disabilities, classroom behavior, and high school graduation (Aizer, Currie, Simon, & Vivier, 2015; M. S. Amato et al., 2012; Evens, 2010; Evens et al., 2015; D. M. Fergusson et al., 1993; D. M. Fergusson, Horwood, & Lynskey, 1997; B. P. Lanphear, Dietrich, & Berger, 2003; B. P. Lanphear, Dietrich, Auinger, & Cox, 2000; H. L. Needleman, Schell, Bellinger, Leviton, & Allred, 1990; Zhang et al., 2013). This relationship remained even after controlling for covariates such as race/ethnicity, gender, maternal education, and poverty. Needleman et al followed up with study participants from their 1979 study as adults (12 years later) and found that adults who experienced high lead exposure (90th percentile) in childhood had “more school failure, reading disabilities, and lower class standing in their final year of high school” than adults who were not exposed to lead (H. Needleman, 2004; H. L. Needleman et al., 1990). A study by Zhang et al examined the long-term effects of early childhood lead exposure (before age 6) on academic achievement in math, science, and reading among 21,281 students attending public elementary and junior high school in Detroit

(grades 3, 5 and 8) (Zhang et al., 2013). Zhang et al found a strong, significant dose-response relationship between childhood blood lead levels and all three test scores, after controlling for grade level, gender, race, language, maternal education, and SES (Zhang et al., 2013). As childhood blood lead increased, the odds of scoring less than proficient on all three tests increased (Zhang et al., 2013).

Two studies by Amato et al examined the relationship between moderate lead exposure before age 3 years old and academic outcomes in almost 4,000 fourth grade students attending Milwaukee Public Schools (M. S. Amato et al., 2012). One study found a significant negative relationship between moderate lead exposure and scores on all 5 subtests of the end-of-grade standardized tests, after controlling for gender, poverty, English language learner status, race/ethnicity, school disciplinary actions, and attendance percentage (M. S. Amato et al., 2012). The other study examined the effects of lead exposure on school suspensions. Amato et al found that children with blood lead levels greater or equal to 10  $\mu\text{g}/\text{dL}$  but less than 20  $\mu\text{g}/\text{dL}$  were twice as likely to be suspended than children with blood lead levels less than 5  $\mu\text{g}/\text{dL}$  (OR=2.66, 95%CI = 2.12,3.32) after controlling for gender, race, and SES (M. S. Amato et al., 2013). Finally, a recent study by Aizer et al investigated if lead exposure could potentially explain part of the black-white gap in educational outcomes for children who attend public school in Rhode Island (Aizer et al., 2015). The study found that a 5  $\mu\text{g}/\text{dL}$  increase in preschool lead levels reduced average reading test scores (from grade 3-8) by 6.4 to 9.6 points or 49-74% of a standard deviation (Aizer et al., 2015). Similar results were found for math test scores (Aizer et al., 2015). The authors concluded that since Rhode Island started a lead hazard reduction program in 1997, lead levels have fallen significantly more in African American children compared to children of other races and the racial gap in child test scores has also declined (Aizer et al.,

2015). Due to the effect lead has on test scores, the authors concluded that the lead reduction program may be partly responsible for the decline in the racial disparity in test scores (Aizer et al., 2015).

Studies have linked academic outcomes with future youth violent offending. Using a nationally representative sample (Add Health), a study by Resnick et al found that repeating a grade, skipping school and learning problems were predictors of youth violence perpetration in males (Resnick et al., 2004). Additionally, the longitudinal study by Loeber et al identified truancy and low school motivation as predictors of violent offending, and being held back in school as one of the 9 predictors of homicide offending among violent offenders (Loeber et al., 2005). Academic factors have also been found to be protective factors against violent offending. The Resnick et al study found high grade point average to be a protective factor for youth violence perpetration in boys (Resnick et al., 2004). A study by Dubrow et al in New York yielded similar results when it found that high educational aspirations at age 19 years provided the strongest risk-buffering protective factors for youth with at least one risk factor for violent offending (Dubow et al., 2016). Finally, a review by Cook and Laub was supportive of these findings. It found that strong school performance was a protective factor for youth violence (Cook & Laub, 2002).

#### *Alcohol and Other Drug Abuse (AODA)*

There is very little research on the relationship between childhood lead exposure and later drug and alcohol abuse. A recent study by Pascale et al followed up with a small cohort of children in Montevideo, Uruguay to investigate if their blood lead levels influenced their drug

use as adolescents (Pascale et al., 2014). The study found that lead levels increased the odds of using tobacco by 2.27, after controlling for the effect of covariates (Pascale et al., 2014).

Unlike lead, far more research has been conducted and found a positive relationship between drug and alcohol use and future violent offending (Bushman et al., 2016; Haegerich & Dahlberg, 2011). A study by Brook et al found that illicit drug use during adolescence is a predictor of violent behavior in early adulthood and that peer delinquency had a direct path to illicit drug use (Brook, Brook, Rubenstone, Zhang, & Saar, 2011). Using data from a longitudinal study of African American and Puerto Rican participants who attended public school in East Harlem in New York City and were followed into adulthood, Brook et al used structural equation modeling to analyze the predictors of adult violent behavior (Brook et al., 2011). The authors found that externalizing behaviors, peer delinquency, illegal drug use and neighborhood crime all predicted violence perpetration in adulthood (Brook et al., 2011). Additionally, a study by Resnick et al using Add Health data found that frequent use of alcohol, marijuana, and/or other illicit drugs is a predictor of youth violence perpetration for boys and girls (Resnick et al., 2004). Marijuana and alcohol use was predictive of future violence for boys, specifically (Resnick et al., 2004). Finally, a longitudinal study by Loeber et al comparing violent and nonviolent male offenders and violent offenders who committed a homicide found that positive attitudes towards substance use and selling hard drugs as predictors of homicide offending by violent offenders (Loeber et al., 2005).

Youth violence and adolescent drug and alcohol use share many of the same correlates (Haegerich & Dahlberg, 2011; Hawkins, Catalano, & Miller, 1992). Correlates of alcohol and drug use include child maltreatment, drug and alcohol use by the family or caregiver, exposure to multiple forms of violence as a child (including sexual, physical and community), high levels of

hyperactivity symptoms and conduct problems, poor school functioning and peer alcohol and drug use (Barkley, Fischer, Smallish, & Fletcher, 2004; Haegerich & Dahlberg, 2011; Hawkins et al., 1992; Hemphill et al., 2011; Lopez et al., 2008; Mannuzza et al., 2008; Weichold, Wiesner, & Silbereisen, 2014; D. R. Wright & Fitzpatrick, 2004). Protective factors against alcohol and drug use include school and family level factors such as academic achievement, positive family interactions and strong parental supervision (Hawkins et al., 1992; Piko & Kovács, 2010; D. R. Wright & Fitzpatrick, 2004).

### *Access to Firearms and Firearm Carrying*

For an individual to be a perpetrator or victim of firearm violence, a firearm is required. According to a study examining the potential driving forces behind the rise and fall of youth violence in the nineties, the dramatic increase in youth homicide, specifically, in the late eighties and early nineties was a result of firearm use, as the rate of homicides where firearms were not used stayed constant during this time (Cook & Laub, 2002). There was an increase in firearm use across all types of homicides, including those associated with felonies, arguments and gang conflicts (Cook & Laub, 2002). Perpetrators of street shootings are most likely to obtain their weapons either on the street, black market or from friends/family (Webster, Meyers, & Buggs, 2014).

A firearm is present in approximately one third of households in the United States (Cole & Johnson, 2005). Up to 50% of parents with firearms in the home keep the firearms unlocked and loaded (Cummings, Koepsell, Grossman, Savarino, & Thompson, 1997). In addition to increasing the risk for unintentional firearm injury or death, having a firearm in the home increases the risk for homicide (and suicide) (Cummings et al., 1997). A study by Resnick et al

found that, for boys only, easy access to firearms in the home is a predictor of youth violence perpetration (Resnick et al., 2004).

In addition to firearm access, firearm carrying increases the risk of lethal outcomes (Webster et al., 2014) and is positively related to local youth violence rates (Cook & Laub, 2002). Studies show that firearm availability is related to firearm carrying. According to a study using YRBSS data, for every 1 percentage increase in firearm ownership, firearm carrying by youth increased by 0.18 percent (Wintemute, 2003). Another study by Webster et al found that firearm availability, criminal behavior, social networks, and fear-driven protection are all determinants of firearm carrying (Webster et al., 2014). Rates of firearm carrying have been found to be higher for violent offenders than non-violent offenders (Webster et al., 2014). The longitudinal study by Loeber et al identified carrying a weapon as a predictor of homicide offending by violent offenders (Loeber et al., 2005).

Firearm carrying is also related to the sale and use of drugs and alcohol. A study by Ruggles and Rajan that analyzed 10 years of YRBSS data found that 8 of the top 10 correlates of firearm carrying were related to drug and alcohol use (Ruggles & Rajan, 2014). Additionally, a study examining adolescent data from a nationally representative sample (2008 National Survey on Drug Use and Health) found illicit drug selling and using to be strongly associated with adolescent firearm carrying (Vaughn et al., 2012). More specifically, youth who sold drugs were 16 times more likely to carry a firearm than those who did not sell drugs (Vaughn et al., 2012).

Being connected to a deviant peer group is associated with increased firearm carrying (Papachristos & Wildeman, 2014). These violent social networks facilitate access to firearms and encourage firearm carrying (Webster et al., 2014). Deviant peer groups may take the form



of gang or crew membership, which also increases an individual's risk for illicit drug selling and use (Webster et al., 2014). Being connected to a high risk social network and participating in gang activity and drug sales all impact an individual's desire to carry a firearm for self-protection purposes and/or violent offending (Webster et al., 2014).

## **Peer Level Factors**

### *Deviant peers*

Exposure to deviant or delinquent peers has consistently been found to be a strong predictor of future violence perpetration and victimization (Pratt & Cullen, 2000; Tracy, Braga, & Papachristos, 2016; Warr, 1993). Only a small group of individuals are responsible for the clear majority of street shootings and those individuals are often connected across similar social networks. A study of the social networks of homicide victims in a predominantly African American, high crime community in Chicago found that firearm violence was concentrated in a small social network of individuals (Papachristos & Wildeman, 2014). Social network analysis revealed that the further a person's social ties were to a homicide victim, the lower their odds of becoming a homicide victim (Papachristos & Wildeman, 2014). More specifically, for each social tie removed from a homicide victim, an individual's risk for their own homicide victimization decreased by 57% (Papachristos & Wildeman, 2014). A study of 602 mostly Latino youth living in Texas found that delinquent peer influences were predictive of violent criminal behavior (Ferguson, San Miguel, & Hartley, 2009). These results were supported in a review of the major long-term predictors of male youth violence by Farrington (Farrington, 1998a). A review of 16 studies published between 1996 and 2015 found that exposure to a victim or perpetrator of firearm violence in one's interpersonal and social networks increases an

individual's risk for firearm violence victimization and perpetration (Tracy et al., 2016). Low peer delinquency or having conventional peer groups have also been found to be protective of youth violence (Bernat, Oakes, Pettingell, & Resnick, 2012; Cook & Laub, 2002).

There is limited research on the correlates of peer delinquency associations. One of the major predictors of deviant peer associations is proximity to delinquent peers (Beaver et al., 2009; Warr, 1993). Kids who are in close contact with antisocial youth in their neighborhoods and schools are more likely to befriend delinquent peers (Beaver et al., 2009; Warr, 1993). Other studies have found that low parental supervision, neighborhoods and schools can all contribute to delinquent peer associations (Beaver et al., 2009). An additional explanation is that kids will seek out kids who are similar to them. Kids who are impulsive or have low self-control will seek out kids who also share those qualities (Matsueda & Anderson, 1998; O'Brien, Daffern, Chu, & Thomas, 2013). A study by Meldrum et al found an interaction between low self-control and peer delinquency (Meldrum, Young, & Weerman, 2009).

### **Family Level Factors**

Family-level factors are some of the most well-established risk factors for youth violence. These risk factors include “harsh and rejecting parents, interparental violence, child abuse and neglect, chaotic family life, inconsistent discipline, and poor monitoring by parents of children showing early signs of aggression” (Bushman et al., 2016, pg. 21). A study of males in the Cambridge Study on Delinquent Development found that growing up in a broken home before the age of 14 years old predicted a violent criminal conviction by the age of 50 (Theobald, Farrington, & Piquero, 2013). The study also found that the broken home-violent conviction relationship was moderated by receiving harsh discipline and mediated by hyperactivity at 14

years old (Theobald et al., 2013). ADHD in childhood may foster behavior that challenges parents who are not fully equipped to manage the behavior constructively (Moffitt, 1993; Pratt et al., 2002). The parent's response may be psychological rejection, verbal abuse or physical punishment - all of which increase a child's risk for future offending (Moffitt, 1993; Pratt et al., 2002). Negative parenting practices have also been found to interact with aggressive child behavior, amplifying the child's aggression (American Psychological Association, 2013). A study examining the social and psychological predictors of violence in young, African American youth living a high crime neighborhood found that family conflict and previous corporal punishment was associated with self-reported use of violence (DuRant, Cadenhead, Pendergrast, Slavens, & Linder, 1994). A review by Farrington supported these findings when it found that family factors such as poor supervision, harsh discipline, a violent parent, large family size, a young mother and a broken family were major long-term predictors of male youth violence (Farrington, 1998a).

Family level factors have also been found to be protective against youth violence. Two studies using Add Health data identified family level protective factors. Resnick et al found that high parental expectations about school (boys only), the ability to discuss problems with parents (boys only), high connectedness with adults outside the family (boys only), frequent family activities, and parental supervision during either waking, arriving home from school, evening mealtime, or when going to bed were all protective of youth violence (Resnick et al., 2004). Brookmeyer et al found that feeling connected to one's parents and school buffered the effect exposure to violence had on violent behavior (Brookmeyer, Fanti, & Henrich, 2006). Finally, a review by Cook and Laub found that strong parental supervision and attachment was a protective factor for youth violence (Cook & Laub, 2002).

## **Multilevel Factors**

### *Socioeconomic Status (SES)*

The socioeconomic status (SES) of a child's family and neighborhood in which they reside affects their likelihood lead exposure (Lidsky & Schneider, 2003). Children with lower family SES are more likely to live in older, rental properties with deteriorating windows and lead paint hazards. Socioeconomically disadvantaged neighborhoods are more likely to have these high-risk properties. SES is also a strong predictor of neurodevelopment (D. C. Bellinger, 2008). For these reasons, SES is typically a potential confounder of the association between lead exposure and a proposed outcome and controlled for in the analysis of this association.

Children of lower SES appear to be more vulnerable to the effects of lead once exposed. For example, a study by Bellinger et al found that both medium (6 to 7  $\mu\text{g}/\text{dL}$ ) and high (greater than 10  $\mu\text{g}/\text{dL}$ ) blood lead levels at 6 months were predictive of deficits in development for children of lower socioeconomic standing (D. C. Bellinger, 2008). Children of higher socioeconomic standing were only affected by high blood lead levels. Studies by Ris et al and Dietrich et al found the negative relationship between lead level and learning/IQ to be exacerbated in families with low SES (K. N. Dietrich, Succop, Berger, Hammond, & Bornschein, 1991; Ris et al., 2004). Other studies have found similar results (D. Bellinger, Leviton, & Sloman, 1990a; Lidsky & Schneider, 2003; Tong, McMichael, & Baghurst, 2000; Winneke & Kraemer, 1984). This increased vulnerability could be for many reasons. Children living in poverty are more likely to have nutrient deficiencies in calcium and iron and one of the ways lead affects child development is through its interaction with these specific minerals (D. C. Bellinger, 2008). Children living in poverty may carry genes that make them more susceptible to

the effects of lead or exposure to lead may trigger the expression of genes that make them more vulnerable to its effects (R. O. Wright & Baccarelli, 2007). Additionally, these children are less likely to grow up in an enriching and stimulating environment, so they may be less resilient to the effects of lead exposure than higher income children growing up in positive environments (D. C. Bellinger, 2008). Finally, living in poverty is stressful for children and they may have limited access to resources to help them cope with that stress (D. C. Bellinger, 2008). Stress can exacerbate the effects of lead exposure (D. C. Bellinger, 2008). Based on these findings, researchers may want to consider examining SES a modifier of lead toxicity, as well as a confounder (D. C. Bellinger, 2008).

SES has long been recognized as a key predictor of street shootings. Most street shootings occur in the neighborhoods with the lowest SES. For example, according to an annual report on homicides and nonfatal shootings in Milwaukee, in 2015, 82% of homicides and 83% of nonfatal shootings in Milwaukee occurred in the zip codes with the lowest SES (Milwaukee Homicide Review Commission, 2015). Lower family SES has also been identified as a predictor of future violent behavior. A longitudinal study by Loeber et al identified low SES and a family being on welfare as predictors of violence, and the family being on welfare as one of the 9 predictors of homicide offending (Loeber et al., 2005). In addition, a longitudinal study by Dubow et al found that lower family SES at ages 8 and 19 years old was a risk factor for adult violent offending (Dubow et al., 2016). Finally, a review by Farrington found that low SES is a major long-term predictor of male youth violence as well (Farrington, 1998a).

### *Exposure to violence*

Children exposed to violence are at increased risk of future firearm violence perpetration and victimization. Studies show that a predictor of future firearm violence perpetration is previous firearm violence exposure (Weaver, Borkowski, & Whitman, 2008). These outcomes were consistent across children who were injured by firearm violence, witnessed firearm violence or lived in neighborhoods with high levels of firearm violence (Garbarino, Bradshaw, & Vorrasi, 2002; Weaver et al., 2008). According to studies by Resnick et al and Brookmeyer et al, a history of violence victimization was one of the strongest predictors of youth violence (Brookmeyer et al., 2006; Resnick et al., 2004). A study examining the social and psychological predictors of violence in young, African American youth living a high crime neighborhood found that personal victimization was associated with self-reported use of violence (DuRant et al., 1994). The survey measuring personal victimization included being a victim of 27 types of violence including gang violence, selling drugs, burglary, police arrests, assaults, physical threats, sexual assaults, weapon carrying, firearm use, and intentional injuries such as stabbings, shootings, suicides, and murders (DuRant et al., 1994).

Firearm violence is concentrated in certain neighborhoods. For example, an annual report on homicides and nonfatal shootings in Milwaukee found that, in 2014, 42% of nonfatal shootings and 33.7% of homicides occurred within an 8.1 sq. mile area (Milwaukee is 97 square miles) (Milwaukee Police Department, 2016). Studies show that just living in those neighborhoods increases children's risk for adult violent offending. A review by Farrington found that living in a high-crime neighborhood was a major long-term predictor of male youth violence (Farrington, 1998a). And a longitudinal study by Loeber identified self-reported bad neighborhood as one of the 11 predictors of violent offending (Loeber et al., 2005).

## **Policy Level Factors**

### *Laws and Regulations*

Lead is regulated at both the federal and state levels. Federally, the U.S. Center for Disease Control and Prevention, Consumer Product Safety Commission, Department of Housing and Urban Development, Environmental Protection Agency, Food and Drug Administration, and the Occupational Safety and Health Administration all have a role in regulating lead (Stark & Shah, 2017). At the state level, the Department of Health Services, Department of Agriculture, Trade and Consumer Protection, Department of Children and Families and Department of Natural Resources all regulate lead further through Wisconsin Statutes and Administrative Codes (Stark & Shah, 2017). These regulations can affect food, product and environmental standards, funding, education, prevention, surveillance, and policy initiatives, all of which could impact lead exposure, abatement and interventions following exposure (Stark & Shah, 2017). For example, the federal government banned the use of lead-based paint in housing in 1978. Therefore, children living in housing built after 1978 are at lower risk for lead exposure. In addition, due to ongoing research on the effects of lead exposure in children, the actionable blood lead levels set by the CDC have been declining since 1960 - 60 µg/dL from 1960-1970, 40 µg/dL from 1970-1975, 30 µg/dL from 1975-1985, 25 from 1985-1991, 10 µg/dL 1991-2012, and 5 µg/dL from 2012 to present (K. N. Dietrich, 2010). These levels determine when a local health department intervenes to prevent further exposure to that child or initial exposure to other children in the home.

A firearm is required for an individual to commit firearm violence. Therefore, it is important to consider the effect firearm laws may have on access to firearms and firearm

violence, in general. Research evaluating this relationship has found that laws allowing more firearms (e.g., “Right to Carry” laws) do not reduce firearm violence. A study by Aneja et al found that “Right to Carry” laws (also known as concealed carry) were associated with increases in aggravated assaults, including firearm assaults (Aneja, Donohue, & Zhang, 2011). In addition, they found evidence that “Right to Carry” laws may increase crimes of rape, robbery, and murder (Aneja et al., 2011). Studies have also found that the number of more restrictive firearm laws can reduce firearm violence. A study by Fleegler et al found a negative correlation between the number of state firearm laws and the rate of firearm violence for suicides, homicides and overall (Fleegler, Lee, Monuteaux, Hemenway, & Mannix, 2013). An investigation by the National Journal found a similar correlation when comparing state firearm laws and firearm user restrictions to rates of shooting deaths (Isenstein, 2015). The greater the number of the following laws and restrictions at the state level - permit or license required to purchase a handgun, background checks extended to private sales, handgun owners required to register or report their handguns, difficulty level of obtaining a concealed carry permit, difficulty to obtain an open carry permit, and waiting period for obtaining handguns - the lower the age-adjusted number of firearm-related deaths per 100,000 residents (Isenstein, 2015). It also found that states without any form of “Stand Your Ground” law (laws that allow armed individuals to use deadly force if they believe they are in imminent danger) had lower average rates of firearm-related homicides than those with such laws (Isenstein, 2015). Other studies have found that specific firearm laws, versus the number of firearm laws, can reduce firearm violence. For example, a study by Lias indicated that laws restricting convicted felons from possessing a handgun impacted homicide rates (Lias, 2015). Additionally, Vigdor and Mercy found that female intimate partner homicide was reduced after states passed laws restricting access to firearms by individuals who are subject



to a restraining order (Vigdor & Mercy, 2006).

Similar to the majority agreement among scientists on global warming, there is scientific consensus that stricter firearm laws reduce firearm violence (Hemenway & Nolan, 2016). According to a poll of firearm researchers, agreement exists on the notion that more firearms and weaker firearm laws cause serious public health problems, that the costs of firearm availability are typically greater than the benefits, and that stronger firearm laws may improve public safety and health (Hemenway & Nolan, 2016).

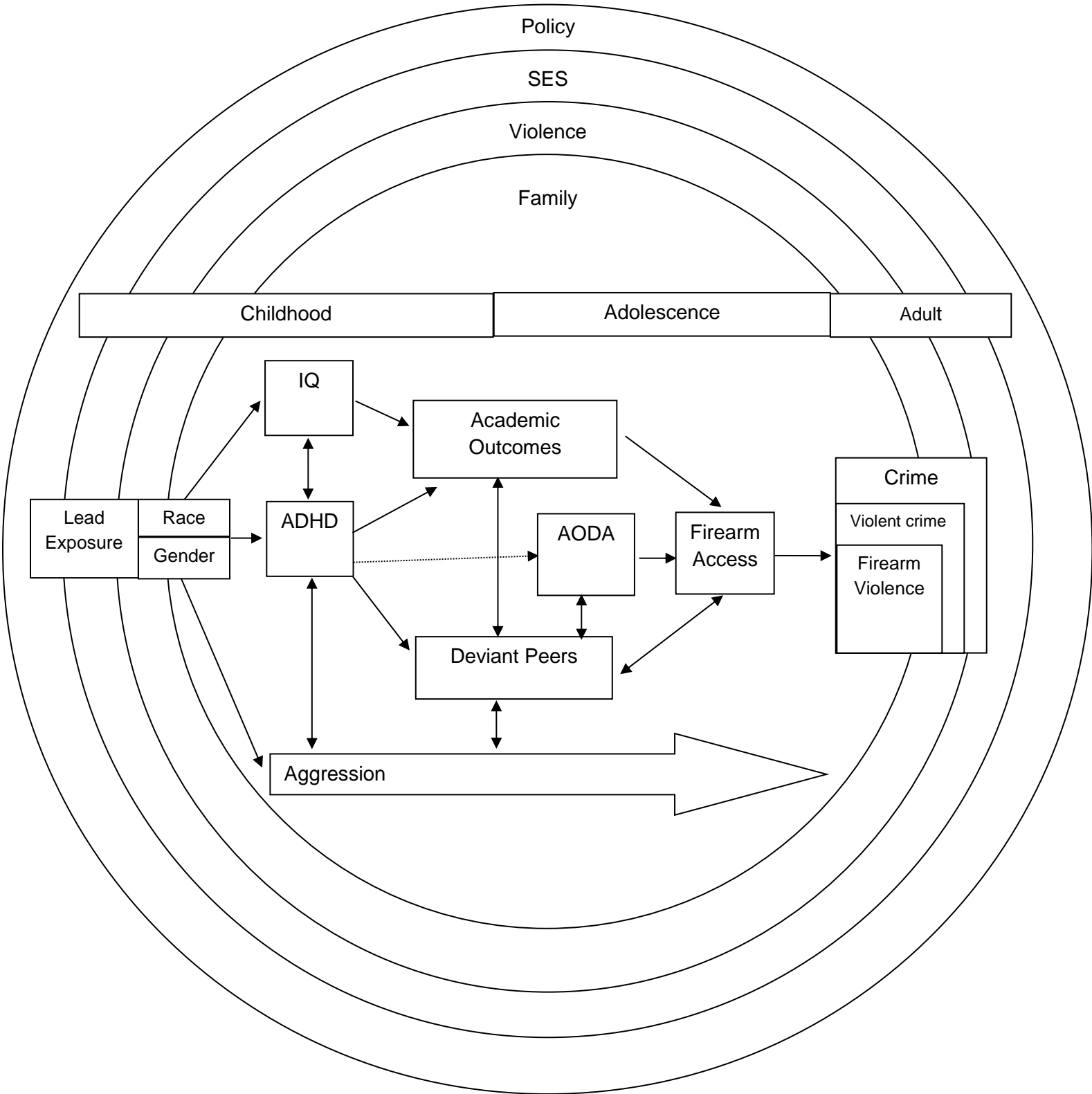
Despite its prevalence and high mortality and morbidity rates, firearm violence research is significantly underfunded (Stark & Shah, 2017). This is particularly apparent when firearm violence funding is compared to funding for other diseases. For example, between 2004-2015, funding for firearm violence research was 0.7% of the allocation for sepsis, a disease with a similar mortality rate (Stark & Shah, 2017). The shortage in funding is related to certain policy-level decisions. In 1996, Congress passed the “Dickey Amendment” that forbid the CDC from spending funds “to advocate or promote firearm control” (Stark & Shah, 2017). At the same time, Congress removed the amount of money the CDC had invested in firearm injury research the year prior (\$2.6 million) from the CDC’s budget and reallocated those funds for traumatic brain injury prevention. Similar restrictions have been extended to other agencies as well (National Institutes of Health) (Stark & Shah, 2017).

## **Discussion**

African American, non-Hispanic children are disproportionately affected by lead exposure compared to children of other races and there is evidence that males and females may be affected differently (Figure 3). Based on the extensive empirical evidence, we can

confidently say that childhood lead exposure is associated with IQ deficits, ADHD diagnosis and symptomatology (i.e. attentional, impulsivity, self-control and hyperactivity dysfunctions), and aggression, all of which are criminogenic. Recent studies have found that the IQ-violent offending relationship may be mediated by poor school performance and that higher IQ is protective against future violent offending. In addition, the ADHD-violent offending relationship may be mediated by academic outcomes, deviant peer association, aggression and/or substance use during childhood and adolescence. Finally, early and persistent aggression is a consistent predictor of violent offending as an adult.

Lead exposed children and violent offenders consistently have poor academic outcomes during childhood and adolescence. They score lower on reading, math and science tests, have lower grades overall, are less likely to graduate high school and have a greater risk of being suspended. This may be related to IQ deficits and/or behavioral issues such as inattention, low



**Figure 3: Conceptual Model of Childhood Lead Exposure and Firearm Violence**

self-control or aggression. Positive academic outcomes such as high grade point average, good school performance, and high educational aspirations have all been found to be protective against youth violence.

Individuals who are involved with firearm violence often associate with delinquent peers. Their relationship with these peers may have started because they were seeking out peers that shared some of their same issues, such as poor academic achievement, impulsivity and aggression, or they may have been introduced to this deviant peer group through their own substance use. Associating with a deviant peer group increases an individual's risk of selling and using drugs and alcohol, both of which are predictive of violent offending. Adolescents who sell drugs are significantly more likely to carry a firearm and they are most likely to acquire firearms through their deviant peer group. Conversely, associating with a conventional peer group has been found to be protective against violent offending.

Family level factors such as broken families, severe punishment and poor supervision play prominent roles before and after lead exposure. Family level factors can increase a child's risk of exposure as well as exacerbate the effects of that exposure. For example, families with lower SES are more likely to live in homes with increased lead hazards, putting a child at greater risk for exposure. Additionally, negative parenting practices may amplify a child's already aggressive behavior or ADHD, both of which are consequences of lead exposure. Family level factors, such as poor supervision and domestic violence, contribute to a child's academic outcomes, their association with deviant peers and their risk of developing substance use issues. Positive family level factors have also shown to be protective against violent offending.

Children and adolescents who are injured by firearm violence, witness firearm violence or live in a neighborhood with high levels of firearm violence are all at increased risk for future violent offending and victimization. Those who are exposed to violence in the home are also at increased risk for developing substance abuse issues. Living in a socioeconomically disadvantaged neighborhood increases the chances of children and adolescents being exposed to any form of violence.

One of the most important factors influencing lead exposure in children, and later firearm violence, is socioeconomic status. Children who are from families with low socioeconomic status and live in socioeconomically disadvantaged neighborhoods are more likely to be exposed to lead. The effects of lead exposure are then exacerbated by their low socioeconomic status, potentially making it a modifier instead of a confounder. For example, the lead-IQ relationship is exacerbated by low SES. Low socioeconomic status also predisposes children to future firearm violence, with higher socioeconomic status working as a protective factor.

Finally, policies at the local, state and federal level can affect lead exposure and firearm violence. Lead regulations can impact lead exposure, prevention efforts and interventions following exposure. More research is needed but there is scientific consensus that stricter firearm laws can reduce firearm violence.

This conceptual model provides a framework to guide future lead and firearm violence research. Future studies can use this model to develop hypotheses, explore the associations between the key constructs, examine potential mediating and modifying relationships, and interpret their results. In addition, researchers and practitioners can use this model to develop future firearm violence prevention interventions.

While the purpose of this review was to develop a more comprehensive, multilevel conceptual model for future research, we recognize that this model will be difficult to test empirically. It will be challenging to find longitudinal datasets that include all or most of these factors. If researchers do not have access to this kind of data, selecting variables or relationships to focus on may prove to be challenging. Due to the complex interplay of the different factors involved, researchers may want to consider using structural equation models to assist with the selection of variables for their analysis.

This conceptual model is more comprehensive than past research, however, it should be viewed as a starting point. Undoubtedly, there are areas where this conceptual model could be expanded. For example, traditionally firearm violence research has focused more on identifying risk factors. Only more recently has it broadened its scope to include protective factors and resiliency (David-Ferdon & Simon, 2014). Due to this, our model favors risk factors and could be strengthened by the inclusion of more protective factors. As more research is published in this area, we encourage researchers to build upon and expand this model as we did with the model developed by Narag et al, especially in regard to protective factors.

## CHAPTER 3: MANUSCRIPT #2

### Association of Childhood Blood Lead Levels with Firearm Violence Perpetration and Victimization in Milwaukee

#### Abstract

While elevated childhood lead levels have been found to be associated with juvenile delinquency, adult criminal behavior and violent offending, researchers have yet to examine the relationship between childhood lead exposure and adult firearm violence, specifically. We estimated the association between childhood lead exposure before age 6 years old and future firearm homicide and nonfatal shooting victimization and perpetration. This retrospective cohort study included 89,129 children born between June 1, 1986 - December 31, 2003 with at least one valid blood lead test reported before age 6 years old and stable residency in Milwaukee. The children were identified using data linked at the individual level from the Milwaukee Health Department and Milwaukee Public Schools. Of these children, 1,000 were identified as victims and 554 were identified as perpetrators of firearm violence by the Milwaukee Homicide Review Commission. We estimated odds ratios of firearm violence victimization and perpetration using logistic regression, adjusting for sex, race, socioeconomic status and year of birth. After adjustment for confounders, our results show that for every 1 $\mu$ g/dL increase in the mean childhood lead level, the odds of an individual becoming a victim of firearm violence increases (OR 1.04, 95% CI 1.03, 1.05) and the odds of an individual becoming a perpetrator of firearm violence increases (OR 1.03, 95% CI 1.02, 1.04). We saw similar results with mean peak childhood lead levels. When childhood blood lead levels were categorized, a significant dose-response relationship was found. This retrospective cohort study confirmed earlier clinical observations and recent research that have linked childhood lead exposure with violent crime,

specifically firearm violence. Both mean and peak childhood lead results were associated with firearm violence victimization and perpetration. Childhood lead exposure prevention needs to be considered in firearm violence prevention efforts.



## **Introduction**

Firearm violence remains a persistent, preventable, public health problem in the United States (U.S.). Over 10,000 people lose their lives and 60,000 people are injured by firearm violence each year (Center for Disease Control and Prevention, 2015a). While mass rampage shootings such as the Orlando nightclub shooting often garner the most media attention, street shootings are the most common types of firearm violence in the U.S. (Bushman et al., 2016). Fatal and nonfatal street shootings disproportionately affect young, African American males living in socially and economically disadvantaged urban areas. In fact, homicide (80% of which is perpetrated with a firearm) is the leading cause of death for young, African American males ages 15-24 and 25-34 years old (Center for Disease Control and Prevention, 2017). This same population also perpetrates the majority of firearm violence (J. L. Lauritsen & Laub, 2007). Studies have found overlapping risk factors for firearm violence perpetration and victimization (Loeber et al., 2012). Victims and perpetrators often have common characteristics because they share similar geography, high risk lifestyles and social networks that put them at greater risk for perpetration as well as victimization (Hindelang, Gottfredson, & Garofalo, 1978).

Identifying key risk and protective factors for firearm violence is paramount to developing effective prevention strategies. One environmental stressor that is a potential key risk factor for future violent crime is childhood lead exposure (Boutwell et al., 2016; Feigenbaum & Muller, 2016; D. M. Fergusson et al., 2008; Mielke & Zahran, 2012; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; Reyes, 2007; P. B. Stretesky & Lynch, 2001; J. P. Wright et al., 2008). Lead is an environmental toxicant that is particularly harmful to children and may cause irreversible, long term damage to a child's brain. Varying degrees of lead exposure during childhood have shown to have deleterious effects on intelligence and cognition,

(Baghurst et al., 1992; Banks et al., 1997; D. C. Bellinger et al., 1992; Canfield et al., 2003; K. N. Dietrich et al., 1993; B. P. Lanphear et al., 2005) behavior, (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; P. B. Stretesky & Lynch, 2004; J. P. Wright et al., 2008) and brain structure (Cecil et al., 2008). Research suggests that the physical, mental and behavioral effects of childhood lead exposure may be conducive to future criminal behavior, including violent crime (Narag et al., 2009). While elevated childhood lead levels have been found to be associated with juvenile delinquency (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; H. L. Needleman et al., 2002; Olympio et al., 2009) and adult criminal behavior and violent offending, (D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004) researchers have yet to disaggregate violent crime as the outcome variable and examine the relationship between childhood lead exposure and adult firearm violence, specifically.

It is important to disaggregate violent crime data because violent crimes differ in many ways, such as by lethality and frequency. Firearm violence is the most lethal of all violent crime. The mean annual case fatality rate (the proportion of individuals injured or infected with a disease who die as a result of that injury or disease) for firearm-related assaults nationally is 19% (calculated for 2010-2012) (Fowler et al., 2015). Additionally, violent crime varies in frequency by type of crime. For example, firearm homicides and nonfatal shootings generally make up a small percentage of overall annual violent crime.

Aggregating violent crime data into one group can produce biased results. A study by Cherry and List found significantly different parameter estimates when they examined the deterrence hypothesis with aggregated and disaggregated crime data (Cherry & List, 2002). The study examined three models: one model aggregated all types of crime into one crime index, one

model disaggregated crime into violent and property crime, and one model completely disaggregated crime by type - murder, rape, robbery, assault, burglary, larceny and motor vehicle. The study found substantial variability in the parameter estimates in each model (Cherry & List, 2002). For example, this occurred when they examined the effect the size of the police force had on crime. The parameter estimates for the aggregated crime index showed that the size of the police force was associated with more crime overall: 0.413, but for the completely disaggregated model, the parameter estimates indicated less crime with an increase in police force, for example, parameters of -0.119 (assault) and -0.395 (larceny) (Cherry & List, 2002).

When firearm violence has been disaggregated from violent crime, prior research has focused predominantly on homicides. While homicide data tends to be more comprehensive because of the amount of resources spent on homicide investigations (e.g., more in-depth investigations, more complete police reports, etc.), homicides are relatively rare events (Papachristos, Wildeman, & Roberto, 2015). Nonfatal shootings occur with far greater frequency than homicides. Despite being far more common and having similar causes and consequences, nonfatal shootings remain significantly understudied (Lee, 2012; Papachristos et al., 2015).

Prior research has found that elevated childhood lead levels are associated with juvenile and adult violent offending (Denno, 1990; D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; H. L. Needleman et al., 2002; R. Nevin, 2000; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004). A study by Pihl & Ervin examined the differences in hair element levels, including lead, between 30 violent and 19 nonviolent males incarcerated in an institute for psychiatrically disturbed criminals (Pihl & Ervin, 1990). Chi-squared analysis revealed that adult male inmates convicted of violent crimes were more likely than adult male inmates

convicted of property crimes to have elevated hair lead levels, but violent crime was aggregated to include murder, assault, armed robbery, and violent rape (Pihl & Ervin, 1990). A longitudinal study by Denno tracked 987 African-American youth born in Philadelphia from birth to age 22 years to try to identify early predictors of juvenile and adult criminal behavior (Denno, 1990). The study found that among males, lead intoxication at age 7 years was one of the strongest predictors of disciplinary problems from ages 13 to 14, juvenile delinquency and adult offenses for male participants (Denno, 1990). The study did not disaggregate juvenile delinquency or adult offenses by type of violent offense. Needleman et al found similar results when they examined the relationship between lead and adjudicated delinquents. In a case control study of 194 adjudicated delinquent juveniles and 146 nondelinquent juveniles, the bone lead concentrations of the adjudicated delinquent juveniles were significantly higher than those of the nondelinquent juveniles after controlling for covariates (H. L. Needleman et al., 2002). Adjudicated delinquent juveniles were four times more likely than controls to have bone lead concentrations above 25ppm (H. L. Needleman et al., 2002). The adjudicated delinquent juveniles could be serving time for violent or nonviolent offenses, but the type of offense was not disaggregated for this study. More recently, Wright et al examined the relationship between early lead exposure (prenatal, mean childhood, and at 6.5 years) and criminal arrests in young adulthood in 250 participants in the Cincinnati Lead Study birth cohort (J. P. Wright et al., 2008). They found that for every 5  $\mu\text{g}/\text{dL}$  increase in prenatal and 6-year-old blood lead concentration, the adjusted total arrest rates were significantly greater (prenatal: RR=1.40, 95% CI 1.07-1.85 and 6.5 years: RR=1.27, 95% CI 1.03-1.57) (J. P. Wright et al., 2008). The adjusted arrest rates for violent crimes were also significantly greater for each 5  $\mu\text{g}/\text{dL}$  increase in mean childhood lead (RR=1.30, 95% CI 1.03-1.64) and 6-year-old blood lead (RR=1.48, 95%

CI 1.15-1.89) (J. P. Wright et al., 2008). Once again, violent arrests were aggregated to include murder, rape, domestic violence, assault, robbery and possession of a firearm (J. P. Wright et al., 2008). Finally, Fergusson and colleagues examined a 21-year longitudinal New Zealand birth cohort to determine if there was a link between dentine lead exposure measured between 6-9 years old and both officially recorded and self-reported criminal convictions between the ages of 14-21 years old (D. M. Fergusson et al., 2008). They found a significant dose-response relationship between childhood lead levels and rates of violent and property crime (officially recorded and self-reported), after controlling for confounding (D. M. Fergusson et al., 2008). While violent crime was disaggregated from nonviolent (property) crime, the study did not examine firearm violence, specifically.

To overcome previous limitations and address current gaps in the research, we examined the relationship between individual level childhood lead exposure and firearm homicide and nonfatal shooting perpetration and victimization risk. Since victims and perpetrators share common characteristics, separate analyses were conducted for both. We hypothesized that higher levels of lead exposure during childhood would lead to higher odds of firearm violence victimization and perpetration, after controlling for potential confounding variables.

## **Methods**

### **Study population**

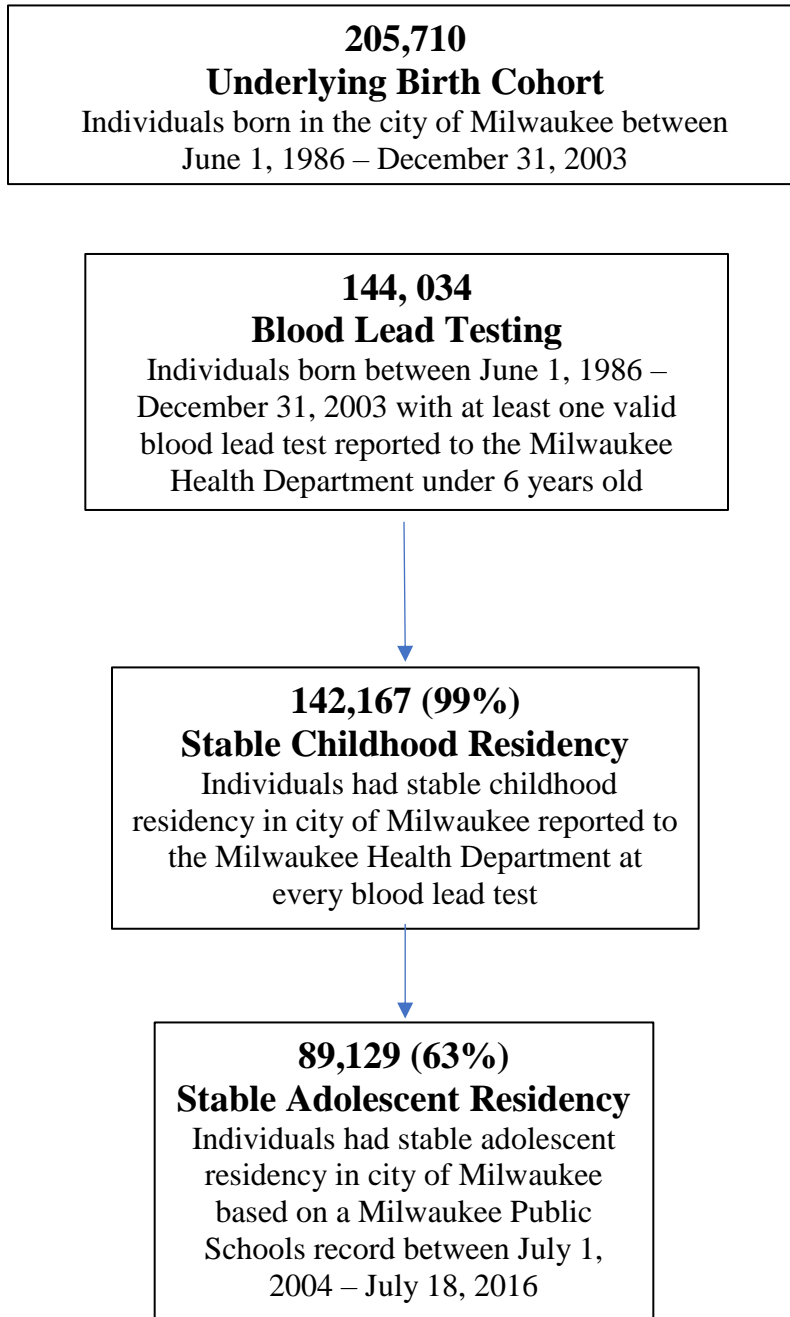
We conducted a cohort study in the city of Milwaukee, Wisconsin by performing individual-level linkages of available datasets on blood lead testing and firearm crime. All data for this study was pulled from an integrated data system called DataShare MKE. DataShare MKE is a multi-sector, multi-disciplinary integrated data system that includes data from the

Milwaukee Health Department, Milwaukee Public Schools and the Milwaukee Homicide Review Commission (among other datasets). Each individual in any of the datasets is assigned a conformed person ID and then this ID is used to link individuals across all of the datasets.

From this resource we constructed a cohort consisting of all children born between June 1, 1986 - December 31, 2003 who had at least one valid blood lead test collected and reported to the Milwaukee Health Department before age 6 years old. To ensure that the individuals in our cohort maintained residential stability, for the purposes of follow-up, they had to have a city of Milwaukee address reported at every blood lead test. Additionally, in order to ensure that these individuals would be included in our outcome dataset if they were a firearm homicide or nonfatal shooting victim or perpetrator and to control for potential education-related confounding, we only included individuals who were present in Milwaukee Public Schools student records between July 1st, 2004 and July 18th, 2016. Milwaukee Public Schools student record data does not include individuals who attend a charter, Montessori, or private school in Milwaukee or live in Milwaukee but attend a school in a different school district by using Wisconsin's open enrollment law. A total of 89129 individuals met these criteria (Figure 1). While we are not able to link the cohorts, the underlying birth cohort (children born in Milwaukee between June 1, 1986 - December 31, 2003) for this study is 205,710 children.

We chose to restrict the sample to individuals born between June 1st, 1986 - December 31st, 2003 because this time period allowed for the construction of a follow-up cohort and the most complete linkage across datasets. Our example is the linkage with the public school data needed to determine adolescent residence: June 1st, 1986 is the earliest an individual could be born and still potentially have a Milwaukee Public Schools student record between July 1st, 2004 - July 18th, 2016 (Milwaukee Public Schools data was only available during that time period).

Individuals born after June 1st, 1986 could be an 18-year-old senior in high school during the 2004-2005 school year. Individuals born before December 31st, 2003 could have a Milwaukee Public Schools student record and would be 12 years old by December 15th, 2015, the latest



**Figure 1: Study Population Flow Diagram**

incident date in the Milwaukee Homicide Review Commission homicide and nonfatal shooting databases. Participants included in this sample must be at least 12 years of age at the time they developed the outcome as this study is only examining juvenile and adult firearm violence victimization and perpetration. There were no perpetrators under the age of 12 years old in the Milwaukee Homicide Review Commission homicide or nonfatal shooting databases. Figure 2 illustrates the different source datasets and timelines.

### **Childhood Lead Exposure**

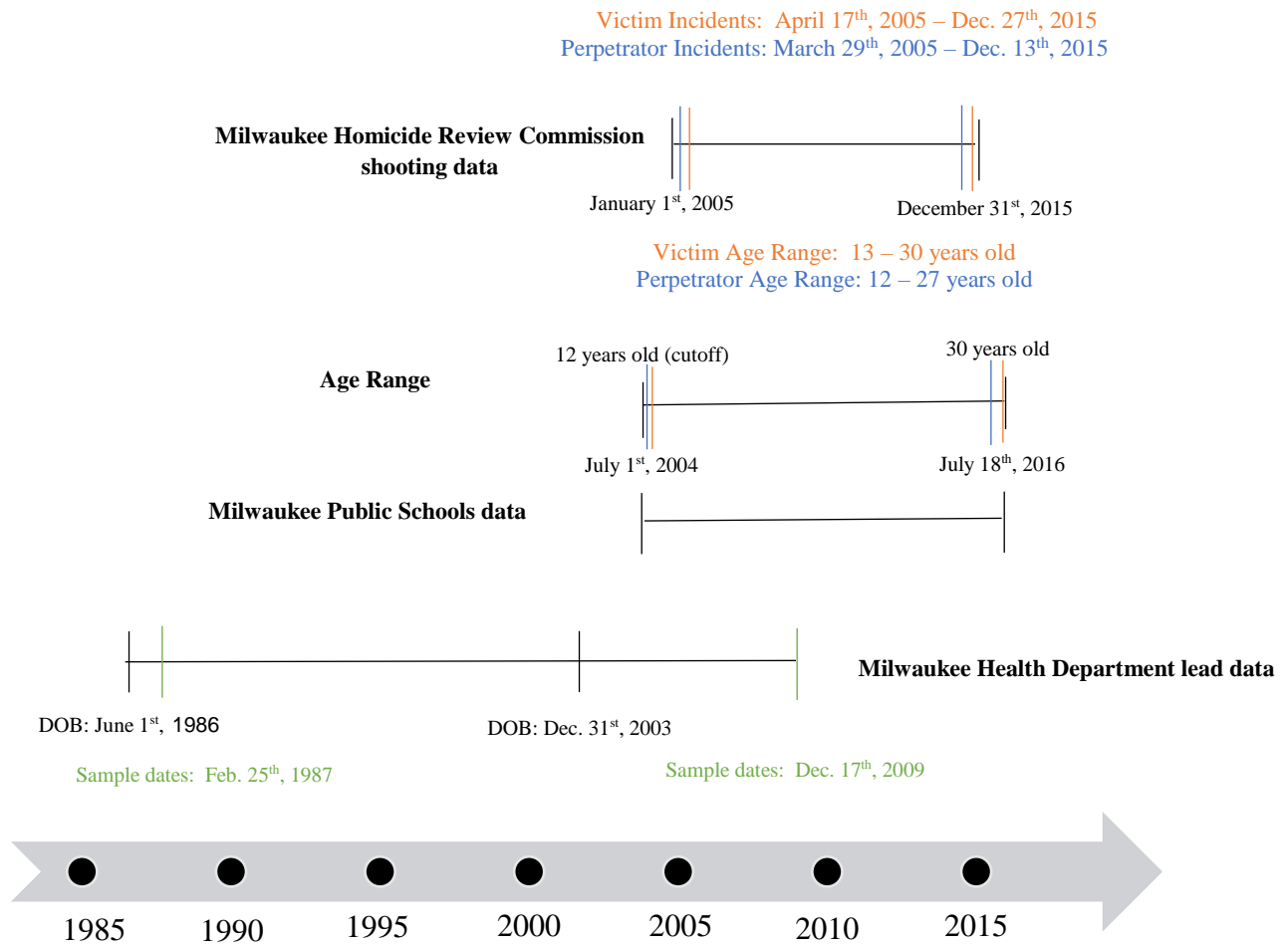
The independent variable for this study is childhood lead exposure. For this study, we will use blood lead data from the Milwaukee Health Department in DataShare MKE to evaluate childhood lead exposure. The Milwaukee Health Department database in DataShare MKE includes all blood lead tests reported to the Milwaukee Health Department between March 25th, 1980 and May 2nd, 2012. Based on the cohort eligibility criteria, we identified individuals born between June 1st, 1986 and December 31st, 2003 who had at least one blood lead test before age 6 years old reported to Milwaukee Health Department and were also present in Milwaukee Public Schools records between July 1st, 2004 and December 31st, 2015.

Blood lead test results were reported as a continuous value in micrograms per deciliter ( $\mu\text{g}/\text{dL}$ ). The actual testing was completed at multiple clinics or labs throughout Milwaukee but then all test results were reported the Milwaukee Health Department. Childhood lead exposure was operationalized in two ways: the mean of all blood lead test results for an individual before age 6 years old and the peak blood lead test result for each individual before age 6 years old. Childhood lead exposure was operationalized in this manner (age cutoff and mean/peak levels) based on previous research (M. S. Amato et al., 2012; M. S. Amato et al., 2013; K. N. Dietrich et



al., 1991; K. N. Dietrich et al., 2001; D. M. Fergusson et al., 2008; J. P. Wright et al., 2008), the age of increased vulnerability (D. C. Bellinger, 2004; Hwang, 2007; H. Needleman, 2004) and the mean age children reported lead tests in the Milwaukee Health Department data. To further investigate the dose-response

**Figure 2: Sample Datasets Timeline**



effect of childhood lead exposure on firearm violence victimization, lead mean and lead peak values were transformed into categorical variables. The categories were chosen based on the

actionable levels set by the Center for Disease Control and Prevention and Wisconsin state regulations (CDC 2012: 5 µg/dL, CDC: 1991 - 10 µg/dL, DHS 254: 5 - 19 µg/dL and 20 µg/dL)

Based on Milwaukee Health Department information on blood lead test validity, blood lead test results above 100 µg/dL (capillary and venous) and capillary tests above 65 µg/dL were removed before the final analysis. We removed 36 blood lead test results above 100 µg/dL and 57 capillary lead test results above 65 µg/dL. Two individuals were dropped from the final sample because one had only one lead test result over 100 µg/dL and the other had only one capillary lead test result over 65 µg/dL.

### **Firearm Violence Victimization**

We defined firearm violence victims (n = 1,000) from within the cohort as individuals who were killed with a firearm (homicides) or shot at (nonfatal shooting) within the boundaries of the city of Milwaukee between January 1st, 2005 and December 31st, 2015. These dates correspond to times when data was available. The potential ages of the cohort members ranged from 12 years old to 30 years old (Figure 2), although observed ages at victimization ranged from 13 years old to 30 years old.

The primary source of data on firearm violence are Milwaukee Police Department homicide files and police reports. Each day, three trained Milwaukee Homicide Review Commission staff (police officer, administrative staff and a researcher) extracted data from these sources and entered it into the Milwaukee Homicide Review Commission homicide and nonfatal shooting database. The data was first entered by the police officer and/or administrative staff and then validated by the researcher. A data collection instrument based on the National Violent Death Reporting System and Uniform Crime Reporting standards was used to ensure that the

information was captured consistently by staff. Any discrepancies in the data were discussed amongst the team and with the Director of the Milwaukee Homicide Review Commission, if necessary. Milwaukee Homicide Review Commission staff would review open cases daily for updated information.

In addition, the Milwaukee Homicide Review Commission convened a multidisciplinary group of criminal justice professionals (law enforcement, prosecutors, corrections agents, etc.) monthly (from 2005 to 2015) to review homicide and nonfatal shooting cases (M. O'Brien, 2007). During this review, additional information not included in the police reports was discussed among partners. Milwaukee Homicide Review Commission staff used this review process to gather additional information to enter into the databases on homicides and nonfatal shootings.

Firearm homicides are coded in the homicide database as homicides caused by a firearm (unknown), handgun, or long gun. A victim was identified as an individual coded as a victim in the Milwaukee Police Department homicide files or incident reports who sustained a gunshot wound. For this study, individuals identified in the Milwaukee Homicide Review Commission homicide (firearm homicides only) and nonfatal shooting databases as victims will serve as firearm violence victims.

### **Firearm Violence Perpetration**

We defined firearm violence perpetrators (n = 554) from within the cohort as individuals who perpetrated a firearm homicide or nonfatal shooting within the boundaries of the city of Milwaukee between January 1st, 2005 and December 31st, 2015. These dates correspond to times when data was available. The potential ages of the cohort members ranged from 12 years

old to 30 years old (Figure 2), although observed ages at perpetration ranged from 12 years old to 27 years old.

As with victims, the primary source of data on firearm violence perpetration are Milwaukee Police Department homicide files, police reports and case reviews. The methods for data collection are outlined above under Firearm Violence Victimization. A suspect was identified as an individual coded as an arrestee, suspect and/or a person of interest with corroborated witness identification or a strong investigative lead in the Milwaukee Police Department homicide files and police reports. For this study, individuals identified in the Milwaukee Homicide Review Commission homicide (firearm homicides only) and nonfatal shooting databases as suspects will serve as firearm violence perpetrators.

It should be noted that there is very little overlap in the victims, perpetrators and incidents in our data and therefore we did not factor that into the final analysis. Eleven individuals were a victim and 35 were a perpetrator of both a firearm homicide and nonfatal shooting. In addition, there were 8 firearm homicide incidents and 21 nonfatal shooting incidents where we had both victim and perpetrator incident data.

### **Covariates**

The covariates for this study included: race, sex, socioeconomic status (SES) and year of birth. All of these factors have been found to be correlated with both lead exposure and violence (D. C. Bellinger, 2004; D. C. Bellinger, 2008; Bushman et al., 2016; David-Ferdon & Simon, 2014; Lidsky & Schneider, 2003; Wisconsin Department of Health Services, 2014). We evaluated the association between each of these covariates and the independent (childhood lead

exposure) and dependent variables (firearm violence perpetration and firearm violence victimization) and included them in the adjusted multivariate analysis.

Race, sex, SES, and year of birth were obtained from the Milwaukee Health Department database. Sex corresponds to the sex of the child and race was the race of the child reported to the Milwaukee Health Department. Due to the limited number of individuals with American Indian/Alaskan Native, Asian, Other, and Unknown listed as their race, these races will be combined into one category (Other). The final race categories were Black/African American, White and Other. SES was not reported to the Milwaukee Health Department, therefore the zip code of the child's residence reported to the Milwaukee Health Department will be used to determine their SES level (lower, middle, or upper). Using 2000 Census data, a study by Vilas et al created an SES index based on the average median income (income) and percentage of people with a bachelor's degree (education) by zip code in Milwaukee.(Vila et al., 2007) The zip codes were ranked and then grouped into four SES levels: lower (53204, 53205, 53206, 53208, 53210, 53212, 53215, 53216, 53218, and 53233), middle (53207, 53209, 53214, 53219, 53220, 53221, 53224, 53225, 53227, 53235) upper (53202, 53203, 53211, 53213, 53217, 53222, 53223, 53226, 53228) and other (53200, 53201, 53234, 53237).(Vila et al., 2007) The other category included zip codes that were PO boxes (53201, 53234, 53237) and of unknown address (53200). Year of birth was the year of birth reported to the Milwaukee Health Department (Table 1).

**Table 1: Study Variables**

Variable Type	Variable	Level of Measurement	Potential Response	Data Source
Dependent	Firearm Violence Perpetration	Dichotomous	Yes/No	Milwaukee Homicide Review Commission
Dependent	Firearm Violence Victimization	Dichotomous	Yes/No	Milwaukee Homicide Review Commission
Independent	Childhood Lead Exposure (Peak and Mean)	Continuous and Categorical	Range: Peak (0, 97 $\mu\text{g}/\text{dL}$ ), Mean (0, 94 $\mu\text{g}/\text{dL}$ ); Categorical - Under 5 $\mu\text{g}/\text{dL}$ , 5-9 $\mu\text{g}/\text{dL}$ , 10-19 $\mu\text{g}/\text{dL}$ , and 20 $\mu\text{g}/\text{dL}$ and above	Milwaukee Health Department
Covariate	Sex	Dichotomous	Male/Female	Milwaukee Health Department
Covariate	Race	Categorical	Black/African American, White, Other (American Indian/Alaskan Native, Asian, Other, Unknown)	Milwaukee Health Department
Covariate	Socioeconomic status (SES)	Categorical	Lower, Middle, Upper, Other SES	Milwaukee Health Department
Covariate	Year of Birth	Categorical	1986 - 2003	Milwaukee Health Department

## **Analysis**

We used logistic regression analysis to quantify the magnitude and precision of the association of childhood lead exposure (independent variable) and firearm violence perpetration (dependent variable) and the association of childhood lead exposure (independent variable) and firearm violence victimization (dependent variable). The adjusted model included childhood lead exposure, race, SES and year of birth. We based the coding of the independent variable and covariates on prior literature, LOESS and AIC results, and ease of interpretation. Chi square, t-tests and ANOVA were used to evaluate the relationship between the covariates and the independent and dependent variables. Odds ratios (OR) were calculated for the unadjusted (no covariates), partially adjusted (sex, race, SES), and fully adjusted (sex, race, SES, and year of birth) models. Only the fully adjusted OR was interpreted as it is the less-confounded estimate. Standard errors were used to construct 95% confidence intervals around each OR for each model. The model was estimated using the “logistic” command in Stata14 (StataCorp, 2015).

## **Results**

A total of 89,129 individuals met our inclusion criteria by date of birth, lead tests reported, and residency. The overall sample was split almost equally by sex, with slightly more males (51%) than females (49%) (Table 2). Two-thirds of the sample was African American (63%), a little under one third was White (27%) and the remainder were Other races (10%). Almost three quarters of the sample fell in the lowest socioeconomic category (74%), followed by just under 20% in the Middle, 4% in the Upper and 3% in the Other category. Overall mean lead levels (Lead Mean: 7.6  $\mu\text{g}/\text{dL}$ , SD: 5.7  $\mu\text{g}/\text{dL}$  and Lead Peak: 9.6  $\mu\text{g}/\text{dL}$ , SD: 9  $\mu\text{g}/\text{dL}$ ) were higher than the current actionable level set by the Center for Disease Control and

Prevention in 2012 (5 µg/dL) but lower than the actionable level set between 1985-1991 (25 µg/dL) and 1991 - 2012 (10 µg/dL). Median lead levels were slightly lower than the means (Lead Mean: 5.5 µg/dL and Lead Peak: 7 µg/dL) but also followed this trend. When lead levels were examined by population characteristics, males had higher mean lead levels than females (Lead Mean for males: 7.7 µg/dL, SD: 5.8 µg/dL vs. for females 7.4 µg/dL, SD: 5.6 µg/dL and Lead Peak for males: 9.8 µg/dL, SD: 8.9 µg/dL vs. for females 9.3 µg/dL, SD: 9.1 µg/dL), African Americans had higher mean lead levels than Whites and Other races (Lead Mean for African Americans: 8.7 µg/dL, SD: 6.2 µg/dL vs. for Whites 5.5 µg/dL, SD: 4 µg/dL vs. for Other races 6.4 µg/dL, SD: 4.5 µg/dL and Lead Peak for African Americans: 11 µg/dL, SD: 9.7 µg/dL vs. for Whites 6.9 µg/dL, SD: 6.9 µg/dL vs. for Other races 8.2 µg/dL, SD: 7.6 µg/dL) and individuals with the lowest socioeconomic status had higher mean lead levels than individuals in all other socioeconomic status categories. Average mean and peak lead levels consistently decreased over time for individuals born between 1986 and 2003. For example, the mean peak lead level for individuals born in 1986 was 21.2 µg/dL, while the mean peak lead level for individuals born in 2003 was 5.9 µg/dL.



**Table 2: Population Characteristics of Sample Population, Victims and Perpetrators**

	<b>All</b>	<b>Victims</b>	<b>Perpetrators</b>	<b>Lead Mean (µg/dL)</b> Mean (Median)	<b>Lead Peak (µg/dL)</b> Mean (Median)
<b>Total</b>	89,129 (100%)	1,000 (1%)	554 (1%)		
<b>Sex</b>					
Male	45,774 (51%)	872 (87%)	529 (95%)	7.7 (5.6)	9.8 (7)
Female	43,355 (49%)	128 (13%)	25 (5%)	7.4 (5.3)	9.3 (6)
<b>Race/Ethnicity</b>					
African American	56,433 (63%)	929 (93%)	506 (91%)	8.7 (6.6)	11 (8)
White	24,351 (27%)	65 (6.5%)	43 (8%)	5.5 (4.5)	6.9 (5)
Other	8,345 (10%)	6 (0.5%)	5 (1%)	6.4 (5)	8.2 (6)
<b>SES</b>					
Lower	65,551 (74%)	869 (87%)	491 (88.5%)	8.5 (6.3)	10.9 (8)
Middle	17,165 (19%)	104 (10%)	48 (9%)	5.3 (4.3)	6.3 (5)
Upper	3,350 (4%)	10 (1%)	6 (1%)	4.6 (4)	5.4 (4)
Other	3,063 (3%)	17 (2%)	9 (1.5%)	5 (4)	5.7 (5)
<b>Year of Birth</b>					
1986	331 (0.4%)	6 (0.6%)	6 (1.1%)	19.3 (17)	21.2 (17)
1987	1264 (1.4%)	34 (3.4%)	18 (3.3%)	16 (14)	17.9 (15)
1988	2003 (2.3%)	63 (6.3%)	35 (6.3%)	15.1 (13)	17.4 (14)
1989	2743 (3.1%)	86 (8.6%)	44 (8%)	14 (13)	16.4 (14)
1990	3696 (4.2%)	113 (11.3%)	69 (12.5%)	12.7 (11)	15.3 (13)
1991	4601 (5.2%)	111 (11.1%)	67 (12.1%)	11.1 (10)	13.8 (11)
1992	5179 (5.8%)	114 (11.4%)	70 (12.7%)	9.9 (8.3)	12.5 (10)

1993	5400 (6.1%)	123 (12.3%)	71 (12.8%)	9 (7.5)	11.5 (9)
1994	5717 (6.4%)	99 (9.9%)	56 (10.1%)	8.2 (7)	10.6 (8)
1995	5866 (6.6%)	94 (9.4%)	52 (9.4%)	7.5 (6)	9.8 (7)
1996	5967 (6.7%)	71 (7.1%)	32 (5.8%)	6.8 (5.5)	9 (7)
1997	6145 (6.9%)	32 (3.2%)	16 (2.9%)	6.4 (5)	8.4 (6)
1998	6450 (7.2%)	22 (2.2%)	12 (2.2%)	6.1 (5)	7.8 (5)
1999	6910 (7.8%)	11 (1.1%)	2 (0.4%)	5.8 (5)	7.4 (5)
2000	6944 (7.8%)	8 (0.8%)	2 (0.4%)	5.4 (4.5)	7 (5)
2001	6834 (7.7%)	4 (0.4%)	1 (0.2%)	5.1 (4.3)	6.6 (5)
2002	6562 (7.4%)	3 (0.3%)	1 (0.2%)	4.9 (4)	6.2 (4)
2003	6517 (7.3%)	6 (0.6%)	0 (0.0%)	4.6 (4)	5.9 (4)

**Lead Mean  
(µg/dL)**

Mean (Median)	7.6 (5.5)	12.7 (11)	12.7 (11)
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**Lead Peak  
(µg/dL)**

Mean (Median)	9.6 (7)	16 (13)	15.8 (13)
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**Lead Mean**

<5	35,066 (39%)	82 (8%)	36 (7%)
5-9	32,609 (37%)	329 (33%)	201 (36%)
10-19	17,399 (19.5%)	421 (42%)	234 (42%)
20+	4,055 (4.5%)	168 (17%)	83 (15%)

**Lead Peak**

<5	23,194 (26%)	55 (6%)	19 (3%)
5-9	35,038 (39%)	244 (24%)	148 (27%)
10-19	21,681 (24%)	419 (42%)	233 (42%)
20+	9,216 (10%)	282 (28%)	154 (28%)

## **Firearm violence victimization**

Of the 89,129 individuals in our sample, 1,000 later were victims of a firearm homicide or nonfatal shooting. Victims were mostly males (87%), African Americans (93%), and individuals in the lowest socioeconomic status category (87%) (Table 2). Prevalence of firearm violence victimization increased by year of birth between 1986 - 1990, plateaued from 1990 - 1993, and then decreased each year until 2003. The greatest proportion of victims were born in 1993 (12.3%). This differed when compared to the overall sample. Mean lead levels for victims of firearm violence (Lead Mean: 12.7, SD: 7.2  $\mu\text{g/dL}$  and Lead Peak: 16, SD: 10.8  $\mu\text{g/dL}$ ) were higher than the actionable levels set by the Center for Disease Control and Prevention in 2012 (5  $\mu\text{g/dL}$ ) and from 1991 - 2012 (10  $\mu\text{g/dL}$ ) but lower than the actionable level between 1985-1991 (25  $\mu\text{g/dL}$ ). Median lead levels for victims were slightly lower than the means (Lead Mean: 11  $\mu\text{g/dL}$  and Lead Peak: 13  $\mu\text{g/dL}$ ).

After adjustment for confounders, our results show that for every 1  $\mu\text{g/dL}$  increase in the mean childhood lead level, the odds of an individual becoming a victim of firearm violence increases (OR 1.04, 95% CI 1.03, 1.05) (Table 3). Similar results were found for each 1  $\mu\text{g/dL}$  increase in peak childhood lead level (OR 1.02, 95% CI 1.01, 1.03). To further investigate the dose-response effect of childhood lead exposure on firearm violence victimization, lead mean and lead peak values were transformed into categorical variables and odds ratios were calculated (Table 3). After adjustment for confounders, the odds of an individual becoming a victim of firearm violence are 1.83 times greater for an individual with a mean childhood lead level between 5 - 9  $\mu\text{g/dL}$  compared to an individual with a mean childhood lead level under 5  $\mu\text{g/dL}$  (95% CI 1.4, 2.4). After adjustment, the odds of an individual becoming a victim of firearm violence are 2.5 times greater for an individual with a mean childhood lead level between 10 - 19

**Table 3: Odds ratios for the association between childhood lead levels and firearm violence victimization and perpetration in Milwaukee**

	<b>Firearm Violence Victimization (95% CI)</b> n = 1,000	<b>Firearm Violence Perpetration (95% CI)</b> n = 554	
<b>Unadjusted<sup>a,b</sup></b>			n = 89,129
Lead Mean	1.094 (1.087, 1.10)	1.09 (1.08, 1.1)	
Lead Peak	1.052 (1.047, 1.057)	1.05 (1.04, 1.06)	
<b>Partially Adjusted<sup>c</sup></b>			n = 89,129
Lead Mean	1.074 (1.066, 1.082)	1.07 (1.06, 1.08)	
Lead Peak	1.04 (1.035, 1.046)	1.04 (1.03, 1.045)	
<b>Fully Adjusted<sup>d</sup></b>			n = 89,129
Lead Mean	1.04 (1.03, 1.045)	1.03 (1.02, 1.04)	
Lead Peak	1.02 (1.01, 1.03)	1.02 (1.01, 1.023)	
<b>99 Unadjusted<sup>e,b</sup></b>			n = 89,129
Lead Mean			
0-4 µg/dL	-	-	
5-9 µg/dL	4.3 (3.4, 5.5)	6.04 (4.2, 8.6)	
10-19 µg/dL	10.6 (8.3, 13.4)	13.3 (9.3, 18.8)	
≥20 µg/dL	18.4 (14.1, 24.1)	20.3 (13.7, 30.1)	
Lead Peak			
0-4 µg/dL	-	-	
5-9 µg/dL	2.95 (2.2, 3.95)	5.2 (3.2, 8.3)	
10-19 µg/dL	8.3 (6.3, 11)	13.3 (8.3, 21.2)	
≥20 µg/dL	13.3 (9.9, 17.7)	20.7 (12.9, 33.4)	

**Partially Adjusted<sup>c</sup>**

n = 89,129

## Lead Mean

0-4 µg/dL	-	-
5-9 µg/dL	3.4 (2.6, 4.3)	4.6 (3.2, 6.7)
10-19 µg/dL	6.8 (5.3, 8.7)	8.3 (5.8, 11.9)
≥20 µg/dL	10.7 (8.1, 14.1)	11.4 (7.6, 17.1)

## Lead Peak

0-4 µg/dL	-	-
5-9 µg/dL	2.4 (1.8, 3.2)	4.1 (2.6, 6.7)
10-19 µg/dL	5.4 (4.03, 7.2)	8.5 (5.3, 13.6)
≥20 µg/dL	7.5 (5.6, 10.2)	11.5 (7.0, 18.7)

**Fully Adjusted<sup>d</sup>**

n = 89,129

## Lead Mean

0-4 µg/dL	-	-
5-9 µg/dL	1.83 (1.4, 2.4)	2.3 (1.6, 3.3)
10-19 µg/dL	2.5 (1.9, 3.2)	2.6 (1.8, 3.8)
≥20 µg/dL	3.4 (2.5, 4.5)	3.0 (1.9, 4.5)

## Lead Peak

0-4 µg/dL	-	-
5-9 µg/dL	1.5 (1.09, 2.0)	2.5 (1.5, 4.0)
10-19 µg/dL	2.2 (1.7, 3.0)	3.2 (2.0, 5.2)
≥20 µg/dL	2.6 (1.9, 3.6)	3.6 (2.2, 5.9)

<sup>a</sup>Continuous models are presented as the change in the odds of firearm violence victimization and perpetration risk for every 1 µg/dL increase in childhood lead level

<sup>b</sup>Unadjusted model includes lead mean and lead peak (separate analyses)

<sup>c</sup>Partially adjusted model includes lead mean and lead peak (separate analyses), as well as sex, race, and SES

<sup>d</sup>Fully adjusted model includes lead mean and lead peak (separate analyses), as well as sex, race, SES, and year of birth

<sup>e</sup>Categorical models are presented as the odds of firearm violence victimization and perpetration at lead levels 2 (5-9 µg/dL), 3 (10-19 µg/dL) and 4 (≥20 µg/dL) compared to level 1 (0-4 µg/dL)

µg/dL compared to an individual with a mean childhood lead level under 5 µg/dL (95% CI 1.9, 3.2). Finally, the adjusted odds of an individual becoming a victim of firearm violence are 3.4 times greater for an individual with a mean childhood lead level 20 µg/dL and above compared to an individual with a mean childhood lead level under 5 µg/dL (95% CI 2.5, 4.5).

We saw similar results with mean peak childhood lead levels. After adjustment for confounders, the odds of an individual becoming a victim of firearm violence are 1.5 times greater for an individual with a peak childhood lead level between 5 - 9 µg/dL compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 1.09, 2.0). The odds of an individual becoming a victim of firearm violence are 2.2 times greater for an individual with a peak childhood lead level between 10 - 19 µg/dL compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 1.7, 3.0). And the odds of an individual becoming a victim of firearm violence are 2.6 times greater for an individual with a peak childhood lead level 20 µg/dL and above compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 1.9, 3.6).

### **Firearm violence perpetration**

Of the 89,129 individuals in our sample, 554 later perpetrated a firearm homicide or nonfatal shooting. Similar to firearm violence victimization, firearm violence was mostly perpetrated by males, African Americans and individuals in the lowest socioeconomic status category (Table 2). Also like victimization, most firearm violence perpetrators were born between 1990 - 1993. Mean lead levels for perpetrators of firearm violence (Lead Mean: 12.7 µg/dL, SD: 7.4 µg/dL and Lead Peak: 15.8 µg/dL, SD: 10.2 µg/dL) were higher than the actionable levels set by the Center for Disease Control and Prevention in 2012 (5 µg/dL) and

between 1991 - 2012 (10 µg/dL) but lower than the actionable level between 1985-1991 (25 µg/dL). Median lead levels for perpetrators were slightly lower than the means (Lead Mean: 11 µg/dL and Lead Peak: 13 µg/dL) and also followed this trend.

After adjustment for confounders, our results show that for every 1µg/dL increase in the mean childhood lead level, the odds of an individual becoming a perpetrator of firearm violence increases (OR 1.03, 95% CI 1.02, 1.04) (Table 3). Similar results were found for each 1µg/dL increase in peak childhood lead level (OR 1.02, 95% CI 1.01, 1.02). When lead exposure was categorized, the odds of an individual becoming a perpetrator of firearm violence are 2.3 times greater for an individual with a mean childhood lead level between 5 - 9 µg/dL compared to an individual with a mean childhood lead level under 5 µg/dL (95% CI 1.6, 3.3), after adjusting for confounding. The adjusted odds of an individual becoming a perpetrator of firearm violence are 2.6 times greater for an individual with a mean childhood lead level between 10 - 19 µg/dL compared to an individual with a mean childhood lead level under 5 µg/dL (95% CI 1.8, 3.8) and 3.0 times greater for an individual with a mean childhood lead level 20 µg/dL and above compared to an individual with a mean childhood lead level under 5 µg/dL (95% CI 1.9, 4.5).

We also categorized mean peak childhood lead levels. After adjustment for confounders, the odds of an individual becoming a perpetrator of firearm violence are 2.5 times greater for an individual with a peak childhood lead level between 5 - 9 µg/dL compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 1.5, 4.0). The adjusted odds of an individual becoming a perpetrator of firearm violence are 3.2 times greater for an individual with a peak childhood lead level between 10 - 19 µg/dL compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 2.0, 5.2). Finally, the adjusted odds of an individual becoming a perpetrator of firearm violence are 3.6 times greater for an individual with a peak childhood

lead level 20 µg/dL and above compared to an individual with a peak childhood lead level under 5 µg/dL (95% CI 2.2, 5.9). Our results were similar when we ran the analysis for individuals who met all but one of the inclusion criteria - continued adolescent city of Milwaukee residency demonstrated with a Milwaukee Public Schools record between July 1st, 2004 - July 18th, 2016.

## **Discussion**

Using blood lead, school and firearm violence data linked at the individual level, we estimated the association between childhood lead exposure before 6 years of age and firearm violence victimization and perpetration. Our multivariate results reveal that lead exposure in the first 6 years of life is positively associated with increased risk for firearm violence victimization and perpetration in early adulthood, after controlling for sex, race, socioeconomic status and year of birth. When mean and peak childhood lead exposure was categorized, our results revealed a dose-response relationship between childhood lead levels and firearm violence victimization and perpetration. These results are consistent with our stated hypothesis that higher levels of lead exposure during childhood will lead to higher odds of firearm violence victimization and perpetration, after controlling for potential confounding variables, suggesting a possible causal linkage in which early lead exposure led to increased risk of firearm violence victimization and perpetration.

Our findings are consistent with prior research that has examined childhood lead levels and juvenile delinquency and criminal behavior. For example, a cohort study by Fergusson et al found a significant dose-response relationship between childhood lead levels and rates of violent and property crime (officially recorded and self-reported), after controlling for confounding (D. M. Fergusson et al., 2008). Additionally, using the Cincinnati Lead Study birth cohort, Wright et al found that for every 5 µg/dL increase in prenatal and 6-year-old blood lead concentration, the



adjusted total arrest rates were significantly greater (prenatal: RR 1.40, 95% CI 1.07-1.85 and 6.5 years: RR 1.27, 95% CI 1.03-1.57). The adjusted arrest rates for violent crimes were also significantly greater for each 5 µg/dL increase in mean childhood lead (RR 1.30, 95% CI 1.03-1.64) and 6-year-old blood lead (RR 1.48, 95% CI 1.15-1.89) (J. P. Wright et al., 2008).

The relationship between childhood lead exposure and firearm violence could be explained by the mental/cognitive deficits and/or externalizing behaviors caused by childhood lead exposure that covary with criminal behaviors. It has been well-established in the literature that childhood lead exposure is associated with IQ deficits, ADHD diagnosis and symptomatology (i.e. attentional, impulsivity, self-control and hyperactivity dysfunctions) and aggression, all of which are criminogenic (American Psychological Association, 2013; Goodlad et al., 2013; Loeber et al., 2012; Marcus et al., 2010; McGloin et al., 2004; Narag et al., 2009; H. L. Needleman & Gatsonis, 1990; Pocock & Ashby, 1985; Pratt et al., 2002; Schwartz, 1994). Other factors could also be mediating the relationship between the cognitive and behavioral effects of childhood lead exposure and firearm violence. For example, poor academic outcomes, association with a deviant peer group and/or an abusive family environment could be mediating the relationship between IQ and/or ADHD and firearm violence (Barkley, 2002; Matsueda & Anderson, 1998; McGloin et al., 2004; Meldrum et al., 2009; Moffitt, 1993; Narag et al., 2009; Pratt et al., 2002; Tracy et al., 2016).

This is the first retrospective longitudinal study to examine the relationship between childhood lead exposure and firearm violence victimization and perpetration using linked individual-level public health, education and criminal justice data. The results of the study have significant implications for firearm violence prevention efforts. Currently, childhood lead exposure prevention is not considered a firearm violence prevention strategy. The results of this

study suggest that lead exposure prevention may deserve attention for playing a role in future firearm violence perpetration and victimization. In addition, while there is little disagreement that childhood lead adversely affects children, the results of this research provide added support for investment in primary and secondary prevention of lead exposure.

This study was able to address several limitations of previous research. It examined the relationship between childhood lead exposure and firearm violence disaggregated from violent crime as well as victimization and perpetration of both firearm homicides and nonfatal shootings using linked data at the individual level. This study also had important limitations. First, using Milwaukee Health Department childhood lead data may affect the validity of the study results. Using blood lead test results to measure exposure may only capture recent exposure. We included the mean of all blood lead test results under 6 years of age in an attempt to better capture childhood lead exposure over time. Additionally, despite guidelines regarding when and how often children should be tested for lead exposure, many children remain untested or under-tested. Children could have been exposed but never tested or exposed but only tested long after the exposure (an exposure a blood lead test would not be able to detect). The fact that blood lead data is potentially missing or not fully representative of a child's exposure could result in a misclassification in their exposure and bias our results. However, the probability of being misclassified is likely constant across our entire sample as we restricted our sample to individuals who had stable childhood and adolescent residency. By doing so, our sample had similar exposure to childhood lead testing campaigns and outreach efforts over similar time periods.

Second, there are limitations to using crime incident data to measure firearm violence perpetration and victimization. The data may be biased due to stereotypes held by law

enforcement. For example, due to racial biases, law enforcement may have identified more African American perpetrators. However, our proportion of African American perpetrators and victims aligned with national trends. Additionally, data may be missing, incomplete, or not systematically collected. This could be due to different policing strategies and/or police distrust (Masho et al., 2016). For example, if clearance rates (the number of crimes that are cleared by arrest by the total number of crimes recorded in a given year) are not prioritized, there is less incentive for officers to thoroughly investigate cases. The less thoroughly a case is investigated, the less information that is collected on that case. The fact that crime incident data could be potentially missing or not fully representative of the outcome could result in a misclassification of victims and perpetrators and bias our results. The Milwaukee Homicide Review Commission has attempted to overcome some of these limitations by creating its own homicide and nonfatal shooting databases and employing consistent data collection and coding methods based on National Violent Death Reporting System and FBI Uniform Crime Reporting standards. Conducting monthly case reviews with criminal justice partners provided added information on homicides and nonfatal shootings that may have been missing from police reports. It should also be noted that law enforcement invests a substantial amount of resources into homicide investigations and there is greater political pressure to close those cases, therefore homicide data is generally more robust than data for other crimes.

Third, there are limitations to using a retrospective cohort study design. Since it is not ethical to randomly expose children to lead, an intervention study is not an option. Therefore, a study of this nature will need to control for as many potential confounders of lead exposure and firearm violence as possible. One limitation of this retrospective cohort study is that the data was collected for a different purpose and so information on all potential confounders was not

collected and cannot be included in the analysis. While we were able to capture some of the important key confounders (sex, race, SES, and year of birth), we cannot exclude the possibility of unobserved confounding (Carlson & Morrison, 2009). For example, we cannot control for other or multiple neurotoxicant exposure. It should be noted that other studies that have controlled for other neurotoxicants and found that lead was the only one that was significant on violent behavior (Denno, 1990). Another limitation is that participants could have moved away from Milwaukee and committed firearm violence (which wouldn't be captured in MPD data) or committed violence outside of the specified date range. Additionally, individuals may have moved out of Milwaukee and returned as children, varying their exposure level. In an attempt to prevent this, we only included individuals who had Milwaukee addresses listed for all of their blood lead tests and were listed in Milwaukee Public Schools records between 2004 - 2016. Despite reducing our sample significantly, we found the Milwaukee Public Schools restriction imperative to ensure residential stability, control for potential academic confounding and reduce the potential for attrition. Finally, because our study population was limited to Milwaukee and only individuals who attended Milwaukee Public Schools, our results may not be generalizable to other populations.

Childhood lead exposure has been on the decline since many of the victims and perpetrators in this cohort were born. Knowing this, we might predict that lead exposure will play less of a role in firearm violence in the future. The reality is that while childhood lead exposure is decreasing in the overall population, children in the United States (and other countries) are still exposed to unacceptably high levels of lead, especially poor children of color. Due to this ongoing issue, we recommend that future research continue to examine the effect of childhood lead exposure on firearm violence disaggregated from other violent crimes, especially

in children at greatest risk for lead exposure.

We also recommend future research focus on the mechanisms by which childhood lead exposure leads to firearm violence victimization and perpetration. For this study, we were not able to evaluate the potential neurobiological or neuropsychological pathways of the relationship between childhood lead exposure and firearm violence, nor were we able study potential mediators and moderators of the relationship. For example, we recommend that future research more closely examine the role of IQ or ADHD in this relationship as well as the potential mediating or moderating effects of factors such as academic outcomes, deviant peer group association and family-level factors.

The effects of lead exposure are permanent but exposure to lead is preventable. This study provides further support for the urgency and prioritization of childhood lead exposure prevention efforts. The nature and severity of the consequences of both childhood lead exposure and firearm violence coupled with its disproportionate effect on African Americans living in socioeconomically disadvantaged neighborhoods makes this is public health crisis with serious social justice implications.

## CHAPTER 4: MANUSCRIPT #3

### Association of Childhood Blood Lead Levels with Firearm Violence Perpetration and Victimization by Shooting Type

#### Abstract

Recent research has demonstrated that fatal and nonfatal shootings differ by the circumstances that lead to the shooting. Shootings during an argument are often described as impulsive in nature. Studies have found differences in the neuropsychological functioning of individuals who commit impulsive violence versus premeditated violence, including lower IQ and executive functioning deficits, both of which are consequences of childhood lead exposure. We estimated the extent to which childhood lead exposure may contribute to argument-related firearm violence victimization or perpetration compared to other types of firearm violence. This study included 1091 individuals identified as victims and 589 individuals identified as perpetrators of firearm violence over the age of 12 years old with at least one blood lead test reported before age 6 years old. The children were identified using data linked at the individual level from the Milwaukee Health Department and Milwaukee Homicide Review Commission. We estimated the relative risk of being a victim or perpetrator of an Argument/Fight-related shooting compared to other shooting types for individuals with higher childhood lead exposure compared to those with lower childhood lead exposure using multinomial logistic regression, adjusting for sex, race, socioeconomic status and age at crime. After adjustment for confounders, our results show that the relative risk of being a victim of an Argument/Fight-related shooting compared to an Other type of shooting is 37% higher for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, holding sex, race, SES, and age constant (RRR 1.37, 95% CI 1.02, 1.85). For perpetrators, the relative risk of being a perpetrator of an Argument/Fight-

related shooting compared to a Retaliation-related shooting is 27% higher for each 5  $\mu\text{g/dL}$  increase in the mean childhood lead level, after adjustment for confounding (RRR 1.27, 95% CI 1.03, 1.55). This study confirmed prior findings that there are differences in victims and perpetrators by shooting type. Childhood lead exposure may be driving victimization and perpetration of Argument/Fight-related shootings more than other shooting types. Additional research is needed to examine the role of other factors, such as deviant peers, in victimization and perpetration of shootings disaggregated by shooting type.

## Introduction

On average, firearm violence is responsible for over 10,000 deaths and 60,000 injuries in the U.S. each year (Center for Disease Control and Prevention, 2015a). For years, researchers have tried to understand the factors contributing to its occurrence. While there is consensus on certain things such as the average characteristics of victims and suspects, e.g. firearm violence disproportionately affects young, African American males living in socioeconomically disadvantaged urban areas, inconsistent findings remain over potential explanations surrounding the causes of firearm violence (Land, McCall, & Cohen, 1990; J. M. Pizarro, 2005; Pridemore, 2002).

One potential explanation for this conflicting evidence is aggregation bias (Cherry & List, 2002; Land et al., 1990; Pridemore, 2002). Aggregation bias occurs when a grouping of something is treated as homogenous, e.g. violent crime, when, in reality, there are real differences within that grouping (e.g. rape, murder, robbery, assault, burglary, larceny, auto theft) (Cherry & List, 2002). Firearm violence has been historically treated as a dichotomous outcome. More recent research has demonstrated that shootings differ by the circumstances that lead to the shooting (Flewelling & Williams, 1999; J. M. Pizarro, 2005; J. M. Pizarro, 2008). For example, a shooting could occur as a result of a drug transaction, a robbery or an argument between intimate partners.

Firearm violence research, including research that has disaggregated by type, has primarily focused on homicides. While homicide data tends to be more comprehensive because of the resources invested in homicide investigations, homicides are relatively rare events (Papachristos et al., 2015). Nonfatal shootings are far more common than homicides and have



similar causes and consequences. It has been argued that, due to these similarities, it would be appropriate to combine firearm homicides and nonfatal shootings in future research (Flewelling & Williams, 1999).

It is important to disaggregate firearm violence by type because understanding what drives different types of shootings will inform firearm violence prevention strategies. Applying a blanket prevention strategy across all shootings may not be the most effective approach. If agencies involved in firearm violence prevention have a clearer understanding of what might be driving different types of shootings, they will be better equipped to develop a more targeted approach and allocate limited resources more efficiently (Miethe, Regoeczi, & Drass, 2004). Expanding our understanding of how shootings differ and what factors influence shootings will also advance our theoretical understanding of firearm violence (J. M. Pizarro, 2008). Additionally, increasing the sample size by including nonfatal shootings in homicide research will improve the power of future research studies. This could lead to more accurate results and consistent findings.

Prior research has disaggregated firearm homicides by shooting type. These studies have looked at gang homicides (Brandt & Russell, 2002; Decker & Curry, 2002; Maxson, Gordon, & Klein, 1985; Papachristos, 2009; J. M. Pizarro & McGloin, 2006), drug homicides (Brandt & Russell, 2002; Goldstein, 1985; Varano, McCluskey, Patchin, & Bynum, 2004), and retaliation (Kubrin & Weitzer, 2003), as well as a variety of homicide types (i.e. altercation/argument/conflict/dispute, felony, robbery, domestic, intimate, gang, drug, revenge)(Cooper & Smith, 2012; Kubrin & Herting, 2003; Kubrin & Wadsworth, 2003; Kubrin, 2003; Parker, 1989; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Roberts, Zgoba, & Shahidullah, 2007; Tita & Griffiths, 2005; Williams & Flewelling, 1988; Zeoli, Grady, Pizarro, & Melde,

2015). The results of this research suggest that shootings are not unitary offenses and support the utility of categorizing shootings into more specific categories in future research (Flewelling & Williams, 1999; J. M. Pizarro, Zgoba, & Jennings, 2011). For example, Kubrin found that neighborhood-level economic disadvantage was associated with all homicide types, but residential instability predicted only felony homicides (Kubrin, 2003). A study by Pizarro and McGloin found that gang homicides were predicted by neighborhood-level poverty but not social disorganization (J. M. Pizarro & McGloin, 2006). Finally, a study by Pizarro examined the relative differences between domestic, drug, dispute, robbery and other homicides (J. M. Pizarro, 2008). The study found that these homicides differed by event characteristics (incident location, weapon, alcohol and drug use, and number of suspects), temporality (incident date and time) and victim/suspect demographics (victim age, victim gender, victim race, suspect age and suspect race) (J. M. Pizarro, 2008).

One of the most common shooting types is firearm violence precipitated by an argument or dispute (Kubrin & Wadsworth, 2003; Kubrin, 2003; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Roberts et al., 2007). In fact, argument-related has been the most common known type of firearm violence nationally since 1980 (Cooper & Smith, 2012). Argument-related firearm violence is often described as “heat-of-the-moment” or impulsive in nature as it can commonly occur during an argument over seemingly trivial issues between the victim and offender (Kubrin & Wadsworth, 2003; Tita & Griffiths, 2005). This is in contrast to other types of shootings where premeditation may be explicit or implied. For example, retaliation-related firearm violence is described as a premeditated response to an earlier or long-standing altercation between the victim and suspect (Kubrin & Weitzer, 2003).

Notable differences have been found in the neuropsychological functioning of individuals who commit impulsive violence versus premeditated violence (Hanlon, Brook, Stratton, Jensen, & Rubin, 2013; Houston, Stanford, Villemarette-Pittman, Conklin, & Helfritz, 2003). A study by Dolan et al found that high impulsive aggressive males had greater executive functioning deficits compared to low impulsive aggressive males (Dolan, Deakin, Roberts, & Anderson, 2002). In addition, a study by Giancola et al found that executive functioning deficits were predictive of impulsive aggression in adolescent boys (Giancola, Mezzich, & Tarter, 1998). Finally, a study by Hanlon et al examined the differences in neuropsychological functioning and intelligence between murderers that killed impulsively and those who killed based on a premeditated plan. Compared to the premeditated murderers, murderers who killed impulsively performed significantly worse on tests of intelligence, memory, attention and executive functions (Hanlon et al., 2013).

Childhood lead exposure is associated with cognitive impairments such as lower IQ, executive functioning deficits and attention-deficit/hyperactivity disorder (ADHD) and symptomatology, all of which are also criminogenic (Narag et al., 2009, pg. 963) Some of the earliest lead studies found that children with higher lead levels had lower IQs and significant attentional, impulsivity and hyperactivity dysfunctions (Byers & Lord, 1943; H. L. Needleman et al., 1979). The negative relationship between childhood lead exposure and child IQ is well-documented in the literature (Al-Saleh et al., 2001; Baghurst et al., 1992; D. C. Bellinger et al., 1992; Canfield et al., 2003; A. Chen et al., 2005; A. Chen et al., 2007; L. Chiodo et al., 2004; de la Burd  & Choate, 1975; K. N. Dietrich et al., 1993; Dudek & Merez, 1997; Factor-Litvak et al., 1999; Kordas et al., 2006; Ris et al., 2004; Rummo et al., 1979; L. Schnaas et al., 2006; L. Schnaas et al., 2000; G. Wasserman et al., 2000; G. A. Wasserman et al., 2003; G. A.

Wasserman et al., 1997). While not quite as extensive as IQ, the positive significant association between childhood lead levels and attention deficit and hyperactivity symptoms (i.e. impulsivity, attentional issues, low self-control) and/or ADHD diagnosis for children in the U.S. and abroad (after controlling for covariates) is also well-established (D. Bellinger et al., 1994; Burns et al., 1999; A. Chen et al., 2007; L. M. Chiodo et al., 2007; L. Chiodo et al., 2004; D. M. Fergusson et al., 1993; Ha et al., 2009; H. L. Needleman et al., 1996; H. L. Needleman et al., 1979; Nicolescu et al., 2010; Nigg et al., 2008; Nigg et al., 2010; Ris et al., 2004; Rummo et al., 1979; Silva et al., 1988; Thomson et al., 1989; Wang et al., 2008). We also know that elevated childhood lead levels have been found to be associated with juvenile delinquency (K. N. Dietrich et al., 2001; H. L. Needleman et al., 1996; H. L. Needleman et al., 2002; Olympio et al., 2009) and adult criminal behavior and violent offending (D. M. Fergusson et al., 2008; Hwang, 2007; Narag et al., 2009; R. Nevin, 2007; Pihl & Ervin, 1990; P. B. Stretesky & Lynch, 2004). Given the effect childhood lead exposure has on behavior and the impulsive nature of argument-related firearm violence, childhood lead exposure may be contributing to argument-related firearm violence relative to other types of firearm violence.

To overcome previous limitations and address current gaps in the research, we will evaluate the extent to which childhood lead exposure may contribute to argument-related firearm violence victimization or perpetration compared to other types of firearm violence. Firearm violence will include both fatal and nonfatal shootings and be disaggregated by type. We hypothesize that individuals with higher childhood lead results are at higher risk than individuals with lower childhood lead results of committing or being a victim of argument-related shootings compared to other shooting types.

## **Methods**

## **Study Population**

We conducted a case-case study in the city of Milwaukee, Wisconsin by performing individual-level linkages of available datasets on blood lead testing and firearm crime. All data for this study was pulled from an integrated data system called DataShare MKE. DataShare MKE is a multi-sector, multi-disciplinary integrated data system that includes data from the Milwaukee Health Department and the Milwaukee Homicide Review Commission (among other datasets). Each individual in any of the datasets is assigned a conformed person ID and then this ID is used to link individuals across all of the datasets.

From this resource, we constructed a sample consisting of individuals who were victims or perpetrators of firearm violence with inclusion and exclusion criteria designed to appropriately link their records to data on blood lead levels. Individuals were identified as victims or perpetrators of firearm violence in the Milwaukee Homicide Review Commission homicide and nonfatal shooting databases in DataShare MKE. Since 2005, the Milwaukee Homicide Review Commission pulled data directly from Milwaukee Police Department homicide files and police reports for its homicide and nonfatal shooting databases. Each day, three trained Milwaukee Homicide Review Commission staff (police officer, administrative staff and a researcher) extracted data from these sources and entered it into the databases. Staff used a data collection instrument based on the National Violent Death Reporting System and Uniform Crime Reporting standards to ensure that the data was captured consistently. Any discrepancies in the data were discussed amongst the team and, if necessary, brought to the Director for review. Staff reviewed open cases daily for additional information and consulted with Milwaukee Police Officers and detectives for updated information. The Milwaukee Homicide Review Commission also assembled a multidisciplinary group of criminal justice professionals (law enforcement,

prosecutors, corrections agents, etc.) monthly (from 2005 to 2015) to review homicide and nonfatal shooting cases (M. O'Brien, 2007). During the reviews, additional information not found in the police reports was discussed among partners. Milwaukee Homicide Review Commission staff used this review process to gather additional information to enter into the databases on homicides and nonfatal shootings.

The Milwaukee Homicide Review Commission homicide database was used to identify individuals who were a victim of a firearm homicide in Milwaukee between January 1st, 2005 and December 31st, 2015 (Milwaukee Homicide Review Commission data was only available during that time period at the time of this study). Firearm homicides are coded in the homicide database as homicides caused by a firearm (unknown), handgun, or long gun. The Milwaukee Homicide Review Commission nonfatal shooting database was used to identify individuals who were a victim of a nonfatal shooting between January 1st, 2006 and December 31st, 2015. A victim was identified as an individual coded as a victim in the Milwaukee Police Department incident report who sustained a gunshot wound. For this study, individuals identified in the Milwaukee Homicide Review Commission homicide (firearm homicides only) and nonfatal shooting databases as victims will serve as firearm violence victims.

The Milwaukee Homicide Review Commission homicide database was used to identify individuals who were a perpetrator of a firearm homicide in Milwaukee between January 1st, 2005 and December 31st, 2015 and the Milwaukee Homicide Review Commission nonfatal shooting database was used to identify individuals who were a perpetrator of a nonfatal shooting between January 1st, 2006 and December 31st, 2015. A suspect was identified as an individual coded as an arrestee, suspect and/or a person of interest with corroborated witness identification or a strong investigative lead in the Milwaukee Police Department incident report. For this

study, individuals identified in the Milwaukee Homicide Review Commission homicide (firearm homicides only) and nonfatal shooting databases as suspects will serve as firearm violence perpetrators.

We will conduct two separate analyses - one for victims and one for perpetrators of firearm violence. Studies have found overlapping risk factors for firearm violence perpetration and victimization (Loeber et al., 2012). Victims and perpetrators often have common characteristics because they share similar geography, high risk lifestyles and social networks that put them at greater risk for perpetration as well as victimization (Hindelang et al., 1978).

Individuals in this sample needed to be at least 12 years of age at the time they developed the outcome as this study only examined juvenile and young adult firearm violence victimization and perpetration. Due to this restriction, 12 individuals were not included in the victim analysis. No perpetrators were excluded from the perpetrator analysis because all perpetrators in the Milwaukee Homicide Review Commission homicide and nonfatal shooting databases were older than 12 years old.

Identified victims and perpetrators also had to have childhood blood lead results reported to the Milwaukee Health Department between March 25th, 1980 - May 2nd, 2012 (Milwaukee Health Department childhood lead data was only available during this time frame). To be included in the sample, they had to have at least one blood lead test collected and reported to the Milwaukee Health Department before age 6 years old. A total of 1103 individuals were a victim and 600 were a perpetrator of a firearm homicide or nonfatal shooting and met the inclusion and exclusion criteria.

### **Study Variables and Covariates**

## **Dependent Variable**

The dependent variable in my analysis is the type of firearm homicide or nonfatal shooting. Each firearm homicide or nonfatal shooting in the Milwaukee Homicide Review Commission databases is coded with a type (circumstances surrounding/leading to the homicide) by a trained staff person using coding guidelines created by the Milwaukee Homicide Review Commission, Milwaukee Police Department and academic experts in firearm violence research (Dr. Mallory O'Brien, Dr. Edmund McGarrell from Michigan State University and Dr. Natalie Hipple from Indiana University) with standards from the National Violent Death Reporting System and FBI Uniform Crime Reporting. The coding was reviewed and validated by three trained Milwaukee Homicide Review Commission staff (police officer, administrative staff and researcher). The shooting type codes are the following: argument/fight, retaliation, drug-related, drug-related robbery, gang-related, robbery, domestic violence, commission of another crime, gambling, negligent handling, possibly self-inflicted, police related, self-defense, other and unknown (Appendix A for codebook). For individuals who were a victim or perpetrator of more than one incident, we took the shooting type of their first shooting incident.

Based on previous literature and small cell counts in some categories, we removed or collapsed some of the shooting type categories for the final analysis. Despite research showing that domestic violence shootings are distinct, (Avakame, 1998; DeJong, Pizarro, & McGarrell, 2011; Kubrin & Herting, 2003; Kubrin & Wadsworth, 2003; Kubrin, 2003; Parker, 1989; Roberts et al., 2007; Tita & Griffiths, 2005; Zeoli et al., 2015) "Domestic Violence" fatal and nonfatal shootings were set to missing because there was such a small number of incidents. "Robbery," "Commission of Another Crime," and "Gambling" were combined into one group (Nondrug Crime) as they are all crime-related but not related to drug crimes. Other studies have



employed similar nondrug crime groupings (Kubrin & Herting, 2003; Kubrin, 2003; Roberts et al., 2007; Tita & Griffiths, 2005). “Drug Related” and “Drug Related Robbery” were combined as they are both related to the drug trade and previous research has shown that drug-related shootings are unique (Brandt & Russell, 2002; Goldstein, 1985; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Tita & Griffiths, 2005; Varano et al., 2004; Zeoli et al., 2015). “Retaliation” and “Gang Related” were not combined with other categories as studies have shown that retaliation (Kubrin & Weitzer, 2003; Zeoli et al., 2015) and gang (Brandt & Russell, 2002; Decker & Curry, 2002; Kubrin & Wadsworth, 2003; Maxson et al., 1985; Papachristos, 2009; J. M. Pizarro & McGloin, 2006; Tita & Griffiths, 2005; Zeoli et al., 2015) shootings are distinct and should remain their own category. “Negligent Handling,” “Police-Related,” “Self-Defense,” “Other” and “Possibly Self-Inflicted” were combined into an “Other” category (fatal and nonfatal shootings that do not fit into the five other categories, have small cells and do not have literature supporting that they are distinct categories), similar to previous research (J. M. Pizarro, 2005; J. M. Pizarro, 2008). Other studies performing similar analysis have removed the “Unknown” category for the final analysis (Kubrin & Wadsworth, 2003; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Zeoli et al., 2015). Due to the high proportion of “Unknown” shootings, especially for victims (28%), we felt it was important to keep it as a separate group for the final analysis. Unfortunately, since there is not enough information about the circumstances surrounding these fatal or nonfatal shootings, we weren’t able to interpret the results or draw conclusions for this group. Based on the results of descriptive statistics analysis, the final categories for this analysis were 1.) Argument/Fight 2.) Retaliation, 2.) Nondrug Crime (Robbery, Commission of a Crime and Gambling), 3.) Drug (Drug-related and Drug Related Robbery), 4.) Gang, 5.) Other (Negligent Handling, Police-Related, Self-Defense, Other and Possibly Self-Inflicted) and 6.)

Unknown.

### **Independent Variable: Childhood Lead Exposure**

The independent variable for this study is childhood lead exposure. We used blood lead data from the Milwaukee Health Department in DataShare MKE to evaluate childhood lead exposure. The Milwaukee Health Department database in DataShare MKE includes all blood lead tests reported to the Milwaukee Health Department between March 25th, 1980 and May 2nd, 2012. Blood lead test results were reported as a continuous value in micrograms per deciliter ( $\mu\text{g}/\text{dL}$ ). The actual testing was completed at multiple clinics or labs throughout Milwaukee but then all test results were reported to the Milwaukee Health Department.

Childhood lead exposure was operationalized in two ways: the mean of all blood lead test results for an individual before age 6 years old and the peak blood lead test result for each individual before age 6 years old. Childhood lead exposure was operationalized in this manner (age cutoff and mean/peak levels) based on previous research (M. S. Amato et al., 2012; M. S. Amato et al., 2013; K. N. Dietrich et al., 1991; K. N. Dietrich et al., 2001; D. M. Fergusson et al., 2008; J. P. Wright et al., 2008), the age of increased vulnerability (D. C. Bellinger, 2004; Hwang, 2007; H. Needleman, 2004) and the average age children reported lead tests in the Milwaukee Health Department data.

### **Covariates**

The covariates for this study are: race, sex, socioeconomic status (SES), and age at crime. These factors have been found to be correlated with both lead exposure and shooting type (D. C. Bellinger, 2004; D. C. Bellinger, 2008; Brandt & Russell, 2002; DeJong et al., 2011; Kubrin, 2003; J. M. Pizarro, 2008; Wisconsin Childhood Lead Poisoning Prevention Program,

2014). Race, sex and socioeconomic status were pulled from the Milwaukee Health Department database. Sex is the sex of the child and race is the race of the child reported to Milwaukee Health Department. Due to the limited number of victims and perpetrators with White, American Indian/Alaskan Native, Asian, Other, and Unknown listed as their race, these races were combined into one category. The final race categories were Black/African American and White/Other. SES was not reported to Milwaukee Health Department, therefore the zip code of the child's residence reported to Milwaukee Health Department at their peak blood lead test result was used to determine their SES level (lower, middle, or upper). Using 2000 Census data, a study by Vilas et al created an SES index based on the average median income (income) and percentage of people with a bachelor's degree (education) by zip code in Milwaukee (Vila et al., 2007). The zip codes were ranked and then grouped into three SES levels: lower (53204, 53205, 53206, 53208, 53210, 53212, 53215, 53216, 53218, and 53233), middle (53207, 53209, 53214, 53219, 53220, 53221, 53224, 53225, 53227, 53235) and upper SES (53202, 53203, 53211, 53213, 53217, 53222, 53223, 53226, 53228).(Vila et al., 2007) There were four addition zip codes in the data that did not fall into the lower, middle and upper categories - 53201, 53234, 53237, and 53200. These zip codes represented PO boxes (53201, 53234, 53237) and unknown addresses (53200 was entered when the zip code was unknown or someone refused to report their address). Due to small cells, the middle, upper and other categories were combined. The final SES categories were Lower and Other (middle, upper and other).

We included the victim/perpetrator age at time of crime in the full model to control for time. We chose this variable over others (incident date and year of birth) after comparing LOESS and cubic spline results. The age of the victim or perpetrator at the time of the firearm homicide or nonfatal shooting incident listed in the Milwaukee Homicide Review Commission

databases was used for the age at crime. For ease of interpretation, this was transformed into a 3-level categorical variable based on the descriptive statistics. Categorizing this variable did not harm the precision. For individuals who were a victim or perpetrator of more than one incident, we took the age at the time of their first shooting incident.

**Table 1: Study Variables**

Variable Type	Variable	Level of Measurement	Potential Response	Data Source
Dependent	Type of Firearm Violence Victimization Incident/Firearm Violence Perpetration Incident	Categorical	Argument/Fight Retaliation, Nondrug Crime (Robbery, Commission of a Crime and Gambling), Drug (Drug-related and Drug Related Robbery), Gang, Other (Negligent Handling, Police-Related, Self-Defense, Other and Possibly Self-Inflicted), Unknown	Milwaukee Homicide Review Commission
Independent	Childhood Lead Exposure (Mean and Peak)	Continuous	Range: Victims Mean - (1, 58 µg/dL), Peak - (1, 65 µg/dL) Perpetrators Mean - (1, 61 µg/dL) Peak - (1, 62 µg/dL)	Milwaukee Health Department
Covariate	Sex	Dichotomous	Male or Female	Milwaukee Health Department

Covariate	Race	Dichotomous	Black/African American, White/Other (White, American Indian/Alaskan Native, Asian, Other, Unknown)	Milwaukee Health Department
Covariate	Socioeconomic status (SES)	Dichotomous	Lower or Other (Middle, Upper or Other SES)	Milwaukee Health Department
Covariate	Victim/Perpetrator age at time of crime	Categorical	Lower, Middle and Upper Age Groupings	Milwaukee Homicide Review Commission

### **Data Analysis Plan**

This is a quantitative secondary data analysis of a case-case study on the effect of childhood lead exposure on firearm violence victimization and perpetration by shooting type. First, we used logistic regression analysis to estimate the magnitude and precision of the association between childhood lead exposure and shooting type (Argument/Fight versus all other types). The adjusted model included childhood lead exposure, race, sex, SES and age at crime. The coding of the independent variable and covariates was based on prior literature, the presence of small cells, LOESS, cubic spline and AIC results, and ease of interpretation. Odds ratios (OR) were calculated for the adjusted model. Only the adjusted OR was interpreted as it is the less-confounded estimate. Standard errors were used to construct 95% confidence intervals around each OR for each model.

We used multinomial logistic regression analysis to estimate the magnitude and precision

of the association between higher childhood lead exposure compared to lower childhood lead exposure (independent variable) and victimization of Argument/Fight-related firearm homicides and nonfatal shootings compared to other types of firearm homicides and nonfatal shootings (dependent variable), with adjustment for race, sex, SES, and age at crime. Relative risk ratios (RRR) were calculated with a RRR greater than one suggesting a higher relative risk of being a victim of an Argument/Fight-related firearm homicide or nonfatal shooting compared to other types for individuals with higher lead exposure compared to those with lower lead exposure, holding race, sex, SES and age at crime constant. The same analytic framework was followed for perpetrators of firearm violence.

A multinomial logit model is appropriate for this analysis. A multinomial logit models the log odds of nominal outcome variables as a linear combination of the predictor variables. (Long, 1997) This model is appropriate because the dependent variable has more than three outcomes that are clearly unordered (Long, 1997). The model was estimated using the “mlogit” command in Stata14(StataCorp, 2015).

## **Results**

### **Victims**

A total of 1103 individuals met our inclusion and exclusion criteria by lead tests reported, age and firearm homicide or nonfatal shooting victimization. Due to setting Domestic Violence incidents to missing, our final sample was 1091 victims. The overall sample of victims was predominantly Male (88%), African American (92%), of lower socioeconomic status (86%) and older than 20 years old (93%) (Table 2). Most firearm homicide and nonfatal shooting incidents were Argument/Fight shootings (31%), followed by Nondrug Crime shootings (18%). Overall

mean and peak lead levels were higher than the current (5 µg/dL in 2012) and former (10 µg/dL from 1991 - 2012) actionable levels set by the Center for Disease Control and Prevention.

When lead levels among victims were examined by sample characteristics, male victims had higher mean lead levels than female victims (Lead Mean for males: 13.4 µg/dL vs. for females 11.4 µg/dL and Lead Peak for males: 16.5 µg/dL vs. for females 14.6 µg/dL), African Americans had higher mean lead levels than victims of Whites/Other races (Lead Mean for African Americans: 13.4 vs. for Whites/Other races 10.1 µg/dL and Lead Peak for African Americans: 16.7 vs. for Whites/Other 11.9 µg/dL) and victims with the lowest socioeconomic status had higher mean lead levels than victims in all other socioeconomic status categories (Lead Mean for lowest: 13.9 vs. for other 8.3 µg/dL and Lead Peak for lowest: 17.3 vs. for other 10 µg/dL). Average mean and peak lead levels were higher for older victims compared to younger victims.

With the exception of the Other group (Police-related, Negligent Handling, Self Defense, Other, and Possibly Self-Inflicted shootings), each shooting type had over 80% male victims. Nondrug Crime and Gang-related shootings had the highest percentage of male victims (95%). All but Gang-related shootings had over 85% African American victims, with Retaliation-related shootings having the highest percentage of African American victims (97%). Additionally, all shootings types except Gang-related had over 80% lower socioeconomic status victims. The Other type of shootings had the highest percentage of low socioeconomic status victims (93%) as well as the greatest percentage of victims under 20 years old (19%). Gang-related shootings had the greatest percentage of victims over 24 years old (64%). When mean lead levels were examined by shooting type, Gang-related and Argument/Fight-related shootings had the highest

mean and peak lead levels compared to other types of shootings. Other shootings had the lowest mean and peak lead levels.

Our logistic regression analysis results show that for every 1  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, the adjusted odds of being a victim of an Argument/Fight-related shooting compared to being a victim of all other shootings was higher but not significant (OR 1.01, 95% CI .995, 1.03) (Table 4). Similar results were found for each 1  $\mu\text{g}/\text{dL}$  increase in peak childhood lead level (OR 1.01, 95% CI .997, 1.02).

The results of our multinomial logistic regression across multiple shooting types showed that the relative risk of being a victim of an Argument/Fight-related shooting compared to an Other shooting is 37% higher for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, holding sex, race, SES, and age constant (RRR 1.37, 95% CI 1.02, 1.85) (Table 4). Lead peak results were similar to mean lead results but not significant (RRR 1.22, 95% CI .99, 1.5). The adjusted relative risk of being a victim of an Argument/Fight-related shooting compared to a Retaliation (RRR 1.07, 95% CI .88, 1.29), Nondrug Crime (RRR 1.05, 95% CI .92, 1.18), or Drug-related (RRR 1.15, 95% CI .96, 1.39) shooting are all higher than 1 for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, but none are significant. We saw similar results for each 5  $\mu\text{g}/\text{dL}$  increase in peak childhood lead level (Retaliation (RRR 1.03, 95% CI .90, 1.16), Nondrug Crime (RRR 1.04, 95% CI .95, 1.13), or Drug-related (RRR 1.10, 95% CI .97, 1.25). The adjusted relative risk of being a victim of an Argument/Fight-related shooting compared to a Gang shooting is below 1 for each 5  $\mu\text{g}/\text{dL}$  increase in the mean (RRR .96, 95% CI .79, 1.16) and peak (RRR .97, 95% CI .85, 1.11) childhood lead level, but these results also included the null.



**Table 2: Population Characteristics of Victims of Firearm Violence in Milwaukee**

	Victims	Argument/Fight	Retaliation	Shooting Type					Lead Mean (µg/dL) Mean (Median)	Lead Peak (µg/dL) Mean (Median)
				Nondrug Crime	Drug	Gang	Other	Unknown		
<b>Total</b>	n = 1091	337 (31%)	78 (7%)	191 (18%)	81 (7%)	58 (5%)	43 (4%)	306 (28%)		
<b>Sex</b>										
Male	960 (88%)	286 (86%)	63 (81%)	182 (95%)	74 (91%)	55 (95%)	32 (74%)	268 (88%)	13.4 (11.2)	16.5 (13)
Female	131 (12%)	48 (14%)	15 (19%)	9 (5%)	7 (9%)	3 (5%)	11 (26%)	38 (12%)	11.4 (9.7)	14.6 (11)
<b>Race/Ethnicity</b>										
African American	1008 (92%)	309 (93%)	76 (97%)	177 (93%)	76 (94%)	43 (74%)	37 (86%)	290 (95%)	13.4 (11.2)	16.7 (13)
White/Other	83 (8%)	25 (7%)	2 (3%)	14 (7%)	5 (6%)	15 (26%)	6 (14%)	16 (5%)	10.1 (8)	11.9 (10)
<b>SES</b>										
Lower	943 (86%)	293 (88%)	68 (87%)	157 (82%)	71 (88%)	44 (76%)	40 (93%)	270 (88%)	13.9 (12)	17.3 (15)
Other	148 (14%)	41 (12%)	10 (13%)	34 (18%)	10 (12%)	14 (24%)	3 (7%)	36 (12%)	8.3 (7.3)	10 (8)
<b>Victim Age</b>										
<20 years	72 (7%)	24 (7%)	11 (14%)	10 (5%)	6 (7%)	1 (2%)	18 (19%)	12 (4%)	8.1 (7.3)	10.7 (10)
20-24 years	470 (43%)	136 (41%)	41 (53%)	79 (41%)	37 (46%)	20 (34%)	23 (53%)	134 (44%)	10.7 (9.5)	14.1 (12)
>24 years	549 (50%)	174 (52%)	26 (33%)	102 (53%)	38 (47%)	37 (64%)	12 (28%)	160 (52%)	15.9 (14)	18.9 (16)
<b>Lead Mean (µg/dL)</b>										
Mean (Median)	13.1 (11)	13.7 (11)	12.1 (11)	13.1 (11)	12.2 (11.5)	13.7 (10.3)	10 (9.5)	13.3 (11)		
<b>Lead Peak (µg/dL)</b>										
Mean (Median)	16.3 (13)	17.1 (14)	15.7 (13)	16.2 (13)	15.1 (13)	16.7 (12.5)	12.7 (11)	16.4 (13)		

**Table 3: Population Characteristics of Perpetrators of Firearm Violence in Milwaukee**

	Shooting Type								Lead Mean (µg/dL)	Lead Peak (µg/dL) Mean (Median)
	Perpetrators	Argument/Fight	Retaliation	Nondrug Crime	Drug	Gang	Other	Unknown	Mean (Median)	Mean (Median)
<b>Total</b>	n = 589	194 (33%)	67 (11%)	123 (21%)	87 (15%)	38 (6%)	38 (6%)	42 (7%)		
<b>Sex</b>						38				
Male	562 (95%)	182 (94%)	63 (94%)	116 (94%)	84 (97%)	(100%)	37 (97%)	42 (100%)	13.2 (11)	16.3 (14)
Female	27 (5%)	12 (6%)	4 (6%)	7 (6%)	3 (3%)	0 (0%)	1 (3%)	0 (0%)	13 (10.5)	16.7 (12)
<b>Race/Ethnicity</b>										
African American	538 (91%)	174 (90%)	65 (97%)	117 (95%)	82 (94%)	28 (74%)	34 (89%)	38 (90%)	13.6 (11.3)	16.6 (14)
White/Other	51 (9%)	20 (10%)	2 (3%)	6 (5%)	5 (6%)	10 (26%)	4 (11%)	4 (10%)	9.6 (9)	13.5 (11)
<b>SES</b>										
Lower	520 (88%)	177 (91%)	59 (88%)	105 (85%)	77 (89%)	34 (89%)	31 (82%)	37 (88%)	13.9 (11.6)	17.2 (15)
Other	69 (12%)	17 (9%)	8 (12%)	18 (15%)	10 (11%)	4 (11%)	7 (18%)	5 (12%)	8.1 (7)	9.8 (9)
<b>Perpetrator Age</b>										
<18 years	155 (26%)	48 (25%)	16 (24%)	30 (24%)	22 (25%)	16 (42%)	13 (34%)	10 (24%)	11 (9)	14.4 (11)
18-20 years	267 (45%)	92 (47%)	23 (34%)	59 (48%)	41 (47%)	20 (53%)	14 (37%)	18 (43%)	12.5 (11)	15.4 (13)
>20 years	167 (28%)	54 (28%)	28 (42%)	34 (28%)	24 (28%)	2 (5%)	11 (29%)	14 (33%)	16.5 (14.3)	19.7 (17)
<b>Lead Mean (µg/dL) Mean (Median)</b>	13.2 (11)	14 (12)	12.2 (11)	12.3 (10.4)	12.9 (11.5)	12.9 (11)	11.6 (8.2)	16.1 (11.5)		
<b>Lead Peak (µg/dL) Mean (Median)</b>	16.4 (13)	17 (14)	15.4 (13)	15.2 (13)	16.6 (15)	16.2 (14)	14 (10)	20.5 (14.5)		

**Table 4: Odds ratios and relative risk ratios for the association between childhood lead levels and shooting type in Milwaukee**

		Shooting Type					
		1 µg/dL					
		Argument/Fight					
<b>Victims<sup>a</sup></b>		n = 337					
n = 1091							
Lead mean		1.01 (.995, 1.03)					
Lead peak		1.01 (.997, 1.02)					
<b>Perpetrators<sup>a</sup></b>		n = 194					
n = 589							
Lead mean		1.02 (.996, 1.04)					
Lead peak		1.01 (.989, 1.02)					
		5 µg/dL					
<b>Victims<sup>bc</sup></b>		Retaliation	Nondrug Crime	Drug	Gang	Other	Unknown
n = 1091		n = 78	n = 191	n = 81	n = 58	n = 43	n = 306
Lead mean		1.07 (.88, 1.29)	1.05 (.92, 1.18)	1.15 (.96, 1.39)	.96 (.79, 1.16)	1.37* (1.02, 1.85)	1.06 (.95, 1.18)
Lead peak		1.03 (.90, 1.16)	1.04 (.95, 1.13)	1.10 (.97, 1.25)	.97 (.85, 1.11)	1.22 (.99, 1.5)	1.05 (.97, 1.13)
<b>Perpetrators<sup>bd</sup></b>		n = 67	n = 123	n = 87	n = 38	n = 38	n = 42
n = 589							
Lead mean		1.27* (1.03, 1.55)	1.16 (.995, 1.37)	1.11 (.95, 1.33)	.91 (.71, 1.18)	1.21 (.91, 1.58)	.86 (.71, 1.05)
Lead peak		1.12 (.95, 1.3)	1.09 (.95, 1.23)	1.01 (.91, 1.15)	.95 (.78, 1.13)	1.15 (.95, 1.4)	.86* (.74, .998)

<sup>a</sup>Continuous models are presented as the change in the odds of firearm violence victimization and perpetration risk for every 1 µg/dL increase in childhood lead level

<sup>b</sup>Relative Risk models are presented as the change in risk of Argument/Fight firearm violence victimization and perpetration relative to other shooting types for every 5 µg/dL increase in childhood lead level

<sup>c</sup>Argument/Fight n = 337

<sup>d</sup>Argument/Fight n = 194

All models are fully adjusted (include lead mean and lead peak - separate analyses - as well as sex, race, SES, and age at crime)

\* denotes p < .05

## Perpetrators

A total of 600 individuals met our inclusion and exclusion criteria by lead tests reported, age, and firearm homicide or nonfatal shooting perpetration. Due to setting Domestic Violence-related shootings to missing, our final sample was 589. The overall sample of perpetrators was predominantly Male (95%), African American (91%), and of lower socioeconomic status (88%) (Table 3). Our perpetrator sample was younger than our victims sample with a quarter (26%) under the age of 18 years old. Similar to victims, most firearm homicide and nonfatal shooting incidents were Argument/Fight shootings (33%), followed by Nondrug crime (21%). Overall mean and peak lead levels were higher than the current (5 µg/dL in 2012) and former (10 µg/dL from 1991 - 2012) actionable levels set by the Center for Disease Control and Prevention.

When lead levels among perpetrators were examined by sample characteristics, males perpetrators had higher mean lead levels (Lead Mean for males: 13.2 vs. for females 13 µg/dL) but lower peak lead levels than female perpetrators (Lead Peak for males : 16.3 vs. for females 16.7 µg/dL), African American perpetrators had higher mean lead levels than perpetrators of Whites/Other races (Lead Mean for African Americans: 13.6 vs. for Whites/Other races 9.6 µg/dL and Lead Peak for African Americans: 16.6 vs. for Whites/Other races 13.5 µg/dL) and perpetrators with the lowest socioeconomic status had higher mean lead levels than perpetrators in all other socioeconomic status categories (Lead Mean for lowest: 13.9 vs. other 8.1 µg/dL and Lead Peak for lowest: 17.2 vs. other 9.8 µg/dL). Average mean and peak lead levels were higher for older perpetrators compared to younger perpetrators.

Each shooting type had over 90% male perpetrators with Gang shootings having the highest percentage of male perpetrators (100%). All but Gang-related shootings had over 89% African American perpetrators and Retaliation-related shootings had the highest percentage of

African American perpetrators (97%). Additionally, every type of shootings had over 80% lower socioeconomic status perpetrators. Argument/Fight-related shootings had the highest percentage of low socioeconomic status perpetrators (91%). Gang-related shootings had the greatest percentage of perpetrators under 18 years old (42%) while Retaliation-related shootings had the greatest percentage of perpetrators over 20 years old (42%). When mean lead levels were examined by known shooting type, Argument/Fight-related shootings had the highest mean and peak lead levels. Other shootings had the lowest mean and peak lead levels.

Our logistic regression analysis results show that for every 1  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, the adjusted odds of being a perpetrator of an Argument/Fight-related shooting compared to being a perpetrator of all other types of shootings increases but is not significant (OR 1.02, 95% CI .996, 1.04) (Table 4). Similar results were found for each 1  $\mu\text{g}/\text{dL}$  increase in peak childhood lead level (OR 1.01, 95% CI .989, 1.02).

The results of our multinomial logistic regression across multiple shooting types showed that the relative risk of being a perpetrator of an Argument/Fight-related shooting compared to a Retaliation-type of shooting is 27% higher for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, holding sex, race, SES, and age constant (RRR 1.27, 95% CI 1.03, 1.55) (Table 4). Lead peak results were similar to mean lead results but not significant (RRR 1.12, 95% CI .95, 1.3). The adjusted relative risk of being a perpetrator of an Argument/Fight-related shooting compared to a Nondrug crime (RRR 1.16, 95% CI .995, 1.37), Drug (RRR 1.11, 95% CI .95, 1.33), or Other (RRR 1.21, 95% CI .91, 1.58) shooting are all higher than 1 for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, but none are significant. The adjusted relative risk of being a perpetrator of an Argument/Fight-related shooting compared to a Gang-related (RRR .91, 95% CI .71, 1.18) shooting falls below 1 for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level,

but the results also include the null.

The relative risk of being a perpetrator of Argument/Fight-related shooting compared to an Unknown-type of shooting is 14% lower for each 5 µg/dL increase in the peak childhood lead level, holding sex, race, SES, and age constant (RRR .86, 95% CI .74, .998). Lead mean results were similar to peak lead results but not significant (RRR .86, 95% CI .71, 1.05). The adjusted relative risk of being a perpetrator of an Argument/Fight-related shooting compared to a Nondrug crime (RRR 1.09, 95% CI .95, 1.23), Drug (RRR 1.01, 95% CI .91, 1.15), or Other (RRR 1.15, 95% CI .95, 1.4) shooting are all higher than 1 for each 5 µg/dL increase in the peak childhood lead level, but none are significant. The adjusted relative risk of being a perpetrator of an Argument/Fight-related shooting compared to a Gang-related (RRR .95, 95% CI .78, 1.13) shooting falls below 1 for each 5 µg/dL increase in the peak childhood lead level, but the results also include the null.

## **Discussion**

Using blood lead and firearm violence data linked at the individual level, we estimated the association between childhood lead exposure before 6 years old and firearm violence victimization and perpetration by shooting type. When we examined the descriptive statistics by shooting type, we saw that victims of Other shootings (Negligent Handling, Police-Related, Self-Defense, Other and Possibly Self-Inflicted) differed from victims of all other shootings. Victims of Other shootings were more female, had lower socioeconomic status, and were younger than victims of alternative shootings. It is difficult to interpret why victims of Other shootings differed from the victims of alternative shooting types because we combined such a diverse grouping of shooting types to create the Other category (shootings that are Police-related, Negligent Handling, Self-Defense, and Other). We cannot use prior literature to guide the

interpretation of these findings because when prior research has included an Other category, the groupings differed from our study. For example, Pizarro combined gang, self-defense, witness tampering, accidents and mental illness-related shootings to make up her Other category (J. M. Pizarro, 2008). Firearm violence can vary across cities (i.e. higher number of gang-related shootings in one city compared to another overall or during the time period of interest) and so it may be more appropriate to group the shootings differently.

Victims of Gang-related shootings also differed from victims of other shooting types. Victims of Gang-related shootings were more White/Other race, Other (Middle, Upper and Other) socioeconomic status and older compared to victims of alternative shooting types. Like victims, perpetrators of Gang-related shootings generally differed from perpetrators of alternative shooting types. Perpetrators of Gang-related shootings were more White/Other race and younger compared to perpetrators of alternative shooting types. These differences could be related to Latin and Asian gangs committing firearm violence in Milwaukee. Again, it is challenging to compare our results on gang-related shootings to prior literature as past studies examined gang-related shootings in different cities. Gang-related firearm violence differs across cities. For example, some cities, such as Los Angeles and Chicago, are known for chronic, long-standing gangs and gang-related firearm violence (J. M. Pizarro & McGloin, 2006). While other cities, such as Newark, NJ, are known as emerging gang cities. Milwaukee falls more in the latter category, but even gangs in similar emerging gang cities differ. A study by Pizarro and McGloin examined gang-related homicides in Newark, NJ. Unlike our study, their sample had a greater proportion of African American victims (87%) and suspects (90%) of gang-related shootings (J. M. Pizarro & McGloin, 2006).

Our hypothesis for our multivariate analysis was that victims of an Argument/Fight-related shooting would have higher lead levels compared to victims of alternative shootings types, holding race, sex, SES and age at crime constant. Our adjusted results reveal that victims of Argument/Fight-related shootings have significantly higher mean blood lead levels compared to victims of Other shootings and perpetrators of Argument/Fight-related shootings have significantly higher peak blood lead levels compared to perpetrators of Retaliation-related shootings. These results are consistent with our stated hypothesis, suggesting a possible causal linkage in which lead exposure led to increased risk of victimization and perpetration of Argument/Fight-related shootings relative to victimization and perpetration of alternative shooting types. While the relative risk of being a victim or a perpetrator of an Argument/Fight-related shooting compared to alternative shooting types (e.g. Nondrug crime, Drugs, etc.) were higher than one but not significant for each 5  $\mu\text{g}/\text{dL}$  increase in the mean or peak childhood lead level, these results are trending in support of our hypotheses. Our results are consistent with previous studies that have disaggregated shootings by type and found significant differences in certain predictors by shooting type (Cooper & Smith, 2012; Kubrin & Herting, 2003; Kubrin & Wadsworth, 2003; Kubrin, 2003; Parker, 1989; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Roberts et al., 2007; Tita & Griffiths, 2005; Williams & Flewelling, 1988; Zeoli et al., 2015).

One result that diverged from our hypothesis across victims and perpetrators was related to Gang shootings. As we saw in the descriptive statistics, victims and perpetrators of Gang-related shootings were generally different than other shootings by race, socioeconomic status and age. While not significant, the relative risk of being involved in an Argument/Fight-related shooting was lower compared to a Gang-related shooting for every 5  $\mu\text{g}/\text{dL}$  increase in mean or peak childhood blood lead for both victims and perpetrators. One potential explanation for this



could be that childhood lead exposure is positively associated with firearm violence victimization and perpetration, but other factors play more of a role in driving Gang-related firearm violence (Land et al., 1990). For example, association with a deviant peer group has consistently been found to be a predictor of future violent offending (Bernat et al., 2012; Farrington, 1998b; Ferguson et al., 2009; Haynie & Osgood, 2005; Loeber et al., 2005). Elevated lead could predispose individuals to firearm violence and then their association with a deviant peer group could influence their participation in Gang-related violence, specifically.

Unknown shootings may have influenced our results, especially for victims of firearm violence. The Unknown shooting category is the second largest for victims. The demographic profiles of victims and perpetrators of Unknown shootings are very similar to those of Argument/Fight-related shootings. Individuals with high lead levels could be victims or perpetrators of Argument/Fight-related shootings but since we don't have enough information from police reports to categorize them as such, they are currently miscategorized as Unknown. The relative risk of being a perpetrator of an Argument/Fight-related shooting was significantly lower compared to Unknown shootings for each 5  $\mu\text{g}/\text{dL}$  increase in the peak childhood blood lead level. The lead mean and lead peak values for perpetrators of Unknown are higher than any other category, so where these perpetrators fall could have a dramatic effect on our results. We recommend revisiting police reports for Unknown shootings to see if any additional information has been added and the shootings can be more accurately categorized for future analysis.

Another point to consider is the potential overlap between impulsive and premeditated firearm violence. We based our hypothesis on the notion that shootings fall into one of these two categories. Argument/Fight-related shootings were impulsive in nature and individuals involved in Argument/Fight-related shootings were more impulsive due to their childhood lead exposure.

In reality, shootings may fall into both of these categories (Declercq & Audenaert, 2011). A perpetrator of a robbery-related shooting could plan to shoot the clerk at a gas station he or she is robbing and then impulsively decide to shoot customers that unexpectedly enter the gas station during the robbery. The potential overlap in impulsive and premeditated shootings should be factored into future research that disaggregates firearm violence by type.

This study was able to address several limitations of previous research. It examined the effect of an environmental stressor on both firearm homicides and nonfatal shootings disaggregated by shooting type using linked data at the individual level. This study had important limitations as well. First, using Milwaukee Health Department childhood lead data may affect the validity of the study results. Blood lead is not able to fully capture long term exposure. To overcome this limitation, we measured the mean of all blood lead test results under 6 years of age in an attempt to better capture childhood lead exposure over time. Additionally, children may not have been tested when they were exposed to lead (blood lead tests detect recent exposure). The fact that blood lead data is potentially missing or not representative of their exposure could result in a misclassification of their exposure and bias our results. However, misclassification is likely constant across our entire sample as we restricted our sample to individuals who consistently lived in the city of Milwaukee throughout childhood and adolescence. By doing so, our sample had similar exposure to childhood lead testing campaigns and outreach efforts during similar time periods.

Second, there are limitations to using crime incident data to evaluate shooting type. Data may be missing or incomplete and it may not employ consistent, systematic reporting methods (Masho et al., 2016). The fact that crime incident data could be potentially missing or not fully representative of the outcome could result in a misclassification of shooting type and bias our

results. This was especially apparent in the high number of “Unknown” shootings. Almost one third of all shootings in our victim-only analysis did not have a known shooting type. This means that there was not enough information in the homicide files or police reports for Milwaukee Homicide Review Commission staff to make a determination on what the circumstances were that led to the shooting. This could be related to Milwaukee’s low shooting clearance rates (Decker, 1996). Clearance rates are calculated by taking the number of crimes that are cleared - suspect identified and charges are laid - by the total number of crimes recorded in a given year. A clearance rate is sometimes used to measure the proportion of crimes solved by the police that year. In 2015, Milwaukee’s homicide clearance rate was approximately 60%, down from 93% in 2008 (Milwaukee Police Department, 2015). That same year, the clearance rate for nonfatal shootings was 31% (Milwaukee Homicide Review Commission, 2017). According to the Milwaukee Homicide Review Commission 2016 annual report, the clearance rate for nonfatal shootings in Milwaukee has ranged between 16% - 34% from 2006 – 2015 (Milwaukee Homicide Review Commission, 2017). Cities with higher clearance rates tend to have more complete police records than those with lower clearance rates (Decker, 1996). In theory, the more information the police have about a shooting, the more likely they are to identify a subject and clear the case. The Milwaukee Homicide Review Commission has attempted to overcome some of these limitations by creating its own homicide and nonfatal shooting databases and employing consistent data collection and coding methods based on National Violent Death Reporting System and FBI Uniform Crime Reporting standards. Conducting monthly case reviews with criminal justice partners provided added information on homicides and nonfatal shootings that may have been missing from police reports. It should also

be noted that law enforcement invests a significant amount of resources into homicide investigations and therefore homicide data is generally more robust than data for other crimes.

Third, our shooting type groupings may have affected our results. We based our groupings on trends found in the literature. Unfortunately, there isn't consensus in the literature on the appropriate groupings. For example, Pizarro defined the "interpersonal dispute" category as "incidents involved nondomestic and nondrug murders that resulted from an argument, physical altercation, or the victim/offender wanting to get even for a past altercation" (J. M. Pizarro, 2008, pg. 330). Based on our definitions, this would mean she combined argument/fight and retaliation-related shootings into one shooting type. Future research may want to consider alternative groupings of shooting types.

This study found that victims and perpetrators with higher childhood lead results were at greater relative risk of being victims or perpetrators of Argument/Fight-related shootings compared to other shootings, warranting further research investigating the differences in shootings disaggregated by type. Childhood lead exposure may be driving certain types of firearm violence in combination with other multilevel factors, such as the association with a deviant peer group. We were not able to evaluate these other factors in this study and therefore recommend including other individual, peer and neighborhood level predictors of disaggregated firearm violence in future research.

## **CHAPTER 5: INTEGRATED DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS**

### **Discussion**

There is a significant public health need to identify risk factors that can be modified by policies and interventions to prevent firearm homicides and nonfatal shootings. Based on my literature review in chapter one, we know that childhood lead exposure is a biologically plausible environmental predictor of firearm violence and an important research area that has not been addressed. To overcome previous limitations and address current gaps in the research outlined in chapter one, I crafted three manuscripts (chapters 2-4) examining the relationship between childhood lead exposure and firearm violence. The first manuscript (chapter 2) reviewed the relevant literature and proposed a conceptual model illustrating the corresponding factors involved in this relationship as well as the mechanisms by which childhood lead exposure leads to firearm violence. The second manuscript (chapter 3) tested this conceptual model quantitatively and evaluated the extent to which childhood lead exposure may contribute to firearm violence perpetration or victimization later in life by estimating the association using logistic regression and controlling for confounding. Finally, for the third manuscript (chapter 4), I took the analysis in manuscript two (chapter 3) a step further and evaluated the extent to which childhood lead exposure is related to certain types of firearm violence perpetration or victimization, compared to other types of firearm violence, by estimating the relative risk using multinomial logistic regression methods and controlling for confounding.

These studies add to a growing body of literature supporting the relationship between childhood lead exposure and violent criminal offending. The first manuscript (chapter 2) integrated the childhood lead exposure and firearm violence literature and created a

conceptual model illustrating the relationship. This model was used to conceptually and methodologically guide the second and third manuscripts (chapter 3 and 4) as well as interpret the results. For example, the model was used to guide the selection of potential confounding variables controlled for in the second and third manuscripts (chapter 3 and 4). I controlled for individual level as well as multi-level factors based on the model and the variables available in the data.

Consistent with prior research, this study found that poor, Black individuals were disproportionately affected by both childhood lead exposure and firearm violence. This was supported both in the literature and our analyses. We learned in manuscript one (chapter 2) that African Americans are more likely be exposed to lead based on where they live. They are more likely to live in areas with elevated lead air concentrations, near highways, industrial zones or smelter sites and in lower income and rental housing, all of which puts them at greater risk of lead exposure (Hird & Reese, 1998; R. Nevin, 2000; P. B. Stretesky, 2003). Due to barriers such economic constraints and restrictive housing policies, African Americans have been less able to move away from these high lead risk areas (Narag et al., 2009; P. B. Stretesky & Lynch, 2004). African Americans are also more likely to have elevated lead levels. National studies found that when compared to children of other races, African American children had significantly higher mean blood lead levels and were more likely to have blood lead levels in elevated ranges (Wheeler & Brown, 2013; White et al., 2016).

We saw this pattern in our second and third manuscripts (chapter 3 and 4). In manuscript two (chapter 3), the overall average lead levels for the entire sample (Lead Mean: 7.6  $\mu\text{g}/\text{dL}$ , SD: 5.7  $\mu\text{g}/\text{dL}$  and Lead Peak: 9.6  $\mu\text{g}/\text{dL}$ , SD: 9  $\mu\text{g}/\text{dL}$ ) were higher than the current actionable level set by the Center for Disease Control and Prevention in 2012 (5  $\mu\text{g}/\text{dL}$ ) but lower than the

actionable level set between 1985-1991 (25 µg/dL) and 1991 - 2012 (10 µg/dL). When we examined the sample by race, African Americans had higher average lead levels than Whites and Other races (Lead Mean for African Americans: 8.7 µg/dL, SD: 6.2 µg/dL vs. for Whites 5.5 µg/dL, SD: 4 µg/dL vs. for Other races 6.4 µg/dL, SD: 4.5 µg/dL and Lead Peak for African Americans: 11 µg/dL, SD: 9.7 µg/dL vs. for Whites 6.9 µg/dL, SD: 6.9 µg/dL vs. for Other races 8.2 µg/dL, SD: 7.6 µg/dL). On average, lead levels for African Americans were higher than the most recent and past actionable levels set by the Center for Disease Control and Prevention in 2012 (5 µg/dL) and 1991 - 2012 (10 µg/dL). When we were only examining victims and perpetrators of firearm violence in manuscript three (chapter 4), African Americans had higher average lead levels than individuals of other races. African American victims had higher average lead levels than Whites/Other races (Lead Mean for African Americans: 13.4 µg/dL vs. for Whites/Other races 10.1 µg/dL and Lead Peak for African Americans: 16.7 µg/dL vs. for Whites/Other races 11.9 µg/dL) as did African American perpetrators (Lead Mean for African Americans: 13.6 µg/dL vs. for Whites/Other races 9.6 µg/dL and Lead Peak for African Americans: 16.6 µg/dL vs. for Whites/Other races 13.5 µg/dL).

We also know that African Americans are disproportionately affected by firearm violence, both as victims and perpetrators. Firearm violence is the leading cause of death for non-Hispanic African American men aged 15-34 years (David-Ferdon & Simon, 2014; Wintemute, 2015) and African Americans are more likely to commit firearm violence than non-Hispanic Whites and Hispanics (Federal Bureau of Investigation, 2015; J. L. Lauritsen et al., 2009; Nielsen et al., 2005). In our second manuscript (chapter 3), we saw that 87% of our victims and 95% of our perpetrators in Milwaukee were African American. Similar results were found in manuscript three (chapter 4).

Also, consistent with the literature, I found that poor individuals were disproportionately affected by both childhood lead exposure and firearm violence (Lidsky & Schneider, 2003). The socioeconomic status (SES) of a child's family and the neighborhood they grew up in affects their risk of lead exposure. Children with lower family SES are more likely to live in older, rental properties with deteriorating windows and lead paint hazards. Socioeconomically disadvantaged neighborhoods are more likely to have these high-risk properties. Additionally, children with lower SES are more vulnerable to the effects of lead exposure potentially due to nutrient deficiencies, potential genetic factors, the stress of living in poverty and growing up in less stimulating environments (D. C. Bellinger, 2008; R. O. Wright & Baccarelli, 2007).

We saw this pattern in our second and third manuscripts (chapter 3 and 4). On average, individuals with the lowest SES had higher mean and peak lead levels than individuals in all other SES categories (Lead Mean for Lower: 8.5  $\mu\text{g/dL}$  vs. for Middle 5.3  $\mu\text{g/dL}$  vs. for Upper 4.6  $\mu\text{g/dL}$  vs. for Other 5  $\mu\text{g/dL}$  and Lead Peak for Lower: 10.9  $\mu\text{g/dL}$  vs. for Middle 6.3  $\mu\text{g/dL}$  vs. for Upper 5.4  $\mu\text{g/dL}$  vs. for Other 5.7  $\mu\text{g/dL}$ ). When we examined only victims and perpetrators of firearm violence in manuscript three (chapter 4), victims and perpetrators with lower SES had higher mean lead and peak lead levels compared to victims (Lead Mean for Lower: 13.9  $\mu\text{g/dL}$  vs. for Other 8.3  $\mu\text{g/dL}$  and Lead Peak for Lower: 17.3  $\mu\text{g/dL}$  vs. for Other 10  $\mu\text{g/dL}$ ) and perpetrators (Lead Mean for Lower: 13.9  $\mu\text{g/dL}$  vs. for Other 8.1  $\mu\text{g/dL}$  and Lead Peak for Lower: 17.2  $\mu\text{g/dL}$  vs. for Other 9.8  $\mu\text{g/dL}$ ) in the Middle, Upper and Other SES categories.

Consistent with the firearm violence literature, we saw that individuals with lower SES were disproportionately affected by firearm violence, both as victims and perpetrators. Most



street shootings occur in low SES neighborhoods and neighborhood and family level SES have been identified as predictors of violent behavior (Dubow et al., 2016; Farrington, 1998b; Loeber et al., 2005). In 2015, 82% of homicides and 83% of nonfatal shootings in Milwaukee occurred in the zip codes with the lowest SES (Milwaukee Homicide Review Commission, 2015). In manuscripts two and three (chapter 3 and 4), we saw that 87% of our victims and 88% of our perpetrators had low SES.

Most importantly, we found an association between childhood lead levels and firearm violence. After adjustment for confounding, our results show that for every 1 $\mu$ g/dL increase in the average childhood lead level, the odds of an individual becoming a victim or a perpetrator of firearm violence significantly increases. Similar results were found for each 1 $\mu$ g/dL increase in peak childhood lead level. After categorizing the lead mean and peak values, we found a dose-response relationship between childhood lead levels and firearm violence victimization and perpetration. These results are consistent with prior cohort studies that found an association between childhood lead levels and violent criminal behavior (D. M. Fergusson et al., 2008; J. P. Wright et al., 2008).

Knowing that childhood lead exposure is associated with firearm violence victimization and perpetration, we took our analysis a step further to see if childhood lead exposure was driving a certain type of firearm violence. We used the conceptual model in manuscript #1 (chapter 2) to guide the development of our hypothesis. Our results show that there is an association between childhood lead levels and firearm violence victimization and perpetration and shooting type. When we looked at victims, specifically, we found that the relative risk of being a victim of an Argument/Fight-related shooting was significantly higher compared to Other shootings for each 5  $\mu$ g/dL increase in the mean childhood lead level, after controlling for

confounding. The relative risk of being a victim of an Argument/Fight-related shooting compared to Retaliation, Nondrug Crime, and Drug-related shootings was higher than one for each 5  $\mu\text{g}/\text{dL}$  increase in the mean or peak childhood lead level, but they all included the null. For perpetrators, we found that the relative risk of being a perpetrator of an Argument/Fight-related shooting was significantly higher compared to Retaliation-related shootings for each 5  $\mu\text{g}/\text{dL}$  increase in the mean childhood lead level, after controlling for confounding. The relative risk of being a perpetrator of an Argument/Fight-related shooting compared to Nondrug Crime, Drug, and Other shootings was higher than one for each 5  $\mu\text{g}/\text{dL}$  increase in the mean or peak childhood blood lead level, but those results also included the null. Our results are consistent with previous studies that have disaggregated shootings and found differences in certain predictors by shooting type (Cooper & Smith, 2012; Kubrin & Herting, 2003; Kubrin & Wadsworth, 2003; Kubrin, 2003; Parker, 1989; J. M. Pizarro, 2005; J. M. Pizarro, 2008; Roberts et al., 2007; Tita & Griffiths, 2005; Williams & Flewelling, 1988; Zeoli et al., 2015). While more research is needed, these results can be used to guide and further refine the conceptual model in manuscript #1 (chapter 2). For example, impulsivity may not only be driving firearm violence (as is illustrated in the conceptual model), but argument/fight-related firearm violence specifically.

For both victims and perpetrators, the relative risk of being involved in an Argument/Fight-related shooting was lower compared to a Gang-related shooting for every 5  $\mu\text{g}/\text{dL}$  increase in mean or peak childhood blood lead, but the results were not significant. I used the conceptual model to look for potential alternative explanations as to why these results may not have been consistent with current literature. The conceptual model identified that deviant peers have consistently been found to be a predictor of future violent offending (Bernat et al.,

2012; Farrington, 1998b; Ferguson et al., 2009; Haynie & Osgood, 2005; Loeber et al., 2005).

Therefore, elevated lead could predispose individuals to firearm violence but then their association with a deviant peer group could influence their participation in Gang-related violence, specifically.

## **Implications**

Before this study, there was little debate that lead exposure in children yields poor health outcomes. Despite this, childhood lead prevention remains a non-priority for many. My hope is that by relating childhood lead exposure to firearm violence, these findings provide further support and urgency for immediate investment in primary prevention of lead exposure in children, especially for those at highest risk.

Primary prevention includes lead abatement through lead painted window updates, repairs and replacement in deteriorating housing and lead water main removal and replacement throughout the city. Landlords who are not remediating lead hazards need to be held accountable, as do lawmakers in charge of funding lead abatement. Currently, the Milwaukee Health Department Childhood Lead Poisoning Prevention Program does remove lead from high risk homes. Unfortunately, funding constraints only allow for 400 remediations per year (City of Milwaukee Health Department, 2016). There are at least 17,000 housing units in Milwaukee where lead paint hazards remain (City of Milwaukee Health Department, 2016). If the funding for lead remediation remains at its current level, it will take Milwaukee 42 years to fully remediate all of these homes (City of Milwaukee Health Department, 2016). We cannot accept this current timeline in good conscience, from a public health and social justice point of view.

Additionally, drinking water is still delivered in Milwaukee through lead lateral service pipes to 70,000 residential properties built before 1951 (Behm, 2016b). Replacing all of the lead

service lines would cost an estimated \$511 to \$756 million (Behm, 2016a). While this is undoubtedly a large amount of money to invest in one program, readers are reminded that the money is available, we just need to prioritize this issue and shift to long-term thinking. Not only is this an important investment for the health and wellbeing of children, studies have shown that reducing lead exposure can result in considerable cost-savings long term. It is estimated that for each 1 microgram/deciliter increase in blood lead exposure, children lose between \$3,000 and \$8,000 in lifetime productivity (Center for Disease Control and Prevention, 2014). Between 2008-2010, the CDC estimates that its state programs helped reduce the number of children 5 years and under with lead levels over 1 mc/dL by 3 million. It attributes a cost-savings of \$26-\$57 billion in lifetime productivity earnings alone from those efforts (Centers for Disease Control and Prevention, 2017). This estimate does not include cost-savings related to other adverse effects such as firearm violence.

Our findings support increased education on individual-level primary prevention strategies, especially for those at greatest risk of exposure. Individuals with children at highest risk need to be aware of the risk and the potential consequences of lead exposure. They also need to be better informed of the possible steps they can take to lower their risk. These steps need to be cost effective or subsidized because those at highest risk have limited resources.

Additionally, our findings offer support and opportunities for secondary prevention efforts. These efforts could include early identification of lead exposed children, the development of comprehensive wraparound services for lead exposed children, and resiliency promotion through the introduction and encouragement of protective factors. One consideration is treating childhood lead exposure like autism. Similar to autism, there is no cure for childhood lead exposure. Children can receive chelation therapy to remove the lead from their body, but

this does not treat the effects of the exposure. Even though there is no cure, we know that early screening and identification of autism as well as early intervention services can improve a child's development (Center for Disease Control and Prevention, 2015b). Extensive research has gone into developing and testing these interventions (Center for Disease Control and Prevention, 2015b). Our findings support the development and testing of similar programs that are appropriate for children exposed to lead. Our conceptual model could be used as a starting point for this type of research. For example, we see from our conceptual model that children exposed to lead are at greater risk for poor academic outcomes (Aizer et al., 2015; M. S. Amato et al., 2012; Evens, 2010; Evens et al., 2015; D. M. Fergusson et al., 1993; D. M. Fergusson et al., 1997; B. P. Lanphear et al., 2003; B. P. Lanphear et al., 2000; H. L. Needleman et al., 1990; Zhang et al., 2013) and that positive academic outcomes can be protective of future firearm violence (Cook & Laub, 2002; Dubow et al., 2016; Resnick et al., 2004). One early intervention for children exposed to lead could be additional support in schools to promote positive academic outcomes.

Due to the link between lead exposure and criminal behaviors, another area to consider is the criminal justice system. We may need to start considering childhood lead exposure when determining appropriate criminal justice interventions. Certain criminal justice interventions may be more or less effective than others for individuals known to have early lead exposure. Applying the same type of criminal justice intervention for individuals with known childhood lead exposure as individuals without lead exposure may just be setting them up to fail. For example, we know from the conceptual model that childhood lead exposure can affect a person's executive functioning, which controls their ability to manage and organize their day-to-day life. This could affect their ability to comply with their community supervision requirements (e.g.,

meeting regularly with his or her agent, performing regular drug tests, etc.). If an agent was made aware of an offender's childhood lead exposure and the potential deficits caused by such exposure when they start working with them, they may be able to provide additional, targeted supports to that person before they are revoked for noncompliance.

Based on our findings, lead exposure prevention also needs to be considered a firearm violence prevention strategy and incorporated into future firearm violence prevention efforts. The Milwaukee Health Department recently developed a city-wide violence prevention strategy (Milwaukee Health Department, 2017). Primary or secondary childhood lead exposure prevention was not included in this plan (Milwaukee Health Department, 2017). The plan does include reaching out to high risk youth, but childhood lead exposure was not included in the list of risk factors for firearm violence involvement. Based on these and previous studies, we need to consider incorporating lead exposure into evidence-based violence prevention efforts.

### **Limitations**

This study was able to address several limitations of previous research. It examined the relationship between childhood lead exposure and firearm violence, specifically, as well as victimization and perpetration risk of both firearm homicides and nonfatal shootings using linked data at the individual level. It then took this analysis a step further and investigated the relationship between childhood lead exposure and disaggregated shooting type.

Despite these advances, this study had important limitations as well. First, using Milwaukee Health Department childhood lead data may affect the validity of the study results. The details of this limitation are discussed in manuscript two (chapter 3) and manuscript three (chapter 4). Second, there are limitations to using crime incident data to measure firearm violence perpetration, victimization and type. This is described more in depth in manuscript two

(chapter 3) and manuscript three (chapter 4). Third, the groupings we chose to collapse the shootings into for manuscript three may have affected our results. This is discussed in manuscript three (chapter 4). Fourth, there are limitations to using a retrospective cohort study design. This is discussed in detail in manuscript two (chapter 3). Fifth, because our study population was limited to Milwaukee and only individuals who attended MPS, the results may not be generalizable to other populations.

### **Recommendations/Conclusions**

This study presents many opportunities for future research. Childhood lead exposure has been on the decline in the years since many of the victims and perpetrators in this cohort were born. Knowing this, we might predict that lead exposure will play less of a role in firearm violence in the future. The reality is that while childhood lead exposure is decreasing in the overall population, children in the United States (and other countries) are still exposed to unacceptably high levels of lead; especially poor, children of color. We witnessed this in Flint, Michigan in 2014. The Flint water crisis affected approximately 99,000 people living in Flint, Michigan. A study by the CDC found that the adjusted probability of children aged <6 years having a blood lead test  $\geq 5 \mu\text{g/dL}$  was 46% higher during the time period when the water source was switched than during the period before the water was switched (Kennedy, 2016). To build upon our work, we recommend further research examining the effect of childhood lead exposure on firearm violence victimization and perpetration disaggregated from other violent crimes, especially in children at greatest risk for lead exposure. This research should consider exploring different methods evaluating this association as well.

More research is needed to expand, validate and update the conceptual model illustrating the relationship between childhood lead exposure and firearm violence. The conceptual model provides a framework to guide future lead and firearm violence research. In our study, we were only able to test a limited number of the relationships between constructs in the model. Future studies should use this model to develop hypotheses, explore the associations between key constructs, examine potential mediating and modifying relationships, and interpret their results. As more research is published in this area, researchers are encouraged to build upon and expand this model as I did with Narag et al's model.

DataShare MKE and other similar integrated data systems are needed further investigate the relationships between health and criminal justice outcomes. One of the reasons why there is limited research on public health risk factors for criminal justice outcomes is the lack of linked longitudinal data available to support this research. The hope is that our study will encourage the development of other integrated data systems like DataShare MKE and promote the use of DataShare MKE for similar multidisciplinary research.

Thinking more broadly, we know that a child's risk of exposure to lead is closely tied to where that child lives. Living in older, deteriorating rental properties increases one's risk for exposure significantly because those properties are most likely to have lead paint hazards. Because children do not control their housing situation, their living situation is dependent on where their family is living.

Milwaukee is one of the most, if not the most, segregated cities in the nation. A history of discriminatory local and federal housing policies has resulted in the racial hyper-segregation of Milwaukee over time. African American residents have been legally banned from living in certain areas, and therefore have not been afforded the same control over where they live that



their White counterparts have enjoyed for many years. At the federal level, the United States Federal Home Loan Bank Board (FHLBB) developed a Residential Security map of Milwaukee County in 1938 that evaluated the riskiness of mortgages based on neighborhood and made it practically impossible for residents get a mortgage in areas they categorized on the map as “red” (Maternowski & Powers, 2017). These areas were categorized as “red” or “hazardous” due to heavy industry, polluted waterways, lack of modern sewage, old housing stock, and the people who lived there (Maternowski & Powers, 2017). Almost all neighborhoods where African Americans lived were categorized as “red” (Maternowski & Powers, 2017). This practice of denying or restricting financial services based on the racial makeup of the neighborhood is called redlining (Chang, Smith, & Gartner, 2016). As a result of redlining, African Americans were restricted from owning homes in their neighborhoods because it was nearly impossible for them to get approved for a mortgage. This policy prevented African Americans from accumulating the wealth that comes along with home ownership.

African Americans also couldn't move and try to own a home in a different neighborhood due to the corresponding existence of discriminatory covenants (Quinn, 1979). These race restrictive covenants were developed in the early 1900s by real estate operators, local real estate boards, financial institutions and title companies in an effort to keep African American and other non-White families from living in certain neighborhoods (Quinn, 1979). The covenants did not allow residential land to be sold or leased to non-White individuals, or African Americans specifically, for a set period of time (Quinn, 1979). By the 1940s, sixteen of Milwaukee County's eighteen suburbs were using racially restrictive covenants that excluded African American families from those residential areas (Quinn, 1979). These racially restrictive covenants were still being used to exclude African Americans from moving to new subdivisions

in 1958 - 10 years after the U.S. Supreme Court outlawed judicial enforcement of them (Quinn, 1979). Many racially restrictive covenants in Milwaukee subdivisions extended into the late 1960's and 1970's, with some extending into the 1980's and even into the 2000's (Quinn, 1979). Redlining and racially restrictive covenants were outlawed in 1968 with the passing of the Fair Housing Act (Maternowski & Powers, 2017).

Even with these policies no longer in place, it is difficult for African Americans in Milwaukee to relocate to different neighborhoods with improved housing stock. African Americans who try to move to more affluent, predominantly White neighborhoods often face racism by neighbors and local police. In a New York Times article on this topic, the father in an African American family that moved to Whitefish Bay, a majority White, more affluent neighborhood, was stopped by the local police about once per week for almost two months shortly after they relocated to the neighborhood (Eligon & Gebeloff, 2016). In addition, he experienced a neighbor bringing her children inside her house when she saw him walking on the street (Eligon & Gebeloff, 2016).

Higher job growth in suburban areas (versus urban areas with high concentrations of African Americans) and a lack of public transportation to these jobs have also inhibited African Americans from accumulating wealth. A study by the University of Wisconsin-Milwaukee Center for Economic Development found that 70% of businesses with strong hiring projections for entry-level work are located in the suburbs of Milwaukee (Rast, 2004). According to the study, almost half of low-income Milwaukee-area families rely on public transportation (Rast, 2004). For low-income families to access these jobs, it is imperative for job opportunities to be accessible by public transportation (Rast, 2004). The study found that when travel times are considered, the percentage of businesses accessible to low-income residents in Milwaukee is low

(Rast, 2004). For example, only 32.4% of businesses with strong hiring projections for entry-level workers are located within a quarter mile of bus lines and reachable within a one-hour commute for residents of the Near South Side neighborhood (Rast, 2004). It is important to note that this problem is not getting better over time as public transportation continues to be cut while investment in highways continues to grow (Rast, 2004).

The concentration of elevated lead levels in low income, African American children in Milwaukee may be a consequence of its history of discrimination towards African Americans. By systematically restricting African Americans to live in certain neighborhoods and limiting their ability move out of these neighborhoods, African American families were forced to live in neighborhoods with housing most likely to have lead paint hazards. African American children were then exposed to lead more than children of other races because of where their families were forced to live. These discriminatory restrictions and the lack of access to job opportunities in suburban areas have also prevented African Americans from accumulating wealth over time. And as we learned from the conceptual model, SES is a major predictor of childhood lead exposure as well as firearm violence perpetration and victimization.

We also need to consider gun control in tandem with childhood lead exposure prevention. We cannot only remove lead, we need to also consider passing stronger gun laws. As we learned in our conceptual model, there is scientific consensus that stricter gun laws reduce gun violence (Hemenway & Nolan, 2016). There is also a shortage of gun violence research. Much of this is due to funding restraints. Compared to other diseases and public health issues, gun violence research has received significantly less funding, despite its prevalence and high mortality and morbidity rates (Stark & Shah, 2017). One reason for the limited funding is related to the “Dickey Amendment,” which does not allow the CDC to fund firearm violence research (Stark

& Shah, 2017). In order to make continued progress in firearm violence research, the Dickey Amendment needs to be repealed and more funding for firearm violence research is needed.

Despite numerous efforts to reduce lead exposure, children, especially low income and minority children, continue to be exposed to dangerous levels of lead. Knowing what we know about the indisputable, irreversible, lifelong negative impacts of childhood lead exposure, policymakers and individuals in charge of resource allocation need to prioritize funding for primary and secondary lead exposure prevention in order to protect the long-term health and wellbeing of children. In addition, Congress needs to lift the restrictions on funding for firearm violence research and increase funding overall. The nature and severity of the consequences of lead exposure and firearm violence coupled with their disproportionate effect on low income, minorities living in socioeconomically disadvantaged neighborhoods makes this a public health crisis with serious social and environmental justice implications.

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## CHAPTER 7: APPENDIX

### Shooting Type Definitions

**Argument/Fight** - A shooting that occurs during or as a result of a dispute; heat of moment in nature; there is continuity in the incident (shooting happens during or at the close of the dispute)

**Retaliation** - A shooting that occurs as a result of an ongoing dispute i.e. multiple disputes over time or leaving the scene of a dispute and returning later to settle the dispute; a shooting that is retribution for prior digressions; not gang related; not drug related

**Drug related** - A shooting that occurs over disputed drug territory; not gang related

**Drug related robbery** - A shooting that occurs during a drug transaction or an incident where an individual is robbed for their drugs/drug money; not gang related

**Gang related** - A shooting that occurs over gang territory or a dispute within or between gangs; not drug related

**Robbery** - A shooting that occurs during the commission of a robbery (i.e. felony murder)

**Domestic Violence** - A shooting that occurs during a dispute between family members, cohabitants, or current/ex-intimate partners

**Commission of another crime** - A shooting that occurs during the commission of another crime

**Gambling** - A shooting that occurs while individuals are gambling (i.e. while playing dice) or over gambling debts/earnings

**Negligent handling** - A shooting that occurs during the negligent operation of a dangerous weapon i.e. gun fired while handling gun

**Possibly Self-Inflicted** - A shooting that appears to be a suicide but they cannot make the determination at the time of the incident; shootings later ruled a suicide are removed from database

**Police-Related** - A shooting where a police officer shoots an individual or engages lethal use of force with a firearm

**Self-Defense** - A shooting that occurs when an individual shoots someone who is threatening to kill them or commit a crime against them

**Other** - A shooting that does not fit the definition of other categories

**Unknown** - A shooting where there is not enough information at the time of incident to code into a shooting category

## CHAPTER 8: CURRICULUM VITAE

LINDSAY R. EMER

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### **Educational Background**

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- Ph.D. Candidate – Dissertator status  
*University of Wisconsin – Milwaukee* Sept. 2013-present  
Public Health – Community and Behavioral Health Promotion Milwaukee, WI  
Joseph J. Zilber School of Public Health
- B.A. *University of Wisconsin – Madison* May 2006  
Major in International Studies Madison, WI

### **Professional Experience**

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- Medical College of Wisconsin* Aug. 2017 - present  
Program Manager Milwaukee, WI
- Provide training and technical assistance for the Enhanced SART Reviews in Sheboygan, Ashland/Bayfield, St. Croix, La Crosse, and Dane County (Department of Justice, Office of Violence Against Women grant)
  - Provide training and technical assistance for the Opioid Overdose Death Reviews in 10 jurisdictions across the state of Wisconsin (Center for Disease Control and Prevention grant)
- Aurora Health Care* Sept. 2015 – Aug. 2017  
Community Research Coordinator Milwaukee, WI
- Coordinate the Milwaukee Sexual Assault Review and the Milwaukee Domestic Violence Review in partnership with the Milwaukee Homicide Review Commission
- Milwaukee Homicide Review Commission* Apr. 2015-Sept. 2015  
Research Assistant Milwaukee, WI
- Work on team in charge of analyzing non-fatal shooting data in Milwaukee, Detroit, St. Louis and Indianapolis to improve our understanding of gun violence (National Institute of Justice Grant)
  - Participate in non-fatal shooting reviews with Milwaukee Police Department, Federal Bureau of Investigation, Bureau of Alcohol, Tobacco, Firearm and Explosions, and District Attorney's office
- UWM Center for Applied Behavioral Health Research* Mar. 2014-Aug. 2014  
Research Assistant Milwaukee, WI
- Perform data collection and analysis for Milwaukee Drug Treatment Court (SAMHSA and U.S. Department of Justice grants, PI: Michael Fendrich, Co-PI: Tom Lebel)
- UWM Joseph J. Zilber School of Public Health* Aug. 2013-Sept 2015

- Research Assistant Milwaukee, WI
- Work on team in charge of analyzing results of a NIH grant titled Pathways Linking Poverty, Food Insecurity, and HIV in Rural Malawi (R01 HD055868): A 5-year study examining the effects on HIV vulnerability of a multi-level intervention involving microfinance, sustainable agriculture training, and food security planning in rural Malawi
  - Work on team in charge of implementing a NIH grant titled Repeat STI Patients: Individually Tailored Socio-Contextual Intervention to Reduce HIV Risk (R01-MH089129) at an urban public STI clinic
- Meta House, Inc.* Apr. 2013-Aug. 2013  
 Manager of Grants and Program Administration Milwaukee, WI
- Oversee operations of Residential, Outpatient and Transitional Housing programs at a substance abuse treatment facility for women
  - Supervise Transportation, Benefits, Peer Support, Case Management, Vocational Education, Transitional Housing and Quality Assurance Department staff
  - Manage federal grant and community partnership compliance
- Meta House, Inc.* Oct. 2010-Apr. 2013  
 Program Administrator Milwaukee, WI
- Oversee operations of Residential, Outpatient and Transitional Housing programs at a substance abuse treatment facility for women
  - Supervise Transportation, Benefits, Quality Assurance, and Transitional Housing staff
- Meta House, Inc.* Aug. 2009-Oct. 2010  
 Residential Program Assistant Milwaukee, WI
- Created and implemented Residential program policies and procedures
  - Secured a partnership with a new community pharmacy; developed new medication procedures and trained all Meta House staff on new system
- AIDS Resource Center of Wisconsin* July 2008-Aug. 2009  
 Prevention Program Operations Coordinator Milwaukee, WI
- Oversaw quality assurance of Needle Exchange and Counseling and Testing Programs
  - Provided public health lectures in schools, treatment facilities, and other community organizations
  - Behavioral Modification Program (Safety Counts) Counselor to first time drug offenders referred by the District Attorney's office for monitoring during their Deferred Prosecution Agreements
- Muscular Dystrophy Association* Feb. 2008-July 2008  
 Project Coordinator (6-month position) Wauwatosa, WI
- Hired for the 2008 MDA Black-n-Blue Ball (raised over \$640,000 on May 2, 2008) and the 5-day MDA events at the Harley-Davidson 105th Anniversary in Milwaukee, WI
  - Recruited, organized and trained over 400 volunteers to work at above events
- United States Peace Corps* Sept 2006-Dec. 2007  
 Environment Volunteer Ghana, West Africa
- Volunteered with a non-governmental organization providing resource management and alternative livelihood education.

- Secured a Ghana AIDS Commission grant, which provided HIV/AIDS education to more than 1,200 Ghanaians in remote villages in the Upper West Region of Ghana
- Leadership Position: Volunteer Advisory Council Representative, Sept. 2006-Dec. 2007

## **Other Skills**

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Proficiency in Microsoft Office – Word, Excel, PowerPoint (Advanced) and Stata (Intermediate)

## **Publications**

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Mkandawire-Valhmu, L., Bauer, W.S., Stevens, P.E., Galvao, L.W., Grande, K., Yerges, A., **Emer, L.**, Mwenyekonde, T. & Weinhardt, L. (in press). Rural Malawian women’s resistance to systematic oppression, violence and abuse by their husbands. *Journal of Interpersonal Violence*.

Weinhardt LS, Galvao LW, Yan AF, Stevens PE, Mwenyekonde T, Ngui E, **Emer L**, Grande KM, Mkandawire-Valhmu L, Watkins, SC. (2016). Mixed-Method Quasi-Experimental Study of Outcomes of a Large-Scale Multilevel Economic and Food Security Intervention on HIV Vulnerability in Rural Malawi. *AIDS and Behavior*, 1-12. doi:10.1007/s10461-016-1455-1

Weinhardt LS, Galvao LW, Mwenyekonde T, Grande KM, Stevens P, Yan AF, Mkandawire-Valhmu L, Masanjala W, Kibicho J, Ngui E, **Emer LR**, Watkins, SC. (2014). Methods and protocol of a mixed method quasi-experiment to evaluate the effects of a structural economic and food security intervention on HIV vulnerability in rural Malawi: the SAGE4Health Study. *SpringerPlus*, 3:296. doi:10.1186/2193-1801-3-296

## **Conference Presentations**

**Emer L**, O’Brien M, Totoraitis M. (2016, November). *Examining the Relative Risk of Victimization by Homicide and Non-fatal Shooting Type in Milwaukee*. Oral Presentation at the American Public Health Association 144<sup>th</sup> Annual Meeting, Denver, CO.

**Emer L**, O’Brien M, Jackson-Obregon A. (2016, November). *Milwaukee Sexual Assault Review: A Public Health Approach to Sexual Assault Prevention in Milwaukee*. Poster Presentation at the American Public Health Association 144<sup>th</sup> Annual Meeting, Denver, CO.

**Emer L**, O’Brien M, Totoraitis M. (2016, May). *A Preliminary Investigation of Crime Guns in Milwaukee*. Poster presentation at the 2016 Wisconsin Public Health Association Conference, Appleton, WI.

**Emer L**, Yan AF, Stevens PE, Grande K, Galvao LW, Ngui E, Mwenyekonde T, Mkandawire-Valhmu L, Saka T, Manda C, Kosamu G, Lemke M, Depete C, Weinhardt L. (2015, November). *Can a large-scale, multilevel economic and food security structural intervention have an impact on gender dynamics in rural central Malawi?*. Oral presentation at the American Public Health Association 143rd Annual Meeting, Chicago, IL.



- Emer L**, Galvao LW, Yan AF, Grande K, Mwenyekonde T, Stevens PE, Mkandawire-Valhmu L, Saka T, Manda C, Kosamu G, Depete C, Ngui E, Lemke M, Weinhardt L. (2014, November). *Can a Combined Structural Microfinance, Food Security, and Gender Empowerment Intervention Impact STI symptoms and Diagnoses in Rural Central Malawi?*. Oral presentation at the American Public Health Association 142nd Annual Meeting, New Orleans, LA.
- Galvao LW, Grande KM, Tagger NS, Yan AF, Mwenyekonde T, Stevens PE, Mkandawire-Valhmu L, Saka T, Manda C, Kosamu G, Depete C, Ngui EM, Lemke M, **Emer L**, Weinhardt L. (2014, July). *Trends in male circumcision awareness and attitudes among adults across six traditional authorities in rural Kasungu District, Central Malawi*. Poster presented at the 20th International AIDS Conference, Melbourne, Australia.
- Galvao LW, Grande K, Yan AF, Mwenyekonde T, **Emer L**, Stevens PE, Mkandawire-Valhmu L, Saka T, Manda C, Kosamu, G, Depete C, Ngui EM, Lemke M, Weinhardt L. (2014, July). *Can a combined structural intervention (microfinance, food security and gender empowerment) impact stigma toward people living with HIV in rural Kasungu District, Central Malawi?*. Poster presented at the 20th International AIDS Conference, Melbourne, Australia.
- Horn, S and Emer, L. (2017, November). *Refusing to Accept the Status Quo: Working Together to Move the Needle on Sexual Assault*. Oral presentation presented at the End Abuse Statewide Conference, Green Bay, WI.
- O'Brien, M and Emer, L. (2017, November). *Firearm Violence Clearance Rates: Fluctuations and Implications for Future Violence*. Oral presentation presented at The American Society of Criminology Annual Meeting, Philadelphia, PA.
- Stevens PE, Yan AF, Galvao LW, Mwenyekonde T, Mkandawire-Valhmu L, Grande KM, Saka T, Manda C, Kosamu G, Depete C, Ngui EM, **Emer LR**, Weinhardt LS. (2014, July). *Improved household resilience to economic shocks: findings from SAGE4Health, a mixed-methods, quasi-experimental, non-equivalent control group effectiveness study of a combined structural intervention in rural central Malawi*. Oral presentation at the 20th International AIDS Conference, Melbourne, Australia.
- Weinhardt LS, Galvao LW, Yan AF, Stevens PE, Ngui, EM, Mwenyekonde T, **Emer L**, Grande KM, Mkandawire-Valhmu L. (2015, November). *Outcomes of a large-scale multilevel economic and food security structural intervention on HIV vulnerability in rural Malawi: The SAGE4Health Study*. Oral presentation at the American Public Health Association 143rd Annual Meeting, Chicago, IL.
- Yan A.F., Galvao, L., Grande, K., **Emer, L.**, Ngui, E., Weinhardt, LS. (2015, November). *Malnutrition in Malawian children: Trends and correlates with lack of access to microfinance, nutritional services, and sustainable agricultural techniques—Results from SAGE4Health Project*. Oral presentation at the American Public Health Association 143rd Annual Meeting, Chicago, IL.

Yan A.F., Ngui, E, Stevens PE, Galvao, L., Grande, K., **Emer, L.**, Mwenyekonde T, Weinhardt, LS. (2015, November). *Development and Validation of Household Asset-Based Socio-Economic Index: Results from SAGE4Health Study in Rural Malawi*. Poster presentation at the American Public Health Association 143rd Annual Meeting, Chicago, IL.

Yan AF, Stevens PE, Ngui E, Galvao LW, Mwenyekonde T, Mkandawire-Valhmu L, Manda C, Kosamu G, Depete C, Saka T, Grande KM, **Emer LR**, Weinhardt LS. (2014, July). *Tempering HIV Vulnerability through Improved Dietary Diversity and Intake: Results of SAGE4Health, a Mixed-methods, Quasi-experimental, Non-equivalent Control Group Effectiveness Study of a Combined Structural Intervention in Rural Central Malawi*. Poster presented at the 20<sup>th</sup> International AIDS Conference, Melbourne, Australia.