PEDIATRIC LEAD POISONING AND THE BUILT ENVIRONMENT

IN KANSAS CITY, MISSOURI 2000-2013

A DISSERTATION IN
Economics
and
Social Science

Presented to the Faculty of the University
of Missouri-Kansas City in partial fulfillment of
the requirements for the degree

DOCTOR OF PHILOSOPHY

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Kansas City, Missouri
2021

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ABSTRACT

This study examines relationships between pediatric lead poisoning and the built environment. Focusing on Kansas City, Missouri between the years 2000 and 2013 this dissertation informs policy options and identifies underexplored lines of inquiry related to pediatric lead poisoning. The dissertation extends the social surplus approach to economic modeling into a discussion of the production of pediatric lead poisoning. This dissertation grounds disparities in pediatric lead poisoning in an interdisciplinary context integrating biology, health effects, exposure pathways, social history, and economic theory into a research agenda. The original contribution of this dissertation is comprised of three interconnected parts: 1) the identification and assembly of an expansive data library for observing lead in the built environment, 2) the development of a warranted geocoding process to match 14 years of pediatric blood lead data to a parcel-level geography which is inconsistent from year to year, 3) and exploratory empirical investigation of the assembled and associated geocoded data. Using a cross section of observational data, I estimate a series of ordinary least squares multiple regression models relating child, housing, and proximity focused explanatory variables to

changes in the blood lead levels in children. The dissertation concludes with a consideration of public policies aimed at preventing pediatric lead poisoning and possible extensions of the assembled data.

APPROVAL PAGE

The faculty listed below, appointed by the Dean of the School of Graduate Studies have examined a dissertation titled "Childhood Lead Poisoning and The Built Environment in Kansas City, Missouri 2000-2013," presented by Neal J. Wilson, a candidate for the Doctor of Philosophy degree and certify that in their opinion it is worthy of acceptance.

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ABBREVIATIONS

AHR	Architectural History Research
bce	Before the Common Era
B_{lead}	Lead-loaded Built Environment
BPb	Blood lead level
B_{safe}	Lead-safe Built Environment
BSN	Bachelor of Science in Nursing
C	Child Focused Variables
CDC	Center for Disease Control
CEI	Center for Economic Information
CMH	Children's Mercy Hospital
CORE	Community Organized Resource Exchange
CORE	Community-Organized Resource Exchange
CPSC	Consumer Product Safety Commission
EBL	Elevated Blood Lead
EPA	Environmental Protection Agency
F	Family
FPL	Federal Poverty Line
ft	Foot
G	Geographic Proximity Variables
GIS	Geographic Information System
GM	Geometric Mean
H	Housing Focused Variables
HIPAA	Health Insurance Portability and Accountability Act
HUD	US Department of Housing and Urban Development
i.e	*Id Est*, that is
KC-HEART	Kansas City-Home Environmental Assessment Research Taskforce
KCHD Pb	Complete BPb dataset from KCMO Health
KCMO Health	Kansas City, Missouri Health Department
L	Labor
LIA	Lead Industries Association
LILZRO	International Lead Zinc Research Organization
llc	Limited Liability Corporation
LoB	Limit of Blank
LoD	Limit of Detection
m	Meter
Abbreviation	Abbreviated

MAGI	Modified Adjusted Gross Income
MARC	Mid-America Regional Council
MAUP	Modifiable Arial Unit Problem
n.b.	*Note Bene*, note well
NHANES	National Health and Nutrition Examination Survey
NHCS	Neighborhood Housing Conditions Survey
NHCS Pb	NHCS subsample of BPb data
NY	New York
OLS	Ordinary Least Squares
OSHA	Occupational Safety Hazard Administration
P	Production Process
Pb	Lead
ppm	Parts Per Million
r_n	Regulatory Regime
RN	Registered Nurse
sd	Standard Deviation
SPb	Soil Lead Level
SSO	Systematic Social Observation
t	Technology
TEL	Tetraethyl Lead
U.S.	United States
UGCoP	Uncertain Geographic Context Problem
UMKC	University of Missouri-Kansas City
µg/dL	Microgram per Deciliter
µgPb/m2/hr	Micrograms of Lead per Square Meter per Hour

ACKNOWLEDGEMENTS

All scholarly work is collaborative, this dissertation no less. I would be mistaken not to mention the encouragement and support of Jordan Ayala and Natalie Kane. Their work set the ethical and scholarly standards I hope to live up to. I must thank Ben Wilson for inviting me into the world of health disparities research. My research would not exist without the Amy Roberts at the Kansas City Health Department. Similarly, my work would not exist without the BPb tests of children suffering from lead poisoning, the goal of this dissertation is to make that sort of collaboration be unnecessary in the future.

In the department of Economics, I must thank the administrators who kept that ship from foundering despite long odds, Deborah Foster, Katie Taylor, Kayla Weru, Jill Folsom. I want to acknowledge Fred Lee and John Henry, each of whom motivated my interest in economics (albeit in very different ways) and neither of whom were here to see me finish this project. I would also like to thank Stephanie Kelton for taking an interest in a non-traditional undergraduate and pointing me on the way.

I want to mention my fellow students by name; Rafed, Usha, Christian, Ruchira, Jonathan, Brian, John, Aqdas, Sudeep, Up, Hussain, Avi, Stephany, Christian (the other one), Muhammed, Ignacio, Brian (the other one), Nicola, Kyle, Ely, Matt, Mitch, Drew, Julia, Alex, Vincent, Sam, George, Laura. Any omissions are accidental. I gained inestimably from learning at your shoulder and I am in your debt.

Throughout this process my family Erin, Kyle, Mike and Charlotte, Jim and Sally, Josh, Sarah, Charlie and Sue, Julie and Tyrel, Sister Teresa Marie) offered their gentle encouragement, support and prayers, listened to me talk nonsensically bashfully and

confidently about what I was learning, and never made any bad economist jokes. I would trade this dissertation to spend another day with Sally and Jim.

Most of all I want to acknowledge Lacey who was not yet my wife when I began writing this book. Her casual grace and literal support make everything I do possible.

For my father who warned me about this.

CHAPTER 1

INTRODUCTION

At first glance childhood lead poisoning might appear to be a solved problem. Among the 1976-1980 cohort the mean blood lead level among children in the U.S. was 12.8µg/dL. In the 2007-2010 cohort, the mean level among that same population was 1.3µg/dL (McClure, Niles, and Kaufman 2016). The principal sources of pediatric lead exposure—lead paint and Tetraethyl Leaded Gasoline—have been banned at the federal level since 1977 and 1986 respectively. There is now an abundance of 'information campaigns' funded at the federal, state, and local levels to educate at risk populations to the dangers of residual lead in the urban environment. Yet the problem of pediatric lead poisoning persists. The more rigorously it is studied the more dangerous it appears, in more ways and at lower levels of exposure.

The facts about lead poisoning as we now know them are grim. The epidemiological literature shows a downward trend in the acceptable level of blood lead such that the contemporary medical consensus admits no safe level of exposure (Lanphear 2017). If lead exposure occurs before 72 months it interferes with the physical development of the brain and central nervous system causing permanent damage (Grandjean and Landrigan 2014). Researchers have identified the latent sequelae of lead exposure persisting decades after initial insult (Reuben et al. 2017). Problems which descend from childhood exposure persist into adulthood. Children of the 1970's, now adults in their 40's, are dealing with the aftereffects of childhood exposure while society at large has had its trajectory twisted by this base element. Still the sources of this problem persist. Lead from paint and gasoline remains bioavailable despite being deposited in the environment a generation ago and the point source emission of

lead is ongoing. In 2020 there remain over 20 million U.S. homes built before 1978 and thus likely to be contaminated with lead paint. Each time one of these homes is remodeled or torn down is potentially a toxic event enabling the effects of environmental lead will to be felt well into the future.

The geography of lead in the built environment complicates this story further. Lead hazards are not spread equally across the urban environment. The communities most affected are the least advantaged in our society. It is the neighborhoods of racial and ethnic minorities as well as the neighborhoods of the poor that are most effected by the presence and legacy of toxic lead. The distribution of lead in the built environment marks a pattern of uneven development in the urban landscape. Sites of lead contamination impair personal development, compound social problems, and tax the best intentions of communities to accumulate wealth and sustain well-being. Helping to understand and thus ameliorate these base facts is the *why* of this dissertation. The work described below has the potential to guide policy, intervention, remediation, regulation and inform the behaviors of families themselves towards better outcomes and hopefully, ultimately, lead free lives.

My research focuses on a particular place and time: the Missouri side of the greater Kansas City, MO metropolitan area between the years 2000 and 2013. This study uses a geographic information system (GIS) to focus on the environment in which children live. Particular attention is placed on observations at the parcel level, the condition of a child's residence and those around it (age and condition of the structure), the volume of demolition activity nearby, an approximation of the lead burden in the surrounding soil (proximity to older roadways and gas stations). These data are assembled into a OLS model where fine-grained observations of the urban environment are associated with individual geolocated pediatric

blood lead levels. All of this to answer my research question; do observations of the built environment, specifically the exterior condition of housing, aid in seeing the problem of pediatric lead poisoning more clearly?

The original contribution of my dissertation is a concatenation of three related activities which make my work of seeing possible. First there is the process of assembling a robust micro-level data set. Second, developing a method for associating this data in a consistent, repeatable, and useful way. Finally, the use of this associated data in such a way that we better understand the pathways of childhood lead exposure. This dissertation assembles a new set of spatialized data, describes a new method for processing the data (the spatialization process), and marks out a trajectory for researching on the basis of such data. I find that looking at the problem of pediatric lead poisoning in a comprehensive manner via OLS produces statistically significant relationships between demolition activity and increases in blood lead levels, and that the interaction of era of home construction and exterior housing conditions provides more robust and statistically significant information than either observation provides on its own. These contributions suggest directions for future research and regulatory scrutiny.

Assembling personal information such as medical records about individuals is difficult. For institutional and empirical reasons the large-scale analysis of chronic disease is seldom done at the level of the housing parcel. The HIPAA privacy rule, designed to safeguard people and their data from being exploited or exposed to undue risk, makes the sharing of individual health records (and particularly the records of children) cumbersome. Obverse to the institutional hurdles are difficulties in obtaining data about the lived environment at the level in which it is lived. Observations of housing conditions that are robust, consistent and extensive enough to be useful in statistical health research generally do not exist. Generating and

maintaining data about the built environment, which by definition exists at the parcel level, is time intensive and expensive. The work of this dissertation is produced in consort with a research consortium, the Kansas City Health CORE, which was designed as a data repository to be used in health disparities research. Demonstrating that these hurdles can be overcome while maintaining the anonymity of the research subjects is a critical outcome of the dissertation process.

While gathering raw data is a first step, it does not guarantee that independent data streams can be usefully merged. The process of spatializing and associating data in a consistent and warranted way is a challenge of its own. Such is the second contribution of the dissertation, the linking of disparate data sets hangs on a multi-stage iterative method designed to match health encounters to a shifting parcel geography using GIS. Despite being designed for use within GIS this multi-stage match method provides value outside the specific parameters of the computing software. My iterative geocoding method is designed to summarize health data at various levels of geography and at different moments in the match process. These various summary levels give researchers investigative flexibility and insight as to if and where bias is introduced in the assembly process. Much contemporary scientific inquiry is collaborative, and the data assembly and match processes are no exception, they were developed in association with fellow researchers Ben Wilson, Natalie Kane, and Jordan Ayala.

A preliminary quantitative analysis of the associated micro-level data is the third contribution of the dissertation. Statistical work on micro level health data, for reasons discussed above, are exploratory. My results open the door to additional statistical investigations. The work at hand involves the use of statistical inference to support the larger

discussion of public policy (involving specifically demolition practices and the upkeep of property) and to refine a trajectory towards a lead-free population.

The task of understanding and describing the conditions, mechanisms, and perpetuation of racial and economic stratification vis a vis childhood blood lead dovetails with the agenda of critical social science. UMKC demands its doctoral dissertations be interdisciplinary and this dissertation meets that demand head on. I draw on GIS, epidemiology, and geography to track chronic disease and risk factors in the urban environment. I draw on medical literature to understand the immediate and long-term effects of pediatric lead exposure. These approaches and their methods are absolutely necessary for the dissertation project to hold together and describe the world in an accurate and useful manner. Questions may come from some quarters as to how this research fits within the coordinating discipline of Economics. Where is the marginal analysis of maximizing agents? What of markets and prices? This work eschews all that to focus instead on the social provisioning process; an exposition of the production of lead poisoned children, a historical process of uneven development perpetuating inequality and masquerading as unintended consequences, the legacy of lead poisoning production of economic, medical, and social outcomes.

Ultimately this dissertation is about seeing the world we have produced. It is about using statistical inference to understand the world better than we already do and using GIS to recognize patterns suggested by the literature. A difficulty is always present when translating from what we can see in one way, say statistically, to what we can see in another way, say via an historical narrative. Eliding this difficulty is part of the work of this dissertation. In a sense, this is a pessimistic work of scholarship. If we skim the history of lead poisoning it is immediately clear that the toxicity of lead has long been known but efforts to remove its risks,

to stop its use, and decontaminate the environment are underfunded or outright stymied—rather than direct action, another study (like this one perhaps) is proposed. Indeed, more lead was put into use in 2019 than ever before. But there is an optimistic vision here as well: data driven policy and a world without childhood lead poisoning.

The structure of this dissertation is as follows. Chapter 2 introduces theoretical and interdisciplinary grounds. Chapter 3 describes the biological nature of lead's toxicity and an overview of its immediate and long-term effects. I spend chapter 4 relating a social history of lead—its use in industrial applications to construct the modern urban environment, the gradual awareness of lead as a public health hazard, and the steps that were taken to address lead in the built environment. Chapter 5 explores the epidemiology of lead poisoning and the GIS methods employed in that work. Chapter 6 discusses the data I have collected as well as the geocoding process used to spatialize the data, and the process of assembling the diverse data sources. Finally, chapter 7 is an analytical chapter which puts the collected information to work in an exploratory statistical analysis. I find that including details about the built environment, particularly the interaction of exterior paint conditions and the era of housing construction, add statistically significant explanatory power to the analyses which rely on just one or the other of these explanations. My findings can be used to design interventions to focus in on housing likely to need lead-safe interventions and thus spare the kids most at risk from housing-born exposure to lead poisoning.

CHAPTER 2

CHILDHOOD LEAD POISONING IN AN ECONOMIC CONTEXT

This chapter places pediatric lead poisoning into an economic context via a social surplus framework. I argue throughout my dissertation that lead poisoning is a health outcome produced largely by previous production decisions regarding the construction of the built environment. Pediatric lead poisoning in turn produces a range of bad health outcomes in those subject to it and social effects in the communities where pediatric lead poisoning has been preponderant. The distribution of these outcomes is largely determined by overlap of the previous production of the built environment and residential segregation by race and class.[1] As such I am talking about a social provisioning process, the production and distribution of the social net product, and the implications of this distribution (Heilbroner 1988; Power 2004). Thus, pediatric lead poisoning is brought into economics. Aspects of this chapter highlight the possibilities of the social surplus approach to inform questions of public policy, environmental justice, and heterodox economic theory.

The substantive relationships between the built environment and health, specifically between housing and lead poisoning underly my specific research question: "Do observations of the built environment, specifically exterior housing conditions, add to our understanding of blood lead levels in children?" The purpose and the method of this 'seeing' are critical methodological issues that will also be addressed in this chapter. Until recently, lead poisoning

[1] I use the word 'largely' purposefully. I do not maintain the built environment is the only source of blood lead poisoning, the private consumption of lead loaded consumer goods is an important source of lead exposure, but it is not the general cause of pediatric lead exposure. Proof is required for these statements and chapters 3-5 of this dissertation are focused on empirically demonstrating this basic claim about the built environment to be true.

in children was not discussed directly by economists. None-the-less, if one looks closely, childhood lead poisoning can be read into economic literature dating back at least to the early 1960's. Considering the general case of exposure that comes with the 'age of lead thesis' a reconsideration of empirical human capital studies could be recommended without appeal to any heterodox literature. [2] One might use the history of lead poisoning to appeal via the Coase Theorem (and the neoclassical approach) for government sponsored regulation and remediation, making a nuanced point about externalities and information (Coase 1960). Contemporary neoclassical considerations of lead poisoning generally, but not entirely, eschew such revisionism focusing instead on parental behaviors in markets for health services. I review this literature not with the intention of debunking but rather complicating and interrogating the standard approach. Then I develop a heterodox approach to lead poisoning with a focus on substantive relationships and cumulative causation informing the distribution of the social surplus. The pecuniary implications of lead poisoning are substantial but cannot be well understood without a discussion of animating ethical frameworks which underly social action. This dissertation investigates childhood lead poisoning in an interdisciplinary context as well, finding critical social science, epidemiology, and urban planning necessary to develop an expansive understanding of the implications of this poisoning in terms of environmental racism, the precautionary principle, and the effects of power in physical space.

[2] The age of lead theory insists that the geologic age defined by the unintentional human alteration of the global environment. sometimes referred to as the Anthropocene, is as typified by unprecedented levels of lead as it is by carbon dioxide emissions. If, as is likely, lead poisoning had an effect on human capability and behavior, and these effects are not accounted for in human capital studies, a general reconsideration of their findings is warranted.

A Reading of Neoclassical Health Literature

Neoclassical economics begins its formal engagement with health via Arrow's "Uncertainty and the Welfare Economics of Medical Care" (Arrow 1963). Arrow notes however that his focus and ours appear to be different, "the subject [of this article] is the medical-care industry, not *health.* The causal factors in health are many, and the provision of medical care is only one" (Arrow 1963, 941). An acknowledgement of a connection between the built environment and health outcomes is present in Arrow's article but it goes unexplored for decades. A child's home, or more precisely, the discrete characteristics of the family home is a causal factor in health; it is common sense and long established in the medical literature (Krieger and Higgins 2002). Social workers since the 19th century have been focused on housing as a type of medical care (Rosen 1958). Recently doctors have picked up the banner. The American Academy of Pediatrics has recommended the expansion, in terms of fiscal and legal support, for interventions in housing conditions (i.e., fixing a leaking roof to stop the growth of black mold, or the removal of lead paint hazards) as a type of preventative medicine (Council on Environmental Health 2016). The National Academies of Science, Engineering and Medicine recommend that health systems "medicalize" social care and address social determinants of health like substandard housing directly (National Academies of Sciences, Engineering, and Medicine 2019). That is, associations of medical professionals suggest eliminating the analytical distinction which Arrow identifies between causes of ill health and the medical-care industry, at least as far as lead poisoning and other chronic disease is concerned. Such an expansive emphasis on preventive medical care was not common at the time Arrow's seminal paper was published.

With regard to medical care, Arrow writes a great deal about the importance of information, how it is usually not available in the way that it is needed, and that asymmetries are doubled with regard to the provision of services.

> I propose here the view that, when the market fails to achieve an optimal state, society will, to some extent at least, recognize the gap, and non-market social institutions will arise attempting to bridge it. Certainly, this process is not necessarily conscious; nor is it uniformly successful in approaching more closely to optimality when the entire range of consequences is considered. …But it is contended here that the special structural characteristics of the medical-care market are largely attempts to overcome the lack of optimality due to the non-marketability of the bearing of suitable risks and the imperfect marketability of information. (Arrow 1963, 947)

He is describing criteria for the emergence of a non-market solution to the lack of information regarding who will get sick with what, when.

Analogizing Arrow's argument with the case of lead poisoning is appealing. We could see his statement, "when the market fails ... society will ... recognize the gap, and non-market institutions will arise attempting to bridge it" as a prophecy from 1963 of a public health regime that would not appear for three more decades. However, considering the longstanding awareness of the problem, the lingering effects of lead poisoning, the persistent resistance to universal lead testing, the chronic underfunding of abatement programs, and the industrial retrenchment against the regulatory institutions of public health, Arrow's model is not well suited to describe the political economy of lead poisoning.

There is a finer methodological question which pediatric lead poisoning brings forward. To fully understand the phenomena of lead poisoning a *de facto* analytical focus on the individual is not appropriate. The phenomena under study should determine the approach used rather than an a priori individual or group wise approach. Although lead poisoning is measured in the individual it is produced through a social process. In my analysis of lead poisoning I

follow Dewey's methodological insight from *Reconstruction in Philosophy,* "[w]hat is needed is specific inquiries into a multitude of specific structures and interactions" (Dewey 2004, 114), rather than the general structure of all interactions. Individual agency is important to respect but it is not methodologically germane to this problem.

A second foundational article in neoclassical health economics is Grossman's "On the Concept of Health Capital and the Demand for Health" (Grossman 1972). In Grossman's article "good health" is modeled as an economic good that individuals produce in accordance with a private utility function in which health capital is distinct from human capital. Health capital, being partly endogenous and partly a function of education in his model, determines the total amount of time once can spend producing money earnings and utility.

> Within the new framework for examining consumer behavior, it is assumed that individuals *inherit an initial stock of health* that depreciates over time – at an increasing rate, at least after some stage in the life cycle – and can be increased by investment. ... Gross investments in health capital are produced by household production functions whose direct inputs include the own time of the consumer and market goods such as medical care, diet, exercise, recreation, *and housing*. The production function also depends on certain 'environmental variables,' … It should be realized that in this model the level of health of an individual is *not* exogenous but depends, at least in part, on the resources allocated to its production. (Grossman 1972, 225)

With the mention of housing and environmental variables a generous reading sees much that is appealing in this description of health capital.[3] There are at least three aspects of Grossman's model agitated by the problems of lead poisoning: the initial stock of health idea, the importance of housing, and the endogeneity of personal health.

The first of these is the idea of an initial stock of health that depreciates over time. What Grossman has in mind is a genetic inheritance subject to the natural process of ageing which

[3] Critiques of neoclassical capital theory are well developed elsewhere (See for example Hodgson 2014) . As my analysis is focused on other issues, the guiding spirit of generosity allows the discussion of these capitals for the purpose of finding value in the work of others.

can be arrested through the active investment is one's health. Lead poisoning does not quite fit into this model because a child can experience lead poisoning in utero. A mother's lifetime burden of lead exposure can be pulled from reservoirs in her body by the experience of pregnancy and subsequently passed to the child before birth. Grossman's model works better for the more general case where a child's health is impaired after birth through exposure to a lead loaded built environment. The active investment in health of Grossman's model is inverted in reality, passive disinvestment in health is a better description of the process of lead poisoning. The importance of housing complicates this point. It is conceivable that well informed parents could avoid disinvestment in their children through the selection of lead safe housing. However, such outcomes assume a degree of choice and the financial means to act upon 'preferences' that does not reflect the experience of race or class in the USA (Swope and Hernández 2019). The arguments around the social determinants of health turn on the question of endogeneity. A household production function for health retains its internal logic where health production is preponderantly endogenous but an examination of the specific structures of lead poisoning point to factors outside of the household. The variation in blood lead levels between urban and suburban children is poorly explained by endogenous variations at the household level (H. L. Needleman, Tuncay, and Shapiro 1972; Griffith et al. 1998; Moody et al. 2016). The social determinants of health literature illustrate the importance of factors like housing and the built environment in the production of health outcomes, such factors are exogenous to the household but typify experience by socioeconomic group (Braveman and Gottlieb 2014; Swope and Hernández 2019). The neoclassical literature, with its emphasis on a choice analytic framework, largely misses the importance of exogenous factors in pediatric lead poisoning.

Central to both Grossman and Arrow's analyses is the importance of information in the process of health-related decision making. Information about lead's toxicity and its preponderance in the built environment is essential for families to make lead safe choices. Making a good lead safe choice is a matter of luck without access to the relevant information.[4] It is thus significant that Grossman's article was published in 1972, the same year as the first modern medical articles on lead poisoning in children are being published. I have no evidence that Grossman was aware of this literature. When he writes about the importance of 'the level of education of the producer' he is arguing about the ability of the individual to make use of available information, attributing responsibility on the poisoned for their health outcomes, albeit with the wrinkle of education. That perspective is in accord with the mid-century statements of the lead paint industry which argued that lead poisoned children we the result of a mixture of bad patenting and the behavior of the child (pica, or the compulsive eating of items like paint flakes that have no nutritional value). Such a line of argumentation founders on the test of reality; it is a young child's lead exposure that has the most deleterious effects, yet a child cannot reasonably be conceived as a purposeful agent in this context. Through the normal process of human development, crawling, hand to mouth behavior, and the passive inhalation of lead dust a lead loaded environment is internalized by any child. Shifting responsibility to the parents does not suffice if the relevant information is unavailable, if some the mechanism impairs the ability to make the relevant choice, or the time frame for choice

[4] Prioritizing individual agency in health decision making necessitates making this knowledge widely available, thus from the 1990's disclosure of known lead hazards in housing has been required by law (T. Dignam et al. 2019).

and exposure are misaligned. Each of these objections applies in the case of lead poisoning.[5]

The choice analytic framework Grossman used is reflected in more recent work by Agee and Crocker. Those authors brough the choice analytic framework and health as a function of family investment to issues associated with lead poisoning (Agee and Crocker 1996a; 1996b). There-in the authors model the value of a lead free child to their parents "by the household's willingness to pay for it ... [derived from] actions taken by parents to avoid lead-induced, chronic health risk in their children" (Agee and Crocker 1996a, 680 - 681). The assumptions that the model relies upon are telling; that all the families have insurance and there are zero out of pocket costs for the therapy[6]; that the value of the time of the parents can be modeled as the inverse of their level of education[7]; that there is a choice that parents can make between reduced lead levels in children and what-ever the parents would have been doing anyway. The parental action the authors focus on is chelation[8], a procedure aimed at reducing acute injury from extreme intoxication. Their text implies that children who receive chelation therapy make a complete recovery from lead poisoning contradicting well established research, for example Byres and Lord (1943). Still, Agee and Crocker insist that "[marginal willingness

[5] At the time Grossman was writing accurate information about the risks of lead poisoning was unavailable. Chapter four chronicles the ongoing revision of our understanding of lead's risks to the point where it is now recognized there is no safe level of pediatric lead exposure. The reality of segregation by race and class and the way that segregation is kept in place through de facto processes underscores the inadequacy of a purely choice analytic framework for the constitution of health where social determinants are relevant. The ability of lead to be transferred from the mother to the child in utero, lead a mother may have been exposed to as a child and stored in the intervening years in her trabecular bone marrow, illustrates a temporal discontinuity with implications for this analysis as well.

[6] We know from the epidemiology literature that lead poisoning is preponderant among the uninsured.

[7] While discretion over one's time is inversely related to level of education.

[8] The authors focus on chelation because it does not produce any other utility in the way that some sort of preventative activity like painting the house might.

to pay] can be expressed as the marginal rate of substitution between the child's lead burden and medical treatment [chelation therapy]" (Agee and Crocker 1996a, 40) and thus an equilibrium is found where the value of a child's lead burden is expressed via the monetary value of the time a parent is willing to give up (either work or leisure) through the experience of chelating their lead poisoned child. However, there is no easy trade off with regard to chelation therapy and lead burden.[9] Chelation is only for acutely lead poisoned; it is an emergency procedure not relevant for the estimation across the margin the authors suggest. Theirs is the sort of claim that interdisciplinary research is designed to prevent.

The importance of an interdisciplinary approach is reflected in "Lead Paint, Toxic Torts, and the Housing Stock: A case study in risk assessment" (R. J. Epstein 1998) which takes a critical approach to some of the epidemiological and benefit-cost literature and criticizes laws which create incentives for seeking legal redress for lead poisoning through litigation.[10] The Epstein article emphasizes the importance of a close reading of the literature upon which epidemiological and cost-benefit decisions are made. Epstein finds an ambiguous result in one seminal paper linking academic performance and blood lead levels and sloppy mathematics in an equally seminal cost benefit report. Epstein's focus turns to the legal structures which emerged to deal with the lead poisoned cities and the effect on real estate markets. Using his own cost benefit analysis he recommends against widespread lead-safe housing remediations

[9] Or any piecemeal strategy. There is a recognition that the reactive methods "such as household education and dust-control measures are not considered effective, especially when not applied in conjunction with other practices that address other sources of lead" (Yeoh et al 2014, Kennedy et al 2016). Furthermore, examining the data Agee and Crocker use to validate their model, we see the mean lead burden of the test population was 15ppm, implying a poisoned population without regard to chelation activity.

[10] Several other articles approach the legal and regulatory incentives associated with legal frameworks designed to limit lead poisoning, Gilligan and Ford (1987) "Investor response to lead Paint Abatement Laws" and Miceli, Panack and Sirmans (1996) "An Economic Analysis of Lead Paint Laws".

and that governments should "limit the incentives for lead litigation against property owners and should avoid mandating large-scale public and private spending programs for lead paint abatement" (R. J. Epstein 1998, 128). In the years since his article was published a king tide of evidence has come in overwhelming doubts about the negative effects of lead at low blood concentration and reinforcing lead's negative impact on neurological function and academic achievement.[11] Aizer and Currie (Aizer et al. 2016) investigate the finding which Epstein rejects, that lead has a substantial effect on academic performance and thus lifetime earnings. Aizer and Currie find a robust connection between increased blood lead levels and poor academic performance (measured in terms of suspension and detention), more critically their analysis finds that traditions OLS methods "considerably underestimates the negative effects of lead" (Aizer et al. 2016, 3).

The Journal of Urban Economics article "Primary prevention and health outcomes: Treatment of residential lead-based paint hazards and the prevalence of childhood lead poisoning" (Jones 2012) looks at remediation in the city of Chicago to examine the contention that focusing on lead remediation activity is not cost effective at the society wide level. Jones finds remediation activity to be highly effective (2.5 cases avoided for every housing unit remediated) and cost effective as well (lower bound estimates of benefits are 2 - 20 times the estimated costs of remediation). The relevant neoclassical trade-off is not simply the cost of remediation but also the value of other returns which could be had with the money dedicated

[11] See chapters 3 and 4 of this dissertation for a lengthy treatment of that literature. Ironically, Epstein's article's critical reading of the epidemiology simultaneously supports one of the major themes of this dissertation, that the living and working conditions in which people are situated has important and measurable effects on their health. Epstein cites the social determinants of health (though he does not use that term) as a relevant factor, "the study did not investigate whether characteristics of the city of residence made any difference [in school performance]" (R. J. Epstein 1998, 130).

to the remediation activity. Jones' article shows that lead hazard remediation activity easily meets this test. An unstated question suggested by this article is why is an economically efficacious health program underfunded? Ethics and economics are not easily separated.

Zivin and Neidell's 2013 article “Environment, Health and Human Capital” reviews the contemporary attitude of neoclassical health economics towards the environment. The authors rearticulate the centrality of the human capital approach to neoclassical health economics stressing the importance of individual optimizing and avoidance behavior for understanding “the relationship between the environment and individual well-being" (Zivin and Neidell 2013, 689). The authors note that environmental hazards can have a negative effect on human capital formation through direct and indirect channels in similar ways to those discussed in Reyes and Currie (2016) and that “[e]stimation of these effects represents an exciting frontier of economic research..” (Zivin and Neidell 2013, 694). Recognizing the importance of substantive relationships generating lead poisoning the authors emphasize the importance of “environmental protection as a national investment, in addition to a consumption good” (Zivin and Neidell 2013, 691). Elsewhere in their article Zivin and Neidell return to the importance of endogeneity, their narrative is based in avoidance or optimizing behavior, when considering the effects of the environment on health outcomes. Quoting their relevant passage, “The key point is that optimizing individuals make trade-offs along multiple dimensions based on the intensity of their preferences for each local attribute, which implies that the characteristics of the neighborhood in which individuals live, including pollution levels, are endogenously determined” (Zivin and Neidell 2013, 702). They similarly note that much epidemiological work does not take endogeneity seriously, then use this as the basis for an appreciation of natural experiments which in their conception, offer a way past the endogenous aporia allowing

researchers to finally observe unbiased choice behavior. Central to a generous reading of their essay is the idea that residential sorting is based on a bundle of neighborhood characteristics, environmental conditions being one among many. They propose a thought experiment where non-asthmatics choose to reside in neighborhoods with significant air pollution and vice versa. Without a recognition of this possibility, it is theoretically possible to observe pollution correlated with better health, or more likely the correlation between pollution and ill-health to be underestimated due to the avoidance behavior of those with the means to choose. The ambiguity of the endogeneity complaint is important but largely irrelevant to the particular facts of pediatric lead poisoning (no child is immune) and the reality of residential segregation restricting options for those with means to choose.

Although the authors have all the pieces to make the argument that environmental effects bare on previous human capital estimations, they stop short. They note that lead has developmental effects, that "pollution should be considered an important factor of production" and that a toxic environment could be "a new form of poverty trap", and suggesting that the arrow of causation of the environmental Kuznets curve could run ‘backwards’ (Zivin and Neidell 2013, 691). From here it is a logical step to call for the re-evaluation of empirical human capital estimates that do not take environmental conditions, particularly lead poisoning considering its ubiquity, into consideration as a confounding factor. Some contemporary economists have taken this argument to similar places, suggesting that environmental policy and regulation should be thought of as social policy (Reyes 2015). Important work remains to be done along these lines, for instance tracing the economic benefits of The Clean Air Act as health and social policy. Implied in these analyses is the work of urban planners to regulate environmental pollution and reconstruct a cleaner environment is justified along vectors for

which individual choice does not apply.

Moore's article "Why Allow Planners to Do What They Do? A Justification from Economic Theory" discusses impediments to the impulses of planners. Here he takes the impulse to regulation to task with reference to the analytical frameworks of the cold war neoclassical economists like Becker, Buchanan, and Coase (Moore 1978). Moore emphasizes that planners (and regulators) must "explicate a comprehensive theoretical framework which meets these criticisms [those of the economists] on their own ground" (Moore 1978, 390) for their work to be accepted as a legitimate use of public power. His position has clear implications for the regulation of lead in the environment. The social history of lead poisoning explicates the existence of a problem—a market failure—a negative externality in the form of widespread urban lead contamination. Moore's article offers the rejoinder that, "the existence of market failure is *not* a sufficient condition for government intervention" (Moore 1978, 393). When searching for a basis in neoclassical economic for regulating the lead industry, ethical commitments are methodologically excluded (Buchanan and Samuels 1975). Moore's article is a fascinating study from a history of science standpoint. The methodologist Larry Laudon describes the evolution of disciplinary paradigms as essentially the process of developing a research tradition such that other rivalrous research traditions are drawn to consider and contend with adversarial argument and analytical framework (Laudan 1981). One could read Moore's article from 1978 as emblematic of the ascendency of the neoliberal paradigm, spreading in a process of economic imperialism from the social sciences into the market based governance of contemporary life (Tullock 1972). However, justifying the regulation of lead need-not resort to meta-theoretical complaints regarding the axiomatic assumptions of competing paradigms. Urban lead toxicity easily qualifies within the neoclassical view in need

of state regulation and intervention—Zivin and Neidell refer to it as such. More critically, the social history of lead illustrates the standard practice of the Lead Industry was to restrict the availability of information available to the general public, while the power of the automobile industry colluded to perpetuate the use of leaded gasoline.

A paucity of information justifying state action is one of the justifications for state action. The importance of information to economic analysis is a red thread stitched through modern economics (Keynes 1964; Shackle 1967; Minsky 1986). A strain of thought, associated with Hayek, argues that market prices function to overcome information limitations. There are however short-comings to Hayek's focus on prices for the transmission of information in society as there are other forms of information that are actively used by the business enterprise in decision making that supersede price in the decision making process (Hayek 1945; Melody 1987). There is oblique reference to problems relevant to lead poisoning in Cowen's focus on uncertainty with regard to information as having important implications for the administration of public goods and market failures (Cowen 1999). These limitations come into focus when we consider the methods for computing the economic value of a statistical life depend on precisely quantified risks (Viscusi 2005) and legal frameworks authorizing environmental regulation generally require the computation of a cost-benefit trade off (Viscusi 1996). If the relevant costs are unknown or misunderstood and risks are understated at the time decisions are made regarding the use or legality of a product, as has been the case with lead, then neoclassical economics has little to add to the discussion. Yet economic regulations are generated in this circumstance all the time, to understand these regulations and their limits requires the introduction of an ethical dimension into the analysis.

The above consideration of neoclassical health economics in light of pediatric lead

poisoning highlights several important issues: health outcomes as something produced by the built environment, the importance of housing as health care and the social determinants of health more generally and using a methodological approach which matches the problem (rather than conforming the problem to an approach). Such issues reinforce the importance of interdisciplinary research as a necessary component of economic analysis. Part of the issue remaining is seeing lead poisoning into the relevant framework, a second part is a justification (economic *and* ethical) for state action to address the issue, and finally an overview of the rest of this analysis.

Towards Another Health Economics

In a paper called "What Should Economists Do" Buchanan writes that the task of the economist is to 'apply core theory to the problems of society'. Buchanan argues along with a tradition of conservative economists like Friedman and Knight, that the analysis of individuals making choices regarding social allocation is the essence core theory (Buchanan 1987). Buchanan acknowledges that what constitutes core theory is contested ground and such is the attitude of this dissertation. The first half of this chapter interrogated the way core neoclassical theory has been applied to the social problem of lead poisoning. In the second half of this chapter my analysis maintains a focus on the individual (it is individuals that become lead poisoned after all) but takes a different approach to the process of allocation within society that mostly elides the choice analytic framework. Furthermore, in contrast to the neoclassical approach, I discuss an ethics which animates my analysis and justifies state action to address pediatric lead poisoning.

Core theory does not begin in *a priori* theoretical or ethical space but through an

encounter with the real world and the investigation of substantive relationships. Modeling decisions and an analytical approach follow from the substantive relationships. As my focus is on lead poisoning it is germane to begin with the relevant model of disease. Sylvia Nobel Tesh describes three paradigmatic frameworks used to conceptualize the causes of disease; germ theory, lifestyle theory, and environmental theory (Tesh 1988). The relevant distinction between these three theories is their causal structures and the relevance of government action to alleviate the disease. Germ theory is not relevant to the case of lead poisoning. Lifestyle theory, with its reduction to individual culpability for the incidence of disease, dovetails with neoclassical analysis but not with the substantive reality of the problem. Environmental theory, which understands health as resulting from production and consumption decisions in which the individual participates but does not delimit, allows a conceptual space flexible enough for real complexity. Writing on this topic in the *Journal of Economic Issues* Haggerty and Johnson summarize this point as follows, "Viewing health as a social issue forces society to define health, examine its underlying causes, and explore the social benefits of preventive medicine. Health as a social phenomenon requires the examination of the means of production and other institutional arrangements in society to see how they interact with and impact our lives" (Haggerty and Johnson 1996, 530).

Approaching core economic theory via the political scientist Sylvia Tesh underscores the importance of an interdisciplinary knowledge base. Throughout this chapter I make reference to the structures of exposure and effects of pediatric lead poisoning; that lead is toxic at any amount of exposure, that children are particularly vulnerable to lead's effects, that particular aspects of the built environment are contaminated with lead, that environmental regulations have been effective at reducing the use of lead in industrial applications, that the

burden of elemental lead is borne unequally in society. As a researcher I cannot assume substantive relationships into being (Lee 2018); Chapters three, four, and five are dedicated to establishing that these substantive relationships reflect the truth of pediatric lead poisoning as it is currently known. The support chapters rely on medical literature, epidemiology, sociology, and history. There are a plurality of disciplines at the core of this analysis. If the goal is to accurately understand pediatric lead poisoning we must inquire into the totality of effects with which it is conceivably related (Peirce 2006). The heterodox social surplus approach uses an evolutionary approach to understand phenomena, valuation based on human life process, and emphasis on causal structures, distributive mechanisms and the application of power. These parameters are not exclusive to an economic approach but rather than territorialize an exclusive space for economics I delight at finding many disciplines and practitioners working in a similar manner.

Substantive Relationships, a Heterodox Social Surplus Approach, Cumulative Causation

I am building a frame in which the particular is seen, a descriptive model of the process of lead poisoning embedded in a vision of social reproduction via the Heterodox Social Surplus approach. All economic models are a "*story* with a specified *structure*" (Varian and Gibbard 1978, 666, italics given). The elements of the model articulate the story through the structure of their relationship, be that structure logical, narrative, mathematical, etc. Models abstract and simplify the world to highlight relationships between the critical features of the phenomenon being studied (Morgan 2012). Consider the schema of figure 2.1. The illustration emphasizes the continuity of the process of lead poisoning, initial steps of mining and refining the natural environment are linked to the myriad of health outcomes which lead poisoning produces.

Figure 2.1: A Schema of Pediatric Lead Poisoning Process

Source: Developed by author from research described in chapters 3 - 5.

Figure 2.1 allows us to see the anthropogenic nature of pediatric lead poisoning. There is no direct connection from the natural environment to the child, lead exposure is always mediated by human action, industrial production and the built environment. Further the schema shows the connection between action anywhere in the production process is eventually linked with the health outcomes which individuals suffer. On the basis of that through line the crusading geochemist Patterson recommended (in 1980!) a "total phasing out, in as short a time as feasible, the manufacture and use of leaded products" (National Research Council (U.S.) 1980, 345). Patterson refers to the monetary impact of lead poisoning as "incalculable". He identifies biological human experience as the only relevant parameter upon which to make value judgments. Patterson's analysis reflects the Institutional Economics of Kapp and hisemphasis on a thorough accounting for social costs in an analysis of the environmental impact of business enterprise (Kapp 1970; 1978).

There is a second vector of value upon which institutional analysis turns, the 'serviceability' of the good or service in question; how well does lead perform in its industrial applications. Lead has been recognized as serviceable for industrial applications for over 3000 years[12] and in recognition of this Patterson, in the same document where he calls for an end in the mining and refining of lead he also calls for an expansion in basic research to replace lead

[12] Lead paint, in terms of the desirable qualities of a paint, was the superior product. Leaded gasoline was the superior product in terms of preventing knocks and increasing the useful life of the engine.

in existing applications. Institutional Economics is well suited to analyze value denominated in the incommensurates of human experience and industrial serviceability (Sturgeon n.d.). Though they do not formally investigate lead poisoning, health economics, or environmental economics, Ayres and Gruchy each articulate how the resolution of tension between serviceability and health or environmental concerns reflects a societies ethical commitments (Ayres 1962; Gruchy 1977). I will return to ethical issues shortly.

Recognizing the production process diagramed in figure 2.1 has become more complicated as acute lead poisoning (manifest as dental lead line, nephrosis, convulsions, death) has decreased and lead poisoning (measured in terms of blood lead levels) has become a proxy for other health outcomes rather than a primary medical condition in itself. Knowledge of the relationship between blood lead levels (BPb) and adverse health outcomes is well known but the actual extent of the problem is unknown due to the absence of universal testing. It is possible for apparently normal children to be lead poisoned, the effects of their exposure to emerge sometime in the future. Figure 2.1 none the less diagrams the connection between the lead exposed child subsequent health outcomes. Chapters three and five establishes links between observed BPb and subsequent negative health outcomes.

Expanded BPb testing to understand the scope of the problem as it currently exists is needed. It is nonetheless deeply problematic to use children as a bellwether for environmental toxicity. One of the advantages of the schema of pediatric lead poisoning is that it lets one visualize how the links between stages are also opportunities for interrupting the lead poisoning process. Patterson saw this clearly when he called for an end to mining and refining lead. If humans stop taking lead out of the ground and using it in industrial applications that will stop the process of 'building the world that kills us' (Rosner and Markowitz 2016). Thinking through

figure 2.1 we begin to understand the temporal complexity of pediatric lead poisoning. Current pathways of human exposure and future manifestations of present exposure are dependent on past decisions regarding extraction and use. Even if mining and milling operations were to stop today there are still hundreds of thousands of tons of lead built into our urban environment. This juncture, where the built environment inherited from times past presents a threat to the child, is where the contributions of my dissertation are found. Mine is an evolutionary inquiry to the degree that the relevant conditions of the subject are inherited from times previous; cumulative causation conditions the life process (Veblen 1919; Kapp 1978).[13] My dissertation is focused on one small piece of understanding and addressing that inheritance, the relationship between the already built environment and health outcomes that occur sometime in the future. It is possible to improve individual health outcomes by improving the condition of the built environment.

The temporal complexities of lead poisoning deserve more emphasis even if it means introducing the shortcomings of figure 2.1. That particular illustration shows a simple through-line from the mine to the child while the process is actually considerably more circuitous and complex. Appendix A reproduces several schemas from the lead poisoning literature which emphasize the complexity of the production process in terms of industrial process (figure A.1), the built environment (figure A.2), and biological processes (figure A.3). Models structure our attention for purposeful action but they are not a pass to ignore the real world (Dewey 2008; Lee 2014). A recourse to the real world must be predicated on the possibility that the model is

[13] A more substantial investigation into what the use of lead has generated in terms of habits of thought still needs to be done. Bruce Nevin's *Lucifer Curves*, and my 2018 presentation at the annual meeting of AFIT, "The Useful Metal, the Aping Disease, and the Institution of Childhood Lead Poisoning", are fitful steps in that direction (Nevin 2016; N. Wilson 2018b).

incorrect, and we are researchers are open to finding contradictory evidence or evidence that the theory upon which the model rests is incomplete (Rutherford 1994).

Several critical aspects of the real world are missing from figure 2.1. There are no agency structures in the model. Though the schema can be used to focus attention, without a mechanism to see the variable effects of human action the model is inert. A lack of agency also marks the model as unrealistic because governmental regulation of industrial processes (agency in the development and implementation of political oversite) has had a substantial effect on the process described by figure 2.1 and the more expansive schemas reproduced in Appendix A. A related criticism focuses on an absence of experiential variation in the model displayed in figure 2.1. In that schema there is one built environment, and everyone is subject to it.[14] In truth every child is not subject to the same degree of lead exposure. The social determinants of health are implied by the 'built environment' term but without variation of exposure the social determinants are not evident. The key insight of the social determinants of health literature is that non-medical factors that are experienced by different groups and subject to social policies (e.g. housing and environmental conditions) which produce systematic differences in health outcomes (Evans, Saltzman, and Cooperman 2001; Bashir 2002; Thomson et al. 2009; Braveman and Gottlieb 2014; Swope and Hernández 2019). Figure 2.1 is an adequate beginning, but another modeling technique is required to begin seeing regulatory agency and processes like social segregation reflected in divergent health outcomes.

What follows is a second step towards developing a heterodox social surplus model that incorporates a vision of health (particularly as a result of lead poisoning) conditioned by

[14] Patterson certainly thought this was the case citing the biologically, evolutionarily normal amount of lead exposure to be 1/1000th of the mean level in 1980. 1/1000th of 12.5µg/dL is far below our contemporary mean pediatric exposure levels.

the built environment as laid out above. Though it is possible to extend this modeling framework to incorporate social determinants of health (Schulman 1996; Todorova 2014; 2015) the absence of a fully articulated social surplus model that incorporates a produced environment in turn producing health outcomes (Holt, Pressman, and Spash 2009; F. S. Lee 2018) means acknowledging this theoretical framework is an open research agenda (Chester and Paton 2013). I propose three small models that begin to position lead poisoning in a social surplus framework. A model where a lead-loaded or lead-safe built environment (B) is produced contingent upon the state regulatory regime (r) of production process (P).

$$\mathrm{P}|r_1 \rightarrow \mathrm{B}_{safe} \qquad 2.1$$

$$\mathrm{P}|r_0 \rightarrow \mathrm{B}_{lead}$$

Model 2.1 assumes the existence of a production process (P). For simplicity the regulatory regime is assumed into a dichotomous perfectly effective and completely ineffective form though this can be expanded by specifying *n* regulatory regimes producing *j* levels of lead in the built environment. This first model is slightly modified to show that with the addition of labor (L) and technology (t) the built environment can be transformed into its lead safe form.

$$\mathrm{B}_{lead} \oplus \mathrm{L}|t \rightarrow \mathrm{B}_{safe} \qquad 2.2$$

Model 2.2 repeats the dichotomous assumption regarding the lead loading of the built environment. My third model shows the family unit (F) interacting with the built environment to produce lead poisoning measured in blood lead levels (BPb).

$$\mathrm{F} \oplus \mathrm{B}_{safe} \rightarrow \mathrm{BPb}_0 \qquad 2.3$$

$$\mathrm{F} \oplus \mathrm{B}_{lead} \rightarrow \mathrm{BPb}_+$$

Model 2.3 does not assume any particular form of the family unit (they may be a bog-standard nuclear family, a single mother with children, children living with extended family or friends,

etc.). Here as elsewhere in my dissertation BPb is a proxy for additional health outcomes. The magnitude of BPb associated with exposure to lead in the built environment is the open research agenda in which the empirical section of my dissertation is nested.

There are several aspects of these interconnected models to be emphasized. Equations 2.1-2.3 maintain the emphasis on the production processes in the construction of the built environment while indicating the parallel processes are creating lead loaded and lead-free environments. The diversity of environmental conditions produces different health outcomes. A telling quote from a New Orleans resident translates this general production process into the lived experience, “I guess there must have been some public health campaigning at one point where they said paint chips are it! Because everybody got that message. It was like Smokey the Bear. But they don't understand that it's so much broader than that... it's in the soil, It's [in] the air. It's in your pipes” (“10 Policies to Prevent and Respond to Childhood Lead Exposure An Assessment of the Risks Communities Face and Key Federal, State, and Local Solutions” 2017, 17). Multiple strategies are required to confront the broad base of contamination and that is reflected in the multiple models; regulation of the production process, active remediation programs, and actualizing a families' ability to choose to reside in a lead-free environment. None of these strategies work on their own, due to the temporality of lead contamination (lead has already been built into housing and the soil, and new vectors of lead are constantly being created and discovered) a multi-vector approach to prevention is necessary.

A regulatory environment is modeled as a variable in equation 2.1. Regulation is part of the production of the basic goods air, water, housing, and food. The regulations can, but need not, be overseen by the state (for instance, the lead industry voluntarily self-regulated the use of lead out of interior paints in 1952). Reality indicates that the degree of regulation

required for lead-safe communities is necessarily a project for the state. Some exercise of state regulatory power is due to the lag time between lead exposure, health outcome and the degree to which the produced health outcomes mimic those produced by other means. The lead loaded environment creates a public in the sense articulated by Dewey in *The Public and its Problems* (Dewey 1927). State action is the product of a healthy political system responding to a public called into being by a common problem. The diffuse and delayed benefits from preventing children from being poisoned by their localized environment are a challenge for private enterprise to capture. A bad equilibrium state where environmental toxicity is the ongoing condition is a fact more than conjecture. Additional exercise of state sponsored activity follows from the social rewards which the regulation and remediation of the lead loaded environment creates. Conceptualizing the production of health in the manner proposed here expands in an instrumental way Waller's conception of the state's role in the provision of economic security (Waller 1992). Similarly the model incorporates Forstater's conceptualization of a roll for the state (to the degree that regulation and remediation prevent cognitive impairment) in creating the conditions for a spontaneous, creative problem solving people (Forstater 2003).

Each of the three equations contains the possibility for some human agency to change outcomes and alter distributions. In equations 2.1 there is the degree of regulation restricting the use of lead in the ongoing production processes. In equation 2.2 there is agency in the amount of labor and technology applied to convert the lead loaded built environment to the lead-free built environment. In equations 2.3 there are lead loaded and lead-free environments which families (ideally) have the ability to choose among. Each of these equations embodies a potential research agenda and should not be considered full specified. My dissertation project is implied in the last equation of model 2.3.

Models are framing devices and necessarily leave out essential content. In this dissertation lead poisoning from food, toys, and other objects are hardly considered. Chelation, nutrition and the complex biological processes post-exposure are absent from these models as well. The explicit processes of creating and converting a lead-loaded environment, demolition, renovation, and remediation, are not treated separately in equation 2.2. There is no time element in the model. It is not clear that aspects of the built environment are produced at different times, nor is it clear how long once built into the environment lead will remain there. Much of my dissertation deals with the spatial aspects of lead in the built environment, and the spatial element of the issue is completely absent from equations 2.1-2.3. Spatial issues are important because of the way lead poisoning is localized. New housing construction is lead free but generally at a remove from older homes and industrial point sources of lead emissions. These lead burdened geographies are just as readily defined by race and class, facts of contemporary existence that must be acknowledged and addressed.

There are three familiar aspects of economic analysis that are by design absent from models 2.1-2.3. The first aspect regards agency in the model; regulation, remediation and exposure to the built environment is not articulated in a utility maximizing framework. discissions about regulation, remediation and housing are guided by law, politics, and de factor realities of segregation by race and class. The second aspect regards the production of lead poisoned children; environmental pollution and subsequent events are not modeled as an externality to some other central activity. The human use of lead creates lead poisoning, indeed there is no other source of lead poisoning than human action. Modeling treating that fact as a special case mis-specify the process in question. Finally, the agency structures in the models are not specified as being governed by pecuniary benefit-cost tradeoffs. Conceiving of social

agency in terms of ethical commitments may be the path to better health outcomes for all.

Ethical and Pecuniary Considerations

The current risk of lead poisoning, the distributional reality of the institution of industrial lead, is not uniform across the population of Kansas City, Missouri nor the national population. Minority and poor populations are more likely to be lead poisoned than more affluent, white populations as has been the case since extensive testing began in the early 1970's (H. L. Needleman, Tuncay, and Shapiro 1972). The demographics of childhood lead poisoning are a clear illustration of environmental racism (Pulido 2000; Morello-Frosch, Pastor Jr, and Sadd 2002; Ringquist 2005; Muller, Sampson, and Winter 2018). Spatial relationships are more than coincidence and that the distribution of childhood lead poisoning is the result of inherently unjust, racist, and inexorably social processes. The framing of agency in neoclassical economics pushes toward understandings based in personal choice and natural endowment which make social processes difficult to see. A task of this dissertation is to see environmental racism reflected through the data and chart a course for environmental justice. Articulating an end in view for the work at hand acknowledges the importance of pre-analytic vision in economic analysis (Forstater 2004b; 2004a). The motivating focus of an ethical framework is not an anomaly, economics is inherently value laden in terms of subject matter, analysis, and conclusions. It should be considered dissembling not to address ethical issues directly (Briggs 1999).

It is a curious aspect of U.S. history that the most efficacious actions to stanch lead poisoning were done without consideration of costs and all recent calculations of benefits from additional lead-safe interventions show benefits to far outweigh costs. The toxic legacy of the

industrial use of lead is a feature of the institutional structure and economic order that nurtured and developed modern society. Challenging this feature required the creation of new legal framework, a Clean Air Act which "specifically excludes the consideration of costs in EPA's setting of national ambient air quality standards" (Viscusi 1996, 121). Before the government reforms that produced the EPA, OSHA, and the CPSC, there was no legal framework for federal intervention to staunch the use of lead in consumer products (Silbergeld 2003). Such federal agencies, their associated research funding and the countervailing power to industry which they embody are rooted in ethical commitments to the health and well-being of the population.

Nonetheless, analysts have considered the costs and returns to the clean-up of the lead loaded environment. Donald A. Lash mentioned the “staggering long term-economic cost” in terms of increased enrollment in special education classes from lead in New York City housing and that the city will “lose the benefit of a great deal of economically useful activity and will lose the taxes these individuals would otherwise have paid (Lash 1997, 305). Bruce Nevin and co-authors estimate that lead-safe window replacements would yield at least $67 billion in net monetary benefits. It would also lower energy costs by 15-25% in pre-1960 homes with single pane windows… [which] could reduce total national residential energy use by 5% or more.” (Nevin et al. 2008, 417). More recently, Billings and Schnepel estimate large returns for spending on lead remediation, ”each $1 spent on lead remediation generating $2.60 in benefits” (Billings and Schnepel 2017b, 69). Elise Gould, an economist at the Economic Policy Institute computed the figures most regularly cited regarding costs and benefits of lead remediation. She finds that “Each dollar invested in lead paint hazard control results in return of $17-221 or a net savings of $181-269 billion” (E. Gould 2009, 1162).

Gould's work is particularly important with regard to the standard complaint of economists towards the "burden" of regulation. Quoting from Kip Viscusi, an expert at the statistical valuation of an economic life, "[f]rom the standpoint of many economists, the centerpiece of the proposals for regulatory reform is the requirement that benefits of the regulation exceed the costs. In its most sweeping form, the requirement for passing a benefit-cost test would become a super-mandate, which overrides any possibly conflicting legislative guidelines" (Viscusi 1996, 127). The neoclassical demand that benefits outweigh the costs both in terms of raw expenditure and in terms of opportunity costs[15] is easily met by the estimates of Gould and others.[16]

Approaching this issue from another perspective highlights the environmental justice aspects of childhood lead poisoning. Consider the skeptical Agee and Crocker (1996) who estimate for the nation[17] a willingness-to-pay for a 1% reduction in their own children's blood lead burden of $2.2 - $2.3 billion in 1980 dollars. Recall from Gould the general unwillingness to pay for comprehensive lead remediation despite the magnitude of estimated benefits. Willingness to pay is estimated to be quite large for one's own family and observed to be inadequately small for society in general. The disconnection between privately motivated action and public need calls for an ethics of state action. Kerr and Newell (2003) discuss the practical effects of such action, the success of state policy to drive technology adoption, rather

[15] In the framework of Lionel Robbins the opportunity cost of housing remediation are the normal returns on alternative uses of the scarce resources committed to that activity (Robbins 1945).

[16] Although Gould does not dwell on these distinctions, it is important to note that the costs associated with lead poisoning are derived in two ways, willingness to pay or stated preference survey is one method, the other major method is hedonic or 'compensating differential'. Viscusi (2005) develops these frameworks clearly, still opportunity costs associated with diminished mental acuity from lead poisoning is difficult to estimate.

[17] These figures assume their sample is representative of the population of a whole and wave away all the other methodological problems that accompany their statistical project.

than prices or consumer preference. Such action is well founded in an economics that sees government activity as a necessary component of a healthy functioning economy (Waller 1992; Mazzucato 2011).[18] The diffuse social nature of the benefits are difficult for an individual party to recover and without state action the tragedy remains. Understanding the monetary benefits of the lead hazard regulation and interventions has been ineffective at motivating activity on the scale needed, an ethics is required to adequately motivate and fund the lead-safe activities suggested in equations 2.1-2.3. Once we acknowledge humans are producing the environment in which we live there follows an ethical obligation to do so purposely (Boulding 1969; Forstater 2004b). There is a related ethical case for eliminating lead poisoning. Questions of how and where to remediate, what technologies to use and how and who will pay are all ethically loaded. I will briefly touch on three related and complimentary ethical framings[19] Sen's Capabilities Approach, Anderson's Democratic Equality, and the relational ethics of public health.

Sen articulates a vision of his approach that shows the interconnection between capability and freedom. “The capability to function is the thing that comes closest to the notion of positive freedom, and if freedom is valued then capability itself can serve as an object of value and moral importance” (Sen 1984, 316). The ability to act unencumbered is predicated on certain other social products. An example Sen uses is valuing people's ability to function with adequate nutrition is dependent on access to food with adequate nutritional characteristics.

[18] On the promise of high social returns of the city of Cleveland, OH has begun to sell social impact bonds to cover the cleanup of 10,000 lead loaded homes (Dealer 2017). The expectation is the cost of the bond payments will be covered by lower demand for social services.

[19] Stating explicitly the distinction between morals and ethics that I am using. Morality refers to the actual beliefs and action of individuals in terms of good and bad. Ethics are a more general framework in which moral belief and actions are justified and judged (Van Staveren 2007).

“If we value capabilities, then that is what we do value, and the possession of goods with the corresponding characteristics is instrumentally and contingently valued only to the extent that it helps in the achievement of the thing that we do value, viz. capabilities” (Sen 1984, 317). From this discussion of nutrition there is a logical path to the conception of those capabilities that follow from a body unexposed to elemental lead. The production of lead poisoned children entails the involuntary degradation of a child's physical integrity, the effect of which is cognitive impairment, damage to the brain and central nervous system, and other negative health outcomes too numerous to report here (Sanders et al. 2009). If one values the capabilities which follow from the ordinary biological functioning of the human body, the instrumental activities of industrial regulation and lead-hazard remediation are valued. Such is the general ethical case for state action against a built environment full of lead.

Eradicating persistent lead poisoning as a source and symptom of inequality calls to mind an egalitarian ethics to motivate action as a social obligation. In such a space we find Anderson expanding on Sen. She articulates a vision of democratic equality which “seeks equality in the capability or effective freedom to achieve functionings that are part of citizenship, broadly construed” (Anderson 1999, 321). The moral imperative of democratic equality is, “the construction of a community of equals, [based on] distribution with the expressive demands of equal respect. … access to the social conditions of their freedom at all times” sentiments similar to what we find in Sen's writing. Anderson's ethics of democratic equality support the redirection of public attention and funds to address lead poisoning (to stop the process and address the effects of poisoning). The basis for such attention derives from what equality requires and does not rest on pecuniary justifications or a paternalistic impulse (which implicitly denies the coeval social status of the recipients of social aid). The division

of labor in contemporary economies means that no one's produce, or consumption is theirs alone. The entanglements of mutual dependency are definitional aspects of contemporary life and an ethics follows.

> The point of view of citizens acting collectively–the political point of view–does not claim authority in virtue of being a possible object of collective willing. Neutral goods are the goods we can reasonably agree to collectively provide, given the fact of pluralism. Thus, the capabilities citizens need to function as equals in civil society count as neutral not because everyone finds these capabilities equally valuable, but because reasonable people can recognize that these form a legitimate basis for making moral claims on one another. (Anderson 1999, 330)

Mutual reliance is the justification for public assistance (in the case of lead poisoning that means regulation, remediation, and an end to de facto segregation) without recourse to feelings of guilt or pity. To gain these neutral goods (like the environment required for good health) some members of society may require more social resources than others.

The problem then becomes, to what degree and by what methods can the help of others be compelled, even at a fraction of the rate that would be offered own children. There are structures in place that distribute the burdens born by at risk groups in society (the social security system is a noticeable one) but there is no similar national program for lead. Unequal demand on resources follows from the unequal distribution of environmental burdens (Naidu, Manolakos, and Hopkins 2013; Banzhaf, Ma, and Timmins 2019). Acknowledging the spatial aspects of action Anderson concludes, “if, on balance, citizens decide that a region should be designated uninhabitable, because the costs of relief are too high, the proper response is not to leave its residents in the lurch but to designate… relief toward helping them relocate. Citizens are not to be deprived of basic capabilities on account of where they live” (Anderson 1999, 323).

There are many ways in which capabilities can be compromised, lead poisoning is but

one, and so a public health ethics with a very broad scope is called for. Public health is definitively what society does collectively to assure the conditions for people to be healthy. Recognizing that people are socially situated and socially constituted Baylis, Kenny, and Sherwin propose 'A relational account of public health ethics'. Their vision of public health is committed to identifying and addressing patterns of systematic disadvantage in giving priority to those who are disadvantaged with respect to their prospects (Baylis, Kenny, and Sherwin 2008). The complimentary ideas of relational personhood (that individuals are social through and through) and relational autonomy (the choices a person can make are socially enabled and can be compromised by society) along with social justice and social solidarity are the foundation of this approach and provide a conceptual bridge to both Sen's capabilities approach and Anderson's ethics of democratic equality (L. M. Lee 2012).

Contextual Overview

The application of core theory in heterodox economics is fundamentally different from the similarly named activity done in the neoclassical tradition. Economic analysis need not adhere to the pre-analytic strictures of the neoclassical approach, other ‘visions and scenarios’ are available (Forstater 2004b). The economic analysis of childhood lead poisoning can be developed through the perspective of social provisioning rather than individual choice behavior. Such an approach entails understanding environmental lead as part of the social surplus, an epiphenomenon of a larger ongoing process. The discussion above illustrates that ethics have a roll in economic discourse which compliments quantitative analysis of costs and valuation based on human experience.

Chapters two through five establish the veracity of relationships described in the

Towards Another Health Economics section, sketched in figure 2.1, and alluded to in equations 2.1-2.3. These early chapters provide context for the research question, 'Do observations of the built environment, specifically housing conditions, aid in seeing the problem of lead poisoning more clearly'. Chapters five through seven investigate this research question as a particular instance of the substantive relationships of equation 2.3 and detail the contribution of this dissertation. Together this dissertation describes the production of health and the environment with regard to lead such that accurate and useful judgements can be made about public health and policy.

Research on the social determinants of health is subject to many daunting challenges. Social phenomena, even when linked to a physical element like lead, do not lend themselves to analysis via the randomized control trials as is the medically standard procedure and there is the additional problem of collecting data which is gathered and housed separately (Braveman and Gottlieb 2014). Add to these challenges an ethical conundrum; the nature of human subject research makes the application of many experimental methods unethical (I. H. Wilson and Wilson 2016). Eliding the challenges, I employ observational methods to research social determinants. Much of the contribution this dissertation makes is related to the task of 'seeing' the spatial footprints of lead in the built environment, lead poisoning in children, and the relationship between the two. The exploratory observational work uses Geographic Information Systems (GIS) to organize, visualize and associate disparate environmental and health data. GIS is a standard approach to understanding social indicators fulfilling a need identified by Hayden nearly 40 years ago (Hayden 1983; Hastings and Miranda 2012; B. Wilson, Wilson, and Martin 2019). I utilize the computing software R for data analysis, statistical, and graphing applications (Wickham and Grolemund 2016). The advantage of the

R platform is it “erases the distinction between user and programmer” thus enabling open ended, adaptive inquiry which suits the question at hand (Giannakouros and Chen 2018, 99), that is: examining the veracity of my theoretical model, suggesting public policy interventions, and opening doors to further research.

CHAPTER 3

THE NATURE OF LEAD'S TOXICITY AND A REVIEW OF ITS EFFECTS

Geology, Human Action, Biology

The human body does not require any lead to work properly (American Academy of Pediatrics 2016) and the incorporation of lead into the human body does not improve the functioning of any biological system. These facts find further emphasis in the case of children, for whom there is no safe level of lead exposure (Dapul and Laraque 2014). It is no surprise to find that a poison serves no useful function and is toxic at any level, but lead is special in that the preponderance of exposure is the unintended result of otherwise purposeful human action.

Lead is a metallic element, naturally occurring in our planet's crust. Lead takes three basic forms: organic lead (meaning it is in a compound containing carbon), inorganic lead (compounds without carbon), and metallic or pure lead. These three basic forms of lead occur in the world at different rates, for different reasons, and have different levels of bioavailability. Meaning, in the abstract, they are not equally dangerous to the human organism. Lead is seldom found naturally occurring in its pure metallic state, instead it is found in a state of oxidation (Pb^{2+}) in combination with elements in other ores (Sanders et al. 2009). Although lead is a naturally occurring element its current prevalence and its chemical form are the byproduct of the modern industrial use of lead (Settle and Patterson 1980; More et al. 2017) with different industrial activities producing the various forms of lead compounds.[1] The categories of organic and inorganic lead are diverse enough to resist easy characterization of their bioavailability,

[1] More on the production of these forms is in Chapter 4, a social history of lead poisoning.

however crucial differences do exist. Organic lead, for example, is more readily absorbed through the skin and in the liver it is actively metabolized (U.S. Department of Health and Human Services 2007). The importance of distinguishing these two forms of lead is apparent once we understand lead poisoning is a social product.

Lead compounds can be produced and spread via natural processes such as volcanic explosions and forest fires or as an evaporate from bodies of saltwater, however, modern levels of organic and inorganic lead, in all regions of the planet bear no resemblance to natural, pre-industrial levels. The geochemist Clair Patterson, after establishing his bona fides computing the geologic age of the earth to be 4.6 billion years, spent much of his career investigating the toxic legacy of human lead usage. His lament, found in a posthumous publication, expresses the primary fact of lead poisoning, "[t]his is the essence of the tragedy of the human world, it has been constructed through mis-developments in engineering technologies determined by malfunctioning social institutions of education. This is the unique character of death among humans compared to all other beings in the earth's biosphere" (Patterson 1998, 181). We do it to ourselves, engineering our own destruction. His critique of 'the mis-developments in engineering' rests on the basic fact that contemporary humans operate with baseline levels of 1000 times more biological lead than our pre-modern ancestors (National Research Council (U.S.) 1980). At the time he was writing the effects of this toxic exposure at the cellular level we unclear.

Understanding the biochemistry of lead, what the modern baseline level entails, aids a robust interdisciplinary understanding of its effects on the human organism.[2] A principal

[2] It may be just as important to understand the ways in which lead is used. Indeed, among lead's nicknames is 'the useful metal' because of the many desirable properties it embodies; it doesn't tarnish; it is pliable; it is not water soluble; it conducts electricity; it is stable, &c. Two principle modern uses for lead are in lead car batteries and

biological reason for lead's damaging effects is that the body has no systemic filter designed to remove lead specifically from the human organism. Lead is subject to bio-purification and accumulation in a manner similar to Calcium, which is a biologically necessary element (Settle and Patterson 1980). The suspicious pairing of biological indifference and biological intolerance suggests humans did not evolve in the proximity to persistent concentrations of lead (Purves, quoted in Warren 2000). Quoting at length form Settle and Patterson clarifies what this implies.

> At present, we are constrained to extrapolate downward through the enormous long-term overexposures to lead … to consider how, on a molecular basis, natural biochemical processes within cells might differ from those existing today. … Contemporary levels of lead in biochemical systems in cells are so excessive for most Americans compared to natural levels that it is probable that numerous perturbations of cellular biochemical processes are being caused by excess lead.
>
> (Settle and Patterson 1980, 1174)

In the nearly 40 years since those words were written the mean level of childhood lead exposure has fallen by 12-fold while the understanding of lead's toxicity has improved dramatically. Yet the basic facts remain. Lead serves no biological function. It has toxic effects in any concentration in the human body and does not improve the function of any biological system (U.S. Department of Health and Human Services 2007).

Contemporary humans are exposed to a thousand times more lead than our biologically modern ancestors of 10,000 years ago. Lead is a naturally occurring element, but our contemporary level of exposure is nearly entirely the accidental result of human action. The body has no system specifically focused on removing lead from the organism. Biologically

solder for circuit boards (Black 2005; Sanders et al. 2009). Two of the animating technologies of modern life are impossible without lead. It is no wonder that Patterson saw little hope for an engineered solution to the problems of lead production. "The long history of lead technology shows that people's past attempts to solve problems created by lead poisoning by means of applied engineering have all failed" (National Research Council (U.S.) 1980, 273).

modern humans most likely evolved without the presence of lead. We have only recently begun to understand the full range of effects of lead on the human body. In the remainder of the chapter reviews what is known about the biological effects of lead and its impact at the cellular level and to various body systems.

How Lead Enters the Body

The pathways for absorbing lead into the body are well understood, it can pass into the body through the lungs, the digestive track, or through the skin. Ingestion is the method of absorption most commonly associated with contemporary childhood lead poisoning—the archetypal 'child eating paint chips.' Factors that influence the rate of absorption through the intestines of inorganic lead are physiological states (age, fasting, nutritional status and pregnancy) and the characteristics of the lead particles (size of particle, dose size, composition, and solubility) (Maddaloni et al. 1998; U.S. Department of Health and Human Services 2007).

Inhalation is an important and overlooked method for transferring lead into the body. Lead can get into the lungs from inhaling tiny particles of inorganic lead suspended in the air or via gaseous concentrations of organic lead. Inorganic lead that is >2.5μm can be transferred by mucus to the digestive track and absorbed as digestive lead. Particles < 2.5μm can be absorbed into the blood directly through the lungs. With regard to inhalation there are two rates to keep in mind. The first is the rate of deposition, not all inorganic lead that enters the lungs will be deposited. Second is the rate of absorption, not all deposited lead will be absorbed. Further, these rates vary by the type and size of inorganic lead compound. Organic lead is more readily absorbed through the lungs. Rates of deposition for organic lead have been documented to be as high as 51% for tetramethyl lead, compared with approximately half that rate for

inorganic lead chloride. Limited studies of tetraethyl, tetramethyl, and tetraallyl lead (three prominent organic lead compounds) find "relatively rapid and near complete absorption" of the lead that is deposited in the respiratory track (U.S. Department of Health and Human Services 2007).

A third method of absorption, through the skin, is similarly overlooked. The family of organic lead compounds are rapidly and extensively absorbed through the skin (Bress and Bidanset 1991). These compounds were used as anti-knock additives in gasoline and stabilizers in industrial productions (in munitions for instance). The research on this toxicity dates back to the early 1920's (Rosner and Markowitz 1985). Inorganic lead, the more frequent result of industrial activity, are not readily absorbed trans-dermally (U.S. Department of Health and Human Services 2007). This fact does not eliminate the risks associated with touching inorganic lead. Material that has accumulated on the skin can be easily transferred to the digestive track, either through direct hand to mouth behavior or through the contamination of food. Similar risks exist for the absorption of lead through secondary contact. Lead deposited on clothing, skin and hair can be transferred to others not initially exposed (Levin et al. 2008).

The practical implications of the variations among methods of transference to the body is that similar quantities of lead encountered in different ways will enter the body at different rates. If equal amounts of lead enter the digestive and respiratory tracks, more will be absorbed through the respiratory track. This supports the importance of accounting for any source that might be associated with airborne lead dust and that lead poisoning, particularly in children can be a passive, or unintended process.

Systemic Damage Once in the Body

A review of the system specific biologic processes associated with lead informs the understanding of how lead does damage to organ systems. The conceptual staging of the toxic process - following exposure, damage at the cellular level, manifesting at the organ and system level - is a useful analogy to staging of poisoning of the individual manifesting at the social level. In such a way it makes the case for the empirical section of this analysis, Chapters 5-7 quantify aspects of lead's movement from the environment to the individual while also describing the movement from an individual to a public.

Most children are asymptomatic when screened for lead, that is they show no outward signs of lead intoxication. The lead not initially expelled from the body is exchanged between the blood, mineralizing tissues (such as bones and teeth), and the soft tissue. An understanding of the internal biological process underway despite a lack of external symptoms underscores the sinister nature of lead's sequelae. The temporal logic of toxicity (exposure –› ingestion –› interference with biological processes –› external manifestations of symptoms) and the temptation to assume a lack of harm when effects are latent, necessitates this research agenda and as I argue in chapter 2 state action. Understanding the biological processes that are involved with lead toxicity underscore the importance of recording biomarkers such as blood lead level (bll) of lead exposure in the absence of overt indications of lead poisoning (Boreland et al. 2015). Finally understanding the biological processes informs the way we design the study and interpret its results.

Once lead is in the body lead is treated similarly to elements of a class with calcium (strontium, barium, etc.). The body relies on the same passive absorption processes that govern the retention of calcium to deal with lead. Just as taking a daily multi-vitamin is more effective

in regulating the body's nutrient level than taking a single massive dose, low level long term exposure to lead in any form seems to be worse than a single large exposure (Warren 2000). Due to similar molecular charges and ionic radii, the body attempts to use lead particles as it would use calcium. This confusion of elements is compounded by a higher affinity for lead (vis a vis calcium) by calcium-activated proteins. Among the effects of this is the disruption of heme production in the mitochondria (Dapul and Laraque 2014). The preference for lead interferes with multiple calcium dependent functions. At the cellular level the malabsorption manifests as oxidative stress, membrane bio-physical alterations, issues with cell signaling, and deranged neurotransmission. Oxidative stress manifests in damage to cellular material and interference with cellular genetics. Lead toxicity follows two pathways to free radical damage: producing reactive oxygen species, and the direct reduction of antioxidant reserves. Additional damage is done when lead binds to those enzymes with sulfhydryl groups, rendering them nonfunctional (Sanders et al. 2009, 13-14). These sub-cellular malfunctions manifest in different organ systems expressing their deleterious effects in distinct ways throughout the body.

The blood is not a long-term sink for lead in the human body, but it is the initial internal contact and the medium through which it is spread throughout the body. In the adult population the half-life of blood lead is between 28 and 36 days, while in the pediatric population, Dignam et al have found that it can take more than a year for blood lead levels >10μg/dL to fall below that benchmark level (T. A. Dignam et al. 2008). The slow fall in BPb can result from an active replenishment of blood lead from an internal source or the active replenishment of blood lead from external environmental sources. Within the blood nearly all of the lead (~99%) is found in the red blood cells with the remaining fraction found in the plasma. However, with acute

blood lead poisoning proportionally more lead is found in the plasma (Settle and Patterson 1980). With acute exposure disruption of mitochondrial heme synthesis lead poisoning can manifest as a blood-based malady, microcytic anemia. Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme synthesis pathway inducing two types of anemia, hypochromic microcytic anemia (small red blood cells that don't carry enough oxygen to the body's tissue and organs) or, at high levels of lead poisoning the anemia may be normocytic (a low number of red blood cells which thus cannot carry enough oxygen) (Dapul and Laraque 2014).

Bones and other mineralizing tissues function as long-term storage for incorporated lead. Lead is not stored in all bones in equal concentration, rather it has the tendency to be deposited in those places where active calcification is ongoing. During childhood, the majority of lead is deposited in trabecular (the soft, web-like, internal section) bone and in deciduous (baby) teeth. Once deposited this lead can remain inert for years or even decades before returning to the blood and/or soft tissue due to a variety of factors: advanced age, broken bones, chronic disease, hyperthyroidism, immobilization (bedridden-ness), kidney disease, lactation, menopause, physiologic stress, or pregnancy. According to the Council on Environmental Health, calcium deficiency will exacerbate any mechanism which returns lead to the processes of the body (Council on Environmental Health 2016). The practical implications are that a store of lead builds in a person and that blood lead levels are not precisely correlated with the timing of lead exposure. Lead can lie dormant in bone reservoirs awaiting a traumatic stimulus and the experience of child rearing can provoke the transfer of lead from mother to child. Blood lead levels are only a first approximation of the blood lead burden that a child is facing.

It is in the soft tissue that lead has its most wide ranging and deleterious effects. There is no known lower bound regarding lead's danger to the kidney where “doubling the bll leads to a significant reduction in the glomerular filtration rate… an important risk factor in chronic renal failure. ... Impaired renal function and kidney disease are reported at high levels of lead exposure, as estimated mainly through concentrations of serum creatinine and rates of creatinine clearance from the body” (ATSDR 2017). Lead has noticeable negative effects on the endocrine system (Ronis, Gandy, and Badger 1998) as well as cardiovascular function (Vaziri 2008). Gastrointestinal distress is among the oldest known symptoms of lead poisoning. In severe cases of lead poisoning lead may manifest as severe cramping abdominal pain, and obstinate constipation (Lin-Fu 1980).

The male and female reproductive systems are both adversely effected by lead which may lower sperm concentration, total sperm counts, and total sperm motility. Low-level lead exposure can result in low birth weight and premature birth (Dapul and Laraque 2014). Furthermore, lead has the ability to cross the placental barrier such that a fetus may be exposed from concentration in the mother's blood stream. The blood lead level of newborn infants has been shown to reflect that of their mother’s (Schell et al. 2003). Recall that pregnancy is one of the factors associated with the release of lead stored in bone tissue. Thus, a mother's early life exposure to lead may be relevant to her child's blood lead level at birth and that child's development.

The principal target for lead toxicity is the central nervous system. The brain is generally protected from toxic substances by the blood brain barrier. The barrier works via physical properties (tight junctions) and metabolic factors (enzymes and specialized transport systems) to restrict the passage of water-soluble substances from the blood stream to the brain

and the central nervous system. Due to its similarity to calcium, and just as in the case of the placental barrier lead is able to cross easily into the brain, concentrating there and in the central nervous system (Sanders et al. 2009). When lead is utilized in place of calcium in the construction of a neurotransmitter it will not properly fulfill the function of transforming an electrical signal into a chemical signal. Technically this means "the direct neurotoxic actions of lead include apoptosis (programmed cell death), excitotoxicity (death by overstimulation) affecting neurotransmitter storage and release, and altering neurotransmitter receptors, mitochondria, second messengers, cerebrovascular endothelial cells, and both astroglia and oligodendroglia" (Sanders et al. 2009, 6). The basic work of a neurotransmitter, the transformation of electrical signals into chemical signals and vice versa, becomes impaired resulting in permanent damage to brain cells. Additional research has shown that childhood lead exposure has a permanent impact on the structure of the white matter of the brain (Brubaker et al. 2009) and that total grey matter is "significantly and inversely associated with mean childhood blood lead concentration" (Cecil et al. 2008, 741).

Lead Exposure is a Particular Problem for Children

Low level lead exposure hasn't always been understood as posing a different class of problems to children than to adults. Understanding the differential effects of lead on children is a relatively recent development. Modern research, beginning with the work of A. J. Turner in Brisbane, Australia (1893) has shown lead to be a different sort of risk to children than adults (Lin-Fu, n.d.). Lead is absorbed more readily and processed differently in children. The behaviors of children, young children in particular, are such that they are more likely to encounter lead in their day-to-day activities (crawling, hand to mouth behavior, etc.). Children

have no say in the degree to which they live in lead poisoned environments (this makes them curious subjects for economic analysis). Children are biologically more vulnerable than adults, they are also developmentally more vulnerable (Medley 1982). Most importantly, the brains and bodies of children under two years of age are actively developing thus the effects of lead toxicity become built into their body's structures in a way that is not necessarily the case with older children. Early exposure has the effect of transforming episodes of acute exposure into chronic health problems.

Looking closer at the differences between children and adult ingestion, here too, children are not simply little adults. Adults typically absorb up to 20% of ingested inorganic lead after a meal and up to 60-80% on an empty stomach. Children absorb about 50% of ingested lead after a meal (ATSDR 2017) and up to 100% on an empty stomach (Control, Prevention, and others 2005). Compared with 94% in adults, only 70% of absorbed lead is deposited in the bones in children. These higher rates of absorption and blood born circulation may partially explain why children are more susceptible to the clinical effects of lead toxicity and why lead contaminated water supply can exacerbate exposure issues. Children fed with baby formula suspended in lead contaminated water can are exposed in a way that has no direct analog in adults. While blood lead level is not an accurate reflection of the total body lead burden, it more closely maps to the level of toxicity in children than in adults (Dapul and Laraque 2014).

Children are more susceptible to lead because they are actively building their body networks, particularly their central nervous systems (Sanders et al. 2009). They are more efficient at digesting and utilizing the mineral content of their food, combine this with the ends to which the minerals are put, children are especially susceptible to the permanent biological

effects of lead exposure (Laidlaw and Filippelli 2008; Akkus and Ozdenerol 2014). Finally, lead exposure can take place within the womb. Pregnancy can resuspend into the blood stream lead accumulated in the trabecular bone of the mother. This lead can readily pass to the fetus through the placenta or to the infant through the mammary glands effecting an intergenerational transfer of lead out of synch with the exposure to lead.

Review of Lead Poisoning and its Latent Effects

A pernicious characteristic of lead poisoning is that at the moment of observation most children do not present as poisoned. Due to this latency of effect an understanding of the diversity of the impacts of lead on society is only emerging. Retrospectively it is difficult to assess a society as lead poisoned though it very well may have been. The variety of symptoms which lead produces, their behavioral nature, and the difficulty of distinguishing lead as the culprit has inspired the nickname 'the aping disease' for the more sanguine plumbism. Although it is well known that the Romans lived in a lead filled environment and that they exhibited signs of lead poisoning, diagnosing Rome as such is problematic without direct evidence (Kizer 1985). *Ex post* diagnoses are speculative medical history in the absence of firsthand observations of bodies. A similar problem arises when we speak about the present and recent past. Due to the nature of behavioral evidence linking it with lead, 'seeing' the connection is a significant challenge. For this reason, the history of lead research is also a history of research design.

The review that follows charts the documentation (and the development of the documentation) of latent behavioral and physical effects. The previous section focused on the biological effects of lead within the body, this section discusses the ways such intra-cellular

disturbances manifest externally, particularly as social phenomena (poor academic performance, lower socio-economic stature, a propensity towards violence).[3] Physical sequelae of lead poisoning, e.g. renal failure and hypertension, have social effects via the stress on public services[4] and the misallocation of resources that such preventable diseases embody in the health infrastructure. Add to this that lead finds its way into the environment by human, née social, action we are looking at a thoroughly social disease.

Plumbism manifests in the body and much work has been done to identify lead's presence there. There is a necessary architecture for understanding childhood lead poisoning as such. There must be a pathway for exposure; it must have been present in the child's environment. It must be shown that the child internalized the lead - there must be some documentation of bodily lead burden. There must be an understanding of the manifestations of the damage wrought by lead poisoning as such; cause and effect must be apprehended and related. This architecture may seem remedial but the ability to 'see' lead poisoning qua lead poisoning, considering its 'aping' qualities, is challenging at each of these stages. A review of the medical literature finds that the ability to understand the manifest damage of lead was originally limited by our ability to know if and the degree to which children were exposed to lead. With the development of accurate methods for measuring a child's blood lead burden[5] the necessity of showing an explicit pathway of exposure falls out of the medical analysis

[3] More perniciously, though not as readily discussed, are the social impacts of lost potential; the *what is not but could have been* contribution of every lead damaged body.

[4] A reasonable comparison here is the stress on public services that accompanies cigarette smoking.

[5] Issues of development and change in technique belong to the social history section but it is impossible to do a thorough literature review of the medical engagement with lead without noticing the importance of developing techniques.

becoming epidemiology and preventative medicine. The focus in the medical literature then turns to associating medical effects (conditions and behaviors) at lower and lower levels of lead burden (Warren 2000; G. E. Markowitz and Rosner 2014). This review of the medical literature shows researchers to be focused on establishing the existence of latent effects while ways of seeing become increasingly sophisticated; moving apace with developments in research design and medical technique to document an increasing range of effects at increasingly lower levels of exposure. Recently, some researchers have shifted focus away from individual experience to the explicitly social manifestations of lead poisoning, this represents an important change in the discussion of lead poisoning.

Early Medical Observations

The modern medical discussion of childhood poisoning begins with Ruddock (1924) and his article "Lead Poisoning in Children". It typifies the state of knowledge in early 20th century knowledge and hints at things to come. The focus of his article is establishing a connection between pica, "a craving for unnatural articles of food", and childhood lead poisoning.[6] In Ruddock's analysis pica initiates a pathway for exposure because, in his words, "A child lives in a world of lead" (Ruddock 1924, 1682). Emphasizing the preponderance of lead, he lists familiar objects, "paint on toys, food colored by lead, from lead printers type, liquid that stood in lead receptacles, improper medication, sucking paint from their mother's face or nursing after the use of lead ointment, or by swallowing lead objects such as BB shot" (Ruddock 1924, 1683). Nevertheless, he writes that doctors may not identify lead poisoning as

[6] Pica plays an important role in the social history of lead poisoning, and the way lead companies dismissed the threat from their products.

such because “the average physician has never had his attention to the fact [that children easily become lead poisoned], and also because the clinical picture is usually very different from that in similar poisoning in adults” (Ruddock 1924, 1684). Despite his emphasis on pica he foreshadows more contemporary research, noting “one case due to inhalation of dust that contained lead.” He then describes the symptoms of lead poisoning “in early cases a change in dispositions often the first symptom noticed. The child becomes fretful, peevish and restless at night; the appetite becomes poor, breathing is foul and frequently hemorrhages occur from the gums. ... convulsive seizure is common … they are very persistent and are attended by a high mortality.” Ruddock is here describing acute plumbism, extreme lead poisoning. To his credit, in comparing lead poisoning in children with the adult variety he noted differences, “colic has been more frequently observed than paralysis, but more common than either are irritability, restlessness and a blue line on the gums” (Ruddock 1924). This ‘blue line’ is the tell-tale Burton lead line which appears in much of the early literature on lead poisoning and can still be used to diagnose instances of very severe poisoning (Pearce 2007). Without the lead line or knowledge of the path of exposure the necessary evidence of lead poisoning “is obtained by the demonstration of lead in the urine and feces.” The article concludes with a prescient warning, “There are many mild cases of lead poisoning in children… the true nature of which are never suspected.” The extent of the unsuspected epidemic would not become clear for some 50 years.

A step towards seeing the epidemic and its effects, can be found in Byers and Lord (1943), “Late Effects of Lead Poisoning on Mental Development”. Here we find the authors document lead's “disastrous effect on mental development”, by means of follow up interviews with children diagnosed with lead poisoning at Children’s Hospital in Boston (Byers and Lord

1943, 471). At that time, the medical consensus held that a child, if treated adequately for lead intoxication, will make a complete recovery. Dr. Lord was skeptical. She was familiar with the literature indicating lead was stored in the bones and released back into the body later in life. This fact led her to suspect there were also long-term effects from lead poisoning. Her hypothesis motivated her to perform follow up investigations with 20 lead-poisoned children to check for latent effects.[7] The findings hint at the scope of later research. At the time of their original treatment for lead poisoning these were "normal children". However, "after recovery from their lead poisoning these 20 children made an extremely poor record in competition with their fellows" (Byers and Lord 1943, 479). To determine the late effects the researchers administered IQ exams and compared the academic progress of the research subjects with that of their peers. On these grounds, they find 'psychoneurological deficits' in the absence of overt evidence of central nervous system trauma. In addition to poor academic performance the authors also document severe behavioral issues; several of the children were excluded from school on the basis of behavior. They note, "their difficulties were in relation to both the intellectual and the emotional spheres" (Byers and Lord 1943, 482). The article concludes with the prophetic statement "it seems probable that lead poisoning of the sort discussed here can at present be recognized in only a small percentage of cases" (Byers and Lord 1943, 484).

[7] The discussion of the method of diagnosis of lead poisoning gives credence to the description, as far back as the 1940's, of lead as the aping disease. First a source of inhaled or ingested lead. The two of the following symptoms: marked pallor or anemia, colic or obstinate constipation, muscular incoordination, peripheral motor paralysis of the most used muscles, basophilic stippling of the red cells, a lead line on the gums and lead in abnormal quantities in the stools and urine. Or three of the longer list of symptoms: "among which were emaciation, pain, loss of strength, head-ache, insomnia, mental lethargy, tremor, dizziness, encephalopathy, hypertension and articular pains." (Byers and Lord 1943, 471–72)

Modern Research Design

Towards the goal of recognizing the breadth of effects from lead poisoning, modern experimental design—specifically the use of control populations and longitudinal data sets—found its way into the literature. De La Burdé and Choate (1972) compare an experimental population of 70 children and a control group of 72. Both groups were from the 'same sorts of background and general area' of Richmond, Virginia. Both groups were from a pediatric population which had been followed since before birth. The authors' preliminary findings state: "lead acquired in quantities insufficient to cause acute clinical symptoms may result in neurologic damage" (de la Burdé and Choate 1972, 1087). The authors performed a series of psychological and physical tests[8], noting a significant difference between the experimental and control populations. They observed that the majority of both populations were of 'average intelligence' but that among the experimental population, a large number of these 'average' children failed at one or more of the other tests. The authors write, "deviations in our over-all behavior ratings occurred almost three times as often in children with lead exposure compared to control subjects. The most frequent combination of behavior characteristics was extreme negativism, distractibility, and constant need for attention" (de la Burdé and Choate 1972, 1088). Modern experimental design had begun to allow researchers to see lead poisoning in ways that previously went unnoticed.

In a review of the state of knowledge about childhood lead poisoning and its historical development up to the year 1980, Jane Lin-Fu emphasizes concern over "'asymptomatic' children with elevated blood lead levels" and "the possible 'subclinical' toxic effects of lead

[8] There is another investigation to be carried out, orthogonal to my own, documenting the varieties of tests given to these children.

in young children." (Lin-Fu 1980) Her article tracks the progress toward observing the latent neurological and behavioral issues that earlier authors had noticed but were not able to illustrate as conclusively related to lead poisoning, and the development of the tools[9] to observe with precision the level of intoxication. That same year, Herbert Needleman and his co-authors published their first paper examining a cohort (of lead exposed children and an associated control group) they would track into adulthood. The behavioral and neurological effects of lead poisoning were, step by step, moving into the light.

Between 1975 and 1978 Needleman and his co-authors collected 3329 baby teeth from children in first and second grades in Somerville, Massachusetts. These teeth were used to determine the level of pediatric lead exposure for children in the sample. From this population two groups were isolated, a high level (top 10% of the lead distribution) and low level (bottom 10%) population. The children were then examined and evaluated to establish relative effects from well-established levels of lead ingestion. The results of the 1979 paper are powerful.[10] Quoting the report at length indicates the range of academic and behavioral effects the authors were able to document.

> As compared with controls, children with high lead levels appeared particularly less competent in areas of verbal performance and auditory processing. … The ability of subjects with high lead levels to sustain attention was clearly impaired, as measured by reaction time at varying intervals of delay. ... Children with high lead levels performed significantly less well on the Wechsler Intelligence Scale, particularly the verbal items, on the measures of auditory and verbal processing, and on attentional performance as measured by reaction time under conditions of varying delay. ... Teacher's reports of classroom behavior showed that children with high lead levels were rated significantly poorer on nine of 11 items and that the sum score of these subjects was lower

[9] These include different types of blood lead testing, Dental lead testing (H. L. Needleman, Tuncay, and Shapiro 1972), and well-designed longitudinal clinical studies.

[10] The effects of the power of Needleman's study are discussed in the section on the social history of lead and can be seen in the fact this work was singled out for scrutiny in Epstein (1998).

> ... Hyperactive behavior is a frequent sequela of lead poisoning and is suspected of being an effect of lead at lower dose. … The deficit of attention in the children with high lead levels demonstrated here may be responsible in part for impaired verbal learning.
>
> (H. L. Needleman et al. 1979, 692)

This is a wider range of effects than had been previously recognized but fits well with what is known now. That such a diverse range of behavioral outcomes could be associated with trace exposure to a mineral years in the past should be alarming. From a cursory review of the modern medical literature on childhood exposure this range of effects should have been expected.

Following up with these same populations four years later, similar but not overwhelming associations were documented, specifically with regard to school performance (D. Bellinger et al. 1984). Returning to this population at the end of their high school career Needleman et al observed that "persistent toxicology of lead was seen to result in significant and serious impairment of academic success" and, similarly, "exposure to lead, even in children who remain asymptomatic, may have an important and enduring effect on the success in life" (H. L. Needleman et al. 1990, 86, 88). Crucial for the legitimacy of their claims the authors describe their work as meeting "six criteria for valid causal inference: proper temporal sequence, strength of association, presence of a biologic gradient, non-spuriousness, consistency, and biologic plausibility" (H. L. Needleman et al. 1990, 87).

Results from around the globe agree with these findings. A study population from NZ—the Christchurch Health and Development Study—has followed a group of 1265 children from birth into middle age. Observing them at birth, 4 months, and annual intervals to the age of 16, then again at 18 and most recently at 38 years, this cohort has been a wellspring of data about the long-term effects of childhood lead exposure. It is widely discussed that children who

experience childhood lead exposure are likely to experience other material disadvantages (low levels of parental education, younger mothers, low socio-economic status, high levels of family conflict, etc.). One task of a well-designed study is to take such confounding factors into account while still focused on seeing the lead-behavioral relationship in the research subject. Taking these potential confounders into account the researchers report "exposure to lead has detectable effects of the subsequent cognitive development and educational achievement" (Fergusson, Horwood, and Lynskey 1997, 477). The results of this study are consistent with Needleman's Somerville study though the observed effects in the New Zealand cohort are less pronounced. The authors suggest this discrepancy may be due to a lower mean level of lead exposure in the New Zealand cohort (6μg/dG) vs. the Somerville cohort (14 μg/dG).

Much contemporary research has examined 'intermediate' phenomena.[11] Several studies have examined the relationship between childhood lead exposure and lead poisoning. Nigg et al (2010) is an example of such work, focusing on the observations associating lead exposure with ADHD. The authors consistently find that even with low-level exposure, "[t]here are reliable relations of blood lead with lifetime symptoms of hyperactivity-impulsivity as assessed by structured clinical interviews with the patient" (Nigg et al. 2010). This finding abides despite, or in addition to, the documented genetic associations with ADHD (Waldman and Gizer 2006). Interestingly, Nigg et al. report that the clinical identifications of ADHD and the teacher reported identifications differ. Childhood lead, even at low levels, is well associated with clinical identifications, while previous teacher reports of ADHD (rather

[11] It may be unfair to refer to a young person experiencing the effects of lead toxicity as 'in an intermediate stage in their life'. To insist such is trivializing their very real, and negative experience. This is particularly the case since we are generally talking about dis-advantaged teenagers, who experience the trivialization and exploitation of their situation as a fact of daily experience.

than lead exposure) were well associated with future teacher identifications ADHD. This supports, albeit indirectly, the intermediate effects hypothesis. After a behavioral 'problem' manifests, the memory of the problem in the heart of the disciplinarian will continue project upon the behavior of child; they have been set on the path to trouble.

As discussed in the section on the various biological manifestations of lead toxicity, latent health effects can be non-behavioral. Two of the frequently mentioned biological effects are hypertension, or consistent high blood pressure, and adverse renal function, or failure of the kidney to effectively remove toxins from the body. Using a measure of lead exposure gleaned from *in vivo* cortical and trabecular lead accumulations and associating them with high blood pressure (systolic blood pressure over 160 mmHg or diastolic blood pressure of 96 mmHg of higher at time of examination) low level lead accumulation was identified as an "independent risk factor for developing hypertension in men in the general population" (Hu et al. 1996). Using the Veterans Administration's longitudinal Normative Aging Study researchers were able to establish that "low level exposure to lead may impair renal function in middle age and older men", additionally low level exposure appeared to accelerate natural age related decline of kidney function (R. Kim et al. 1996).

A crucial question in any statistical study is the magnitude of the effect. Establishing the statistical significance of an effect is an important step but not enough to make the case. Focusing on the magnitude of effects is one of the critical insights of the McClosky and Zilliak stalking horse *The Cult of Statistical Significance* (Ziliak and McCloskey 2008). Not all significant causes are equal in magnitude. What researchers are looking for is, in McCloskey's memorable phrase, 'Oomph'. The legitimacy of any statistical oomph depends on a regular relationship between what is measured and what is claimed about the measurement. An area

of research that embodies the confusion of measurement with the measured, is IQ. There has been a lively debate about the nature of IQ as an accurate measure of intelligence (Gould 1982), one can look to the controversy surrounding *The Bell Curve* to observe the miss-use IQ can be put to.[12] One might prefer to set IQ aside from scholarly consideration but the fact remains it is frequently used in studies such as those reviewed here as a proxy for intelligence and is has been used as the basis for elementary school placement, to determine college placement, opportunities for job placement and advancement, etc. Furthermore, there is a literature referring to IQ as a proxy for the development of white and gray brain matter with. As such, examining the relationship between lead loading and whatever it is that we are measuring when we measure IQ has been repeatedly examined and associations have been made. Lanphear et al. work on the connection between high levels of lead and diminished IQ found a statistically robust connection (Lanphear, Matte, et al. 1998). Needleman's remark from 1990 points out that lead poisoning's effect "in many studies is about 4 to 6 IQ points. … We have shown that a shift of this magnitude [the insult from childhood lead exposure] predicts a 4-fold increase in the rate of severely impaired children (IQ <80)" (H. L. Needleman et al. 1990). Conversely this implies a fall in the rate of children in the upper tail of the IQ distribution.

A very thorough study looked at preschool children with blood lead levels above 10µg/dL (the 'level of concern' identified by the CDC at the time this research) and found an average 7.4 point loss in IQ vs. a child with 1µg/dL bll (Canfield et al. 2003). It was unclear from that research if cognitive defects would occur at blood lead levels below 10. Later studies set out to measure lower levels of exposure, taking account of potential confounders to try and

[12] Nevin investigates how, by ignoring the unequal exposure of African American populations to the negative effects of environmental lead, the arguments of *The Bell Curve* are illegitimate on its own terms (Nevin 2012).

determine what a maximum allowable blood lead level would be. Concurrent measures of lead and IQ found children in the 5 - 10 µg/dL range had a Full Scale IQ mean 3.7 points lower than the <5 µg/dL group, and 5.5 points lower in Performance IQ[13] (Jusko et al. 2007). Using a non-linear model, they found "IQ decreased by approximately 1.2, .32, and .15 points per 1 µg/dL in peak blood lead over the range of 2.1 - 10, 10 - 20, and 20 -30 respectively" (Jusko et al. 2007, 245). Stated differently, increments of blood lead at lower levels have a greater average effect on cognitive function than the same increments at high levels of blood lead loading.

A Turn Toward Social Effects

A recent article returned to the New Zealand cohort to see if the IQ effects[14] observed in subject children persisted into adulthood lead (Reuben et al. 2017). The researchers found that among "this cohort born in New Zealand in 1972-1973, childhood lead exposure was associated with lower cognitive function and socioeconomic status at age 38 years and with declines in IQ and with downward social mobility" (Reuben et al. 2017, 1250). This is not a surprising result, although it is important to have documentation of the lifelong impact of lead. The surprising and notable finding of this study is that childhood lead exposure, even taking IQ into account as a cofounder, is associated with lower levels of socioeconomic status than would otherwise be expected. That is to say, the effects of lead can be statistically disentangled from other confounding variables and the effect of lead poisoning has more pronounced negative effects than would be expected from lower IQ, or any of the other environmental

[13] See note in footnote 8, above.

[14] A complete review of the work on IQ and lead may be required considering complaints of spurious results in the regulatory literature (Epstein 1998).

variables, on their own.

As damning as connections between lead and IQ, are connections between childhood lead exposure, violence, and anti-social behavior. There are over twenty years of research into this connection dating back, at least, to Needleman et al. (1996). In other expressions of lead poisoning (i.e., renal failure or ADHD) the social aspect is implied: the demands on the lead-stricken society for assistance are secondary to the biological phenomenon themselves; the missing social benefit of the lives impaired by lead poisoning is secondary to the impairment itself. In the case of violence and other anti-social behaviors lead toxicity is inextricably interpersonal. There is no second step from biological manifestation to a social result. Tracking the association between lead poisoning and anti-social behavior has become something of a cottage industry for Bruce Nevin (Nevin 2000; 2007; 2009; Carpenter and Nevin 2010). Studying the correlation between instances of violent crime and lagged childhood bll (so that the crimes of lead poisoned children can be observed) Nevin is able to attribute so many pernicious social trends to lead poisoning that he has taken to calling its legacy "*Lucifer:* a toxic image caused by a lead-contaminated environment" (Nevin 2016, itallics in original).

The Nevin work is done at the national scale, and though there is value in terms of establishing the veracity of the lead correlation as a cause of violent crime, the scale at which it is done leaves much to desired with regard to the mapping issues of MAUP and the ecological fallacy.[15] Milke and Zaharan tack out another course to see the social effects of lead. They shift the scale of their analysis to the city level, choosing 6 cities of distinct climate, size and socioeconomics, then focus on aggravated assault, a crime readily associated with impulse control for methodological reasons, as the proper statistical measure. After accounting for

[15] For more on the Modifiable Arial Unit Problem (MAUP) and the Ecological Fallacy see Chapter 5.

confounding variables, looking at all six cities together, they find that "for every metric ton of Pb released 22 years prior, a latent increase of 1.59 aggravated assaults (95% CI, 1.36 to 1.83, $p<0.001$) per 100,000 [residents] were reported" (Mielke and Zahran 2012). For the city of Chicago, a city of approximately 3 million, which regularly released 3000 metric tons of lead into the air, 'back of the envelope' calculations imply over 100,000 additional aggravated assaults, in the city of Chicago alone, per year can be laid at the feet of lead. To the victims of these crimes, who may not suffer from lead poisoning themselves, the toxic insult none-the-less is manifest in their lives. In this way lead manifests, socially as an active disinvestment in well-being.

A Summing Up

This section has reviewed some of the literature into the effects of lead poisoning. It should now be clear what is at stake. Over 100 years of associations and 40 years of intensive study have shown the ways that lead is toxic. We have experienced the effects in our cities. The literature review above and in the previous section reflect a slim fraction of the research on this topic, the CDC (ATSDR 2017) lists over three hundred relevant medical reports any of which are systematic reviews of the extended literature. It is important to re-emphasize that a small amount of lead can have serious toxic effects (Aizer et al. 2016), that the latency of these effects is the rule rather than the exception, and lead poisoning can manifest as in social outcomes.

Combining some of the insights of this section and the previous: Lead poisoning is a human made problem and children are particularly susceptible to its worst effects. Except in cases of acute exposure, those exposed will show no immediate signs. Lead is pernicious in

part because a significant portion of lead exposure is unintentional, following from the normal behavior of children. Though lead toxicity effects individual people at the sub-cellular level it has had severe observable effects that are social in nature. Settle and Patterson's words from 1980 were prophetic, we had no idea the problems lead created at the cellular level. As the work proceeds, putting number and word to these problems we also came to know that environmental lead as a social product.

CHAPTER 4

A SOCIAL HISTORY OF LEAD POISONING

> Sometime in the near future it probably will be shown that the older urban areas of the United States have been rendered more or less uninhabitable by the millions of tons of poisonous industrial lead residues that have accumulated in cities during the past century.
>
> Clair Patterson, *Lead in the Human Environment*

Compared to the way historians talk about the Iron and Bronze age, Lead is a poor cousin. Along a geologic timescale, contemporary thinkers have taken to dating the birth of the Anthropocene—the geologic epoch defined by human impact—with the deposition of a fine layer of carbon across the globe. A layer begun, of course, with Newcomen's steam engine designed to aid in the extraction of coal from coal mines in the English Midlands and taking off with in the 20th century with cars, petrochemicals, and the modern power plant. This dating of the Anthropocene may also be mistaken.

In the 1950's the geochemist Clair Patterson was tasked by his dissertation advisor with determining the age of the earth. Modern chemistry had revealed that Uranium decays into lead at a steady rate though at geologic time scales. Dating the age of the oldest stones on Earth is thus as simple as making two very accurate measurements, one of lead the other of uranium and comparing the two measurements to find the age of the planet. Patterson's measurements of uranium content were consistent and in line with expectations, but the amount of lead Patterson found was too high by a factor of 1000. The re-calibration of instruments and the resampling of source material did not help. Patterson suspected the samples were becoming contaminated with undue atmospheric lead. To staunch this contamination, he pioneered the

ultra-clean lab and promptly dated the earth to the authoritative age of 4.8 billion years. This experience with lead contamination inspired him to seek out and document that an unnatural amount of lead had become the normal background level (Tilton 1998).[1] That is how Patterson discovered we have been living in the Lead Age.[2]

Patterson dates the dawn of the lead age to "at least 5000 years ago" (1980). Refined lead almost certainly was an accidental discovery, perhaps the result of the inclusion of galena stone in a campfire. The first substantial production of lead qua lead, emerges with the cupellation process for refining silver from lead bearing galena.[3] There is material record of lead in pre-dynastic Egypt (4000 bce) and in Cretan tombs (3000 - 2500 bce). "The infamous Laurium mine on which the wealth of Athens was founded produced about 130 ounces [of Silver] to the ton" (Waldron 1973, 393). The process of refining process produced great amount of lead slag which was noted by ancient authors Pliny and Vitruvius as being toxic (Waldron 1973). The earliest known uses of lead were as pigments for face paints. The full flowering of the ancient Mediterranean world used in great quantities for construction, not only in the aqueducts but also in piers, steps, and in the Hanging Gardens of Babylon. (Waldron 1973). The full flourishing of the Roman republic demanded ~80,000 tons of lead a year (Settle and Patterson 1980). Numerous authors wrote about the dangers from lead. For instance Dioscirides wrote *"lead makes the mind give way"* and Nicander of Colophon, an expert in poisons and snakebites, wrote about the toxicity of lead in the first and second century BC

[1] He looked in the remote corners of the world and he looked at Greenland, remote mountain jungles, the deep water of our largest oceans, to document the unnatural levels of lead everywhere. The clincher may have been the comparison between lead levels in Egyptian and Peruvian mummies and contemporary humans (Patterson 1965).

[2] Lead doesn't go away; it lingers in the environment on time scales that are unfamiliar to human experience.

[3] This history from Ancient Athens implies that the use of metallic currency is indistinguishable from a lead intoxicated environment and that environmental lead poisoning has always been bound to issues of class.

respectively (Lin-Fu 1980). The lead poisoning referenced by those authors is the acute plumbism today, found only in cases of industrial poisoning and extreme accidental exposure. Despite its legendary use in Roman Plumbing the major pathway of exposure is thought to be via wine and cooking utensils (Reddy and Braun 2010). The alkaline water of ancient Rome deposited an impermeable layer of minerals sealing the lead in pipes in place. Lead's ability to disrupt enzymatic activity (one of the biological effects of lead discussed in chapter 3) was utilized for the production of sweet wine across the Hellenic and Roman world. Similarly, lead glazes were used on plates and bowls which, when acidic dishes were served in them, leached lead into the food and then into the body. An examination of the historical record indicates the likelihood of multiple epidemics of lead poisoning around the ancient world that followed cooking, serving and preparing acidic food or wine and spirits in lead lined containers (Waldron 1973).

Contemporary support for the Age of Lead thesis comes from research into a trio of glaciers in the Swiss Alps. These glaciers are downwind from lead mines in western Europe which have been in continuous use since for 3000 years. By examining the strata of ice cores from the glaciers researchers are able to observe the deposition of annual layers of lead particles with two critical and telling pauses. “Our findings imply that what were once believed to be background Pb levels represent, in fact, a significant anthropogenic component of the atmosphere over the past ~ 2000 years” (More et al. 2017). Their findings show that only in the plague years of the Black Death 1349-1352 did lead deposits in glacial ice return to natural background levels.

Such studies, indicating that atmospheric lead can be bourn aloft on air currents, have also been performed on the Greenland and Antarctic Ice Caps. Transitioning to the modern

moment, “near the north pole, lead concentrations apparently have increased about 400% between 1750 and 1940. Since 1940, there has been another, sharper increase - about 300%” (Mack 1973, 40). This first period, the take-off of the industrial revolution, saw a tremendous increase in adult plumbism. Lead is, as much as anything, a useful metal, workable and stabile. Lead usage takes flight as industrialized economies transformed the way we live. The second period, the flowering of the modern industrialized world, sees more lead used on a year-by-year basis while the knowledge of its dangers grows apace.

The discussion of lead poisoning as an occupational disease dates to at least the 16th century, with Paracelsus describing the conditions commonly found among lead miners. Bernardino Ramazzini described lead poisoning as a hazard of painters due to their habit of cleaning small brushes between the teeth (Mack 1973). Ben Franklin, in a letter of 1786, discusses lead poisoning as a chronic and well-known occupational condition among printers following the heating of lead type. He refers to it as “a kind of obscure pain that I had sometimes felt as it were in the Bones of my hand” (Franklin 1786). With regard to this knowledge he wrote, “the opinion of this mischievous effect from Lead, is at lead above Sixty years old; and you will observe with Concern how long a useful truth may be known and exist, before it is generally reciv’d and practis’d on” (Franklin 1786). Which can also be read as a general thought about the joint stock of knowledge. Heading into the 20th century the dangers of lead poisoning were well enough understood that labor codes were adopted to prevent women from working in leaded occupations with the understanding that still birth, miscarriage, and neurologically deformed children would follow (Tepper 2007).

This past is prolog to the drama of the 20th century; two great and now outlawed uses of lead—paint and gasoline, ‘industrial hygiene’ protecting the workers that encounter lead at

their jobs, the Kehoe rule protecting the industrial use of dangerous materials, and battles over attempts by public health professionals to mitigate the threat from the human environment threaded through with useful, toxic lead. The more recent trends, falling BPb levels at the same time a consensus is emerging that there is no safe level of exposure particularly in children.[4] The rest of this chapter is focused on the use of lead paint and tetraethyl lead as well the regulatory environment and intervention practices developed to deal with their legacies.

Lead Paint

It is important to understand the development of industrial processes and the development of relatively safe industrial processes are separate events. The push for developing safe processes had to wait for clear evidence that standard practices were unsafe.[5] By 1921 Edward Cornish, president of the National Lead Company writes, "lead manufacturers, as a result of "fifty to sixty years" experience, agreed that "lead is a poison ..." (G. Markowitz and Rosner 2000, 35). The lead industry is one of the classic case studies for the recognition of dangerous practices and the engineering of methods to make it safer. Warren writes about the invention of industrial hygiene, which set standards for contamination, practices for safety, and established occupational safety measures like proper ventilation and protective gear for workers (Warren 2000). Such were the happy outcome of the interaction of external experts and an ongoing machine process. Sadly, it set a dangerous precedent based on

[4] There are other significant social uses of lead that go undiscussed in my narrative, lead in cosmetics, lead in food (the lead solder in canned food and lead in spices), lead in the water supply. These are all well discussed elsewhere (Renner 2009; Gleick 2010; Hanna-Attisha et al. 2016a).

[5] Scaling up the traditional 'dutch' method for adding lead powder to paint as pigment meant creating factories characterized by clouds of lead dust in which workers lived while on the job (Warren 2000).

incomplete evidence: the total effect from lead was not equal to the observed effects; that nothing crucial is missing at the moment when decisions are made is a common mistake. My review of the effects of lead poisoning is a summary of knowledge largely unknown in the 1920's. A misrepresentation of the dangers of lead and the success of industrial hygiene at preventing acute illness in the workplace led to an exponential expansion of the use of lead in paint and in gasoline the effects of which are still to be dealt with.

Franklin's letter mentions lead paint as having toxic effects, that much was already known. The earliest discussion of lead's toxicity to children in the medical literature comes from Australia, again it is acute toxicity rather than the contemporary forms. However, “by the mid-1920's, there was strong and ample evidence of the toxicity of lead paint to children, to painters and to others who worked with lead as studies detailed the harm caused by lead dust, the dangers of cumulative doses of lead, the special vulnerability of children, and the harm lead caused to the nervous system in particular” (Markowitz and Rosner 2000, 37). In many European countries this knowledge led to bans or restrictions on the use of lead paint (G. E. Markowitz and Rosner 2014). Alternatives to lead pigment had been developed and experts called for the removal of such paint from the environment of the child. However, “despite the accumulating evidence of lead paint's dangers to young children, the industry did nothing to discourage the use of lead paint on walls and woodwork or to warn the general public or public health authorities of the dangers inherent in the product. In fact, it did the opposite: it engaged in an energetic promotion of lead paint for both exterior and interior uses from the 1920's through the Second World War” (Rosner and Markowitz 1985). This expansion may be due to the largest paint companies being part of a vertically integrated industry which controlled

production from the mine to the consumer. Such companies had no profit incentive to discover, share, or respond to the extent of lead's toxicity.

The industry advertised its products health qualities, marketed to schools, and health departments for interior use until under thread of regulation they voluntarily lowered the concentration of lead in interior paints (Warren 2000). Lead as a strong product could stand up to the wear and tear of indoor public space. Marketing was directed to children – the dutch boy logo (referencing the dutch process not the country) – and for their rooms (G. Markowitz and Rosner 2000). In the 1970s the stakes shifted, as did the stakeholders. From 1971 to 1980, the US Government began to invest in environmental health research, including research on lead poisoning. Under the leadership of David Rall, the National Institute of Environmental Health Sciences supplanted LIA and ILZRO as a major source of research funds, with no strings attached. As scientific research certified and refined the state of understanding regarding the hazards of lead poisoning government began to restrict the use of lead in gasoline and paint. The institution of regulation, however, are imperfectly designed for the task of monitoring, directing, and funding the wholesale remediation of the urban environment (Silbergeld 2003).

None the less pressure was mounting with regard to the negative effects of lead paint on children. The ultimate conclusion of Byers and Lord's 1943 paper is a normative sentence, “Lead Poisoning is a serious disease developing from entirely man-made hazards, which should be controlled by appropriate legislation” (Byers and Lord 1943, 484). For this, the Lead Industries Association (LIA) attacked Rudolph Byers in the media (Dr. Elizabeth Lord had recently died) and threatened legal action (Silbergeld 1995). The industry insisted up into the 1950's that “white lead adds more desirable qualities to paint than any other white pigment and had practically no undesirable qualities to nullify its advantages” (LIA quoted in Markowitz

and Rosner 2000).[6] Despite its deep pockets for lawsuits and insistence on the safety of its products the industry stopped using lead in interior paints in the late 1940's though lead based interior paint was sold well into the 1950's. At the same time the LIA shifted its strategy to blaming the victim and the adoption of voluntary warning labels. As news of the negative effects of lead proliferated the industry deflected such that lead poisonings were talked about as "essentially a problem of slum dwellings and ignorant parents" (Markowitz and Rosner 2000, 44). The victim blaming focused on pica behavior and attempted to make an argument about the endogeneity of the effects attributed to lead poisoning[7] (Warren 2000). LIA's voluntary labels were designed to avoid using phrases dictated by governments agencies, seeking specifically avoiding the use of the word poison on the paint labels (Markowitz and Rosner 2000).

The second half of the 20th century was characterized by suburbanization of the American metropolis and the residential sorting of the poor and racial minorities into older homes. That movement can be recast as the segregation of the poor and racial minorities into those homes most likely to contain residue of lead paint and the movement to the suburbs as the accidental escape to a less poisonous built environment. The Residential Lead-Based Paint Reduction Act (Title-X) of 1996 was an attempt at the federal level to require the disclosure of lead-based hazards in housing (for sale or for lease), the emphasis there was on addressing potential difference in knowledge about the safety of the home between a buyer/lessee and a seller/lessor (Griffith et al. 1998). The U.S. Department of Housing and Urban development

[6] It may be startling to recognize that this is 1/2 correct. Lead paint remains the superior product when judged only on its merits as paint. Incorporating the biological experience casts lead paint as a toxic menace.

[7] i.e., The lead poisoned would have had their observed problems without the influence of lead in their lives

begins addressing known housing-based lead hazards via grants to local institutions for remediation in the mid-1990's. In 2008 the EPA articulates the Lead-Based Paint Renovation, Repair and Painting rule to address the potential of creating lead-based hazards in the process of removing lead from the built environment. In 2011 it was estimated that approximately 23 million housing units remain significantly lead-contaminated by lead paint in the United States (T. Dignam et al. 2019).

Gasoline, Ethyl

The second major pathway of lead exposure specifically associated with the 20th century is leaded gasoline. By the early 1920's the US. automobile industry was making millions of modern cars each year. However, there was an issue with the operation of the gasoline engines—the premature ignition of gasoline in the engine, or knocks, causing poor performance and unnecessary engine wear. An exhaustive search for a gasoline additive to stabilize gasoline in the engine resulted in the use of tetraethyl lead (TEL) (Nriagu 1990). Fractional additions of this organic lead compound to gasoline radically expanded the power and flexibility of the internal combustion engine with the additional benefit of serving as a valve lubricant thus preventing engine damage while improving performance. TEL was so effective in expanding the effectiveness of the engine that one engineer working of the project called it “A gift from god” (Rosner and Markowitz 1985). In 1924 General Motors and DuPont formed the Ethyl Corporation to produce and market leaded gasoline. The only thing preventing the widespread use of this new fuel additive was that it was a known nerve toxin, it had been used as such in World War I. Worse for the Ethyl corporation, workers at the factories which made this product were being driven to insanity and death by their workplace exposure.

The initial response to this deadly hurdle was similar to that in the paint industry: the introduction of 'industrial hygiene'. Taken together with the example of lead paint, this improvement in workplace safety marks a major success of the progressive era (Warren 2000). While workplace improvements were taking place there were public hearings regarding the suitability of the widespread use of TEL. Alice Hamilton, the first woman appointed to the faculty of Harvard University, and Yandell Henderson, director of the Yale Laboratory of Applied Physiology, testified before Congress recommending against tetraethyl lead. For their troubles Hamilton and Henderson were vilified in the press as hysterical and anti-progress.[8] The congressional hearing did not stop the adoption of TEL, it merely precipitated a change in marketing strategy. Tetraethyl lead would be marketed with the trade name Ethyl (Nriagu 1990). Generations of drivers would talk of 'getting the lead out' not with regard to removing TEL from gasoline but rather pushing lead particulates through the automotive tail pipe and out into the world.

LIA and the successor organization, the International Lead Zinc Research Organization (ILZRO), adopted a two-sided strategy for the acceptance of their products. The first was to attack and discredit those scientists and the research that could constrain their business; only 'proven science' would come to bare on the action of private industry. The same tactics were used by cigarette companies, any hint of uncertainty in unfavorable results would disqualify such research from influencing private industry (Silbergeld 2003). The second tactic was to

[8] This pattern of *ad hominin* attacks sponsored by the lead industry visited epidemiologists like Byers and Lord after their 1943 article associating lead poisoning with mental impairment, Herbert Needleman after his series of critical articles associating lead poisoning with intellectual impairment, and Clair Patterson for his work challenging the state of knowledge regarding background rates and prevalence of lead exposure (Warren 2000; G. E. Markowitz and Rosner 2014; Rosner 2015)

sponsor and monopolize the research into their own product. The man tapped by the lead industry to do this work was Robert Kehoe. The effect of this approach was to preempt the adoption of the precautionary principle and make lead poisoning the standard condition of modern life.

Kehoe would articulate a vision of corporate responsibility for environmental disaster that assumed a harmony of interests between corporations and the public, and a skepticism towards the possibility of generalized harm. At the close of the surgeon general's meeting on TEL he said, “If it can be shown ... that an actual danger to the public is had as a result of the treatment of the gasoline with lead in, it will be discontinued from that moment. ... [B]ut ... when a material is found to be of this importance for the conservation of fuel and for increasing the efficiency of the automobile it is not a thing which may be thrown into the discard on the basis of opinions. It is a thing which should be treated solely on the basis of facts” (Loeb 1999). It was a small step from here to Charlie Wilson's famous syllogism from 1953, ‘What is good for General Motors is good for the USA’ Unfortunately for the world, the syllogism does not run both ways. Bringing research effectively in-house, such that only the industry itself could know that its product was hazardous, no one could challenge the industry's claim that “potential health hazards in the use of leaded gasoline ... while well worth investigating, were hypothetical in character” (Nriagu 1990).

LIA and later ILZRO funded lead research – at Harvard, Johns Hopkins, and the University of Cincinnati – to such an extent that until the 1970's their support dominated the field of lead research. “Long before big tobacco, the lead industry understood the inestimable value of purchasing ‘good science’. To that end the Lead Industries Association as well as precursors and subsequent lead industry organizations, made substantial and continuing

investment in research and researchers to ensure its hegemony over medical opinion" (Silbergeld 2003,164). At the same time LIA developed to an art the use of the bullying response to intimidate independent researchers. The use of lead in paint and gasoline in the presence of doubts about its safety cannot be understood without reference to "the enormous resources the lead industry devoted to allaying public health concerns from the 1920's through the early 1950s. … The lead industry as a sponsor and clearing house of information about lead, was positioned to be in the forefront of efforts to prevent lead exposure in children. Instead, the industry placed its own economic interests ahead of the welfare of the nation's children" (Markowitz and Rosner 2000, 36).

Awareness of Problems, Regulation of Products

The diligence with which Patterson investigated and scientifically disproved each of the complaints the lead industry brought against his work is testament to this tenacity. Still, the effectiveness of his scientific work at communicating the effects of the lead on the environment at large was readily eclipsed by two classics of the 1960's. The first was Rachael Carson's *Silent Spring* which polemically documented the damage wrought by the chemical industry on the natural environment. It is a masterpiece of persuasion, describing the catastrophic, widespread, latent, and unintended effects of the chemical industry in the natural environment. The other was Ralph Nader's *Unsafe at Any Speed*, in which he showed that the big three automobile manufacturers had developed a product that filtered noxious particulates from the exhaust (the catalytic converter) but were colluding to prevent anyone from bringing this technology to market.

These books coupled with unmistakable environmental degradation—the flaming Cuyahoga river and perpetual smog in Los Angeles—alerted the public to the degree of the man-made problem. There was a history of municipal and state governmental attempts to prevent lead paint from being sold (Warren 2000), but there was no overarching legal structure by which national standards could be imposed. Eventually political action in the form of the Clean Air Act and the Lead Based Paint Reduction Act (1971) came to pass. These two acts were the beginning of the end for lead in gasoline and paint, but not the use of lead in industrial applications. It is important to note that the tide was turned without either the medical or the economic experts carrying the load on these issues. The end of leaded gasoline, which comes after a gradual phase out in the 1980's, was the consequence of the EPA's ability to "prohibit any fuel or fuel additive that will ... impair to a significant degree the performance of any emission control device or system which is in general use" (*Clean Air Act* 1967). Because lead gas damaged the catalytic converter, the EPA could force the abandonment of leaded gas.[9]

None-the-less the lead industry kept to its play book, attacking the work of scientists and the quality of the work they had done. In 1975 J. F. Cole wrote on behalf of the lead industry, "... despite the tremendous amount of activity and research, the issues remain much what they were in the beginning. The search for a solid, factual, scientific basis for claims against lead has produced nothing of substance. ... Scientific evidence does not support the premise that lead in gasoline poses a health hazard to the public, either now or in the foreseeable future" (Nriagu 1990). Meanwhile researchers were expanding the forefront of knowledge about lead poisoning and showing how mistaken the understandings of lead in the

[9] This history has been well covered elsewhere a detailed economic accounting of the benefits of this legislation with a focus on pediatric lead poisoning is left for another investigation.

atmosphere, the oceans, plant and animal life were. Patterson wrote that “despite more than 40 years of study… agencies lacked the ability to correctly monitor the extent of lead pollution” (Settle and Patterson 1980, 1168). Patterson's findings and methods have been internalized by the scientific establishment and the state of all our health is better for it. By 1990, despite aggressive challenges to his scientific integrity leveled by the lead industry Herbert Needleman was able to declare, “the association between lead and [impaired neurological function] here meets six criteria for valid causal inference: proper temporal sequence, strength of association, presence of a biologic gradient, non-spuriousness, consistency, and biologic plausibility” (H. L. Needleman et al. 1990, 87). The Kehoe rule, where scientific evidence would be enough to support the cessation of lead's use in gasoline, despite the work of the lead industry to prevent it, could finally be invoked.

Unequal Exposure

Before the clean air act, during the 1960's, there had been the alarming discovery of ‘lead belts’, sections of urban centers of the United States characterized by high levels of observed pediatric blood lead levels (BPb). Beginning in Chicago and taken up promptly in New York, Baltimore, and other industrial cities, large scale lead screening programs were initiated by doctors and community leaders who had been alerted to the dangers of lead paint by public service announcements concomitant with Lyndon Johnson's War on Poverty. In Chicago, researchers found 5-15% of the children screened had a BPb over 50µg/dL. A composite look at several large cities found that, 25-45% of the children, aged 1 - 6, living in high-risk areas had BPb exceeding 40 µg/dL (Berney 1993). As expected in light of the discussion in chapter 3, most of these children were asymptomatic for lead poisoning. The

discovery of the extent of toxicity facilitated a shift in focus with regard to lead poisoning from treatment of acute toxicity, to calls for mass screening and prevention. New techniques for understanding lead poisoning are pioneered in the early 1970's, specifically Needleman's technique of examining the lead content of deciduous teeth, a non-invasive method of assessing levels of pediatric exposure.[10]

Needleman's 1972 article added an extra layer of description to the problem, an urban/suburban difference in exposure. Despite oversampling nearly 2 to 1 the teeth of the urban children (n=425) Needleman was not able to find any of those children with a lead level below 2 ppm, while 13 of the suburban children had lead levels there or below. Meanwhile, the "mean tooth lead for suburban controls is 11.1 +/- 14.8 ppm., and for children from the ghetto 51.1 +/- 109 ppm" (H. L. Needleman, Tuncay, and Shapiro 1972). Though there is no disaggregation of the sample population by race, the statistical contrast between urban and suburban populations suggests the African American population was, by the mid 1960's, disproportionately lead poisoned. With the second National Health and Nutrition Examination Survey (NHANES) conducted from 1976 - 1980 a decomposition by race and ethnicity was possible and it was found that "the median level in all children was 15µg/dL; in black children it was 20µg/dL" (Berney 1993). A discrepancy in mean BPb by race persists to the present day in Kansas City, MO as well as in the national data.

This difference in exposure by race is observed in Jane Lin-Fu's 1980 article "Lead

[10] Needleman's first major paper examined the difference in exposure between urban and suburban children while pioneering the use of deciduous teeth to evaluate the degree of childhood lead exposure. In the earlier literature, there is a consistent problem in determine the degree of lead exposure. Recall the biology of lead exposure, the majority of ingested lead is stored in the bones and teeth, so that tooth exposure is a good method for gauging the degree of childhood exposure. Although not demonstrating the effects of lead exposure the paper's findings well demonstrate the difference in environmental exposure.

poisoning and undue exposure in children: History and current status" and it is evident in Dignam et al “Control of Lead Sources in the United States, 1970-2017”, published some 39 years later (Lin-Fu 1980; T. Dignam et al. 2019). Older homes are more likely to contain lead pipes or lead-containing fixtures of solder (Troesken and Beeson 2003). Older homes are also more likely to contain lead paint (Rogers et al. 2014). Older homes are more likely to be found in urban areas and disproportionately house poor and/or African American populations (Berney 1993; Warren 2000; Filippelli et al. 2005). Regulation largely eliminated housing-based lead hazards in new construction by the early 1990's confining pediatric lead poisoning tobe a racialized disease of urban poverty. Lead contaminated neighborhoods and homes are not the result of choices made by residents, but rather an inheritance from a time before lead was regulated; a significant amount of lead was inherent to the housing stock or, in the case of TEL, the result of action beyond their own control. After regulation of lead paint and TEL the legacy of lead lost its urgency in the national agenda. This process is rearticulated in Needleman's surmise that the lack of action is racially motivated. “[There is] a widely held belief that lead poisoning is a problem exclusively affecting African American children. As the current attitude of indifference [to] the poor and minorities developed, the attack on lead exposure lost its urgency” (H. Needleman 1998). In the years since Needleman's exhortation the U.S. department Housing and Urban Development has spent hundreds of millions of dollars neutralizing housing-based lead hazards and much work remains.

Deeper Understandings and Remediation Strategies

Just as there was an awakening to the fact that any lead in the built environment is the result of human activities there is a narrative traced through the medical literature of our

understanding of how much lead is too much lead. Jerome Nriagu writes, "Until the early 1970s, it was generally assumed that increased lead absorption was of little clinical significance if there was no recognizable symptoms of *acute* poisoning (such as renal damage, peripheral neuropathy, anemia, neurological dysfunction etc.) and the BPb were below [50 or 60 µg/dL]" (Nriagu 1990, 22). This evolving understanding is another place where the social comes into play, the definition of what constitutes lead poisoning is a socially produced standard. The standard for most of the 20th century for blood lead poisoning was set by the level of adult industrial plumbism. As the state of knowledge regarding lead's effects on children has developed the definition of lead poisoning has changed. This has given rise to some complaints from authors sympathetic to the lead industry. As the blood lead level indexed to medical lead poisoning has fallen the number of children identified as lead poisoned has risen, at the same time mean blood lead levels are falling and the magnitude of these population wide trends are obscured in the headline number of children identified as lead poisoned (English 2001).

This cry of shifting baselines has some merit. The condition called lead poisoning (plumbism) at the beginning of the 20th century is a different beast from what we know today as lead poisoning, though they are both predicated on biological responses to lead intoxication. As medical professionals have gotten better at measuring the effects and existence of lead in the body, the definition of what it means to be lead poisoned has changed. When childhood lead poisoning was spoken of in the 1920's the symptoms of exposure were acute: catastrophic trembling, overt neurological damage, and death. Children still are struck by intense lead poisoning, but overt and immediate symptoms are no longer exclusively implied by lead poisoning. On the basis of cohort studies of children it is apparent that a wider range of effects

are produced by lower levels of blood lead intoxication than was conceivable at the beginning of the 20th century (H. L. Needleman et al. 1990; Sonderman et al. 2013; Lanphear et al. 2018).[11]

The criteria level of concern for pediatric blood lead has shifted over the course of the 20th century from 65µg.dL in the 1960's when the silent epidemic was discovered to 30 µg/dL in the late 1970's when the first modern medical studies were performed (Needleman et al. 1979). In 1991 the CDC lowered the standard BPb level of concern to 10µg/dL (CDC 1991) where is stood until 2012. The current standard is defined as the top 2.5% of the distribution of childhood BPb reported by the NHANES. Because there is no level at which no damage occurs, any measured blood lead level should be concerning (Lanphear 2017) the contemporary terminology refers to this statistical standard as a "reference value" (Center for Disease Control 2020). Maintaining a reference value at the 97.5th percentile of the distribution indicates a commitment to those bearing the greatest burden while tacitly acknowledging the importance of eliminating any level of pediatric lead exposure.[12]

A remediation literature and practice followed the implementation of HUD's intervention strategy. The understanding of lead's pathways (peeling paint, windowsills, dust and soil, waterways, soil, etc.) from the built environment into the body is also an understanding of opportunities for intervention and prevention. Much of the early intervention literature and actives were focused on lead paint (Medley 1982; Binder and Falk 1991; Lash

[11] Investigating the development of the field of public health is a warranted line of inquiry. "How those in each field [environmental, pediatric, and occupational] perceived and engaged the others. Surely a most important trajectory of the post-World War II history of lead is how seemingly settled boundaries between these realms had to be challenged and redrawn, both because of this toxin's own straying and because lead Poisoning burst out of rarefied professional discussions into public and legislative debate" (Sellers, 825).

[12] The reference value is arbitrary. Recall Patterson's exhortation that contemporary levels of exposure are still 1000x greater than natural pre-industrial levels.

1997). It was obvious to researchers in the 1970's that "that the dust lead standards for floors and interior windowsills should be lowered to adequately protect children" (Lin-Fu 1980). Remediation attention was also focused on soil and water infrastructure (Mielke and Reagan 1998; Renner 2009).

There has been some success eliminating many sources of lead, from cosmetics, ceramics and canned food, by way of new regulatory statues (T. Dignam et al. 2019). In the late 1970's and early 1980's there was a call for universal lead screening for children in the USA. Save a handful of cohort studies, knowledge of population specific lead poisoning comes from targeted testing and the voluntary screening of children as part of regular medical check-ups and Medicaid related testing in particular. Universal screening of all children will be useful for improving the state of our knowledge and planning public health interventions while follow up screenings would clarify which interventions are working as well as aid in medical research. The regulations of leaded paint and gasoline have triumphed in a way, following the elimination of Leaded Gas and leaded paint average BPb in children levels fell by 78% from 1976 to 1991, but the momentum for universal screening died under Reagan (G. E. Markowitz and Rosner 2014). There is a recognition that, though important, reactive methods such as household education and dust-control measures are not effective long term measures, especially when not applied in conjunction with other practices that address other sources of lead (Yeoh et al. 2006; C. Kennedy et al. 2016).[13]

[13] Two additional dour notes. 1) Though our knowledge of the harms from lead poisoning is more robust than ever and our ability to regulate its use has been effective, humanity is using lead in ever greater volumes. There is ever more lead contributing to building our environment. 2) Lead poisoning, in its contemporary form, is a 100-year-old problem. There are new and possibly more robust environmental contaminants being distributed through our web of global industrial production that needs the attention of health researchers. There is no time for rest.

Summing Up: Lead Poisoning as Social Phenomena

Many scholars argue that pediatric lead poisoning is best understood as an association of social phenomena (Warren 2000; English 2001; Muller, Sampson, and Winter 2018). This chapter has charted how evolving definition of what it is meant by 'lead poisoned' was generated through a social process, just as it is a social process to use that definition to set policy. Chapter 3 discussed seeing lead poisoning as a biological reaction to the body's exposure to lead and because this exposure is the result of human action there again a social process is in evidence. Also discussed in chapter 3 are the inherently social outcomes from lead poisoning; a preponderance of poor educational outcomes, behavioral problems, and medial issues. All of which demand resources to address. Functionally this is what unequal development looks like.

The social aspect of the environment becoming lead loaded calls another social unit into being. In this we find a world of unintended consequences; "For the essence of the consequences which call a public into being is the fact that they expand beyond those directly engaged in producing them" (Dewey 1927, 27). It was the effect of social action that polluted the world and an inescapable fact the modern world to be exposed to it. People that work in the mines and the factories, the people that run the operations, sell the paint, shop for paint under the legal framework that oversees the product, apply the paint, these are not where the effects of the paint end. A public of lead poisoned children becomes through shared effect. We can similarly extend this social fabric to ethyl gasoline and embrace the entire population. To this degree, when we talk about action and the inescapable relation of effects we are talking (ultimately) about the experience of the entire society. The rest of this dissertation disaggregates our lead poisoned society. Looking closely at Kansas City, Missouri and

answering questions like who is lead poisoned, by how much, and what is associated with lead poisoning. By looking closely with intention, a path towards a society that is not lead poisoned begins to come into focus.

CHAPTER 5

GIS, EPIDEMIOLOGY, MODELING RISK FACTORS

The remaining chapters of this dissertation are about developing a way of seeing relationships between the built environment and pediatric lead poisoning quantitatively at the level of experience in Kansas City, Missouri. This chapter is about how Geographic Information Systems (GIS) are used in health research. This chapter is also about epidemiological research connecting the built environment with pediatric lead poisoning. There is some discussion of GIS as a way of seeing and what can be seen with the tool, particularly the racialized and class bound geography of environmental hazards. The permanent and severe effects of children's exposure to lead means the causation of Pb poisoning vis a vis the built environment is very difficult without utilizing an unethical experimental research design.[1] Therefore conclusions about health effects of Pb exposure and its association with different aspects of the built environment depend on indirect and observational studies and rely further on the constancy and magnitude of observed relationships.

The three contributions of my dissertation begin with the development of a new data archive. The contributions of assembling the archive and manipulating its data in GIS are discussed in chapter 6, and the contribution embodying the statistical investigation of the assembled data comprises chapter 7. Before the data can be assembled there needs to be some

[1] The history of medical research is well populated with investigations that violate the basic dictate of medical ethics, "do no harm." Investigations to understand lead poisoning and how to adequately fight it have found their way onto that ignominious list of unethical research (H. Epstein 2013). More damning is the tacit acceptance of the racialized and class bound geography of lead poisoning, a society wide experiment in the systematic poisoning of the population that should be considered ongoing.

guidance as to what data is relevant, additional guidance is necessary for the pre-analytical modeling of assembled data. To set out on gathering and assembling the data without guidance is an invitation to engage in ad-hoc justifications of convenient operations.

A reading of the relevant history is the first guide, though not an infallible one, for gathering data and its use. The preceding chapter demonstrates the importance of a historically grounded investigation underscoring the importance of leaded paint and leaded gasoline in any lead poisoning related research.[2] At the outset of my dissertation project, as part of the KC-HEART initiative, there was a brainstorming session with experts at Children's Mercy Hospital (CMH) developing a list of risk factors for lead poisoning, this list is reproduced as appendix B. With the input of medical professionals from CMH, The Kansas City, Missouri, Health Department, the list of risk factors includes individual focused data, family focused data, housing, environmental, and neighborhood focused data. Appendix B includes 53 separate risk factors to account for in the modeling of pediatric lead poisoning.

Knowing which data to gather is far afield from a plan for dealing with the data once it is gathered, what techniques are useful for understanding the data, and for prioritizing among the identified risk factors. To make these judgments recent review articles of the epidemiological literature are consulted (Akkus and Ozdenerol 2014; Rogers et al. 2014; Council on Environmental Health 2016; ATSDR 2017). These articles facilitate a prioritization of which data to acquire and more critically points to a literature to review for background on best practices for modeling these data. Knowing what to look is paired with ways of seeing. Before reviewing the epidemiological data, the primary tool for visualization and association,

[2] There is no account to these factors being considered obvious. Their risks were not obvious enough to prevent their widespread use during the 20th century. Much progress involves channeling the power of the obvious towards obscure ends.

GIS, is discussed in general, with respect to the health research, and ultimately lead research. The way of seeing that is employed in the statistical section is introduced later in this chapter and discussed in greater depth along with the statistical analysis in chapter 7.

Geographic Information Systems (GIS)

Identifying the source of lead in a child's body is essential for direct intervention and regulation (removing the sources and preventing new sources of lead poisoning from emerging). With the realization that lead poisoning is a pervasive problem, researchers noted that the distribution of lead poisoning had distinct spatial character. The spatial distribution of lead poisoning was referred to as a 'lead belt' in much of the early literature, including the seminal Needleman et al. (H. L. Needleman, Tuncay, and Shapiro 1972). Spatially the lead belt encompassed several states, a kind of regional identifier akin to the Rust Belt that identifies a geography of urban industrial production in the US. A different geography of lead poisoning appears within the city. To see the variety of spatial distributions hinted at early in the research required the development of GIS. Understanding how GIS has been used in health research and more specifically in the investigation of the links between a child's blood lead burden and the built environment clarifies the contributions of this distribution, all of which are centered around seeing the problem with clarity at the parcel level.

GIS is merely a tool for 'observing the world' and one tool among many. There is a literature, embodied in Jackson (2008), critical of the use of GIS. In light of such criticisms, it should be acknowledged, understandings derived from a tool or technique may obscure those understandings that can only follow from the use of a different tool. It is not obvious to the tool user what they may have learned from using a different tool or technique. A version of

this thought is found in Eiman Zein-Elabdin's discussion of culture in the context of Institutional Economics (Zein-Elabdin 2009). The thought, that important knowledge lives in cultural practices that 'become' invisible or irrelevant when subjected to certain tools for knowing, is implicit in Jackson's lament for the decline of community embedded mapping practices. The implicit tradeoff is this: in examining a social process, do the results of actions that are not deliberate yet pervasive and on a personal level passive,[3] outweigh what is gained by the deliberate action taken at some remove from the community of study (this is the GIS technician working at a remove from their office)? This is the implicit tradeoff made with the development of GIS based research. The promise of this dissertation is in the technical precision and flexibility of this technology allowing us to see what was previously obscure and to relate this knowledge in an effective and persuasive manner. Additionally GIS is not invariably dis-embedded from the community, the ubiquity of cellphones and open source activism has created an 'open GIS' movement, the purpose of which is to erase the distinction between researcher in the lab and subject in the field (Sui 2014).

Another critical issue in the GIS literature is the question scale of analysis, an issue which distinguished this dissertation. There is a lively discussion of the importance of analytical scale in the geospatial literature (Openshaw 1983; Zandbergen 2009; Briant, Combes, and Lafourcade 2010; Jaquez 2012). The critical point in this context is the degree to which observations are aggregated to administrative boundaries. For example: when we have a health observation of a child, where is that child located in the conceptual GIS space? Are the observations associated with a neighborhood, a zip code, a census tract or city; are they a

[3] I am referring to the face-to-face transfer of knowledge that comes from embedding a mapping practice directly in the community.

point along a road network; are they associated with a home or the parcel on which it sits? The standard practice is to perform the statistical analysis at the scale of a standardized administrative geography. For instance the census tract, zip code, and commuting zones used by Raj Chetty in his work on spatialized inequality (Chetty et al. 2014; 2016). The principal reasons for performing statistical analysis at the administrative level are straight forward: identifiers for administrative geographies are generally associated with observational data. Using aggregative geographies is also the logical outcome of an interest in privacy, particularly when it comes to personal health and economic data. Census data, which is collected at the household level, is generally only available at anonymizing levels of aggregation. Conversely, some information is simply not collected at a suitably fine level of aggregation for analysis to take place at the relevant scale; this is the case for many environmental conditions.

These aggregative administrative geographies engender several methodological critiques of GIS and spatial analysis more generally. The modifiable areal unit problem or MAUP (Openshaw 1983), the ecological fallacy (Robinson 2009), the uncertain geographic context problem or UGCoP (Kwan 2012). The MAUP arises from the arbitrariness of administrative boundaries aggregating observations that exist at a lower level. In the case of the MAUP, changing the unit of aggregation - the administrative geography used to organize data - alters the understanding of the underlying phenomenon. One finds the MAUP in the different visions of city life one forms when looking at data grouped at the zip code level versus data grouped at the neighborhood level. Neighborhood data is more reflective of the lived experience of a city's residents as zip codes relate to the convenience of delivering mail rather than living a human life. The ecological fallacy is derived from the tendency to confuse aggregations for the individuals that comprise the aggregation. While relatively harmless when

describing things like electoral outcomes (where 'majority rules') the ecological fallacy describes the inaccuracies which follow from the use of aggregated data for the purpose of individual level inference.[4] The UGCoP follows from the ability of people to move between administrative geographies without notice. The classic example here is a child that spends 1/3 of their day at school, 2/3 at home. Valid inference in this case will be limited if characteristics of both environments are not acknowledged or addressed in the analysis. These three issues are endemic to geographic analysis.[5]

There is another group of issues associated with the choice of administrative geography related to entering data into GIS. These issues focus on the trade-off between match rates - the percentage of observations successfully located in the GIS - and precision - the accuracy with which an observation is located in the GIS. The use of aggregate administrative geographies are advantageous for data availability and generally produce high match rates (Rushton et al. 2006). These high-level aggregate geographies have a drawback in the form of an unnecessary degree of placement error. For example, observations are geocoded to the centroid of a zip code rather than in the precise location within the zip code. It is possible to geocode to a street network geography (street centerlines) in GIS, but this too introduces unnecessary placement error (Rushton et al. 2006; Edwards, Miranda, and Strauss 2014). When geocoding to the address level for health research it is well recognized that the parcel centroid geography introduces less distortion than the street centerline geography (Jaquez 2012; Manson et al.

[4] Consider the case where your neighbor receives a million-dollar windfall. Average wealth on the block increases but your wealth has not changed. To use the description of your block's finances to describe your personal finances embodies the ecological fallacy.

[5] The principal contribution of this analysis is related to the MAUP and performing health research at the correct geographic level of analysis. Problems related to the ecological fallacy and UGCoP are addressed implicitly in the geocoding process and in the structure of the statistical model used.

2009; Rushton et al. 2006). Some authors note that this level of aggregation can induce cost concerns, lower match rates, and privacy issues (Rushton 2003). Thus much lead focused GIS work is done either at an aggregate administrative level (Bailey, Sargent, and Blake 1998) or done using multiple maps of the same location for visual comparison of characteristic densities (Akkus and Ozdenerol 2014).[6] Other researchers defend the use of higher level geographies like neighborhoods for privacy reasons and as appropriate because of the UGCoP, movement of people throughout their neighborhood as a course of living implies a shared experience of risk factors by all neighbors (Oyana and Margai 2010). Chapter 6 describes a process for geocoding to multiple geographic levels and utilizing GIS to understand aspects of lead poisoning at multiple levels.

GIS has become a standard tool in lead research for visualizing health observations and relevant risk factors. Visualization is an investigative tool in its own right and can reveal surprising results and anchor a strong narrative. The power of mapping as a research tool is as old as epidemiology itself, relating back to John Snow's mapping of cases in SoHo during the 1854 Cholera epidemic (Beyer 2016). Eye opening early work on the problems with the water in Flint, Michigan utilized GIS (Hanna-Attisha et al. 2016a), similarly early work on Covid-19 in New York City used GIS to explore a relationship between outbreaks and the public transportation system (Harris 2020). GIS as a visualization tool has frequently uncovered surprising patterns just from a mapping of relevant data; Reissman and her co-authors identified a systematic mis-allocation of health resources through a mapping operation (Reissman et al. 2001) and Haley and Talbot demonstrated that the urban geography of upstate

[6] A more thorough discussion of these issues follows in chapter 6.

New York experienced more severe lead poisoning than New York City did, though The City because of its larger population experienced more poisoning (Haley and Talbot 2004).

Combining observations from multiple layers and disparate sources through spatial association has shown GIS to be a useful tool for processing data that otherwise lack a common indexing field. Visualizations and processing becomes the ordinary least squares analysis more familiar in statistical work (Bailey, Sargent, and Blake 1998; Thomas Reibling et al. 2001; Haley and Talbot 2004; Morales, Gutierrez, and Escarce 2005). There are other statistical approaches that are endemic to spatial analysis; spatial kriging (Griffith et al. 1998), Moran's I (Zhang et al. 2008; Oyana and Margai 2010), local incidence of spatial autocorrelation (Anselin 1999; Proost and Thisse 2019). Though not explored in this research these techniques can be applied to the data gathered as one of the three contributions of this dissertation.

Facilitating the assembly of spatial data from multiple geographic levels lends GIS to public health task of constructing indices for lead risk. These indexes combine socio-economic risk factors at the neighborhood or census geography (concentration of minority population, level of parental education, median household income) with parcel level risk factors (age of housing). The promise of these indices is the more effective direction of public health resources to the places and population most likely to need them. Lead risk indices are in use that target both the neighborhood level (Vaidyanathan et al. 2009; Shao, Zhang, and Zhen 2017) and the parcel level (Holton 2002; Miranda, Dolinoy, and Overstreet 2002; Roberts et al. 2003; D. Kim et al. 2008; Hastings and Miranda 2012; Schuch, Curtis, and Davidson 2017). The principal parcel level data serving as a proxy for lead paint risk is year of building construction (though occasionally assessed tax value or owner occupancy status are incorporated into the models) which can foil the risk model in places where there is little variation between homes. Schuch

et all avoid this issue by look at condition of the home rather than the age or value of the home (Schuch, Curtis, and Davidson 2017).

Epidemiology and Risk Factor Modeling

Returning to appendix B indicates two related aspects of the relevant data, what are the risk factors and how should the risk factors be modeled. This lays out twin objectives for the review of the epidemiology. First, what data are to be collected. Second, how to model the data in the statistical analysis. Get either of these steps wrong and the entire analysis is foiled. Appendix B considers five ways to model variables: binary, multinomial, continuous, count, and 'other'. This last category is both open ended and realistic, underscoring the necessity of consulting the established research and understanding the collected data. Appendix B also groups risk factors into five categories: individual, family, housing, environmental, neighborhood. This dissertation tracks a similar path by grouping variables into child-focused (the appendix B categories of individual and family), housing-focused, and geography (appendix B categories of environmental and neighborhood). However much the researcher may wish to, it is seldom possible to gather health data as one would shop at the store, bringing a list and return with everything on that list. Much of what one would like to have is not available and what is available can only be accessed after much relationship building and legal oversite. Chapter 6 discusses the steps involved in gathering health data for statistical analysis and the characteristics of the gathered data.[7]

[7] Separating the data focused activities of chapters 5 and 6 implies that all the work in chapter 5 comes before any of the work in chapter 6, but that implication is incorrect. The activities described in these two chapters are largely coterminous. The separation into distinct chapters is done for clarity of exposition. Chapter 6 could be placed before chapter 5 with no significant change to the logic or the realism of the exposition.

Acknowledging the limits of data collection is important, shortcomings should be kept in perspective. Wilson and Wilson discuss the effect of uncontrolled confounding in some lead research—a practice that, though they do not use the term, can lead to omitted variable bias (I. H. Wilson and Wilson 2016). Some degree of omitted variable bias is inevitable in lead poisoning research so long as 'wrap around' observations of individual behavior and the environment are unavailable. The task then is to make a sincere effort to gather all the relevant data and be transparent about what it is and isn't.

Child Focused Variables

Appendix B lists twenty-one possible risk factors associated with the individual child subset into 'Individual' and 'Family' groupings. It is not possible to gather data on each of the family subset of risk factors listed in appendix B, the list was intended as a brainstorming device and as a set of aspirational goal. The list of family risk factors includes: Family history of (lead poisoning, thumb sucking, pica), History of traditional high-risk practices (make up, jewelry, spices, dirt eating, gum sharing), Marriage status, Income, Insurance type, Race/ethnicity, Maternal IQ, Family violence, Socioeconomic status, Sibling with lead poisoning, Family members with high-risk occupation. Although any of the listed variables are plausible risk factors, each of them can be found in the epidemiology literature (Berney 1993; Whelan et al. 1997; Morales, Gutierrez, and Escarce 2005; D. C. Bellinger 2008; I. H. Wilson and Wilson 2016; Muller, Sampson, and Winter 2018), they are not equally important as risk factors. For instance, 'marriage status' is linked to lead poisoning through a relation to poverty which is linked to lower quality housing which is linked to lead paint which is linked to lead poisoning. Meanwhile, 'family history of lead poisoning' is directly connected to an

observation of lead poisoning. Furthermore, when we consider the causal chains connecting 'family' risk factors to lead poisoning most of them move through the epidemiologically identified exposure vectors of housing and soil. Only the risk factors of 'high-risk practices' (the use of make-up or spices containing lead) and 'family member with high-risk occupation' (an occupation that brings one in direct connection with lead particulates) represent vectors of lead exposure not covered by housing, soil, or community district.[8] The modifying risk factors associated with the family category are good objects for a research agenda but their absence from this study does not devastate the statistical inference. The absence of socio-economic and race/ethnicity data is troublesome for methodological reasons; it is important to disaggregate by race and class to describe our social divisions regarding lead poisoning quantitatively. This dissertation was designed to serve as a base from which such investigations can be made.

The 'Individual' subset of the child focused variables can give a behavioral or biological clue to actions that typically increase exposure to lead. The list in appendix B runs ten deep: Age, Race/ethnicity (increased risk in AA, increased risk with some traditional practices, increased risk of renal involvement in some races?), Gender, Obesity, Asthma, Developmental Delay, Persistent hand to mouth behavior, Pica, IQ, Foreign born. Each of these variables are well represented in the epidemiology literature (Nevin 2000; Morales, Gutierrez, and Escarce 2005; Sandel et al. 2010; Nigg et al. 2010; Dapul and Laraque 2014; Fatima et al. 2016; Muller, Sampson, and Winter 2018). Some of these variables (Asthma, Developmental Delay, IQ, etc.) are understood as outcomes of lead poisoning rather than causes. The causal

[8] Those risk factors are characteristic of individual agency that, though focused on individual outcomes, is largely lacking from this analysis. This lack is a shortcoming of this analysis.

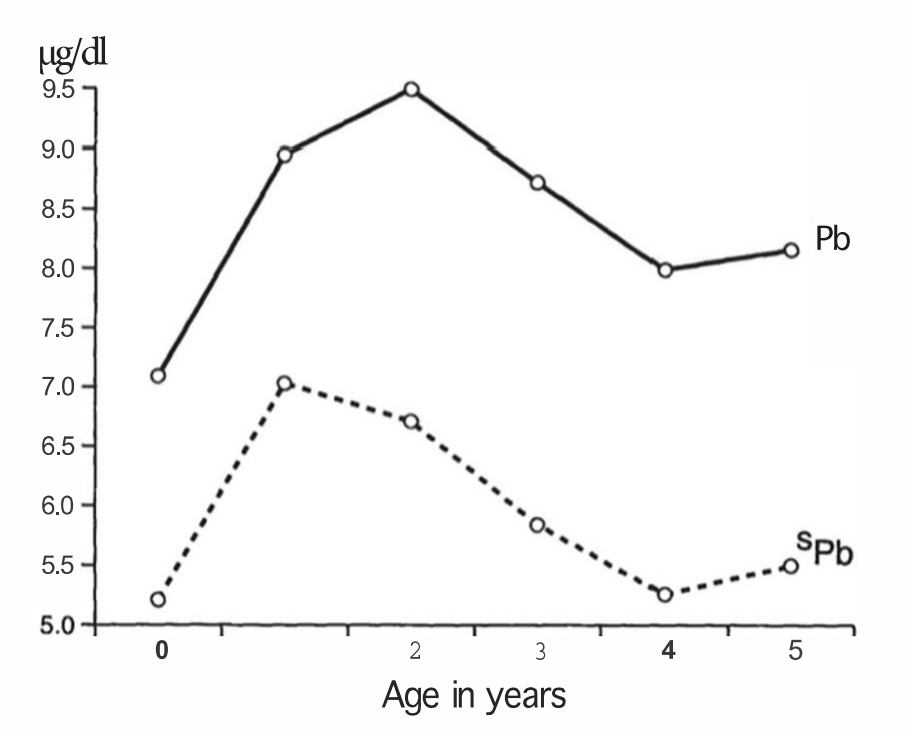
relationship is however irrelevant when gathering data. One of the contributions of this dissertation is a dataset intended to be investigated with an eye to new research agenda.[9]

Four child focused variables are gathered for statistical analysis, two of which, though they can be found in the epidemiology literature, are not found in appendix B. The variables are age (in months), sex, test type, and year of test. What follows is a brief review of how each of these variables are treated in the literature and how they will be treated in the statistical section of chapter 7.

As discussed in chapter 3, the susceptibility of a child to lead poisoning changes with their biological development. Moving from being completely dependent on parents, passing from crawling to walking, building the growing body in the unique manner of early childhood, gradually ageing out of the habit of putting hands and all things (potentially covered or consisting of bioavailable lead) into the mouth, these behaviors which overlap and periodize a child's life are reflected in mean blood lead levels. An age-wise pattern has been observed in the lead epidemiology since at least 1979. Figure 5.1 reproduces a typical graph of this relationship (Griffith et al. 1998). Other graphical representations of this relationship displaying the same pattern are found throughout the literature (Billick, Curran, and Shier 1979; Lanphear et al. 2002; S. Clark et al. 2011). Figure 5.2 demonstrates a similar pattern: increasing blood lead levels reaching a peak around 24 months, falling levels to about 48 months and then slow growth again towards a steady level.

[9] The Obesity variable, for instance, is related to nutrition and food insecurity, a research vector with implications for public policy. Researching for potential biological differences in renal involvement is important to rectify potential issues of medical racism regarding lead poisoning - though some researchers insist there is no medical reason to include race as an explanatory variable (Jones 2012).

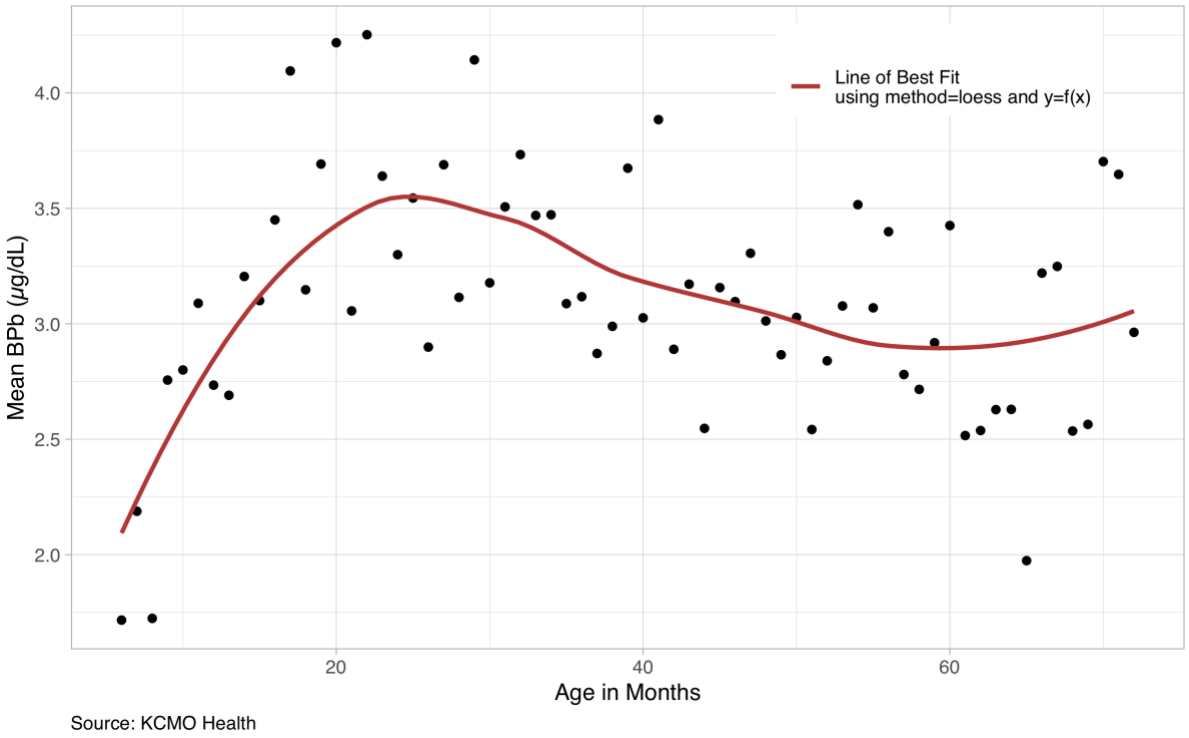
Figure 5.1: Variation in BPb by Age in Griffith et al. (1998)



Pediatric age variation in screening test results for Onondaga County: average (Pb) and standard deviation of µg/dl blood-lead level.

Recognizing a pattern exists leads to issues of how to model the data such that the pattern is accurately represented in the statistical investigation. There are two modeling decision regarding the age of child that are common and have been mirrored in this analysis. The first is to focus only on pediatric cases, the second is to mirror the pattern observed in figures 5.1 and 5.2. There are several reasons to look only at pediatric cases. The developmental aspect of young children makes the pediatric experience particularly important; the behavioral limitations of children means it is plausible to model their experience in terms of their environment without special consideration of individual agency, there are medical and legal guidelines that generate BPb tests for the pediatric population whereas blood lead observations outside of the pediatric range reflect a likelihood of elevated blood lead. Taken

Figure 5.2: Variation in BPb by Age in KCMO Health Data



together it is common for studies to terminate at 72 months (Billick, Curran, and Shier 1979; Griffith et al. 1998; Rabito et al. 2007; Oyana and Margai 2010; Jean Brown et al. 2011) and begin at 6 months (Lanphear et al. 2002; Rabito et al. 2007; S. Clark et al. 2011) or 12 months (Billick, Curran, and Shier 1979; Caldwell et al. 2017). In my Investigation the data provided by KCMO Health are analyzed between the ages of 6 and 72 months.

After selecting an age range the question becomes how to model the age of child such that it can be understood in the context of an ordinary lead squares (OLS) model. Figures 5.1 and 5.2 demonstrate the non-linear relationship between BPb and Age, though age in months is a continuous variable it is inappropriate to model it as such. Still some have modeled age as a continuous variable (Zahran et al. 2013). It is far more common for researchers to divide the age variable into several dummy variables. Two strategies are generally used to create these

dummies; intervals of equal length, for example 0-12 months, 13-24, 25-36, 37-48, 49-60, 61-72 (Billick, Curran, and Shier 1980; Lanphear, Burgoon, et al. 1998; Lanphear et al. 2005; Oyana and Margai 2010), or intervals of varying length that reflect the developmental stages children move through, for example 6-11 months, 12-35, 35-72 (Rabito et al. 2007; DHD 2017). A third strategy, and the one utilized in the statistical analysis in chapter 7, models age of child with a cubic specification (where a single measure of the child's age becomes three variables age, age^2, and age^3 thus reflecting the relationship observed in figures 5.1 and 5.2. Though less common than the dummy variable approach the cubic specification of child's age can be found in well cited peer reviewed literature (S. Clark et al. 2011). My analysis is exploratory and modeling decisions such as this are subject to judgment on the basis of their veracity (judged by out of sample predictions, etc.).

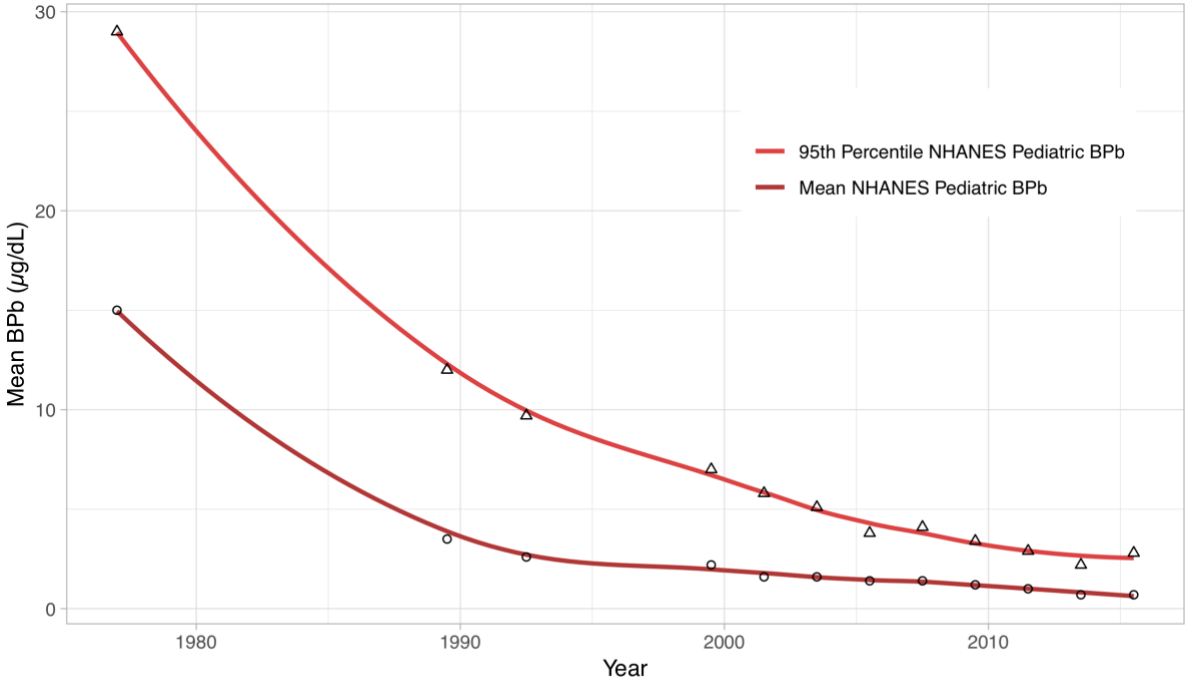
It is common for the sex of the child to be observed and included with observations of the blood lead levels. There is no evidence that any observed difference between the sexes regarding blood lead levels is based in biology (Lanphear et al. 1996; Lanphear, Burgoon, et al. 1998). Nevertheless a dummy variable indicating the sex of child is frequently included as part of statistical analysis (Romieu et al. 1992; Lanphear, Burgoon, et al. 1998; Lanphear et al. 2005; Rabito et al. 2007; S. Clark et al. 2011). Sex being included in the KCMO Health data and the penalty for including it being minimal a dummy variable for sex of child is included in my statistical analysis.

The BPb test type, capillary or venous exam, is included in the KCMO Health data. The inclusion of this data may reflect Missouri state guidelines that differentiate between the two test types, “If the initial test is obtained by Capillary fingerstick, and the result is 10µg/dL or greater, it MUST be confirmed by venous blood…” (MO Department of Health and Senior

Services 2008, emphasis orriginal). The differentiation exists in the guidelines because the potential for lead on the skin to be mixed in with the capillary blood sample, not because there is a difference in the sensitivity or a bias in the results of either exam type (Schlenker 1994; Parsons, Reilly, and Esernio-Jenssen 1997). The availability of data on test type and the minimal penalty for including an additional variable support the inclusion of a dummy variable indicating test type. Because a large number of observations of test type are missing (~8%) a second dummy variable is included to indicate a missing observation of blood lead test type. The inclusion of these dummy variables reflects the heterogeneity of the KCMO Health lead data.

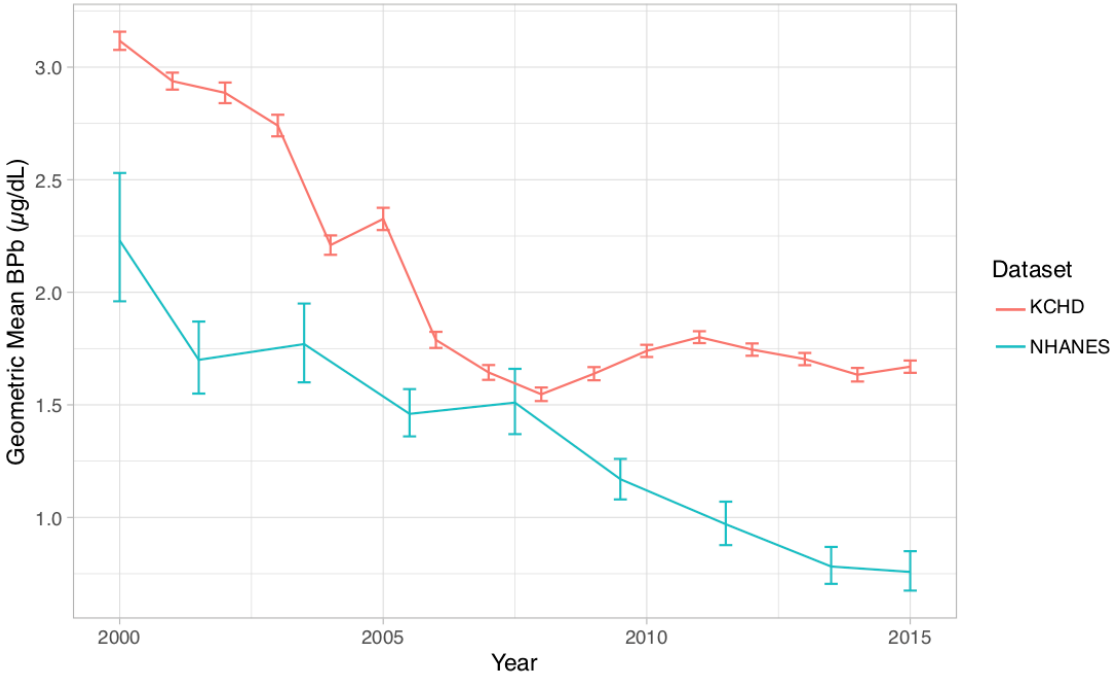
Additional heterogeneity follows from the length of the study period, fourteen years is longer than most observational studies. The fall in mean pediatric blood lead levels over time demonstrates is associated with the success of lead regulations. Figure 5.3 illustrates the decrease in BPb at the national level for the 1976-2016 period while figure 5.4 illustrates the same phenomena for the Kansas City geography for the 2000-2013 period. This trend, though persistently downward, is not best represented by a straight line, it follows a mitigated path of exponential decay. It is important to account for this trend even if the state of our knowledge about its persistence is that it reflects aggregate unobserved factors that drive down elevated blood lead levels homogenously across space over time. Several scholars have noted the importance of the year over year fall in population BPb mean for modeling outcomes (Lanphear, Matte, et al. 1998; S. Clark et al. 2011) however, when a study is focused on only a few years of observation it is uncommon to observe the year of test modeled explicitly. Among the research that takes account of the year of testing the most common practice is to

Figure 5.3: Falling BPb levels in NHANES Data



Source: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

Figure 5.4: Falling BPb in KCMO Health Data



Source: National Health and Nutrition Examination Survey, Kansas City Health Department

separate the analysis by year, running multiple statistical tests one for each year (Billick, Curran, and Shier 1979; Oyana and Margai 2010). Others incorporate year of test through a series of dummy variables (Jones 2012). In modeling the year of test, I transform the year variable to reflect the observed relationship using a cubic specification. As noted with regard to year of test, my analysis is exploratory and open to revision on the basis of warranted judgements.

Housing Focused Variables

Although the manufacture and distribution of lead paint and leaded gasoline are both effectively prohibited in the United States (1977 and 1986 respectively) there is no consensus regarding which source's environmental legacy (C. S. Clark et al. 1985; Jacobs 1995; Lanphear et al. 1996) is more responsible for ongoing lead poisoning. Sorting through this issue is important when designing intervention policy (is it more important to focus on remediating soil or housing?). It is also important for legal liability reasons. Recent legal judgments against the manufacturers of lead paint have turned on the capability of tracing the chemical fingerprint of ingested lead to either lead paint or leaded gasoline (Savage 2018). Appendix B separates paint and soil into different categories. Lead paint is grouped into the housing category as are many other lead poisoning correlates. Some of these variables require a more intensive observational regime than is possible when analyzing an entire city over the course of 14 years (date moved in, how many nights per week at this address, pets, flooring, clutter inside). Other housing related variables potentially could be gathered (type–single, duplex, etc., registered rental, basement, mixed use, yard condition, clutter outside) but are peripheral to the focus this analysis. A final selection of variables (age, structural integrity, renovation/repair history) are

well represented in the epidemiological literature and are included in this analysis though they are not referred to in those terms.

Age of housing is the most widely used variable to identify and describe the risk of lead poisoning from the built environment (T. Dignam et al. 2019). The literature establishing its risk to children[10] from housing is voluminous and rather than attempt a review here I will instead highlight the diversity of ways age of housing has been modeled with the end in view of choosing a modeling approach. The US CDC and the Environmental Protection Agency, in their public statements, describe 1978 as the year before with housing is "likely to contain lead paint" and do not further subdivide risks by age of construction (Center for Disease Control 2020).[11] Many scholarly publications use 1978 as the cutoff year to construct a dichotomous variable for age of housing (Patridge et al. 2006; Gulson and Taylor 2017). It is more common among those researchers who use a single dichotomous variable for era of construction to use an earlier date, 1950, associated with the end of the use of interior lead paint (Davies, Watt, and Thornton 1987; Sargent et al. 1995; Diorio 1999; Farfel et al. 2003; 2005; Patridge et al. 2006; Jones 2012). Still others use different specifications for the critical year dividing the era of construction into discrete groups such as the end of World War II (C. S. Clark et al. 1985) or the year 1940 (Mielke et al. 1997).[12] Some researchers have chosen to use several variables to subdivide era of housing construction, the issue then becomes which years divide the era of

[10] n.b. The establishment of this relationship is associative, from consistency of results rather than through designed experimental results.

[11] This is reasonable with an eye to the public purpose of these declarations to warn the public and advise people to take additional steps for their own safety. A distinction between risks is not relevant here as the same advice is relevant for all homes built before 1978.

[12] In analysis performed at the census tract level 1940 is a popular variable due to its availability in the US Census.

construction meaningfully and thus define the variables. 1950 and 1978 would seem the relevant dates though some researchers use 1940 (Jacobs et al. 2002) to divide the early years and others choose to subdivide year of housing construction into more than three era (Jacobs and Nevin 2006). The reason for subdividing age of construction into distinct eras is clear; though some researchers model age of construction as a continuous variable (Rabito et al. 2007) the association between age and lead hazard does not change continuously but rather leaps at well-defined moments in consort with regulatory changes in the lead paint industry. Whether the changes are voluntary as was the case with the lead paint industry choosing to end the production of lead-based interior paint in 1952, or from government regulation as in the prohibition of exterior lead based paint for housing in 1978, there are clearly demarcated moments when the degree to which lead was built into housing changed significantly.[13] Those two years, 1952 and 1978, are chosen to construct three binomial variables identifying various eras of lead-intensive housing construction.

A relationship between the renovation of homes with lead paint and elevated pediatric blood lead levels has been identified in the literature (S. Clark et al. 2004). The causal chain relates renovation or remediation activities to the disturbance the dormant toxin making it acutely (and for the child passively) bioavailable leading potentially to lead poisoning. Such observations were incorporated into the EPA Lead-Based Renovation, Repair and Painting Rule which establishes training and certification programs to stem the threat from such activities (Advisory Committee on Childhood Lead Poisoning Prevention 2012). The expected

[13] A third era might be defined by 1986, the year lead water service lines were formally banned in the United States. Lead service lines are different enough from lead paint, in terms of the way they age and the way their 'secure' lead becomes agitated to pose a threat to children, nevertheless they are not considered in this analysis. That is not to imply that they are not a potentially significant lead hazard (Jacobs et al. 2010).

association between renovation and BPb is ambiguous. There is the potential for skilled contractors to perform the relevant work safely while simultaneously there is no guarantee that such skilled contractors will be hired. This is a reasonable topic for investigation which a paucity of data impairs; there is no comprehensive method for tracking home renovations while observing the qualifications of the people doing the rehabilitation work. But there is at least one instance where observations of renovation activity do exist, public housing constructed during the earliest era of lead paint was largely renovated to completely remove lead paint hazards in Kansas City during the middle 1990's (Milstien 2017). The importance of this classification for researchers is reflected by David Jacobs and coauthors who stipulated in their research that renovated public housing should be considered a distinct class of housing regarding its status as a lead hazard (Jacobs 1995). While I do not designate homes in renovated public housing as a distinct era of housing in this analysis, information about renovations contributes to the allocation of homes into the three eras of housing as identified in the previous paragraph.[14]

The literature relating lead poisoning to housing conditions is vast, what follows is an overview of significant papers with an eye towards the interaction of age of construction and condition of structure. Critical questions include: are those variables considered in consort, how is condition of home observed, are there other significant modeling decisions that underly the analysis?

Clark et al (1985) observe patterns in blood lead levels charted against condition and type of housing. The authors also include a rough age of housing variable to help describe the housing in question. They find, “the highest blood lead levels were found in children living in

[14] For more on the process of determining era of housing construction see chapter 6.

pre-World War II housing in deteriorated or dilapidated condition, intermediate levels were detected in children in rehabilitated housing or other well-maintained older housing, and the lowest levels were found in children living in public housing and other recently built private housing" (Clark et al. 1985, 51). In this study the condition of the home was determined on the basis of a 'windshield' study. Clark et at (1985) also note that age of housing is not a pure condition in its own right, "In the subsidized rehabilitated houses, older woodwork was almost always replaced by standard grade new lumber, and all rehabilitation work was performed while the building was unoccupied" (Clark et al. 1985, 48). By this they mean that rehabilitated public housing constitutes a different class of housing than unrehabilitated housing. It is also an acknowledgement, early in the history of the literature, that year of construction and condition of structure are informative when combined. Chisolm et al. (1985) come to a similar conclusion when observing recovery patterns in children with elevated blood lead levels after chelation therapy (Chisolm, Mellits, and Quaskey 1985).

Developing a method to identify communities at risk for lead poisoning without having to wait for children to be identified as lead poisoned motivates much of the research connecting housing and lead. Sargent et al. (1995) utilize the high rates (58%) of testing in early 1990'w Massachusetts to develop a census track level analysis that could be used to pick out at risk communities. They look at children from urban, rural, and suburban communities and find "lead poisoning in all types of communities". The authors note that "children living in communities with high rates of poverty, single parent families, and pre-1950's housing and low rates of home ownership were 7-10 times more likely to have lead poisoning." (Sargent et al. 1995, 531) One census variable, % of children 5 or younger living in poverty, identifies 71% of all lead poisoned children in their data. The authors note the importance of deteriorating

lead paint and owner occupied status, writing "we suggest that old homes that are not owner occupied are more likely to be allowed to deteriorate and to release lead-based paint into the environment" (Sargent et al. 1995, 532), but they do not incorporate observations of paint condition into their work.

Jacobs (1995) reviews three types of scientific papers (case study reports of the effect of disturbing or abating lead-based paint, studies of environmental correlates of blood lead levels in children, and analytical source identification studies through use of stable isotope ratio techniques) supporting the importance of lead paint vis a vis lead poisoning. His article informs strategies to reduce or eliminate exposure as well as legal reasoning regarding responsibility for the cleanup of toxic material. The author uses strong language to describe the importance of the interaction of era of construction and lead paint.

> It should be fairly obvious that intact paint does not stay that way. In fact, there are a number of deteriorated paint conditions formally used by the painting and decorating industry to describe routine paint failure. … The idea that old lead-based paint will remain intact forever and not become available for ingestion, especially in dilapidated housing is naïve at best. … The current weight of the scientific evidence indicates that failure to control lead-based paint in older dwellings will result in continued exposure to lead for a large number of children. … lead-based paint will eventually deteriorate or be removed through renovation and repainting activities and re-enter the pathways, presenting an immediate hazard.
>
> (Jacobs 1995, 182)

The author notes that so long as lead paint remains in housing vigilance is required lest some child suffer needlessly from lead exposure.

Focusing on quantifying a relationship between lead in house dust and elevated BPb (≥10μg/dL) Lanphear et al examine a cohort from Rochester, NY. After accounting for significant covariates (race, paint condition, parental educational level, soil lead level) they determine elevated blood lead levels to be found among children in homes that passed HUD's

post abatement lead-dust clearance level (Lanphear et al. 1996). Elsewhere Bruce Lanphear and co-authors estimate the importance of several pathways (water, soil, paint and dust) bringing lead into the child's body. With regard to paint condition the authors find that "the percentage of children with elevated blood lead levels (ie ≥ 10µg/dL) increased significantly as the condition of painted surfaces deteriorated."(Lanphear, Burgoon, et al. 1998) 15.4% of the children with "good" condition paint were observed to have elevated blood lead versus 39% with paint in poor condition. The authors suggest that paint condition may be a better indicator of health risk than the lead content of the paint alone.

Another article pools 12 previous studies into a single regression estimating the importance of lead-loaded interior dust. Variables considered include exposure from exterior soil, maximum observed interior paint lead content, household water lead level, paint condition (dummy variable), child's race, child's age, family socio-economic status, child's mouthing behavior on blood lead levels. The authors find that "in the multivariate regression, floor dust lead loading was the most significant environmental predictor of children's blood lead levels. To a lesser extent, lead-contaminated soil contributed to children's lead intake. Child's age, mouthing behaviors, and race were also significant predictors of children's blood lead levels" (Lanphear, Matte, et al. 1998, 57). The authors note the imperfect nature of their associative model (versus a mechanistic model) for estimating environmental impact on lead levels, while also noting the importance of lead from car exhaust is not included, nor is the general downward trend in BPb accounted for directly. The authors conclude by emphasizing their findings that understanding housing conditions are crucial for identifying the principal cause of lead poisoning.

Inspired by the surfeit of evidence connecting lead in housing to lead poisoning, Jacobs et al estimate the number of homes with significant lead to be 24 million (±2.7m) from a nationally representative sample of U.S. Housing (Jacobs et al. 2002). They find no distinction between urban, small urban and rural settings regarding likelihood of lead exposure. Rental status is slightly more likely to indicate a lead-based hazard. Critically for this dissertation they find soil lead levels to be related to deteriorated exterior paint, "comparing units with and without deteriorated exterior lead-based paint, the percentage of units with bare soil levels ≥ 1200ppm from 24% to 4% respectively" (Jacobs et al. 2002, A603). They credit trends in demolition, housing rehabilitation, lead hazard control, and 'other factors' for the decrease in number of homes with lead hazards before closing with a discussion of the costs and benefits of remediation. They find that the benefits of remediation out way the costs of lead poisoning nearly four to one.

Lanphear, Hornung, and Ho (2005) use housing-based observations—lead in dust, condition of the home, and rental status—to develop a predictive model which can be used to identify homes with lead hazards. Their motivation is to develop a technique for identifying and remediating lead-based hazard in homes without waiting for children first to become lead poisoned. Their variable for housing condition is bimodal, good/bad, and it is not clear what their criteria for judgment is. They found, "By itself, poor housing condition identified 47% of children with a blood lead level ≥10 μg/dL" (Lanphear, Hornung, and Ho 2005, 307). The authors interpret their finding to imply a tradeoff between housing-based lead-hazard interventions and lead poisoned children. When funding these programs, the relevant consideration then is how many lead poisoned children are you willing to allow. But the authors note this tradeoff is an accurate description of existing lead policy. The emphasis on housing

condition adds a wrinkle however as it implies the relationships between housing and poisoning are not stable. Housing conditions in their analysis matter no less than era of construction.

The effectiveness of seven interior housing-based lead hazard remediation strategies are reviewed for the US Department of Housing and Urban Development (HUD) in Clark et al. (2011). The authors discuss the effectiveness of the intervention strategies finding the largest decrease in BPb coming with those children that had the highest BPb levels, those in the lowest range of BPb had no observable improvement with intervention, and more extensive lead removal results in statistically lower BPb rates. The confounding factors of age, seasonality, housing condition (bimodal based on visual on-site assessment), as well as neighborhood pollutants are also included in the analysis. Their study observed the importance of exterior lead paint on long term BPb. With or without interior interventions exterior paint conditions are associated with higher observed BPb, without modifying the analysis by era of home construction.

Jones (2012) is concerned with understanding the impact of HUD sponsored lead-hazard interventions on blood lead levels and subsequent health outcomes. Understanding interventions will help understand the relative importance of hazards in the build environment. His census track level analysis shows that HUD remediations yields a return on expenditure between 2:1 as a lower bound and 20:1 at the upper bound. His findings track with all the published reports (Stefanek, Diorio, and Frisch 2005; Nevin et al. 2008; E. Gould 2009; Billings and Schnepel 2017a; Zhou et al. 2017). Jones writes that, “benefits from remediations and the resulting reduced lead exposure in children far outweigh the costs of the necessary lead hazard treatments” (Jones 2012, 151). In calculating the benefits to society his estimates

emphasize lost earnings and medical care while the costs identified are the price of intervention activity. Jones notes that there is a general decrease in bll over time and includes a dummy variable for the year in which the test took place. To assess the condition of housing at the census tract level he computes a 'health of housing stock' statistic by taking the number of pre-1978 housing units that have been remediated and dividing by the total number of pre-1978 housing units by tract and by year. He estimates several regression models where the dependent variables are a count of children with elevated blood lead levels at the census tract level. He arrives at a conclusion regarding age and housing condition that is germane to my dissertation.

> Lead poisoning increased with the age of the housing stock but then decreased in areas with the highest percentage of older housing, perhaps reflecting the greater wealth concentrated in areas with very old housing in Chicago. The finding also provides evidence that the condition of the housing is far more important than the age, particularly in a city where the majority of housing units were built prior to 1950 and contain some amount of lead-based paint. The age variable was most likely acting as a proxy for condition in other lead studies that found large, significant effects.
>
> (Jones 2012, 163)

Here, Jones confirms two facts that inform the modeling of housing variables: homogeneity of housing age by geography and the necessity of interacting age and housing condition.

Explaining variation in BPb levels with regard to the housing portion of the built environment requires more than an age of construction variable. In many neighborhoods there is not adequate variation in age of house for the variable to be informative. Second, while all housing built during a particular era of can be thought of in the abstract as posing a uniform risk of lead-exposure, the maintenance of a home makes a substantial difference in the risk posed to inhabitants. Familiar lead risk correlates like household income, property value, and rental status should be understood as instruments for housing condition. Previous attempts to incorporate housing conditions have been hampered by the difficulty of gathering housing

condition data relying, for instance, on small samples (C. S. Clark et al. 1985), aggregate geographic boundaries (Jones 2012), or the observational overlay mapping of density surfaces (Schuch, Curtis, and Davidson 2017). One of the unique aspects of my analysis is the volume of parcel level housing conditions data I have to draw upon via the CEI's neighborhood housing conditions survey (NHCS). The NHCS has observations about five different housing conditions, utilizing a multi-level ordinal scale to describe each housing condition.[15] The major contribution of this dissertation is to model the importance of housing conditions on pediatric BPb through the interaction of era of housing and age of housing at the parcel level. I do this by interacting the dichotomous era of housing variables with three dichotomous variables reflecting exterior paint conditions. Multiplying these dichotomous variables creates an additional six dichotomous variables which describe the confluence of era of construction and exterior paint condition for each home. Also included in the analysis are dichotomous housing condition variables which have not been interacted with era of housing describing the condition of windows and doors as well as the condition of porches. In total I use 14 housing focused variables to describe the housing-based lead risk each child experiences in the built environment which surrounds them.

Geography Variables

The two categories mentioned in appendix B, Environmental and Neighborhood risk factors, are analogous to the variables grouped in this dissertation under the heading geography variables. Soil as a medium for lead poisoning is now a prominent area of focus for health

[15] The NHCS utilizes a five-level scale in its observational practice. This five-level scale is reduced to a three-level ordinal scale for use in my analysis. For much more about the NHCS and a discussion of the transformation of the five-level scale to a three-level scale see chapter 6.

researchers (Mielke and Reagan 1998), deserving the sobriquet ‘the elephant in the playground’ (Filippelli and Laidlaw 2010). Most of the Environmental and Neighborhood risk factors mentioned in appendix B can refer in some way to the possibility of lead loaded soil (street condition, land use, rural/urban, nearby road work or construction, landscaping, yard condition, proximity to busy streets, vacant lots). A second grouping describes the active creation and exposure to new sources of lead poisoning (Air pollution, demolition, distance to school, neighborhood socioeconomic status). For the first subset of geography variables, an ideal measure is the direct observation of soil lead levels (Markus and McBratney 2001). Direct observation of lead in soil is the basis for research in Baltimore, MD (Schwarz et al. 2012), New Orleans, LA (Zahran et al. 2013), Oakland, CA (McClintock 2015), and elsewhere (Markus and McBratney 2001). Unfortunately, systematic city-wide observations of soil lead levels do not exist for Kansas City, MO, and collecting such information is outside the scope of this dissertation. I turn instead to indirect observations of lead in soil, looking at geographic proximity to historic sources of lead in soil, structural demolition activity, seasonality (which affects the bioavailability of lead in soil), and community district.

Automobile traffic, Gas Stations, Community Districts, and Seasonality

In the 1970's environmental scientists determined conclusively that leaded gasoline or TEL (Tetraethyl Lead gasoline) had increased the amount of lead in the built environment by several orders of magnitude over pre-industrial levels. Significant work took place to show that the concentration of automobile traffic was able to predict differences in lead levels between census tracks that were otherwise similar (Mielke et al. 1989). Other work has shown the necessity of augmenting studies of lead dust levels with a measure automobile traffic (Sutton

et al. 1995). TEL was a major scientific breakthrough which helped unlock the potential of the internal combustion engine. It allowed for larger, more powerful engines. Following its introduction in 1923 the automobile took on its character as one of the transformative technologies of the 20th century. The efforts to understand, regulate and ultimately ban TEL is an 80-year story (discussed in chapter 3). As Nriagu (1990) notes, TEL is one of the few environmentally unsafe products to be forced entirely out of the marketplace. The footprint of this environmental toxin is not intuitively obvious. In the absence of documented lead soil measurements many researches have worked towards useful proxy measures for lead soil levels.

Early studies (Lagerwerff and Specht 1970; Motto et al. 1970) looked at the relationship between Pb and traffic with a different emphasis than later work. The preponderance of evidence had not yet accumulated and so sentences like "[h]azards due to environmental Pb have variously been emphasized (Patterson, 1965) and deemphasized (Kehoe 1961) and are clearly subject to controversy" (Lagerwerff and Specht 1970, 583) were still warranted. The emphasis in these early articles was on establishing the source of lead in soil and plant life and describing the relationships. Because these articles are from before Clair Patterson established the modern scientific clean room standards (Settle and Patterson 1980) the exact figures in these studies are suspect, however, they demonstrate that highways and the lead pollution from exhaust are the principle source of lead in the surrounding soil. Similarly, the early articles helped establish that lead in the environment increases with the volume of traffic and in proximity to the roadway. These tests were performed during the era of leaded gasoline, so there was no reference to a normal level of exposure nor any accurate measurement

of a lower bound to exposure. Such findings would have to wait for the adoption of contemporary laboratory procedures and the end of the TEL epoch.

In 1988 just after TEL was phased out of use in the USA, Howard Mielke and co-authors began to study census tract level relationships between BPb, traffic density, age of housing, and size of city. Their report indicates that traffic volume and density together were a better indication of elevated BPb than age of housing on its own (Mielke et al. 1989). The article suggests that the traffic flow observations from a single state, Minnesota, are applicable to the rest of united states. The article also includes careful language that describes how paint condition of individual homes cannot be a causal factor for population wide lead poisoning. The prevalence of poisoned children living in homes far from lead painted housing can be explained via pollution from automobile exhaust. This distinction is relevant to the fine-grained geospatial work that my research embodies.

A study of BPb in children in Bangladesh, from before that country phased out Leaded Gasoline in the early 2000's, adds information to our understanding. Dhaka, where this study was centered had some of the highest atmospheric lead levels of any city in the world. Of the children studied ~90% had BPb levels at or higher than 10µg/dl (The level then marked as elevated by the CDC). The authors note, "of the 10 associations examined, age, low parental education, close proximity of home to highway or intersection of major roads, and soil eating showed positive associations with crude and adjusted data" (Kaiser et al. 2001, 564). This work demonstrates the warranted use of proximity to roadways as an instrument for exposure to lead from TEL.

By 1997 Mielke could state authoritatively, “the legacy of accumulated lead in the soil of the urban environment is an especially important potential source of exposure to children”

(Mielke et al. 1997, 953). This Mielke et al. study looking at associations between soil lead (SPb) and childhood BPb develops a logic of geographic proximity connecting ambient sources of lead with soil lead levels. The authors conclude that busy urban intersections are associated with more soil lead particulates than rural intersections. Through regression analysis they compare census tract level soil lead (SPb), percent of housing built before 1940, and BPb. They find SPb and housing age both to be correlated in a statistically significant manner with BPb but that the BPb "response is 12 orders of magnitude stronger for soils than for age of housing" (Mielke et al. 1997, 953). This Mielke et al. article emphasizes the importance of modeling sources of lead in soil in addition to age of housing considerations and demonstrates the SPb modeling can be done with reference to distance and type of roadway.

A Danish study modeled the relationship between dental lead levels (measured in baby teeth) and proximity to traffic while also accounting for additional confounders. The researches find, "children with a high lead burden resided significantly more often in heavily-traveled streets than children with a low burden, but only during their first 3 years" (Lyngbye, Hansen, and Grandjean 1990, 419). The authors strike a familiar note acknowledging their modeled relationship "was based on limited theoretical considerations, but such difficulties in creating descriptive models for lead exposure from observational data are not unique, as lead exposures are chronic and irregular, and originate from several sources" (Lyngbye, Hansen, and Grandjean 1990, 421). Despite data limitations they report a robust relationship through an odds ratio of elevated BPb considering three categories of traffic volume (601 - 1500 cars per day, 1501 - 5000 cars per day, 5000 and up) versus a control of less than 600 cars per day. The researches also include in their statistical analysis several measures of socio-economic status, educational level of the mother, marital status of the family, occupational history, tobacco

smoking, and whether the child had a mouthing habit. None of the confounders they tested yield the dramatic relationship they found with intensity of traffic. The geographic distance between a home and the roadway used by the authors is ≤100m.

A pilot study of Mexico City children, published in 1992, also sought to establish the use of residential geographic proximity to traffic as a proxy for pediatric exposure to lead (Romieu et al 1992). They use OLS regression incorporating confounding factors (age, mouthing behavior, playing in dirt, eating canned food, time spent in traffic, sex of the child, drinking juice in lead glazed vessels) to investigate relationships between blood lead levels and the built environment. The authors find the main determinant of blood lead level was geography of residency. Children who lived on residential streets had significantly lower blood-lead levels (5µg/dL, sd2.1) than those who lived on large streets (7µg/dL, sd 1.5), or those who resided near a main road (7.5µg/dL, sd 1.9) (all at $p = .001$). This geographic variable accounted for 27% of the variability of blood lead levels in the population. Their result was unchanged after age and other factors were adjusted for (Romieu et al. 1992, 247).

Pioneering work on the relationship between BPb and TEL can be found in Billick, Curran, and Shier (1980), an article focused on the relationship between the volume of lead used in automobile fuels and mean blood lead level in New York City. The authors find "a highly significant association between geometric mean blood lead levels and the amount of lead present in gasoline sold" (Billick, Curran, and Shier 1980, 213). Their analysis divides a sample population to look separately at trends by sex and race such that the mean BPb of reasonably homogenous groups can be compared with the volume of lead in gasoline. Despite the strong relationship these authors find between TEL and BPb they insist total lead in the environment (TEL plus all other factors) is the relevant magnitude to focus on.

Other authors are more strident about the connection between SPb and BPb. Mielke, Laidlaw, and Gonzales (2010) insists that TEL is the principal source of lead in soil, and that soil is the principal source of BPb. Supporting their analysis is an estimation of the volume of lead emitted by automobiles for several major U.S. urban centers during the years 1950-1982. They then subdivide further by location within the city. The goal of their analysis, as with much epidemiological work, is to develop a map of the soil lead load which can in turn be used for prevention, planning, and remediation. Their paper also reviews the literature supporting the need for a comprehensive SPb mapping to begin addressing issues of environmental justice from the unequal distribution of lead in soil. Comprehensive mapping of SPb levels will facilitate the investigation of lead paint vs. TEL on soil lead levels, health effects of SPb including school performance, the seasonality of SPb exposure, and most important the possibility of arresting the process.

Without systematic observations of SPb in Kansas City my research utilizes geography-based proxy variables which capture some of the risks in the built environment associated with SPb. This proxy variable approach is in keeping with the Wang and Zhang (2018) who note there is no standard technique for predicting soil lead levels short of soil samples. Research which tries to incorporate the soil exposure vector but does not have access to soil samples must make warranted modeling choices informed by publications such as those discussed above. There is a literature, exemplified by Teichman et al. (1993), which quantifies soil lead levels by distance from highways while taking into account weather conditions like the direction of prevailing winds and changes in elevation. Such investigations connect with recent work on lead in soil using GIS to describe the spaces produced by elevated SPb (Markus and

McBratney 2001; Lejano and Ericson 2005; Schwarz et al. 2012; Mielke et al. 2013; McClintock 2015).

Soil lead as the product of human activity and a pathway of pediatric exposure finds expression in my dissertation via three geographic proximity focused variables. The first is a single dummy variable which indicates if the home of the child meets any of the following criteria: within 100m of a highway, 100m of a gas station from the era of TEL, 30m of a major road.[16] The second suite of dummy variables indicates the community district in which the child lives. The community district variables follows from the earlier discussion of the relevant level of analysis (Bailey, Sargent, and Blake 1998; D. Kim et al. 2008) and the GIS intensive literature on lead and soil. A third geographic variable reflects the seasonality of exposure via lead in nearby soil. If the ground is frozen or saturated with water the resuspension of lead loaded soil is unlikely. A seasonal pattern is observed in test results and commented on in the literature dating to the early 1970's (Billick, Curran, and Shier 1979; 1980). Much of the housing and soil focused work incorporates a variable to indicate the season in which the BPb test took place (Griffith et al. 1998; Haley and Talbot 2004; Laidlaw et al. 2005). The work explicitly focused on seasonality notes that the patterns of seasonality in lead poisoning are different depending on climate (Laidlaw and Filippelli 2008; Zahran et al. 2013) and that weather can be successfully incorporated in a model of SPb risk. In my dissertation seasonality is modeled via a single dummy variable indicating if the BPb test took place outside of the winter months December, January, February, March. These three geographic variables represent a first pass at modeling lead soil risk in Kansas City. A more sophisticated

[16] The inclusion of proximity to old gas stations as an exposure vector is inspired by Davies, Watt, and Thornton (1987). The choice of these particular distances is inspired by the breadth of the literature reviewed, there is no single standard distance though 100m can be found as a reference distance in the demolition literature.

investigation and modeling of these variables embodies research agenda somewhat distinct from my own. To the degree my dissertation adds to the reviewed research tradition it is via the assembly of data and professional relationships which could facilitate a more complete modeling of lead in soil.[17]

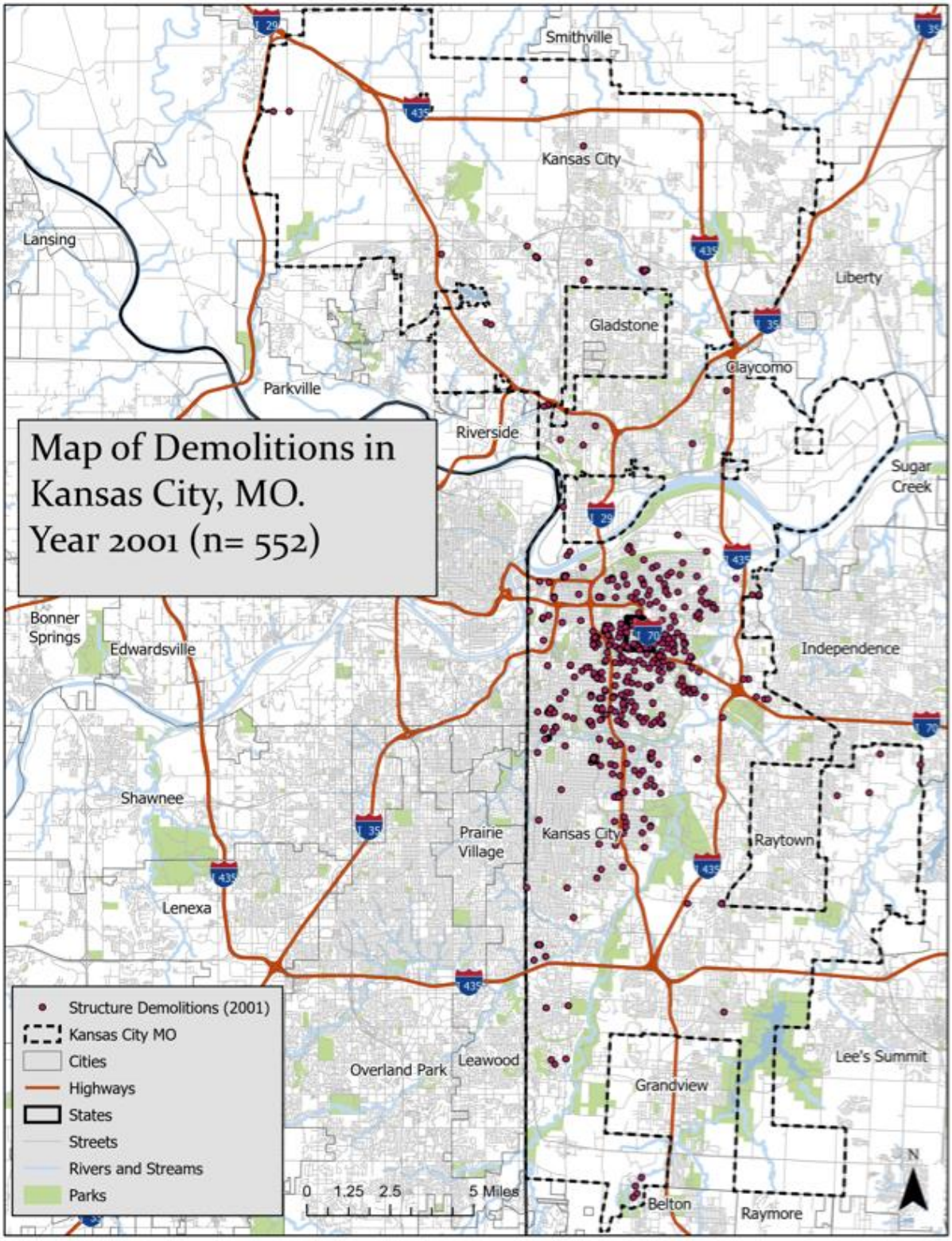
Geographic Variable: Structure Demolitions

A fourth geographic variable, hinted at in the appendix B brainstorming, is an indicator variable reflecting a structural demolition within 100m of the child's home address. As with the three proxy variables discussed above, the demolition variable I use in my statistical analysis is a first pass at modeling a complex phenomenon. I use a dummy variable to indicate if any demolition has taken place within 100m of the child's home within the child's lifetime. Initial investigations of the KCMO health data indicated its importance[18] and map 5.1 demonstrates that demolition activity is not evenly spread across the city in the year 2001. Map 5.2 demonstrates that demolition activity is not evenly spread across the city for the years 1990-2013. Chapter 6 demonstrates that this unequal distribution of demolition activity reflects the underlying geographies of race and class in Kansas City. For these reasons a larger literature review follows than might otherwise be expected. The incorporation of the demolition variable, even as a point of departure for later investigations, is an important contribution to the existing body of knowledge regarding lead hazards in Kansas City and urban environments more generally.

[17] Chapter 6 discusses the gathered data and data sharing partnerships in some detail. For a taste of weather modeling related to chronic disease in Kansas City see Kane (2020).

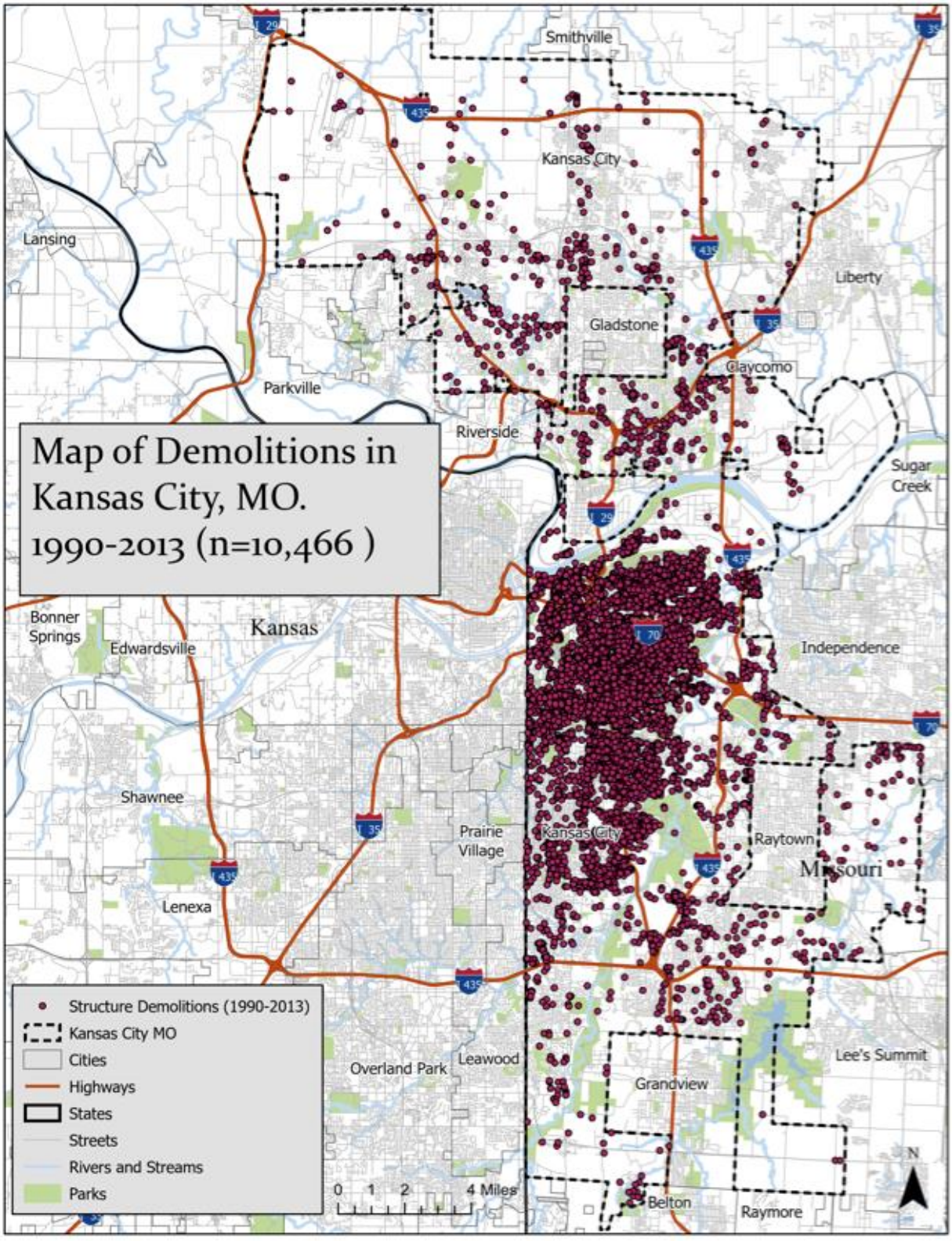
[18] CEI working paper 1801-03 (N. Wilson 2018a) and appendix H contain the preliminary investigations which early indicated the importance of the Demo variable.

Map 5.1: Structure Demolitions (2001)



Map created by author.

Figure 5.2: Structure Demolitions (1990-2013)



Map created by author.

The demolition of older housing stock can be a significant source of fugitive lead dust and lead-loaded dust can persist long after the demolition process has ended. The demo variable in my statistical analysis is a proxy for lead in air and for the lead loading of soil. Like all fugitive material it is possible for demolition rooted lead to migrate from original resting places to nearby homes and businesses—a phenomena first noted in the medical literature in 1903 ("The Dust of Buildings in Process of Demolition and Its Prevention" 1903). As part of a multi-dimensional study of lead pathways, Davies, Watt, and Thornton (1987) document an increase in lead in garden soil within ¼ mile of a demolition site. Their study hypothesizes that dust can move into homes (where the majority of exposure takes place) through open windows and doors or be tracked in on books, clothes and house pets, thus a positive correlation between exterior lead dust levels and interior levels will be found. Thirty years later, Gulson and Taylor (2017) tested this hypothesis using long duration petri dish samples (the technique relies on the passive collection of interior dust fall) to estimate BPb with precision. From these studies we can expect that demolitions and the lead loaded environment that follows will be associated with elevated pediatric blood lead levels.

Diorio (1999) indicates that, in the United States, there was no standard best practice to wet down the demolition site before 1999. The author also reports that demolition increases fugitive lead dust on neighboring houses by 109% even in the presence of surface wetting during demolition. Absent the wetting procedure amount of lead-dust on neighboring houses was nearly 5 fold (491% higher) baseline levels (Diorio 1999). Articulating a similar line of reasoning Farfet et al. (2003) write that an "[i]ncrease in lead concentration during demolitions [10m from a block of row houses] was statistically significant. The increase in lead concentration during debris removal compared with baseline was of borderline statistical

significance" (Farfel et al. 2003, 1232). More precisely Farfel et al report that, "Geometric mean lead dust fall increased [from 10µgPb/m2/hr] to 410µg Pb/m2/hr on an hourly basis and (from 62µg Pb/m2/sampling day) to 2700 µg Pb/m2/sampling day on a cumulative basis. An increase of more than *40 fold* above baseline. … During Debris removal, the lead dust-fall rate increased to 61 and to 440 for cumulative lead dust fall (a more than *6-fold* increase)" (Farfel et al. 2003, 1233, emphasis orriginal). Examining dust 100m from demolitions during demolition Farfel et al. (2005) report, "GM (geometric mean) lead loadings immediately after demolition were 200%, 138% and 26% higher that baseline levels for streets, alleys and sidewalks. … at site 1, 18% 18% and 29% higher" (Farfel et al. 2005, 211). The authors also find that levels of lead on surfaces 1 month after demolition exceed the EPA standards for interior floors by some 40-fold. There is no standard operating procedure regulating lead-based housing demolitions. Note that there may be heterogeneity among demolition events, reflecting variation in type of structure, weather conditions during the event, and the number and size of structures demolished. Note as well, demolition removal may spread lead dust along the disposal route from the job site to the disposal site.

Despite some early work on the subject, the process of developing our present state of knowledge about the relationship between demolition dust and BPb has been circuitous. A quotation about how this developed in Saint Louis is informative.

> In 2002, St. Louis City Lead detoxification crews began reporting to St. Louis City public health officials that children near demolition sites were showing higher than normal rates of elevated blood lead levels. Crewmembers stated that children, who had undergone Chelation Therapy to reduce blood lead levels, would return home, only to be back in the hospital within two weeks. Since most of their residences had undergone some form of abatement during the child's absence, they strongly suspected that airborne lead from demolitions was the culprit.
>
> (Patridge et al. 2006, 8)

Front-line health workers observed a pattern and formed a hypothesis which was then tested using GIS to associate elevated BPb (then defined as ≥10μg/dL) with any housing demolitions within a ¼ mile (~400m). At the 5% level of significance the authors were able to reject a hypothesis that blood lead test results are independent of demolition observations. Their result holds for areas where housing was predominantly from the era of lead paint, it is unclear if the study examined demolition practices where housing is from the most recent era of lead-free construction.

These same data are modeled more rigorously in Rabito et al (2007). The authors model relationships between BPb and demolitions, age of child, age of housing, race of child, and sex of child. The BPb and demolition are associated in space (census block) and time (within 45 days of the test) then modeled in two ways: as a dummy variable (any demolitions vs. no demolitions), and as ordered variable (no exposure/one exposure/many exposures). The BPb variable was log transformed in this analysis due to its non-normal distribution. Being exposed to a demolition was found to be associated with an increase in blood lead level (coefficient= .096; 95% CI = .009, .183; p-val =.031) But when they tested the other demolition specification, they found one exposure not to be correlated with higher BPb in a statistically significant manner (coefficient .07, 95% =.02, .16, p-val =.155) while many exposures are statistically significant with higher blood lead level (coefficient =.0296, 95%=.08, .51, P-val .007). In making sense of their results the authors mention five shortcomings of the analysis: the need for direct observation of housing condition, the lack of data on neighborhood characteristics, an inadequately simple spatial association between housing and demolitions, an overly simplistic model of dust dispersal, and the choice to select the highest BPb test result (in the case a child received multiple tests) potentially introducing bias into the analysis. The

identification of these short comings is a step towards building a research agenda focused on demolition activities.

A study in Chicago used the same sampling technique as Farfel et al. (2003) situated closer to the demolition site to observe the volume of lead in dustfall from the demolition of scattered, single family homes. The authors were able to observe a difference in background lead levels between Baltimore and Chicago without a ready explanation for the difference. With the demolition of single homes (rather than multiple homes as was the case in the Baltimore row houses) a non-linear decrease in the volume of the lead dust fall was observed. A 20-fold decrease was expected while the observed decrease was only 7-fold. The data gathered in the study is noisy, there is high variation in the measurements of dustfall at demolition sites (the dustfall range was 1.3-3902.5, mean 64.1, SD 776.2 µgPb/m^2/hr). (Mucha et al. 2009). The authors note, as we observe in the Kansas City demolition data, residential demolitions are not evenly spread through the city. The analysis was not able to distinguish between the lead dust produced by different types of homes.

Another study of single family demolitions in Chicago was published in 2012 (Jacobs et al. 2012). This study found the critical distance (beyond which there was no statistically significant fugitive lead dust) to be 400ft (120m). The authors also sampled for other heavy metals finding, "GM lead and cadmium were significantly greater in demolition samples than in background samples, and dust fall in µg/ft^2/hr was significantly higher for arsenic, chromium, copper, iron, lead and manganese in demolition samples" (Jacobs et al 2012, 458). Observations of air quality during demolition of housing following Hurricane Katrina indicate that lead is not the only pernicious substance to consider, Barium and Arsenic may be found in higher concentrations than allowed by relevant air quality statutes, also the pesticide Ziram,

as well as dangerous bacteria and fungi (Ravikrishna et al. 2010). We should be circumspect about generalizing too much from the specific post-Katrina housing conditions while also acknowledging the research in New Orleans supports the dangers of a demolition derived dust cloud.

The Health Department of Detroit, Michigan issued a report which tested for the impact of housing demolitions on elevated blood lead levels using several spatial/temporal specifications. The researchers used a model with repeated observations to show that a child exposed to a single demolition has, within 45 days, 20% higher odds of moving from low blood lead to moderate BPb, or moderate to high BPb. Multiple demolitions push the numbers to 38% - as compared to an unexposed child. The difference between exposed and unexposed children accounts for a population attributable risk fraction of 2.39%. This is a dose response relationship between demolitions and EBL increasing with proximity in both space and time. They report that after 45 days and beyond 400 feet the effects of demolitions on EBL dissipate. They conclude "our findings suggest that demolition activity may increase risk of EBL during the summer months when children are most likely to be exposed to environmental lead release from demolition activity. However, demolition activity does not appear to be a major driver of EBL among children on a population level" (DHD 2017, 17). Considering lead is toxic at any level of exposure this analysis demonstrates a vector of exposure. The question then turns on the political ability and desire to intercede in making the demolitions less risky.

With an eye towards dust suppression there is some discussion and investigation of variation in demolition technique (Rabito et al. 2007). Demolition of structures that contain lead paint are subject to the EPA's Renovation, Repair, and Paint (RRP) rule and required by the US Occupational Safety Hazard Administration to have an engineering plan a take basic

safety precautions (Olen 2019). However, the regulatory guidance is focused on the protection of workers involved in demolition activities and residents where the RRP is taking place, rather than the public in proximity to the demolitions (US EPA 2013; OSHA and US Department of Labor n.d.). In those cities that have adopted a regulatory structure to mitigate risk from demolition activities enforcement is intermittent despite evidence that simple steps can have a substantial impact on dustfall. A report on best practices from a Baltimore test study noted that the use of up to 4 fire hoses (rather than the typical single hose) and a full-time dust suppression manager, resulted in "geometric mean dustfall of 7.9µg and over 66% of the dustfall levels being below detection limits" (AECF 2011). It is possible to reduce the lead fallout from demolitions to levels indistinguishable from background levels. Another article lists the dust suppression and risk mitigation techniques developed in Baltimore in collaboration with neighborhood advocacy groups; these include training all demolition workers in lead-safe work practices, designation of a full time dust suppression manager, provision of walk-off mats and high efficiency particulate air vacuums for residents near the periphery of the demolition zone, landscaping and greening of lots, regular street and sidewalk cleaning, environmental monitoring; installation of jersey barrios and fencing covered with plastic to limit entry and help contain dust, sediment control, and perhaps most importantly, the extensive use of fire hoses, with one wetting the roof and building exterior and the second wetting the debris on the ground (Jacobs et al. 2012, 459). The requisite knowledge exists to make demolition activity safer, the will to develop enforceable guidelines is lacking.

The absence of nationwide guidelines and enforcement mechanisms to address fugitive lead dust is vexing. An article in The Lancet from 1903 recommend the "simple expedient of watering" during demolition to decrease the dust cloud that accompanies demolition ("The

Dust of Buildings in Process of Demolition and Its Prevention" 1903). 100 yeas on a new regime may be on the march. On the strength of the anti-lead poisoning cohort community leaders, researchers, health professionals and outreach groups Baltimore adopted its first comprehensive city ordinance designed to make demolitions more lead safe (AECF 2011). Inspired by the Baltimore protocols the Health Department of the City of Detroit recommended a more advanced set of recommendations covering pre, during, and post demolitions though money for enforcement and implementation remains an issue (DHD 2017).

There are two modeling issues present in this demolition literature, one regards quantifying the relationship between demolition activity and BPb, the other is about modeling the costs of demolition. The first modeling issue is closer to the focus of this dissertation, but the second issue deserves explication because the issues are related. As a connection is established between BPb and demolition activity, attention turns to prevention. Critical for justifying the costs of prevention is an understanding of the costs of demolition and the framing of structure demolitions as an act of production (producing an empty lot, producing construction debris, producing a cloud of lead dust and a landscape peppered with lead, producing lead poisoned children). Knitting these social costs into the costs of demolition can be accomplished in a sense by embracing the recommendations derived from Baltimore and Detroit. The East Baltimore Development Initiative estimates that their guidelines for demolition increase the cost of demolitions by about 25% (AECF 2011). It is an open research question if such an increase in costs is adequate to balance the costs of demolition.

Recapitulation of Modeling Decisions

The previous three chapters have established the veracity of the models and schema advanced in chapter 2. This chapter has discussed GIS and epidemiology, the ground on which my statistical analysis is built. The chapter has also reviewed the literature (and professionally informed brainstorming sessions) on which data gathering and modeling decisions are made. Clarity follows from a brief review of the modeling decisions grouped by the focus of each variable.

Two of the child focused variables are modeled as dichotomous, sex of child and blood lead test. In the absence of an observation for either variable an additional dichotomous variable is added reflecting if the relevant value is known or unknown for that child. Two of the child focused variables are modeled as being continuous non-linear variables. In each case the decision to model the variable as such is based on the distribution of the variable in reference with the relevant epidemiological literature. The year of BPb test variable is modeled as a square function and the age of child in months variable is modeled as taking on a cubic shape. With reference to both epidemiologic literature and medical testing recommendations the data set is subset to the range 6 to 72 months.

The Housing Focused Variables are derived from observations of era of home construction and exterior housing condition. Era of home construction is represented by dichotomous variables reflecting how lead paint was regulated when the home was built. The relevant dates, 1952 and 1978, are based in the history of the lead paint industry and are typically reflected in epidemiological literature. Similarly present in the epidemiology literature is an understanding that the condition of housing is a proxy for the lead-based risk the home poses to the children that reside there. Following this housing condition literature, a

series of dichotomous variables are derived from the CEI's NHCS, each variable reflecting the condition of the home. Combining age and housing condition into a single analysis is the major analytical contribution of this dissertation. Consistent with this contribution, a suite of variables are developed based on the interaction of era and exterior paint condition dummy variables.[19]

The Geographic Variables are all dichotomous variables that reflect in different ways lead in the built environment. The demolition (demo) variable indicates if there have been any structures demolished within 100m of the child's residence during the life of the child. The in_prox variable describes the geographic proximity of the home to lead in the environment derived from TEL gasoline. The emission sources (major roads, highways, and old gas stations) as well as distances from these sources (140ft, 100m, 100m) are inspired by the epidemiology literature. The use of a seasonality dummy variable to reflect if lead in soil is likely to be re-suspensible and thus bioavailable to young children is familiar from the epidemiology literature on lead poisoning from the built environment. The community district variables dichotomously associate those children who live within the same cluster of neighborhoods and thus similarly exposed to lead in ways that are not explicitly modeled in other ways in this dissertation.

[19] The variables indicate is a house is from the earliest era of lead paint with substandard exterior paint, the earliest era with severely deteriorated paint, the middle era substandard paint, and so forth.

CHAPTER 6

Data, Geocoding, Assembly

Chapters six and seven describe the original contributions of this dissertation, they are focused on understanding the relationship between pediatric blood lead and the built environment. In chapter 7 I use a linear regression model to investigate relationships between observations of the built environment surrounding a child and observations of the child's blood lead level (BPb). Equation 6.1 presents a model that reflects the grouping of variables first discussed in chapter 5.

$$BPb_i = \alpha + \sum_{h=1}^{8} \beta_h C_{hi} + \sum_{h=9}^{20} \beta_h H_{hi} + \sum_{h=21}^{25} \beta_h G_{hi} + \epsilon_i \quad (6.1)$$

Where BPb_i is the dependent variable, α is an intercept term, and the 25 β_h terms are coefficients relating changes in the independent variables with changes in BPb. ε_i is a vector of random disturbances modeled as normally distributed with a mean of 0 and a variance of σ^2. Eight child focused independent variables are indicated by C_{hi}. Twelve housing focused independent variables are indicated by H_{hi}. Five geography variables are indicated by G_{hi}. The modeling decisions reflected in equation 6.1 have been discussed in chapter 5 along with relevant epidemiological work. A discussion of each variable used in equation 6.1, including the interaction terms, introduces the statistical analysis in chapter 7. Successful statistical work relies on the preliminary activities of gathering, cleaning, visualizing, (in some cases) estimating, and associating data. That work and the first two original contributions of this

dissertation are described in this chapter: gathering the necessary data from disparate sources and developing the geocoding procedure used for the spatial association of the gathered data.

Relationships, Organizations, Resources

The seed for this dissertation was planted in 2014 by the Kansas City-Home Environmental Assessment Research Taskforce (KC-HEART). The KC-HEART heart project was organized with a United States Office of Housing and Urban Development (HUD) Healthy Homes Technical Studies Grant # FR-5500-N-15 (OMB approval # 2501-0017). Three aims organized the activities of the KC-HEART project; aim 1 created a geospatial exposure model using physical housing conditions and socio-economic data correlated with ICD-9 diagnosis codes and health insurance claims data; aim 2 developed correlations between observations of exterior and observations of interior housing conditions; aim 3 focused on developing additional data about the experience of asthma and the environment in which children live to be associated with the analysis in aims 1 & 2.

Several important aspects of this dissertation project grew from the seed of KC-HEART. The first regards the use of HIPAA protected health information.[1] KC-HEART utilized deidentified patient level health records for the geospatial exposure model, this required developing warranted protocols for receiving, storing, working with, and sharing private patient data. These protocols were developed and approved by the Internal Review Board of Children's Mercy Hospital (CMH) of Kansas City with a covering agreement

[1] The Health Insurance Portability and Accountability Act of 1996 created a national set of standards for the protection of individually identifiable health information. The act addressed the use and disclosure of 'protected health information' (health records) by 'covered entities' (doctors, hospitals, insurance companies, etc.). The goal of the act is to "assure the protection of individual health privacy while allowing the flow of health information to promote healthcare and protect public health and wellbeing" (Office for Civil Rights (OCR) 2008).

accepted by the University of Missouri Kansas City (UMKC). Without this agreement, the protocols on which it was based, and the trust built by proven coordination between CMH and UMKC this dissertation could not have taken place.

Two additional successes of the KC-HEART project deserve mention. The first is the collaboration with medical professionals at CMH. Drs. Jay Portnoy and Charles Barnes were active in Aim 1 brainstorming sessions about health outcomes and Dr. Portnoy provided expertise for interpreting the ICD-9 diagnosis codes used by hospitals to classify hospital events.[2] The mutually beneficial experience led to the development of KC Health-CORE and encouraged additional collaborations by CMH affiliated doctors and other health professionals with the research agenda growing out of KC-HEART. The second success of the KC-HEART program is more technical in nature: a process for geocoding (or address matching) health encounters with a specified parcel geography. The geocoding process developed as part of this project has been integral to several works of scholarship (N. Wilson 2018a; Kane and Eaton 2018; B. Wilson et al. 2018; B. Wilson, Wilson, and Martin 2019; Kane 2020).

KC Health Community-Organized Resource Exchange (hereafter CORE) was designed to expand the type of research capabilities embodied by the KC-HEART project while adding significant capabilities. CORE benefited from encouragement and funding by the Kansas City Health Forward Foundation. Additional funding and in-kind support for the CORE project has been provided by UMKC, CMH, and the Global Institute for Sustainable Prosperity. Briefly, the CORE project embodied three interrelated aspects: a community outreach component, a

[2] Appendix B reproduces a document from a KC HEART brainstorming session listing potential correlates of lead poisoning. Appendix B is discussed more completely in chapter 5. Not all of the ideas in that expansive document are discussed in this dissertation underscoring this work as a point of departure in a larger research agenda.

data repository component, and a research component. All three of these aspects are relevant to the agenda of this dissertation. The community outreach component can facilitate the sharing of my research findings directly with those communities affected by lead poisoning and allows these same communities to request and direct my future research attention to their immediate concerns.[3] My pediatric lead poisoning focused dissertation functions as a type of proof-of-concept for the data repository and research components of CORE. CORE research into the social determinants of health and the development of health indicators is designed to be expansive, encompassing adult health, observations of the built environment, crime, income and employment. The IRB approval from KC HEART passed to CORE as did the data sharing agreements. Professional relationships and data sharing agreements are ongoing between CORE and the Kansas City, Missouri Health Department, and the online data platform MySidewalk.

The Institutional home at UMKC for both KC HEART and CORE is the Center for Economic Information (CEI). CEI was founded in the early 1990's as a core agency of the Missouri State Census Data Center and whose original mission was "to use advancing network information technology to inform research and policy in the public, private, and non-profit sectors." From the very beginning CEI focused on providing data services across sectors. over time CEI evolved a focus on using GIS for anchoring its analyses and services. The CEI has contracted for the cities of Kansas City, Missouri and Kansas City, Kansas, The US Department of Housing and Urban Development, The Kansas City Neighborhood Alliance as well as others. In 2010 CEI joined the National Neighborhood Indicators Partnership, a peer

[3] This direction/research sharing happened with regard to the Blue Hills neighborhood committee which wanted additional information to leverage in discussions with the city regarding the safe demolition of a neighborhood school.

learning network coordinated by the Urban Institute. Critical among the institutional relationships CEI maintains locally is with the Mid-America Regional Council, an umbrella group focused on regional planning in the bi-state Kansas City metropolitan area. In addition to institutional support and its professional track record, by being nestled a public university CEI is able to function as a non-profit neutral platform for health subject research.

This Dissertation owes a significant debt to each of those projects and organizations. The Geocoding process, initial funding and IRB protocol are descended from the KC-HEART project. Significant data resources, connections to professionals and community, as well as research funding has been provided by CORE. Tying things together are the geospatial resources and institutional home of the CEI. This research makes use of resources originated in state and private institutions, resources that are typically kept in separate silos. The CEI as an institution within a public research university is uniquely situated to gather and analyze data from these disparate sources without challenging the revenue frameworks that maintain the institutions or violate the HIPAA protections patients have a right to by law.

In addition to the necessary funding, institutional support, and other resources mentioned above the collaborators associated with KC-Heart, CORE, and the CEI itself all contributed to the data repository from which this dissertation relies. A comprehensive discussion of these data follows apace from the listing of data objects, their sources, and the process of their organization.

CEI supplied a catalog of GIS data focused on the Kansas City Metropolitan area in addition to the computing power and software required to make use of those data. The most important geospatial data provided by the CEI are the Neighborhood Housing Conditions Surveys. Additional data sets include line shapefiles for highways, major roads, railroads,

rivers and streams, and polygon shapefiles for states, counties, cities and incorporated places, parcels, neighborhoods, community districts, parks, water features, census tracts, census block groups, census blocks, and zip codes.

The Kansas City Missouri Health Department (KCMO Health) supplied observations of lead poisoning for the Missouri side of the metropolitan area.[4] These data were shared via an encrypted and password protected thumb drive. Due to legal and administrative restrictions comprehensive observations of blood lead levels from the Kansas side of the metropolitan were not made available. The paucity of observations from Kansas inspired their omission from further consideration in this dissertation.

MySidewalk provided data from the Decennial Census of the USA and the 5-year American Community Survey. The benefit provided by MySidewalk is the association of census data with the several administrative geographies: census blocks, block groups, tracts, zip codes, neighborhoods, community districts, cities and incorporated places.

The City of Kansas City, Missouri has maintained an 'open data portal' since January of 2013. The stated purposes of the portal are "transparency", a "better-informed" citizenry participating in the democratic process, "better organizational efficiency" of city programs, encouraging "civic entrepreneurs to more easily build technological solutions to improve City services" ("Open Data | City of Kansas City, Missouri Mayor's Office" n.d.). The Kansas City open data portal provided structure demolition data dated back to 2000. Following a request to the Kansas City Missouri office of City Planning and Development I received demolitions data dating back to 1990.

[4] Amy Roberts RN, BSN, program manager for Healthy Homes and Childhood Lead Poisoning Prevention at KCMO Health participated in the brainstorming session referenced in Appendix B and expertise and support throughout the dissertation project.

MARC supplied information related to its role in regional planning operations. They provided a line shape file of street centerline data indicating the range of addresses associated with a particular street segment (as opposed to distinct addresses associated with discrete places on a map). The MARC centerline file is the standard resource used in development, emergency response and other public planning activities. MARC also provided a polygon shape file indicating the year in which structures at the parcel level were constructed across the metropolitan area. Such information is typically kept by the separate county level administrations.

Two remaining data sources will be discussed in greater detail in conjunction with the age of housing on parcel data and geocoding process. Relevant to geocoding is a comprehensive list of gas stations in Kansas City, Missouri from the twilight of the leaded gasoline era (1976). This list was compiled using the Polk City Directories for Kansas City in the Budd Special Collections room at UMKC's Miller-Nichols Library. Enhanced information about the date of construction and renovation for a subset of parcels is provided by Architectural & Historical Research, LLC of Kansas City, Missouri.

Table 6.1 provides a summary of the data gathered to make my dissertation possible. Establishing and maintaining relationships among data partners, developing the infrastructure to securely store and process their data, and gathering it in one location—all this together is the first of the three original contributions of this dissertation. It is an inherently collaborative contribution. I was part of a team which made it happen. The significance of assembling these data is multifaceted. These data were kept in isolated repositories where they could not be used in conjunction. The combination of the data in table 6.1 is the basis for the statistical analysis in chapter 7 and the geocoding section in chapter 6 below. Table 6.1 is a template for what

Table 6.1: Data Gathered and Source

Data Object	Data Source	Research Program
Kansas City Metropolitan Area (KCMa) Basemap Shapefiles: highways, major roads, railroads, rivers and streams, states, counties, cities and incorporated places, neighborhoods, community districts, parks, water features, zip codes, parcels	CEI	CEI General
KCMa Census Geography Shapefiles: blocks, block groups, tracts	CEI	CEI General
Neighborhood Housing Conditions Survey	CEI	CEI General
Deidentified Blood Lead Observations	KCMO HD	CORE
US Census Data for KCMa: 2000 and 2010Decenial Census data associated at the neighborhood and community district geography.	mySidewalk	CORE
KCMO Structural Demolition Records	KCMO HD	CORE
KCMa Street Centerline shapefile	MARC	KC HEART
KCMa Age of Structure on Parcel Shapefile	MARC	CORE
Gas Station Locations (1976)	UMKC Miller-Nichols Library	CORE
Supplementary Parcel Information	AHR llc.	CORE
IRB approval	CMH	KC HEART

must be gathered to reproduce this analysis in other locations and time frames, it also serves as the basis for other research into the built environment (bringing in lead in soil, point sources of pollution, content of lead in soil), other geographies of lead poisoning (centerline address, neighborhood, or community district geography), or the expansion beyond lead and the built environment to health outcomes and the social determinants of health more generally.

Data Descriptions, Geocoding, Processing and Association

Neighborhood Housing Conditions Survey

The CEI's Neighborhood Housing Conditions Survey (NHCS) is one of two critical data sets that distinguish my analysis. Using the NHCS in conjunction with era of housing construction generates an interaction term that is ubique in the literature. Statistical

investigation of these interaction terms is a major part of the contribution of this dissertation. The NHCS is actually 22 separate survey initiatives. These surveys use a common methodology to generate ratings that are consistent over time and space, and among analysts. The CEI's survey is a type of Systematic Social Observation (SSO), an observational approach sociologist Albert Reiss described as, "observation and recoding done according to explicit procedures which permit replication and that rules are followed which permit the use of the logic of scientific inference" (Reiss 1971, 4). The SSO approach can be applied wherever observations of public space are relevant to social phenomena (Sampson and Raudenbush 1999; Wei et al. 2005; King 2015; Baggetta and Bredenkamp 2019). The Sponsors of the NHCS surveys are various. The initial survey was commissioned by the City of Kansas City, Missouri. Other surveys were sponsored by The Unified Government of Wyandotte County, the National Institutes of Health, The American Recovery and Reinvestment Act, Greater Kansas City Local Initiatives Support Corporation, The Department of Housing and Urban Development, among others. The diversity of funders underscores the variety of uses which a consistent set of observations of housing conditions can be put to.

The initial development of the NHCS was funded in 1997 by a HUD Community Outreach Partnership Center Grant. Ed Linnebur of the Kansas City Neighborhood alliance designed the initial survey and students from the UMKC Urban Affairs program implemented his design. The pilot form of the NHCS was called GPLAN (Geographic Planner) and it focused on the 49/63 neighborhood in Kansas City, Missouri. The completion of GPLAN, detailed in the "Final Report for the City of Kansas City Missouri Contract No. 1999-032", translated into a complete residential parcel survey of 116 of the 246 KCMO neighborhoods (Bowles 2000). A report about that survey articulates a clear vision for the NHCS, "*It is a*

powerful planning tool and it establishes a baseline that can be used to evaluate the effectiveness of housing policy" (Eaton, Hernandez, and Olson, n.d., italics orriginal). An early report on the NHCS lists eight distinct development agendas in Kansas City and suggests the NHCS can be used to evaluate each agenda on a consistent basis (Bowles 2000).

The discussion of indicators and benchmarking in the guidebook of the National Neighborhoods Indicators Partnership guidebook, "Building and Operating Neighborhood Indicators Systems", frames the significance of the NHCS. An emphasis of the NHCS is on developing benchmarks, or "a reference point or criterion against which to judge one's own performance", which exist at the finest level of detail that is cost-effective to produce in terms of labor time and money ("Building and Operating Neighborhood Indicator Systems: A Guidebook" 1999, 18). There are many ways to put such a benchmark to work; from directing civic infrastructure spending to directing private investment spending; from characterizing neighborhood conditions in physical terms to driving research into the nexus of health and the built environment. These systematic housing conditions surveys can be the basis of a robust warehouse of neighborhood level indicators and a core piece of a data driven neighborhood development model (Bowles and Eaton 2010).

Beginning in 2000 and continuing through 2014 the CEI carried out 22 separate housing conditions surveys in the Kansas City metropolitan area. Table 6.2 describes the NHCS by program, year, date of survey and number of parcels surveyed. Several surveys took place over several years, and multiple surveys could take place during the same calendar year. Some surveys we quite large while others were relatively small, the first KCMO housing conditions survey (program 03) looked at 82,081 parcels while the Sugar Creek survey only

Table 6.2: NHCS Overview—Programs, Years, # Surveyed

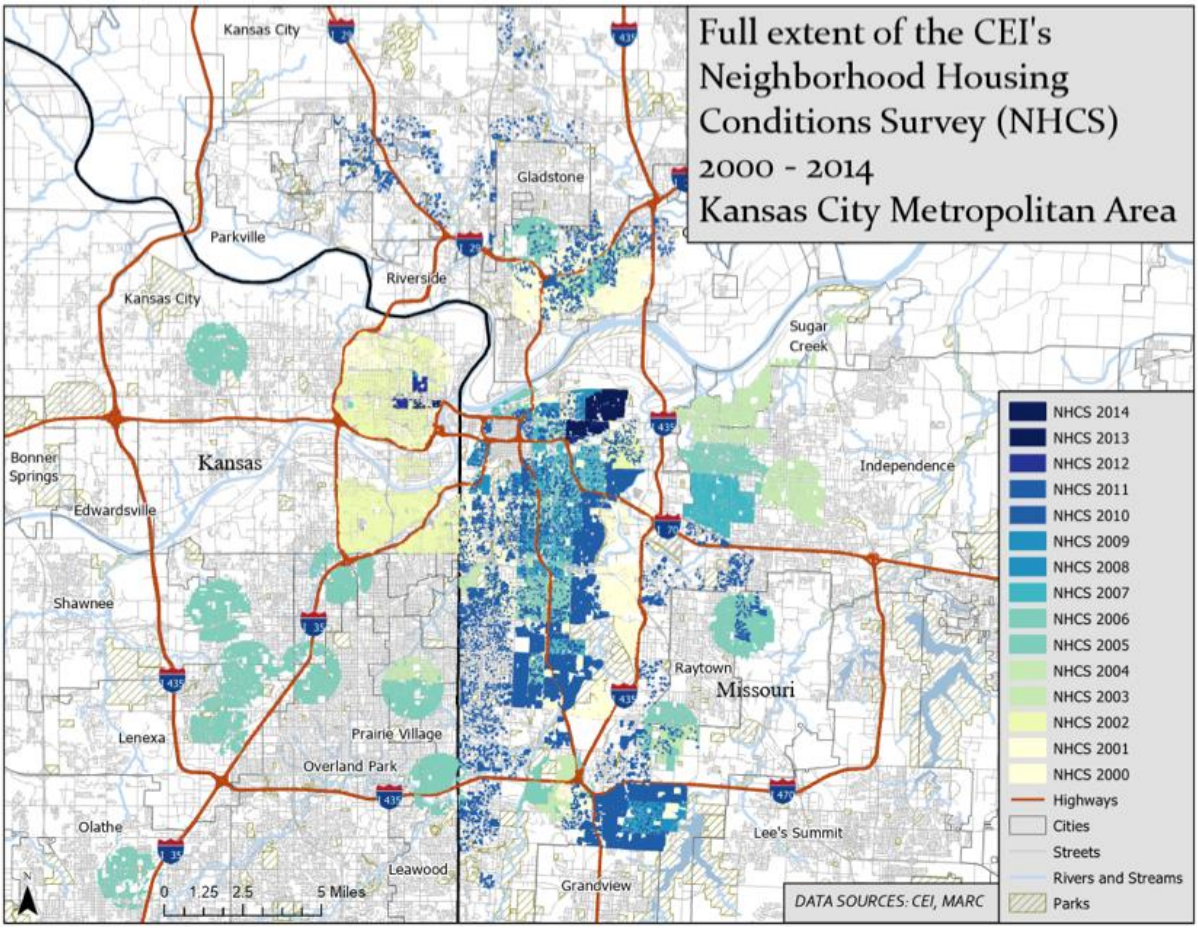
Program #	Name	Year	First Inspection	Last Inspection	Parcels Surveyed
3	KCMO Housing Survey	2000	4/6/00	12/10/00	34,893
3	KCMO Housing Survey	2001	1/10/01	12/24/01	47,091
3	KCMO Housing Survey	2002	1/7/02	2/25/02	97
6	UMKC HRG Toxic Environment Pilot (residential)	2001	4/8/01	8/10/01	1,139
8	KCKs/Wyandotte County Unified Gov't Housing Survey	2001	11/10/01	12/31/01	7,230
8	KCKs/Wyandotte County Unified Gov't Housing Survey	2002	1/2/02	12/21/02	24,591
8	KCKs/Wyandotte County Unified Gov't Housing Survey	2003	3/1/03	12/12/03	11
12	Blue Valley NHCS 2002	2002	11/8/02	11/21/02	3,377
13	Sugar Creek	2003	3/21/03	6/6/03	1,790
14	Independence	2003	3/6/03	9/24/03	1,964
15	Northland Neighborhoods (Chaumiere, Winwood)	2003	4/11/03	8/3/03	1,583
16	Blue Hills NHCS 2003	2003	12/8/03	12/31/03	813
16	Blue Hills NHCS 2003	2004	1/9/04	3/9/04	2,378
18	NIH03 Surveys 1&2 (NACS; Survey 1: NHCS/Res; Survey 2: DSI/Nonres)	2004	3/4/04	12/31/04	9,777
18	NIH03 Surveys 1&2 (NACS; Survey 1: NHCS/Res; Survey 2: DSI/Nonres)	2005	1/2/05	12/16/05	30,888
18	NIH03 Surveys 1&2 (NACS; Survey 1: NHCS/Res; Survey 2: DSI/Nonres)	2006	1/13/06	3/14/06	276
19	Independence 2004 (area 2)	2004	3/16/04	6/18/04	1,653
21	Independence 2005	2005	3/5/05	5/12/05	3,973
22	cg003 Vineyard	2005	4/22/05	6/9/05	2,001
24	KCMO Housing Survey	2006	6/28/06	12/12/06	15,473
25	Independence Housing Survey	2007	5/1/05	6/19/07	4,705
26	KCMO Housing Survey (2007)	2007	7/26/07	11/21/07	15,442
27	KCKs Housing Survey (2008)	2007	9/19/07	9/20/07	484
28	KCMO Housing Survey (2008)	2008	7/7/05	11/9/08	15,612
29	Green Impact Zone NHCS (2009 - 2010)	2009	10/6/09	12/2/09	3756
30	KCMO Housing Survey (2010)	2010	6/18/10	10/28/10	15,991
31	KCMO Housing Survey (2011)	2011	7/9/11	8/18/11	8101
32	LISC CSI Grant funded Housing Survey (2012)	2012	10/10/12	12/14/12	899
32	LISC CSI Grant funded Housing Survey (2012)	2013	7/28/13	7/28/13	111
32	LISC CSI Grant funded Housing Survey (2012)	2014	7/21/14	7/23/14	446
33	KCMO Blight Study-Lykins/Indian Mound (2012)	2012	11/10/12	12/20/12	329
33	KCMO Blight Study-Lykins/Indian Mound (2012)	2013	1/10/13	9/13/13	5,368
Total	22 programs	2000 - 2014	4/6/00	7/23/14	262242

n.b. The total number of parcels surveyed includes 3,499 parcels with no associated housing conditions observations.

1,790 parcels were rated (program 13). In the 15 years of the survey (2000-2014) 258,743 parcels received ratings.

Kansas and Missouri are both included in the NHCS. The method for determining which parcels were to be rated changed from program to program, all programs focused on residential parcels though some gathered auxiliary data about commercial properties as well (18, 29). Some programs only looked at particular neighborhoods (12 - 16, 22, 24, 25), some looked at many neighborhoods while targeting those expected to have a preponderance of substandard conditions (26, 27). One program focused on Kansas City, MO drew a random

Map 6.1: Full Extent of CEI's NHCS (2000-2014)



Map created by author.

sample of parcels from the city (30), another program looked at all the houses within randomly drawn circles of 1-mile diameter scattered across the metro area (18). Map 6.1 illustrates the effect the NHCS being composed of 22 programs each of a different size and scope, parcel surveys create a patchwork quilt across the metropolitan landscape. The volume of the parcel level observations changes from year to year. Some neighborhoods (e.g. KCMO's East Side and Historic North East neighborhoods) were rated several times while other neighborhoods and incorporated places (e.g. Edwardsville, Riverside, Liberty, Lees Summit) received little or no survey attention. Observed variability by year and geography is attributed to funding and motivations changing from program by program. Despite its patchwork nature the NHCS is a useful resource for studying the built environment in much of the Kansas City Metropolitan Area during the 15 years it was carried out.

Considered as a single dataset the NHCS is a large collection of observations for surveys of its type—19 observations about 258,743 parcels is nearly 5 million data points—part of what makes it useful is the geographic level at which these data points reside. Chapter 5 established the importance of health research being done at the geography of experience and the parcel level emphasis here corresponds with that insight. The NHCS is comprised of observations of residential and non-residential parcels. It contains unique observations of property boundaries for parcels with and without a structure. An aspect of the NHCS that makes it useful for applications such as health research (applications for which it was not designed but fall within the bounds of its general usefulness) is that it gathers objective structural classifications as well as conditions data about the structures themselves. Structural classification data include precise street address, use type, structure profile, and whether there is a visible address associated with any structures on the property. Some programs gathered

auxiliary information about the parcels such as whether it was a rental property or not, the city and county parcel id, and whether the owner of the parcel lives at the address. This auxiliary information, because it was not collected uniformly for all programs, is left out of table 6.3 the data dictionary for the complete NHCS.

Table 6.3: NHCS Data Dictionary

Field Name	Description
OBJECTID	Index number generated by ArcMap
SHAPE	type of shape file
PID	Foreign Key from City/County (like kiva PIN, not unique)
PIDSRC	Unclear from Support Materials
STRNO	Address Field: Street Number
STRDIR	Address Field: Street direction
STRNAME	Address Field: Street Name
STRTYPE	Address Field: Street Type
LANDUSE	Description of Land Use from Government Agency
LUSRC	Unclear from Support Materials
PRGM	Housing Condition Survey Program Number
YEAR	Year of Housing Survey
INSPDATE	Date of Housing Condition Survey for which results are below
VISADDR	Visible Address
STYPE	Structure Type
UTYPE	Use Type
RESTYPE	Residential Type
SPROFILE	Structure Profile
ROOF	Roof Condition
FOUNDATION	Condition of Foundation and Walls
WINDOORS	Condition of Windows and Doors
PORCH	Condition of Porches
EXTPAINT	Condition of Exterior Paint
PVTWALKS	Condition of Private Sidewalks and Driveways
LAWN	Condition of the Lawn and Shrubs
VEHICLES	Status of Vehicles Parked on the Property
LITTER	Assessment of Litter on the Property
OPENSTRG	Assessment of Open Storage on the Property
PUBWALKS	Condition of Public Sidewalks

Table 6.3 (continued)

Field Name	Description
CURB	Condition of Curbs Adjacent to the Property
STRLIGHTS	Condition of the Streetlights Adjacent to the Property
CATCHBSN	Condition of the Catch Basins Adjacent to the Property
STRCON	Condition of the Street Adjacent to the Property
Length	GIS Field: Length of Parcel Perimeter on which Home is Located
Area	GIS Field: Area of the Parcel on which House is Located
PGM_id	Primary Key for all NHCS parcels

My dissertation uses 8 of the of the 35 fields listed in table 6.3. Of the 15 ratings conditions contained in the NHCS this dissertation focuses on 3 of the 5 structure condition ratings.[5] Neither the conditions of the parcel grounds (private sidewalks and driveways, lawn and shrubs, vehicles, litter, and open storage) nor the conditions of the public infrastructure (public sidewalks, curbs, streetlights, catch basins, and street conditions) were used in my statistical analysis of the built environment. Though my dissertation is limited to the structure conditions, none-the-less, such information is assembled and ready to answer future research questions.

Each of the housing conditions (roof, foundation and walls, windows and doors, porches, and exterior paint) is rated using a five-level ordinal ranking. A score of 1 is the lowest, 5 the highest. A score of 6 indicates that the condition is not applicable to the parcel in question, implying either the lack of a structure on the parcel or the absence of that particular facet of housing (e.g. a home without a porch would receiving a rating of 6 as would a vacant lot). Thus, the percentages of parcels with 6 ratings reported in table 6.4 vary slightly from

[5] The Roof and Foundation and Walls structural conditions were incorporated into preliminary models but excluded from my final analysis due to information loss associated with the difficulty of observing these housing conditions. Statistical results for the model containing all five housing-focused structural conditions are available upon request.

roof to porch and so forth. The roof rating contains an additional score, 7, which indicates either a flat roof or an obstructed view of the roof. Ratings of 4 and 5 indicate good and excellent conditions, respectively. There is more variation in the rating system among substandard or worse conditions. Across all conditions a score of 3 or less indicates a substandard housing condition while scores of 1 and 2 indicate severely deteriorated and seriously deteriorated conditions, respectively. A detailed breakdown of the ratings system of the NHCS classifications and conditions is in appendix C, a ratings guide summary from a 2006 report generated associated with a survey the CEI did for the National Institutes of Health. Appendix C distinguishes between each of the ratings for the housing, grounds, and infrastructure conditions as well as enumerating the different objective classifications of housing type.

Table 6.4: Percentage of Housing Conditions Receiving Each Rating

	Rating						
	1	2	3	4	5	6	7
Roof	0.87%	4.92%	28.59%	33.96%	13.03%	16.22%	2.76%
Foundations and Walls	0.33%	0.87%	6.26%	33.76%	42.87%	15.83%	0.00%
Windows and Doors	0.80%	1.63%	8.09%	31.23%	42.35%	15.90%	0.00%
Porch	0.57%	2.29%	13.26%	30.41%	36.17%	17.30%	0.00%
Exterior Paint	1.64%	3.88%	18.66%	34.03%	25.87%	15.93%	0.00%
Private Walks	4.61%	5.27%	20.04%	33.24%	22.22%	14.62%	0.00%
Lawn	1.75%	2.13%	10.83%	27.52%	57.77%	0.00%	0.00%
Vehicles	0.29%	0.73%	2.92%	5.54%	90.53%	0.00%	0.00%
Litter	0.53%	1.21%	5.34%	13.60%	79.32%	0.00%	0.00%
Open Storage	0.44%	0.83%	4.23%	11.42%	83.08%	0.00%	0.00%
Public Walks	5.46%	4.20%	10.26%	20.15%	20.07%	39.86%	0.00%
Curbs	5.83%	8.45%	12.19%	31.38%	25.16%	16.99%	0.00%
Streetlights	1.22%	0.21%	0.38%	3.07%	95.11%	0.01%	0.00%
Catch Basins	0.20%	0.26%	0.66%	1.56%	4.77%	92.54%	0.00%
Street Condition	0.72%	0.69%	5.73%	43.04%	49.37%	0.45%	0.00%

Reviewing the data in table 6.4 it is important to be circumspect; these percentages refer to the housing conditions survey in total. Many parcels have been surveyed more than once, several many times, and still more parcels in the metropolitan area were not surveyed at all. There is no reason to expect the individual neighborhoods and community districts to reflect the percentages reported above. Still, general trends in housing conditions are apparent. About 16% of the parcels surveyed are vacant lots and 2.75% of the roofs were un-ratable for one reason or another. Roof ratings also diverge from the rest of other conditions in that it does not contain a preponderance of excellent ratings. Foundation and Walls conditions have the highest percentage of good and excellent ratings and the lowest percentage of ratings substandard or below. All housing conditions exhibit a narrow range (30%-34%) of parcels with good ratings. Critically, the percentage of parcels with substandard or worse rating is less than 35% of the total number of surveyed parcels. The outlier again being roof conditions with no other conditions within 8% of its figure. The percentage of deteriorated parcels is uniformly less than 6% with exterior paint exhibiting the largest percentage of ratings observed to be severely deteriorated. We should be circumspect when interpreting the characteristic ratings of the individual housing conditions because the NHCS uses an ordinal rating system. The ratings themselves are shorthand for qualitative judgements. It is straightforward to recognize that the difference between substandard and good will not be exactly the same as the difference between severely deteriorated and seriously deteriorated. The housing conditions scores, though consistent, are not equidistant (i.e. the difference between ratings 1 to 2 is not necessarily the distance between 3 and 4).

The housing conditions surveys themselves were performed by pairs of observers. It is a ‘windshield’ or ‘eyeball’ survey. Rating parcels involves driving slowly and systematically

through residential neighborhoods and recoding first person observations of the actual conditions of the homes. The cars preforming the survey are clearly marked to indicate they are a survey vehicle. The earliest surveys were recorded on paper but quickly the process was transformed to be done via PDA. The surveys are mapped against municipal property boundary maps which are themselves updated by surveyors in the field.

To ensure the ratings are consistent from program to program and researcher to researcher every one that performs a survey is required to pass a two-part classroom-and-field training regimen. The classroom session is focused on learning the ratings guide reproduced in appendix C. To pass their field examinations potential raters must return ratings with scores that correspond to the scores of the same parcels by their trainers. A housing condition survey was designed so that a rating of 3 or lower indicates that aspect of that structure is in violation of Kansas City building codes. After certification surveyors go out in the field to record their observations.[6] Following the collection of surveys the results are certified by randomly selecting and resurveying 2 blocks of at least 20 parcels. Ratings must be repeated if original surveys differ significantly from the resurvey. The original housing condition survey running from 2000-2003 cost approximately $350,000 for a survey of 82,081 parcels (about $4.25 per

[6] From an unnamed internal CEI document on the NHCS: "The following quality control procedures are performed. Two blocks with a minimum of 20 residential parcels per block are randomly selected from the survey data for each survey team. These parcels are then inspected by the survey developer/trainer to provide a set of standard results for each parcel. For categorical variables (visible address, structure type, use type, residential type, and structure profile) a direct a comparison is made. For ratings variables, an overall average difference per rating is calculated. This is calculated by taking the absolute value of the difference between the original surveyor rating and the standard rating for each parcel and summing over the total number of parcels surveyed, then dividing that value by the total number of parcels surveyed. In cases where the surveyor rates an item as un-ratable (a value of 6) and the standard rating was anything other than un-ratable, or vice versa, the rating is not counted in the average. An average difference under one is considered passing and means that over all the ratings fall within the range of -1 to +1 from the standard rating. Any average difference greater than one is considered as failing." Surveyors failing their field training are returned to the classroom for additional training. After the initial in-the-field assessments are made they are analyzed for consistency and omitted data. In the event that anomalous or missing data a mop-up team returns to re-survey relevant housing parcels.

parcel). In 2014 the cost of an additional survey of 2,559 parcels was estimated at approximately $20,000, about $7.50 per parcel. These costs include University overhead for facilities and administration as well as all the other costs incurred by the CEI for the implementation of the survey (database management, etc.) which benefit from economies of scale. Smaller surveys are more expensive on a per parcel basis than larger surveys. The most recent incarnation of the NHCS is a 'social block' level[7] survey which records the conditions for a city block (conceptualizing it as the relevant social geography) rather than the parcel level analysis of the first 15 years of surveys.

There is work remaining to more completely understand the information contained in the collected NHCS data. For instance, selecting only those parcels that are rated in more than one program to compute a rate of change analysis for the several housing conditions over a variety of geographies and temporalities. Comprehensive correlation analysis between the several housing, grounds and infrastructure ratings should be performed for the individual homes within the variety of geographies and temporalities. Local incidence of spatial autocorrelation can be performed to discover patterns in the housing, grounds, and infrastructure ratings between parcels. An algorithm can be devised to associate the parcel level observations of the early years of the NHCS with the more recent block level NHCS. The 'eyeball' nature of the housing conditions survey implies it may be possible to automate future

[7] A social block is defined in contrast to a census block. A similar geography has been used in criminal justice literature (Davison and Smith 2001; Wei et al. 2005) and public health literature (Caughy, O'Campo, and Patterson 2001; Hirsch and Hillier 2013). Social blocks are comprised of homes that face each other across a public street. Census blocks comprise homes facing each other via back yards or across alleys, a geography bound by multiple public streets. The social block is hypothesized to better reflect lived experience than the census block geography. (Bowles 2005)

surveys through a combination of remote sensing observations and artificial intelligence ratings.

There are several advantages to the NHCS as designed and implemented. The parcel level observations of housing, grounds and infrastructure conditions are at a usefully precise geographic level resolving major issues around the modifiable aerial unit problem (discussed in chapter 5). The process of actively looking at the city can create a feedback loop which can allowing researchers, in essence, to find what they did not set out looking for. Surveying every parcel in a specified geography allows researchers to update municipal records. These updates are relevant to vacant parcels and postal addresses but are less relevant to updating vacant structures. The NHCS as it has been carried out is a labor-intensive operation, but this can be understood as an advantage to the degree to which it produces detailed, useful information that is otherwise unavailable. Surveying the entire metropolitan area on a regular basis could be the basis of a 'shovel-ready jobs program' while the results of the survey could feed into targeted employment programs.

No program is perfect and there are limitations to the NHCS which must be noted. The NHCS is limited by design to exterior conditions limiting its explanatory power with regard to health outcomes. The housing conditions survey developed with funding from HUD specified observations of interior and exterior conditions in all but the most severely deteriorated homes (Breysse et al. 2008; Dixon et al. 2008). The rate of change of the various housing conditions has not been estimated limiting the applicability of the NHCS to health encounters in the same year as observations take place. As a 'windshield' survey there are visibility limitations (vegetation, inaccessible streets, flat roofs) that impact the completeness of the survey. Also,

more robust quality control measures can be implemented[8] to take advantage of the methods generated by other parcel level surveys that have come online since the NHCS was developed (Zenk et al. 2007; Paquet et al. 2010). Finally, given the deleterious uses to which the legibility of housing conditions have been used by governments in the 20th century (Gotham 2014; Rothstein 2017), it is important the NHCS is not used as a tool of discrimination and displacement. Although it easily could be, the NHCS is explicitly not a codes enforcement tool. The housing conditions surveys which the CEI performs are to direct funds to under-reported areas of need and assess the progress that has been made by institutions with regard to that mandate. However, in keeping with contemporary open data practices where public information is provided for all takers without proscription or restrictions on its use, the CEI has never formally restricted the uses to which the NHCS can be put in its data sharing agreements. The surfeit of data in our contemporary world can cut both ways.

It has long been understood that poor housing is associated with poor health (Lin-Fu 1980) and, in order to understand the nature of that association, systematic parcel level surveys can be an effective tool (Krieger and Higgins 2002). Kevin Kennedy's 2011 master's in public health thesis, "Housing and Health: Making the connection Using Geographic Information Systems" was an attempt to make an explicit connection between the housing conditions surveys that the CEI carried out and health outcomes (K. Kennedy 2011). Kennedy continues to advocate for this framework and in the years since his initial report that the NHCS has been a part of multiple successful public health grants and scholarly publications.

[8] Summary statistics regarding quality control activities are a relevant starting point.

Geocoding Process

The complete NHCS, all 22 programs, is the basis for the second contribution of this dissertation—an iterative geocoding approach to be used to match health observations to observations of the built environment. An earlier version of this process was developed in conjunction with Benjamin Wilson and applied to observations of pediatric asthma as part of the KC Heart research agenda (B. Wilson, Wilson, and Martin 2019). Applying that geocoding algorithm to pediatric lead observations demonstrates the useful ness of the approach.

The multi-year patch-work character of the individual NHCS surveys structure the geocoding process based upon it differently than a similar process based on comprehensive housing survey carried out ins a single year. As illustrated in map 6.1 the surveyed extent of the Kansas City metropolitan area changes with each year of the survey. I refer to these changes as a shifting geography. Recall the NHCS surveyors begin with a municipal geography layer that they update through in person observation. To match accurately at the parcel level to the NHCS you must use the NHCS geography. Although some parcels are surveyed more than once in the NHCS the surveys cannot tell us on their own what conditions are between surveys. Furthermore, in some years multiple programs took place and some programs spanned multiple years, there is no out of survey information. These facts underscore the time component of the geocoding process. The year is selected as the relevant temporal unit instead of the program. To address the challenges created by these facets—the shortcomings of the NHCS in terms of extent and yearly consistency—we designed an iterative geocoding process which can be generalized to solve difficulties associated with address matching parcel level observations to a shifting geography.

In the development of a scholarly literature focused on geospatial processes several criteria for judging the quality of a geocoding operation are discussed (Beale et al. 2008). There are five inter-related metrics used to judge the quality of a geocoding process: completeness, accuracy, repeatability, flexibility, and cost (Whitsel 2004; Jaquez 2012; B. Wilson, Wilson, and Martin 2019). Completeness describes the percentage of the data matched with a target geography. It is usually measured with a match rate statistic. Several issues can interfere with the completeness of a geocoding process. There may be issues with individual addresses, they may not contain complete information (i.e., 'no address'), they may refer to locations not indicated on the target geography (i.e., an address not listed as an official address), their information may be incorrect or mis-specified (misspellings or inaccurate zip code). Typically, there is a trade-off between the precision of the target geography and the completeness of the geocoding. It is easier to be more complete using a less precise geography while pushing for a more complete geocoding, in terms of correcting for the issues with individual addresses, increases the cost of the operation (Rushton 2003; Edwards, Miranda, and Strauss 2014). Geocoding to the NHCS presents dual challenges for geocoding, first is the shifting geography over time, the second is the issue of devising a warranted match rate. I will return to the match rate issue shortly.

Accuracy is defined by the relationship between the actual location of the target address in map space and the location determined by the geocoding operation. Considering geocoding to the zip code level is instructive. For polygon matches the geocoding procedure locates the encounter at the centroid of the target geography, in this example that mean all encounters matched to a zip code are placed at the same point in the center of the geography. Clearly all the homes in the zip code are not located in the same place, so there is in this example a low

degree of accuracy. Different issues arise with centerline matches which positions encounters offset from a street centerline. The centerline match process references only the street and not the surrounding parcels, it is rare for these geographies to match as accurately. Location accuracy is a separate issue from the MAUP issues discussed in chapter 5, though the solution of matching to the parcel level is the same. The current standard for epidemiology and public health is to match to the parcel level (Rushton et al. 2006; Manson et al. 2009; Jaquez 2012). Accuracy, like completeness, implies a tradeoff with cost due to the extra labor time involved. Accuracy also introduces potential privacy issues as undisguised addresses could be used to reverse engineer the individuals violating their rights under HIPAA (McElroy et al. 2003). For this reason, though they are used in analysis, point address layer maps are never produced as part of any final reports.

Repeatability is a critical issue for empirical research. The ideal in geocoding processes, as with any statistical process, is to be transparent enough for anyone given the appropriate tools and training to be able to reproduce the output with minimum variation. Resolving issues of completeness and accuracy can require the informed judgment of geocoding technicians. It is thus critical for the geocoding process to contain clear guidelines to follow and limitations on the interpretive prerogative of the technicians. Cost is often a limiting rather than a descriptive aspect of the geocoding process. Budgets are fixed and the process is defined within its limits. In such cases repeatability often contrasts with completeness and accuracy. Technicians are not given unlimited budgets with which to manually match each encounter, just as time limits on the underlying investigation prevents this from happening. Variable cost for ‘in-house’ geocoding is technician labor time and fixed costs come from computing power. Alternatively, geocoding can be contracted through third

party services, though such cases still require technician labor time to prepare the data and manually check the results. In-house methods can be more accurate and less time consuming than third party services (Faure et al. 2017).

A 2019 article added a fifth criteria, flexibility, for judging the quality of a geocoding process (B. Wilson, Wilson, and Martin 2019). Flexibility in this context refers to the variety of uses to which the geocoded output can be put without significant additional effort. Flexibility is a useful characteristic in geocoded output, recall the use of the NHCS in health research is due to the flexibility of the underlying data. Contemporary data science turns on the flexibility of established data to be used for alternative applications. It is critical for data, particularly geocoded data, to be ready for unforeseen applications and the flexibility criteria accounts for this in a positive manner. After introducing the iterative geocoding process the example of pediatric lead poisoning, I will return to these criteria and apply them to the process.

The iterative approach to geocoding described here is an extension and development of the standard multi-stage approach (Goldberg et al. 2008; Sonderman et al. 2013). As the name implies the iterative approach applies the similar processes repeatedly to create the final geocoded dataset. There are three phases in the iterative approach, phase 1 is the preparation of data for geocoding, phase 2 is the geocoding process, phase 3 is the re-assembly of the geocoded data. These three phases are slightly different than those described in Wilson, Wilson & Martin (2019).[9] The three phases of geocoding are recursive such that a phase 2 geocoding

[9] Similarly, the exact steps of this process are slightly different than those described in Wilson, Wilson & Martin (2019). The difference in processes are due to the contents the data geocoded. The 2019 paper describes the application of the process to pediatric asthma data from a children's hospital, the process here is focused on blood lead observations from a health department. The asthma data has an associated DCN number which facilitated the revision of unmatched zip codes when that field was incomplete in the lead data creating a slightly different zip code revision process.

operation becomes a phase 1 preparation for an additional phase 2 geocoding operation. After this recursive activity the multiple phase 2 operations are collected in a phase 3 data assembly which itself is a phase 1 preparation for additional geocoding action. Appendix D reproduces a diagram schematizing the operations and intermediate data objects created in the process of converting the KCMO Health lead data (supplied to me as an excel file) into a shapefile of health encounters geocoded to the parcel level.

Step 1: Prepare the source material for the address locators. The address locator is the reference function used by the GIS software as the basis for the geocoding operation. The iterative geocoding uses a series of address locators. For street centerline geocoding (steps 4-6) the source material is the Kansas City metropolitan area street centerline shapefile provided by MARC. Parcel level geocoding uses the NHCS subdivided by year of survey (not survey program). While there is only one reference file for street centerline geocoding there are fourteen reference files for the parcel geocoding. In addition to separating the NHCS by year minor adjustments are needed to make the address fields internally consistent (such as making sure there is only one abbreviation for avenue in the street type field).

Step 2: Create address locators. Address locators are created by the ArcMap function Create Address Locator through dialog box. There are many parameters in the dialog box, the misspecification of any of these leads to a non-functional address locator. To create the street centerline address locator select as address type, US Address - Dual Ranges, then specify which fields in the MARC centerline file contain the range of address on each side of the centerline, the street name associated with the centerline, and the zip code on each side of the centerline. To create a parcel level address locator, select, US Address - Single House, as address type. Then specify which field in the target year's NHCS file contains the house

number, street prefix, street name, street type and zip code. The parcel level locator requires more precise inputs than the centerline version. Proper specification of the address locator is essential, it requires the manual alignment of reference categories with referent fields. After the address locator has been created it is tested on well understood data, the output is scrutinized, and the match rate examined all as a quality control procedure.

Step 3: Separate, clean and observe health encounter data. To keep the matched health encounter within a calendar year of the housing conditions survey, separate the BPb data by year. Remove any encounters from the data that cannot be geocoded. Data cannot be geocoded for several reasons; the cell may be blank; it may have an explicit value such as "bad" or "unknown" containing no useful information; the associated address may be a post office box. Also removed are observations that are not relevant to the research agenda, in the example of pediatric lead poisoning that means blood lead observations of individuals older than 216 months (18 years). Table 6.5 Preparing Blood Lead Data for Geocoding describes this preparatory step. Note that the number of tests doubled from the beginning of the period to the end of the period and the inverse trends in bad addresses and encounters of patients over 216 months.[10]

Step 4: First round street centerline geocoding. Use the street centerline address locator developed in step 2 with the default settings on one year of data prepared in step 3. This automated geocoding process creates an output indexed in three ways: matched, tied, and unmatched. Matched output has been successfully geocoded and is ready for later use. Output identified as tied has been ambiguously geocoded, the automated process has placed it in more than one location. This issue

[10] I discuss the BPb data in detail immediately following the discussion of the geocoding process.

will be addressed later. Tied output is merged with matched output and is ready for later use. The third level of output from the first round of geocoding is unmatched,

Table 6.5: Preparing Blood Lead Data for Geocoding

Year	All Encounters	Over 216 Months	Bad Addresses	# encounters under 216 months with addresses suitable for geocoding
2000	9245	18	3906	5321
2001	9914	258	4177	5479
2002	8982	449	1120	7413
2003	8552	524	823	7205
2004	10055	520	2541	6994
2005	8108	533	1724	5851
2006	14576	1144	195	13292
2007	15617	1067	86	14477
2008	17666	1887	175	15697
2009	20284	2421	183	17784
2010	20188	2483	182	17618
2011	20693	2866	149	17741
2012	18928	2470	106	16391
2013	18783	2284	98	16438
Totals	201591	18924	15465	167701

n.b. Patients over 216 months (18 years) are excluded from the geocoding process. "Bad Addresses" includes P.O. box addresses and other field values ("bad address", "no address", etc.) that are not suitable to parcel geocoding.
Source: Developed by author in the geocoding process

these encounters are isolated from the rest of the output and prepared for the next step in the iterative geocoding process.

Step 5: Second round street centerline geocoding. The unmatched encounters from step 4 are the input for a second pass at street centerline geocoding. For this step in the ‘geocoding options’ tab that appears as part of the ‘geocode addresses’ process lower the three criteria (spelling sensitivity, minimum candidate score and minimum match score) from their default

settings (85-75-80) to 70-65-70. This slightly relaxes the requirements for a successful address match to incorporate the marginal encounters excluded by the first-round process. As with step 4, matched and tied addresses are passed along to the next step in the geocoding process. Those encounters that are still unmatched after two rounds of geocoding are filtered by address and zip code to catch any addresses that suffer from incorrect zip codes. Any addresses that remain unmatched after two rounds of centerline geocoding are isolated from the rest of the output and do not pass on to the next steps in the geocoding process.[11] Table 6.6 reports the match rate of two rounds of street centerline geocoding for the 14 years of this analysis. Note the number of parcels in the second round are an order of magnitude.

Table 6.6: Street Centerline Matches (2 rounds) Reported by Year

Year	Round	Number	Matched	Tied	Unmatched	Match Rate
	1	5321	4836	54	431	91%
2000	2	431	76	6	349	92%
	total		4912	60	349	92%
	1	5479	5083	62	334	93%
2001	2	334	95	10	226	95%
	total		5178	72	226	95%
	1	7413	6964	56	393	94%
2002	2	393	135	7	251	96%
	total		7099	63	251	96%
	1	7205	6681	77	447	93%
2003	2	447	142	6	299	95%
	total		6823	83	299	95%
	1	6994	6523	59	412	93%
2004	2	412	154	12	246	96%
	total		6677	71	246	95%
	1	5834	5521	40	290	94%
2005	2	290	113	1	176	97%
	total		5634	41	176	97%
	1	13292	12741	56	495	96%
2006	2	495	220	8	267	98%
	total		12961	64	267	98%

[11] Future versions of the iterative geocoding process would do well by including after this step a zip code level geocoding operation. Step 5 includes a zip code correction process so the match rate would be higher than if the process were run earlier. Further the zip code level address matching could be used as a first filter for encounters that lie far afield from the target NHCS geography.

Table 6.6 (continued)

Year	Round	Number	Matched	Tied	Unmatched	Match Rate
	1	14477	13980	32	465	97%
2007	2	465	233	6	226	98%
	total		14213	38	226	98%
	1	15697	15223	45	429	97%
2008	2	429	241	2	186	99%
	total		15464	47	186	99%
	1	17784	17206	60	518	97%
2009	2	518	276	4	238	98%
	total		17482	64	238	98%
	1	17618	17079	46	493	97%
2010	2	493	259	3	231	98%
	total		17338	49	231	98%
	1	17741	17188	40	513	97%
2011	2	513	280	3	230	98%
	total		17468	43	230	98%
	1	16391	15871	44	476	97%
2012	2	476	259	2	215	98%
	total		16130	46	215	98%
	1	16438	15934	40	464	97%
2013	2	464	235	3	226	98%
	total		16169	43	226	98%
Totals:		167684	163548	1525	3366	98%

Source: Developed by author in geocoding process.

Step 6: Merge and Select Relevant Addresses from the centerline match process. To prepare for parcel level geocoding the matched and tied encounters from the centerline process are merged into a single file. This new file is then subset by proximity (100m) to the NHCS geography of the year in question. The selected encounters are the centerline matches likely to be matched to the parcel geography. These selected encounters are passed along to step 7. The entire merged centerline file is retained separately for uses outside of this investigation.

Step 7: Automated parcel level geocoding. The matched, merged and selected encounters from step 6 are exported from the .shp file format to a database format (e.g. .csv, .xls, etc.) in preparation for the parcel level geocoding. The exported encounters are the input for an initial geocoding via an address locator based on one year of the NHCS. The matched encounters have been successfully geocoded to the NHCS parcel geography; these encounters

pass along to step 9. For this the parcel level geography of the iterative geocoding process the tied and the unmatched encounters are passed together to step 8 of the geocoding process.

Step 8: Manual geocoding. Each encounter address from step 7 is examined individually and cross referenced with a limited set of auxiliary address records. The permitted auxiliary address records are the "legal" field contained in the expanded collection of fields associated with some of the NHCS programs, the GIS parcel viewer maintained at the county level. Care must be taken in the search of auxiliary records such that the identification of the child's location cannot be identified through deductive methods. When investigating online sources do not type the exact address you are searching for into the search bar, rather an address at the end of the block in question, then manually investigate the parcel you investigate adding additional noise to the investigatory footprint. When all the addresses have been manually investigated pass the matched encounters on to step 9 and discard the unmatched encounters from further consideration. Table 6.7 records the results of the step 7 and step 8, typical match rates are 75% and higher. Divergence from these high match rates are from two types of issues, small samples sizes as in the case of these programs and the random character of program 31 which did not utilize the neighborhood geography but rather took a random sample from the city as a whole. The select by location function used to identify likely matches is not an efficient technique for identifying possible matches to the diffuse program 31 parcel layer.

Step 9: Assemble the results of the geocoding operations. At the conclusion of step 8 the encounters for one year will be geocoded to both the street centerline and the NHCS parcel level geography. Add a unique id field from the NHCS parcels to the corresponding parcel level encounters for future reference. After finishing year 2000 proceed to year 2001 and so forth. Tables 6.7 and 6.8 record how each year's process contains a series of four distinct

geocoding operations. Taken together the 14 years in question embody 56 distinct geocoding operations. After all of these operations are complete merge the pediatric lead encounters matched to the street centerline into a single file and merge the pediatric lead encounters matched to the parcel level into a separate geography. The parcel layer encounters are ready for use with other spatial data.

Table 6.7: Parcel Level Match Rate by Year and Program

Year	Programs	Selected in Proximity	Automated Matches	Automated Tie	Automated Unmatched	Manual Matches	Total Matches	Match Rate
2000	3	1,771	1,372	5	394	206	1,578	94%
2001	3, 6	1,544	882	7	1	5	1,270	82%
2002	3, 12	717	599	0	118	61	660	92%
2003	13, 14, 15, 16	95	75	0	20	1	76	89%
2004	16, 18, 19	208	143	5	60	22	165	82%
2005	18, 21, 22	154	85	11	58	30	115	75%
2006	24	1,130	726	2	402	177	903	85%
2007	25, 26	1,327	1,001	3	323	186	1,187	91%
2008	28	2,047	1,527	3	517	378	1,905	94%
2009	29	243	160	0	83	35	195	91%
2010	30	1,081	722	2	357	216	959	89%
2011	31	2,247	258	0	1,989	72	330	15%
2012	33	143	47	0	96	0	47	34%
2013	33	788	670	1	117	47	717	91%
Totals		13,495	8,267	39	4,535	1,436	10,345	75%

Source: Developed by author in geocoding process.

After applying the iterative geocoding process to the KCHD pediatric lead data enough information is compiled to judge the quality of the process. Table 6.6 displays match rate information about the street centerline geography. At the Street centerline level, the process is complete—a total match rate of over 98%. Note that the match rates vary across years with match rates in the early years of the study systematically lower than those in the later years. Judging the quality of the parcel level geocoding operation is a more difficult task due to the

shifting geography issue. The 'Geocoding Address Match Rate' column indicates the rate of encounters passed through for parcel level geocoding that are matched to the parcel level, 98% of these are matched to the centerline level but only 6% are matched to the parcel level. However, the majority of these encounters are irrelevant to the parcel level geocoding process. In half of the years less that 750 encounters are within 100m of an NHCS parcel while the number of health encounters can number over 20,000, and in no year are even 50% of the health encounters within the relevant proximity. An important aspect of the iterative geocoding process developed for this dissertation is that it produces a 'warranted' match rate, one that only takes into account those encounters likely to be part of the shifting geography of the NHCS. Three years of the study have warranted match rates under 80%. The target geographies of those anomalous years are comprised of diffuse parcels with no coherent neighborhood geography suitable to the sub-setting process in step 6.

There are no established standards for what qualifies as an acceptable match rate. For instance a multi-level study with filtering to a restricted geography published results based on a raw match rate as low as 4.3% (Richmond-Bryant et al. 2013). That study did not involve any manual geocoding and so the warranted match rate was not listed. As is the case in my dissertation justifications for the match rate are clearly stated. Other studies similarly focused on large accumulations of address level data exhibit match rates between 69% and 97.4% (McElroy et al. 2003; Goldberg et al. 2008; Oyana and Margai 2010; Sonderman et al. 2013). Another way to think about the completeness of the geocoding operation is as a sampling process. It is ideal to use the entire population. Given limits in time, money, and subject privacy, no geocoding process will be perfect there is a trade-off between match rates, accuracy

and cost thus transparency regarding match rates is key (Rushton et al. 2006; Zandbergen 2009).

Geocoding to less precise geography can increase the completeness of a geocoding operation. For instance, the entire KCMO Health lead data set could reasonably be geocoded to the geography of the Kansas City metropolitan area. If we were to do that all the encounters would be placed in the same location stacked at the center of the centroid of the shape of the metro area. Clearly, completeness in that case comes at the expense of accuracy. Alternatively, much health research takes place at the level of an administrative geography (zip code, census tract, etc.) to protect the anonymity of patient, this too introduces issues of accuracy when the unit of analysis is the individual. When working with address level health data the parcel level, the parcel's centroid, is preferable to the street centerline level. Centerline matches, logically, are based on the centerline geography and not based on the parcel geography. Centerline matches introduce positional errors that should be avoided if possible. On the basis of parcel level matches of the type produced by my iterative method it is possible to generate precise spatial associations.[12] It is critical to never display/share these precise results in a way that would allow the identity of the identification of an individual.

A minimum standard for scientific research is reproducibility. In the context of the iterative geocoding process reproducibility is ensured by placing restrictions on the discretion and judgement of technicians in the match process, thus the explicit parameters in the automated match process described in steps 4 and 5, as well as the specifications of source material in step 8. From the perspective of process, the reproducibility of my iterative

[12] It is possible to generate more precise address locators that place an encounter in a structure, at an elevation in the structure, even in a room in that structure. Such precision can is useful for some health applications but is unnecessary for this analysis.

geocoding process is demonstrated by its application to multiple health data sets, pediatric asthma and pediatric lead, utilized in this dissertation and that of Natalie Kane (2020).

The flexibility criterion suggested in 'Using GIS to Advance Social Economics Research' (2019) is judged with regard to the variety of applications the output of a geocoding process can be put to. Parcel level data is ideal for detailed health research, but it includes only a subsection of the data. Centerline data is appropriate for mapping the density of outcomes across the extent of the metropolitan area down to the community district geography. Additionally, it is appropriate to aggregate street centerline matches up to more general geographies (i.e. census geographies, neighborhoods, community districts etc.) when an investigation calls for such operations.

The largest share of the cost for inhouse geocoding is the time of the technician. The iterative geocoding process is designed to reduce costs in the manual geocoding process by limiting the number of encounters to be considered in the manual process while adding the street centerline process in the process. Table 6.8 reports the difference between the total number of centerline matches (164,332) and the number of automated parcel matches (8,267) is 156,065 encounters, while the number of unmatched encounters examined in the manual geocoding process (from table 6.8) is 4,535. To perform a manual geocoding operation resulting in the same number of match encounters would on a cost nearly 34 times as much. The other option would have been to design the iterative process without the centerline steps and focusing only on the parcel geography. Losing the centerline matches would have negative implications for completeness of the operation (the warranted match rate would be much lower, and the number of matches would be lower due to the specificity of the parcel level address

locator) the flexibility of the output would also be negatively affected, and the output would only be suitable for my research agenda.

Table 6.8: Comparison of Final Match Rates

Year	All Encounters	Encounters Under 216 Months Less	Total Centerline Matches and Ties	Centerline Matches in Proximity to	Total Parcel Matches	Raw Match Rate	Geocoding Address Match Rate	Centerline Match Rate	Warranted Match Rate
2000	9,245	5,321	4,972	1,771	1,578	18%	31%	34%	94%
2001	9,914	5,479	5,250	1,544	1,270	13%	23%	24%	82%
2002	8,982	7,313	7,162	717	660	7%	9%	9%	92%
2003	8,552	7,205	6,906	95	76	1%	1%	1%	89%
2004	10,055	6,994	6,748	208	165	2%	2%	3%	82%
2005	8,108	5,851	5,675	154	115	1%	2%	2%	75%
2006	14,576	13,292	13,025	1,130	903	7%	7%	7%	85%
2007	15,617	14,477	14,251	1,327	1,187	8%	8%	9%	91%
2008	17,666	15,697	15,511	2,047	1,905	11%	12%	12%	94%
2009	20,284	17,784	17,546	243	195	1%	1%	1%	91%
2010	20,188	17,618	17,387	1,081	938	5%	5%	6%	89%
2011	20,693	17,741	17,511	2,247	330	2%	2%	2%	15%
2012	18,928	16,391	16,176	143	47	0%	0%	0%	34%
2013	18,783	16,438	16,212	788	717	4%	4%	4%	91%
Totals	201,591	167701	164332	13,495	10086	5%	6%	6%	77%

Source: Developed by author in geocoding process.

At a manual re-match rate of 50 encounters per hour the iterative rematch process took approximately 90 hours at $20 per hour. On these terms the iterative process saved an estimated $59,862, a prohibitively large figure. The iterative geocoding process, the second contribution of this dissertation, allowed this research to take place in a cost-effective manner.

Blood Lead Data

The datasets distinguishing my analysis are the de-identified address level blood lead testing data provided by the Health Department of Kansas City Missouri. These data are kept

in accordance with Chapter 710.326 of the Missouri State code, originally passed in 1992 and most recently updated in August of 2001. The relevant section of the statute reads, "The department of health and senior services shall establish and maintain a lead poisoning information system which shall include a record of lead poisoning cases which occur in Missouri along with the information concerning these cases which is deemed necessary and appropriate to conduct comprehensive epidemiologic studies of lead poisoning in the state and to evaluate the appropriateness of lead abatement programs" (Missouri Revisor of Statutes 2001). The updated 2001 legislation mandated universal pediatric lead screening and required the creation of a statewide lead screening plan which in turn identified risk criteria on the basis of geography and socio-economic status. Critically, universal screening does not entail universal lead testing. Only those children flagged via risk assessment criteria are required to be tested. However, if the screening criteria were followed to the letter it would lead to universal testing. There are three types of lead screening criteria: spatial, economic, and behavioral.

If a child resides, spends at least 10 hours per week or attends a day care facility in an identified high-risk area, guidelines stipulate that child should have their blood lead levels tested every year. Missouri is the top lead mining state in the US, as a result there are 35 counties the entirety of which are classified as high risk. There are an additional 7 counties sections of which are identified as high-risk. Jackson county, the county at the center of the Kansas City metropolitan area, is one of these counties. Kansas City, MO and greater Jackson county contain 16 zip codes identified as high-risk areas ("Missouri Childhood Lead Poisoning Prevention Program" 2018). These spatial guidelines should, in the high-risk zip codes, lead to universal testing.

The socio-economic criteria for lead testing is all MO Healthnet Division *eligible* children are required to take a test at 12 and 24 months ("Missouri Childhood Lead Poisoning Prevention Program" 2018). Children are MO Healthnet Division eligible (regardless of their family's insurance status) if their the monthly family Modified Adjusted Gross Income (MAGI) meets one of the following criteria, 196% of the federal poverty line (FPL) for children under 1, and 148% FPL for children 1-18 years.[13] Additional criteria apply to uninsured children; a family MAGI up to 150% FPL, or if the family MAGI is 150% - 300% FPL the child may be eligible for subsidized premium coverage ("MO HealthNet for Kids | Mydss.Mo.Gov" n.d.). There is considerable overlap between the spatial and socio-economic screening criteria, together they should cast a fairly wide net.

The behavioral criteria are determined through a lead risk assessment questionnaire which asks 9 questions. An affirmative question to any of these questions will trigger a BPb test. One of the assessments on the questionnaire, is the child "Between 12 & 72 months and has never received a blood lead test?" ("Healthy Children and Youth Lead Risk Assessment Guide" 2009), implies that a thorough application of the questionnaire would create a universal testing database on a 5-year rolling basis. According to the data provided by KCMO Health the 5-year pediatric population testing rate for the 109 zip codes in the MO side of the metropolitan area was 44.35%. The testing rate for the 16 high-risk zip code in Kansas City and Jackson county was higher, 58.44%. The numbers are far worse for the single year 2010 where the rolling average was 19.57% for the entire metro with the high-risk zip codes doing

[13] In 2000, the first year of this study the FPL for a family of four was $17,050. In 2013, the last year of the statistical analysis that number was $23,550. 300% of the FPL was $51,150 and $70,650, respectively. ("Prior HHS Poverty Guidelines and Federal Register References" 2015)

somewhat better at 25.03%. Recall the spatial and socio-economic guidelines stipulate annual testing for at-risk children.[14]

There are two different blood lead tests in general use, capillary and venous exams. There are significant differences between these tests, most critical among them is the possibility of residual lead contamination making its way from the skin to the sample in the case of the capillary (or finger prick) exam. The possibility of residual contamination creating erroneously high-test results spurred state and national guidelines recommending any child with an elevated capillary-based test result[15] be subject to a confirmatory via the venous method. For test results that are significantly higher than the reference level it is recommended that a follow up exam be performed within three months. The technologies for performing the analysis on capillary and venous exams is significantly different and will be discussed in the section on the limitations of the BPb testing data.

The finger prick (or capillary) exam is quick, relatively inexpensive, non-invasive, and can be performed in the field as part of a targeted testing program. The principal concern with the technique is the possibility of sample contamination from lead present on the skin of the child (Harvey 2005). However, research has indicated that there is no inherent reason that capillary blood draws will lead to higher lead results. In a laboratory environment the mean difference between simultaneously drawn venous and capillary samples is less than 1µg/dL (Schlenker 1994). Nonetheless, due to the possibility of sample contamination, the CDC

[14] Further discussion of the short comings of Missouri's testing regime follows in the section on the limitations of the blood lead data. The causes of these low testing rates are beyond the scope of this dissertation, but it should be noted that there is significant heterogeneity among the high-risk zip codes.

[15] Defined as a test result over the reference value, currently 5µg/dL. For the first 10 years of my analysis 10µg/dL was referred to as the level of concern.

recommends that any child testing above the reference level (currently 5µg/dL) in a capillary exam be retested using a venous exam. The intravenous blood draw has the advantages of lower chance for contamination and the possibility to be paired with other blood-based health exams. The drawbacks of the venous exam are cost (the recommendation that high capillary exams be re-tested makes detection of higher levels of lead poisoning more expensive though more exact), time (results are lab based rather than available from a dedicated mobile machine and not available on the spot), loss of participation (taking a child to a clinic or hospital is an investment of time that may be unreasonable for undocumented or otherwise precarious families), and invasiveness. This last point is underdiscussed in the scholarly literature but well understood to practitioners, the experience of having blood drawn can be traumatic to children and a non-starter for parents who may project a fear of needles on to their children. Indeed, a study of testing rates in a high-risk community indicates that switching to capillary exams from venous draws can have a substantial impact on screening rates, particularly among the very young. One study tracking a change in testing practices among birth cohorts observed the proportion of children tested before 24 months of age to increase from 63% to 98% and one year testing rates increased from 39% to 75 from 2008 to 2012 (Boreland et al. 2015).

The de-identified blood lead data provided by KCMO Health contains information about the child and the conditions of their test. Table 6.9 is a data dictionary from the BPb dataset. My analysis makes use of the age of child in months, sex, address information, jurisdiction, and collection date fields in addition to the test type and test result fields.

Table 6.9: Lead Data Dictionary

Field Name	Data Type	Definition
Collect_Date	date	Date on which BPb sample was taken m/d/y
DOB	date	Done on which child was born m/d/y
DCN	numeric	Department Client Number. Unique Identifier: 8-digits
GENDER	character	Sex of child
STREET_ADDRESS	character	Street address, single field
CITY	character	City of address
STATE	character	State of address
ZIP_PLUS_4	numeric	Zip plus 4 of address
JURISDICTION	character	Jurisdiction of address: Health Department
COUNTY	character	County of Address: Clay, Jackson, Platte
COUNTY_FIPS_CODE	numeric	Numeric code of County: 47, 95, 165
TEST_RESULT	numeric	Measured BPb level, test result in micrograms per deciliter (µg/dL)
AGE_AT_TEST_MONTHS	numeric	Age of child in months
SAMPLE_TYPE	character	Type of blood lead test: Venous, Capillary, or Unknown
SC_UOM	character	Unit of measure of blood test: micrograms per deciliter (µg/dL)

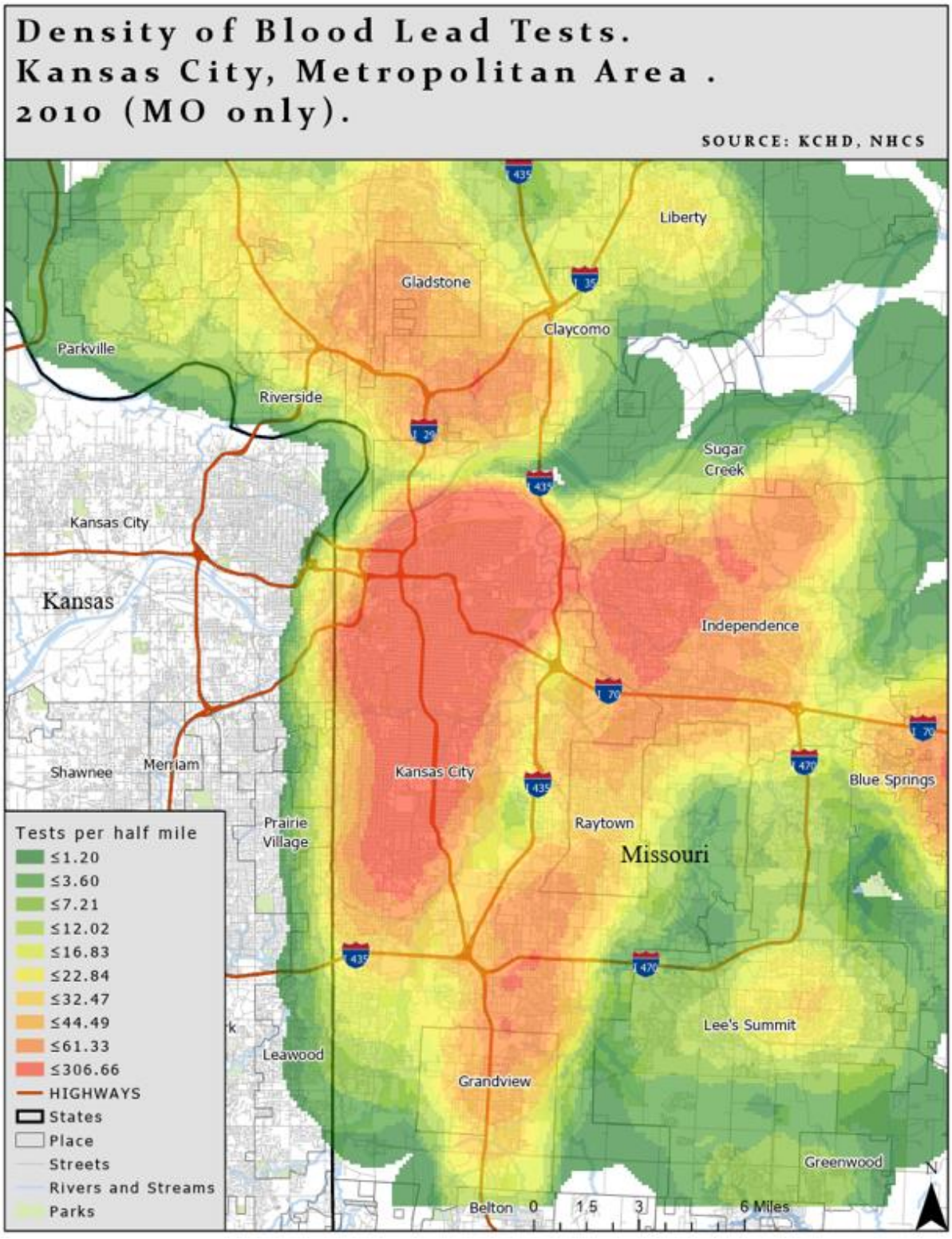
Source: KCMO Health Department

The DCN field may be useful to future pediatric lead researchers. Anyone who receives a service from the Missouri Department of Social Services has a DCN (department client number) assigned to them, and anyone born in the state of Missouri since 1994 has been automatically assigned a DCN (MO DSS 2013). It is Missouri's equivalent of the federal Social Security number. The mandatory pediatric lead test reporting (from which the KCMO Health data owes its provenance) uses the DCN to ensure that only one test per child per year is reported. In the event that a child is tested more than once in a year only the highest test value is kept in the state archive. In my analysis each test result is modeled as an independent observation (the same child may be observed in multiple years) in keeping with the biology of lead poisoning. Lead is detectable in the blood within 15 minutes of inhalation. Within a day of oral ingestion the subject's blood will contain over 50% of the ingested lead (Leggett 1993).

Studies featuring repeated BPb testing of children over a six month period observed relatively stable blood lead levels due to consistent exposure to lead in the environment (M. E. Markowitz et al. 1996). The removal of lead from the body is complicated, but a baseline estimate for the half-life of lead in the blood stream is 35 days (Lidsky and Schneider 2003). These articles imply that repeated observations of a child's blood lead level may produce similar test results, but these test results are indicative of the continual exposure to a toxic environment rather than the legacy of a single encounter with a toxic substance.

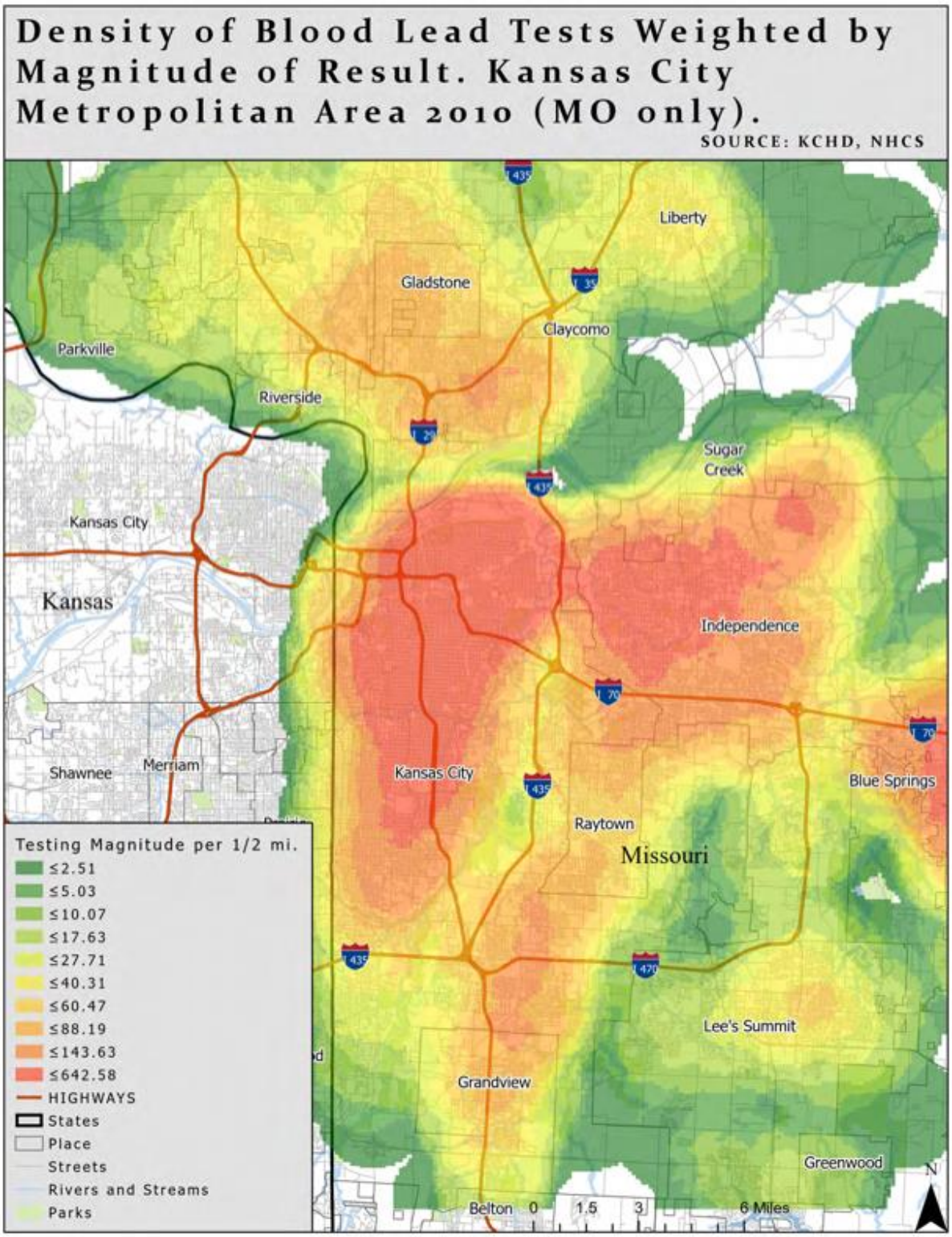
The address fields facilitate the spatialization through GIS of the pediatric lead test data. Street level addresses allow for geocoding to the NHCS data and to the street centerline geography. Though it is not explored in this dissertation the street level addresses will allow for the selection of particularly high-risk children; those that are living in temporary housing, motels, women's shelters, etc. Maps 6.2 and 6.3 put the address level data to work aggregating all the encounters from one year and by doing so reveal the spatial pattern of lead poisoning in Kansas City. The individual addresses can be used together in a way that provides insight into lead poisoning while protecting the anonymity of the individual that has been tested. Though these maps only consider the year 2010, the patterns produced here are reproduced in each year of the study. These maps describe a consistent geography of blood lead poisoning and testing. Combining magnitude of the test results in combination with the number of tests given, map 6.3 illustrated minor differences with map 6.2 which is a map of testing densities along. The area of Kansas City, Missouri near Prairie Village, Kansas experiences less intense lead poisoning than is expected from map 6.2 and the north eastern sections of Independence towards Sugar Creek experiences more intense lead poisoning than would be expected by looking at map 6.2 alone.

Map 6.2: Blood Lead Tests Per 1/2 Mile



Map created by Author

Map 6.3: Blood Lead Tests Per 1/2 Mile Normalized by Magnitude of Test



Map created by author.

For the 14 years studied in this dissertation (2000-2013) the mean pediatric blood lead level was 2.46µg/dL (sd 2.36µg/dL). Table 6.10 indicates that blood lead levels fell by nearly 50% during the period of analysis from a mean of 3.86µg/dL in 2000 to 1.99µg/dL in 2013. Meanwhile the distribution of test results narrowed more considerably as measured by the standard deviation from 3.54µg/dL to 1.45µg/dL. Looking at the mean and standard deviation of the KCMO Health data all together and over time tells us about the situation in Kansas City but it gives little context to these figures beyond what we already know about pediatric blood lead levels.[16] The fifth column of table 6.10 reports a reference value for the 97.5th quantile of each year in my study. A national reference value of 5 µg/dL (which replaced the 10µg/dL level of concern which had been set in 2010) was set in 2010 on the basis of the distribution of NHANES BPb test results (97.5% of the tests in that cohort had a test result at that level or lower) ("Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention" 2012). Recall, the reference level is not intended as a biological threshold or level beneath which exposure is medically.[17] It is clear by inspection that the reference level for the KCMO Health data is higher for every year of the study than the nation reference level.

A more consistent comparison with the national data can be had by comparing the geometric mean (and the 95% confidence interval around the mean) of the KCMO Health data with that of the two-year NHANES surveys covering the period 2000-2015. Note that the comparison here is between *geometric* means $\left(\sqrt[n]{x_1 * x_2 * \ldots * x_n}\right)$ rather than *arithmetic*

[16] For instance, any amount of blood lead is too much (Flegal and Smith 1992) and BPb test results have generally been falling since the late 1970's (T. Dignam et al. 2019).

[17] The level at which interventions are performed to ameliorate lead poisoning is determined by the resources and priorities of communities and individuals ("Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention" 2012) because there is no safe level of pediatric blood lead exposure.

Table 6.10: Mean BPb by Year

Year	Observations	Mean	SD	Max	97.5th Quantile
2000	8,772	3.86	3.55	63	12
2001	9,120	3.55	3.06	86	11
2002	8,020	3.69	3.38	81	12
2003	7,554	3.54	3.14	49	11
2004	9,075	2.92	2.89	46	10
2005	7,056	3.09	3.08	49	10
2006	12,686	2.34	2.30	57	8
2007	13,387	2.09	2.14	58	7
2008	14,770	1.87	1.68	40.4	6
2009	16,776	2.02	1.67	39	6
2010	16,639	2.11	1.65	74	5
2011	16,800	2.11	1.61	66.9	5
2012	15,535	2.05	1.53	35	5
2013	15,636	1.99	1.45	42	4.4
all	171,826	2.46	2.36	86	8

source: KCMO Health Department

means $((x_1 + x_2 + \cdots + x_n)/n)$. The geometric mean is always equal to or less than the arithmetic mean value. It is common practice to use geometric means when comparing changes to observations that are measured on different scales because the geometric mean normalizes changes to the mean value. For instance, a 20% change in a rating that goes from a magnitude of 5 to 6 has the same effect on the geometric mean as a 20% change in a rating that goes from a magnitude of 100 to 120. Blood lead observations from NHANES data are summarized using the geometric mean. The comparison with NHANES figures is the only place in which the geometric mean will be used in this dissertation. More about the choice and computation of mean statistics follows shortly.

Apparent upon inspection of the national trend (and echoed in the local trend of falling blood lead levels) in figures 5.2 and 5.3 in chapter 5 is a persistent decline in BPb. That trend

is also clear in table 6.10 and the cited literature. A close look at these figure reveals how persistently higher than the national sample the KCMO Health sample is. Only in the 2006-2007 cohort is the KCMO Health sample within the 95% confidence interval of the NHANES sample. The geometric means begin the period far apart, ~1µg/dL (26% higher) in 2000 and appear to be moving towards convergence through the 2006-2007 period. After 2006-2007 the NHANES data continues on its downward trend while the KCMO Health sample reports a stubborn stasis in blood lead levels. At the end of the 2015 period the KCMO Health sample is farther from the NHANES sample in absolute and proportional terms (~1.5µg/dL, ~2x higher) than it was in 2000.

There are important shortcomings in the KCMO Health data that must be acknowledged. The calculation of the geometric mean brings attention to the technical issues surrounding the limit of detection calculated for a particular test. The calculation of a geometric mean statistic does not allow for zero values, a single zero value in a list, no matter how long, produces yields a geometric mean of zero. During the 14 years of the survey period the quality of testing procedures improved such that increasingly minute amounts of blood lead are identifiable by the testing equipment (Caldwell et al. 2017). The enhanced resolution brings into question the ability of the test to accurately identify blood lead in trace amounts below some lower limit.[18] Do null values indicate the absence of blood lead is a question as much about the technology of measurement as it is about the BPb being measured. As a question for the technology, the issue is whether the machine can consistently return 0 results if the test is being run on a blank sample, this is the limit of blank ($LoB = mean_{blank} + 1.645(SD_{blank})$).

[18] This technical issue is separate from the question of whether it is realistic to say that in the contemporary lead saturated environment it is possible for children to have a zero BPb.

the limit of detection is the lowest concentration likely to be reliably distinguished from the limit of blank and at which detection is feasible (Armbruster and Pry 2008). The limit of detection is always higher than the limit of blank ($LoD = LoB + 1.645(SD_{low\ concentration\ sample})$). The NHANES lab work documents state that recorded measures below the limit of detection are to be reported as $LoD/\sqrt{2}$ for analytical purposes (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS) 2018) thus resolving issues that could arise in computing the geometric mean and accounting for the potential imprecision of the test device.

To compute the geometric mean for the KCMO Health data I undertake a similar substitution, revealing another shortcoming of the BPb data. Although it does report the type of procedure used to sample the child's blood the KCMO Health dataset does not report the exact testing equipment was used to run the tests. I do not have access to the equipment specific LoD figures, nor do I have access to information about the general precision of the testing equipment. To compute the statistics reported in figure 5.4 I use the reported LoD levels in the NHANES literature.[19] The figure 5.4 estimates are the only statistics which utilizes this substitution. By not using this substitution in my other estimations I am making a different set of assumptions, accepting that it is possible for a child to have zero blood lead and that the testing procedure is exacting enough to distinguish a null result from an inclemently higher number. I also assume that any difference in the precision of the test types is immaterial to the statistical analysis which I preformed.

[19] These limits of detection range from .6µg/dL in 2000 to .07µg/dL in 2015 (Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCHS) 2001; Caldwell et al. 2017).

There are problems with the address data as provided. The most critical problem for this study a lack of length of tenancy information. Looking at the data provided it is unclear if a particular child has lived in the home for a single year, for their entire life, or if their family moved from the home immediately after the blood lead test. Thus, some assumptions are required for the address match process. The focus of this analysis being on the relationship between lead poisoning and the built environment, I made a semi-restrictive assumption to match only KCMO Health observations to NHCS observations that took place in the same calendar year. More restrictive or inclusive assumption about tenancy will change the number of warranted KCMO Health observations and the outcome of the geocoding process.[20] The majority of the clerical errors in the recording addresses are corrected in the match process. One type of clerical error is more serious. In the year 2000, 232 geocoded encounters were associated with the same single-family street address on Drury avenue, an impossibility. This same clerical error associated with the same address is present in each of the first 5 years of the study. The cause of this anomalous address attribution is unknown. In total 626 encounters are eliminated from consideration in the statistical model due to clerical error, lowering the number of parcel level matches from 10,086 to 9,460.

Demographic data is missing from the KCMO Health data making it impossible to disaggregate the data by race or class. Social history and epidemiological literature connect race and ethnicity with lead poisoning ("Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention" 2012) thus the absence of demographic information in the KCMO Health data is problematic. The lack of demographic data may have negative

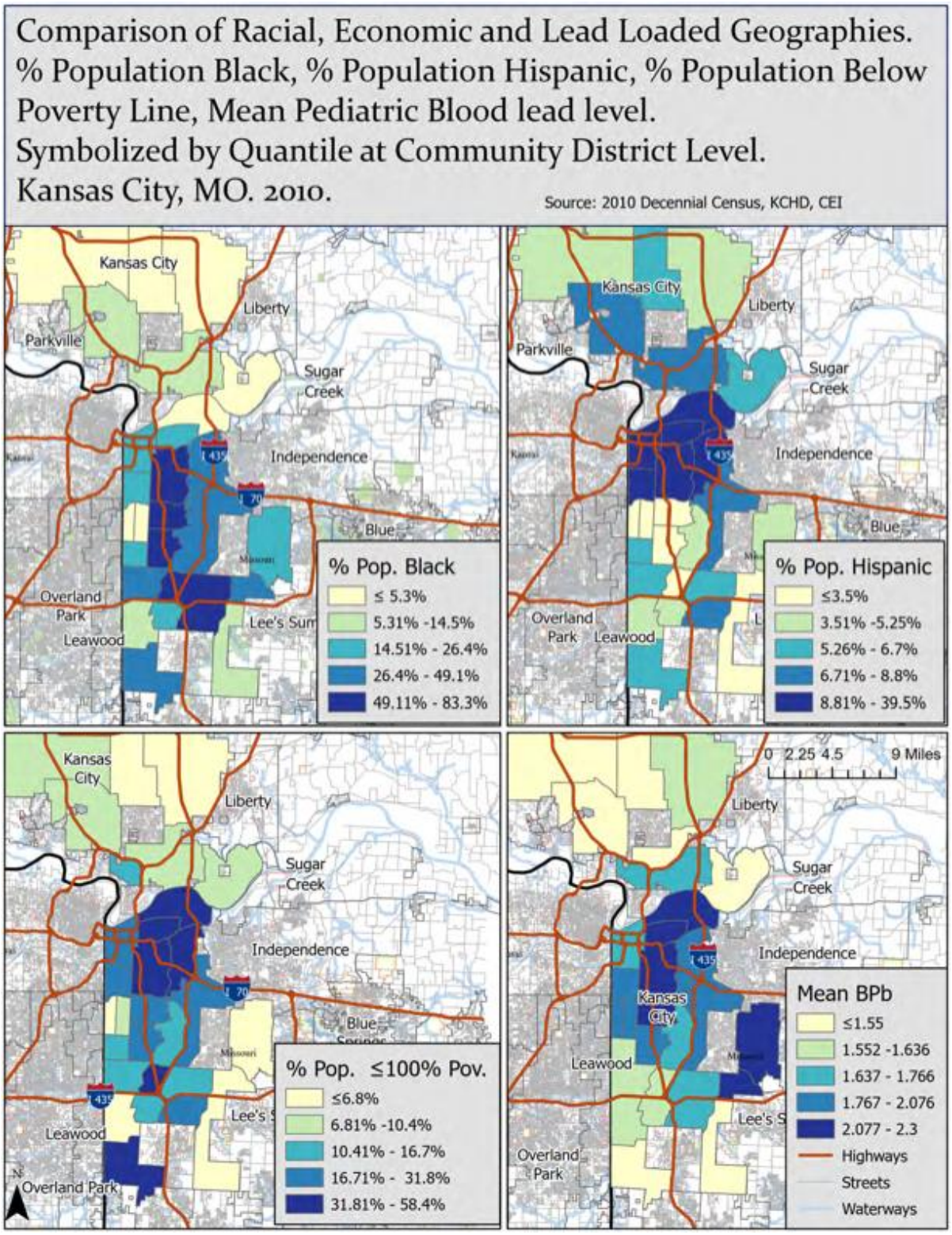
[20] Issues of tenancy are related to the uncertain geographic context problem (UGCP) discussed in Chapter 5 (Kwan 2012).

implications for my statistical analysis due to omission of an important confounder (I. H. Wilson and Wilson 2016). Equally troublesome are the negative implications for any investigation of environmental poisoning does not explicitly include race or ethnicity in its narrative of experience and social effects. An analogous set of complaints can be made about the lack of socio-economic indicators in the KCMO Health data. Another issue arises from the absence of information about parental employment status.[21] Though individual level demographic information is preferable, this study elides the weight of these concerns through the introduction of community district[22] level variables (Fortson and Sanbonmatsu 2010; Vivier et al. 2011; Moody et al. 2016). The inclusion of community district indicator variables uses the hyper segregated nature of the Kansas City metropolitan area to account for some the experience of race and class and follows from the observation, found in the 1992 Residential Lead Paint Hazard Reduction Act –Title X (and elsewhere), that lead-based environmental hazards harry *communities* and that "low income and minority communities [are] disproportionately affected [by lead hazards]" (*Residential Lead-Based Paint Hazard Reduction Act of 1992 –- Title X* 1992). Map 6.4 shows 4 critical geographies simultaneously; it shows at the community district level the portion of the local population that is Black, Hispanic, living below 100% of the federal poverty line, and the mean BPb level. The geography of segregation in Kansas City is apparent by inspection with clear majorities of the Black and Hispanic population living in distinct neighborhoods from each other and the white

[21] Parents that work in industries rife with lead have children with higher blood lead levels than the background population. (Rinsky et al. 2018)

[22] Community districts are agglomerations of neighborhoods. There are 246 neighborhoods in Kansas City, Missouri, and 32 Community Districts (24 of which are represented in statistical analysis). Appendix E contains a map of Kansas City, MO, community districts and anther map demonstrating how the neighborhoods and community districts relate to each other. The assembly health and environmental data (the first major contribution of the dissertation) includes unique identifiers for community district and neighborhood geographies.

Map 6.4: Four Critical Geographies



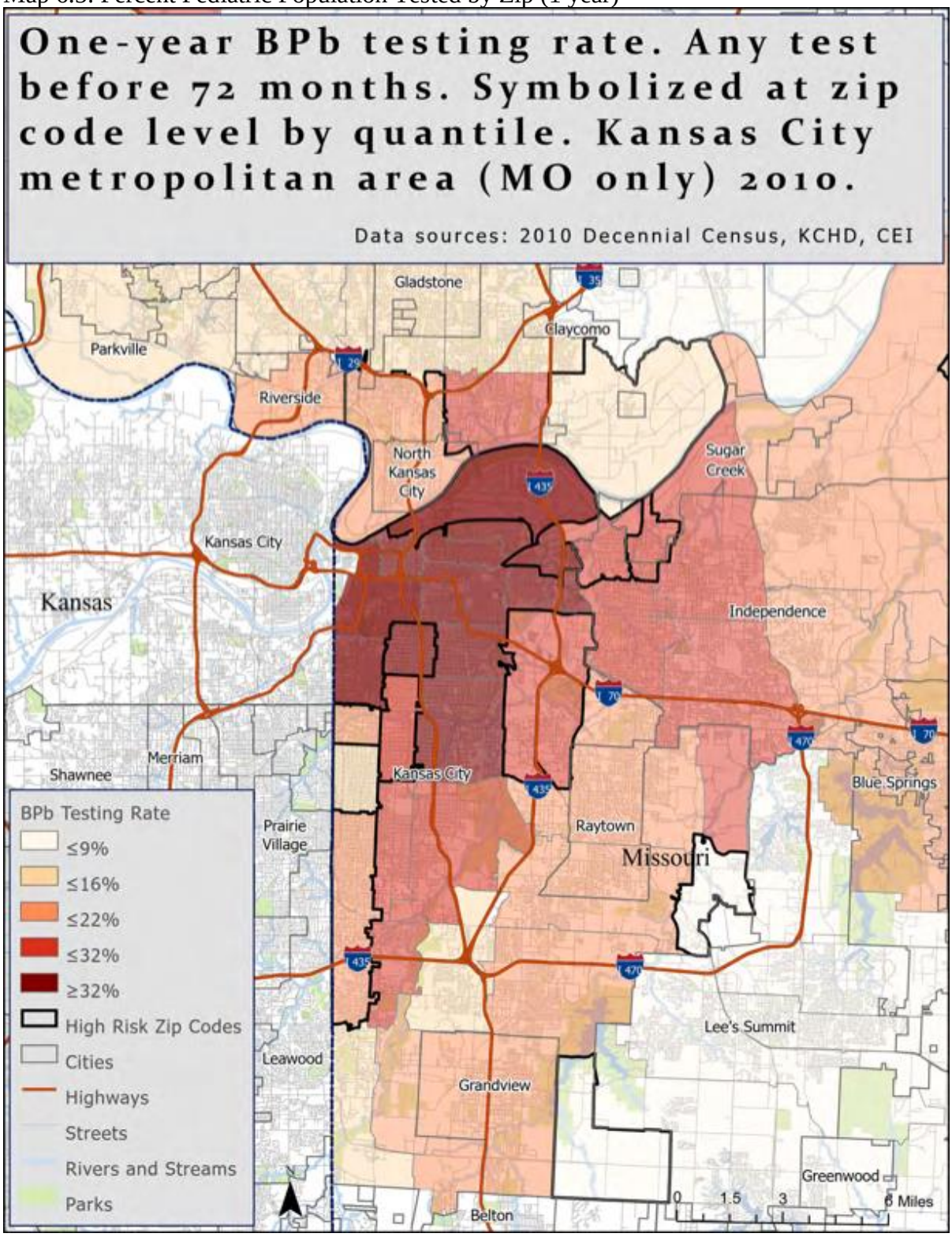
Map created by author.

population. The clustering of the low-income population (illustrated here as households making below 100% of the federal poverty line) significantly overlaps the racialized geographies. Mean blood lead levels track the racial and economic geographies well. The quartet of maps supports the practice of grouping via community district specific indicator variables. It is a useful (though not ideal) practice to capture some of the environmental context of social reality (O'Campo 2003). Visual comparison alone does not translate with statistical precision the magnitude of association between race, class, and pediatric blood lead levels. These community district variables are best understood as the beginning of a line of inquiry the aim in view being an incorporation of observations of race, class and educational status by community district and neighborhood, allowing us to quantify how these socio-economic factors correspond to the lead-based environmental risk of the spaces in which people live.

There are other issues with the KCMO Health data that must be addressed. The Sample Type variable is missing from 716 observations, and this issue is particularly pronounced in the early years of the study although there are unknown sample types in every year of this study. The prevalence is high enough that unknown test type is added as an explanatory variable in my omnibus model. Map 6.6 shows a rolling average testing rates of children under 6 for 2005-2010 by zip code. 2010 is chosen as the baseline year for the population to reference decennial census counts rather than the estimated values of the American Community Survey. Map 6.5 shows the testing rates for just the year 2010.

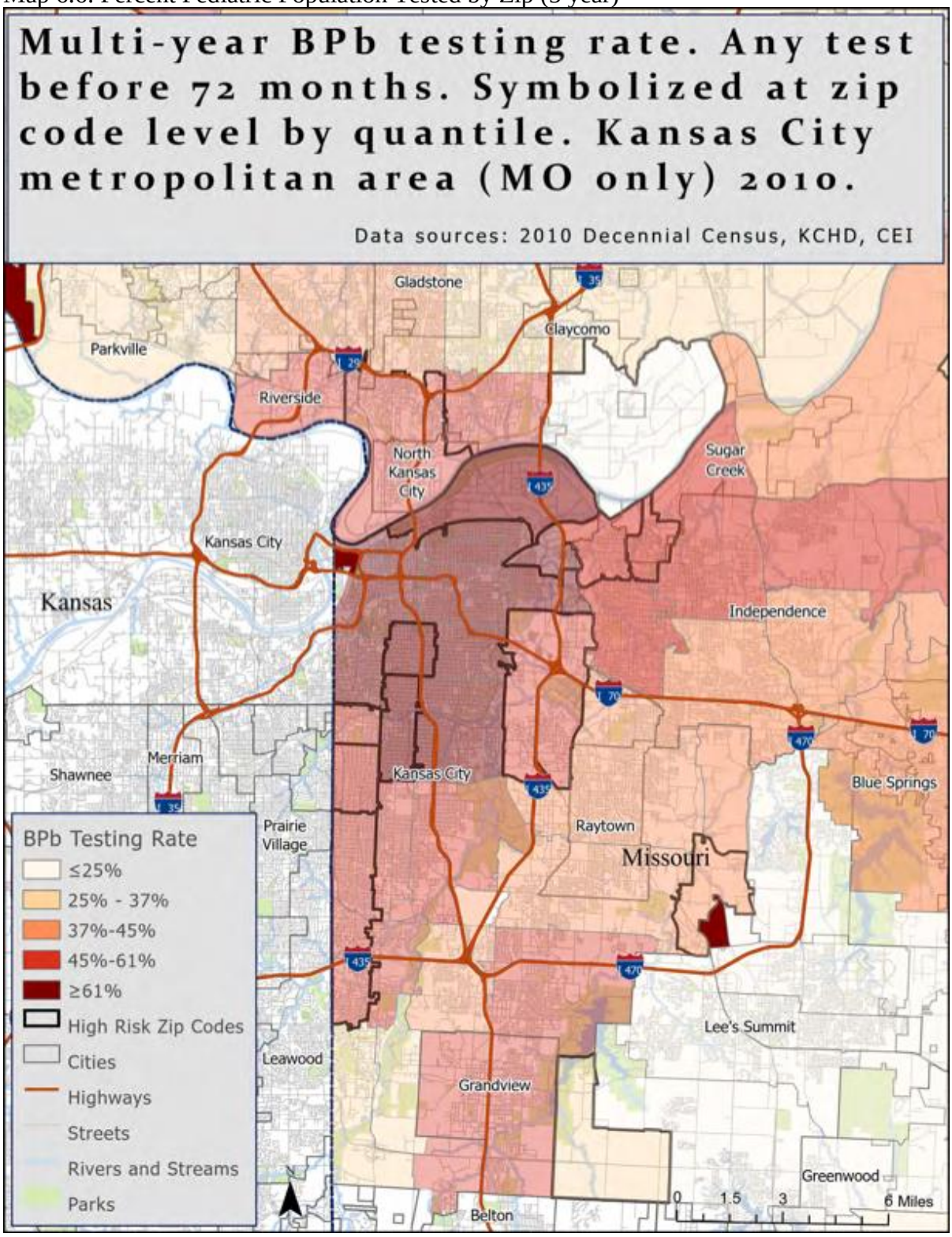
The absence of universal pediatric blood lead testing is a major problem. It rases problems for statistical analysis due to a lack of observations of children with null results, potentially introducing bias into the sample. Instead of reporting the mean blood lead level for a neighborhood or zip code, the incomplete data requires that results are reported with the

Map 6.5: Percent Pediatric Population Tested by Zip (1 year)



Map created by author.

Map 6.6: Percent Pediatric Population Tested by Zip (5 year)



Map created by author.

phrase, ‘Among the data available…’. My aim is not only robust statistical work and appropriately worded reports, the goal of pediatric lead testing is to prevent and ameliorate lead poisoning. Most critically, without universal testing the true distribution of lead poisoning is unknown. This means that children with lead poisoning grow up without knowing the nature of their condition and public health officials lack the information to fully address the problem, clusters of blood lead poisoning may persist undetected and the course of lives will be altered. Without universal testing it is unknown how many children from all walks of life experience low level blood lead poisoning. Common cause across society remains elusive. Lead poisoning continues to be a problem for the poor and the marginalized to sort out on their own with limited resources.

Testing guidelines as written would lead to universal testing of children 5 and under, however that is not what happens. Comparing 2010 census data with aggregated testing data reveals a geography of lead testing where children are far more likely to be tested if they live in the center of the city of Kansas City, Missouri. Maps 6.5 and 6.6 describe this geography clearly and show the pattern is not mitigated by the state identified ‘High Risk Zip Codes’ (“Missouri Childhood Lead Poisoning Prevention Program” 2018). Map 6.5 describes the 1-year rate of pediatric lead testing in the year 2010 by zip code, while map 6.6 shows the 5-year rate of pediatric lead testing ending in the year 2010. Comparing the geography of testing rates with the geographies in Map 6.4 illustrates that being tested for lead poisoning is as bound by racial and class divisions as are the effects of pediatric lead poisoning. There is a logic to this testing, screening the population which is most likely to have relatively high levels of lead poisoning could be an efficient use of limited testing funds. There are several problems with the testing regime. First, a test for lead poisoning is not necessarily associated with remediation

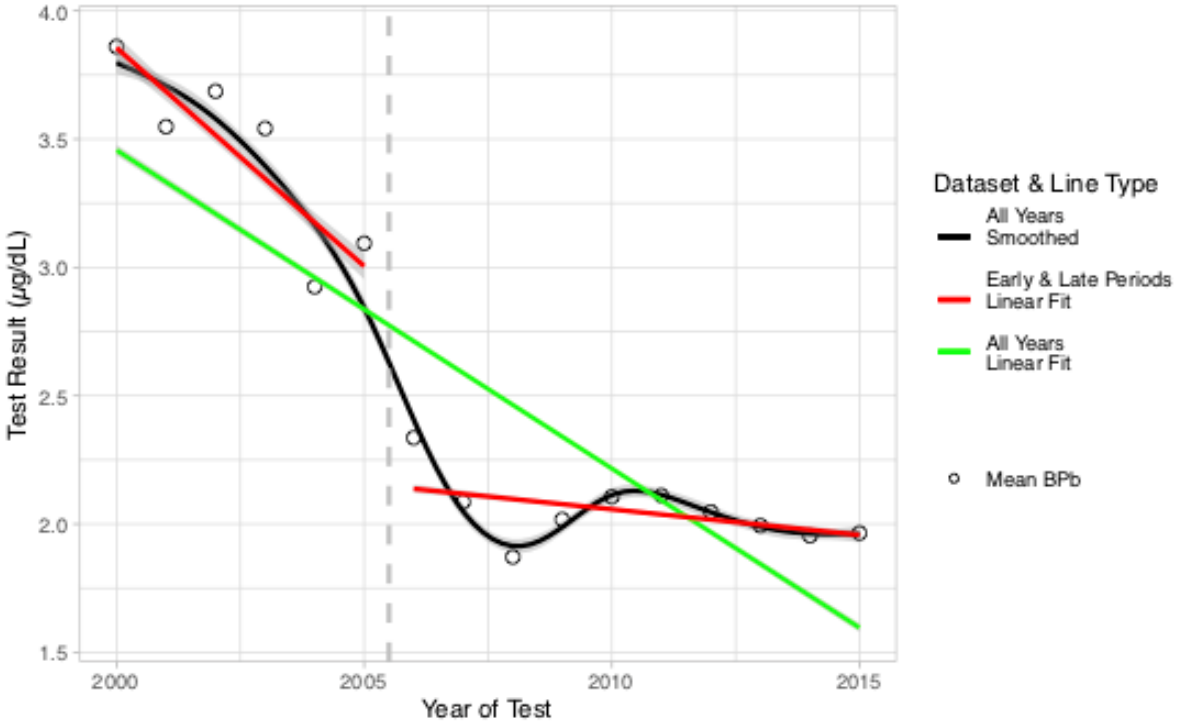
activity.[23] Second, the regime as designed will not find what it is not looking for and lead poisoning will remain a racialized problem rather than a social problem. If the only acceptable level of lead poisoning is zero then everyone should be tested until the modal BPb is zero. Only then will targeted testing make sense medically. The conceptual isolation of lead poisoning creates problems for political action by isolating the most powerful social actors from the shadow of effects. Third, the absence of universal testing creates a problem for the statistical analysis of lead poisoning. The current testing regime does not reflect the residential pattern of the Kansas City metropolitan area, thus there is the possibility that testing practices have created a sample which is biased toward those individuals that are lead poisoned thus introducing bias into any statistical analysis which the data are used for.[24]

Among the observations provided by KCMO Health there is evidence of a change in testing regimes during the study period. Table 6.6 indicated the volume of testing nearly doubles between 2005 and 2006 from 8,108 observations to 14,576 observations. Accompanying the increase in tests is a change in the trend of mean test results over time. Such a trend was in evidence in figure 5.4 where the convergence between NHANES and Kansas City BPb is shown to reverse in the later period. Figure 6.1 focuses only on the Kansas City BPb data illustrating a change in trend between the early years (2000-2005) and the later years

[23] The MO side of the metropolitan area averages well over 15,000 positive BPb test per year. Though a test can be associated with remediation, it is most likely associated with information awareness and the distribution of advisory pamphlets such as "Blood Lead Levels in Children" (Center for Disease Control n.d.).

[24] Indeed, the over sampling of the poisoned is in some ways the purpose of the data set. Furthermore, the solution I advocate is not necessarily a testing regime more amenable to statistical analysis. Though a truly universal testing regime would be acceptable the end in view is a regulatory regime that eliminates the need for lead poisoning.

Figure 6.1: Discontinuity in BPb Over Time



Source: Kansas City Health Department

(2006-2013) of the analysis. In figure 6.1 the black line is a best fit line running a non-linear path through the arithmetic mean of BPb test results for the years 2000-2013. The bright green line traces the linear trend through the same range. The two red lines trace the linear trends for the periods 2000-2005 and 2006-2013. A divergence in the slope of the early period and the late period lines is apparent upon visual inspection and a chow test confirms the discontinuity.[25]

[25] A standard technique to statistically establish a discontinuity in the data trends is to use a Chow Test. This statistic allows us to test if two groups of data come from the same population by using the sum of square errors (SSE) from the complete survey period and the SSE from the two sub periods normalized by degrees of freedom in a type of F test. The model I use is not suitable for a Chow test due to an ambiguity in degrees of freedom in multi-level modeling (for more on this issue see chapter 6). For the purpose of inspection, I estimate a chow test using a simple toy model where blood lead levels are estimated merely as a function of the year of the test.

$$BPb_{all} = f(Year_{all})$$

The model is then subset by the two periods of analysis BPb_1 (2000-2005) and BPb_2 (2006-2013). The formula for the Chow Test is:

$$\frac{(SSE_{all} - SSE_1 - SSE_2)/K}{(SSE_1 + SSE_2)/(n + m - 2K)} \sim F_{K,n+m-2K}$$

Where:

SSE_{all} is the sum of squared errors for the complete population BPb_{all}, years 2000-2013

It is unclear what to make of this anomaly, there is evidence of a significant discontinuity in trend in blood lead levels, but the cause of this change in trend is not clear. It could be the result of a change in testing guidelines (Binns et al. 2007), it could be a statistical artifact of expanding the volume of tests; it could be the result of a change in lead-related regulation; it could be the result of another yet to be discovered factor; and it could be a combination of any of these factors.

Uncertainty provokes a question, do patterns in the raw data provided by KCMO Health persist to the data geocoded to the NHCS parcel geography. Geocoding to the NHCS layer is a subsetting process with the potential to introduce selection bias into the sample. Table 6.11 displays a side-by-side comparison of the KCMO Health data (KCHD Pb) with the data set which results from the geocoding process (NHCS Pb).[26] The mean test result of the NHCS Pb subsample is 20% higher than the KCHD Pb data while the standard deviation of NHCS Pb results are 43% larger than that of KCHD Pb. The division of tests by gender and sample type

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SSE_1 is the sum of squared errors for the first population subset BPb_1, years 2000-2005
SSE_2 is the sum of squared errors for the second population subset BPb_2, years 2006-2013
K is the number of restricted coefficients
n is the number of degrees of freedom in the first population subset
m in the number of degrees of freedom in the second population subset

To carry out this test we compare the computed Chow statistic from the left side of the equation with the critical statistic given by the right side of the equation.

$$H_0: \beta_1 = \beta_2$$
$$H_1: \beta_1 \neq \beta_2$$

Chow statistic = 1971.154
Critical F statistic = 4.6052 at the 99% confidence level.
1971.154 > 4.06
The Chow statistic is greater than the critical F statistic, thus we can reject the null hypothesis that these populations subsets come from the same population. Lending credence to idea that a change in testing regime occurred between 2005 and 2006.

[26] Recall, an additional 626 encounters with a mis-specified address have been removed, reducing the subsample from 10086 to 9460.

are fairly similar, however the mean and standard deviation of Age in the NHCS subsample are significantly higher than those in the complete KCMO Health dataset.

Table 6.11: Comparison of KCMO Health and NHCS Samples

	KCHD Pb	NHCS Pb
n ()	171,826	9,460
Mean Test Result	2.464	3.055
sd Test Result	2.357	3.384
%Male	51.39%	51.07%
% Capillary	35.14%	30.54%
% Venous	58.43%	61.67%
Mean Age	30.338	41.159
sd Age	18.151	30.694

source: KCMO Health Department

Reviewing issues with the KCMO Health blood lead data: Lack of complete information about the testing equipment means we cannot adjust low level test results to compensate for limit of detection issues. There is no length of occupancy information requiring an assumption to facilitate matching to the NHCS. There is no demographic data about the test subjects, no economic data about the test subjects, and no employment information about the household limiting the ability to make use of those important observations. Every child does not receive a blood lead test, it appears that children living in minority and less affluent neighborhoods are more likely to receive a blood lead test, all of which implies that a child is more likely to get tested if they are likely to have a higher blood lead level. There is an inadequately explained discontinuity in the test results. Finally, the geocoding process is a selection process that has observable effects on the statistical characteristics of the sub-sample population. These are important issues. They cannot be glossed over, even if their effect on the statistical analysis is ambiguous. It is likely that these shortcomings alone or in combination

has some effect on the properties of the NHCS associated sub-sample. The total effect of these issues is not terminal so long as we are circumspect and judicious when drawing conclusions from statistical analysis drawing on the data.

Additional Explanatory Data

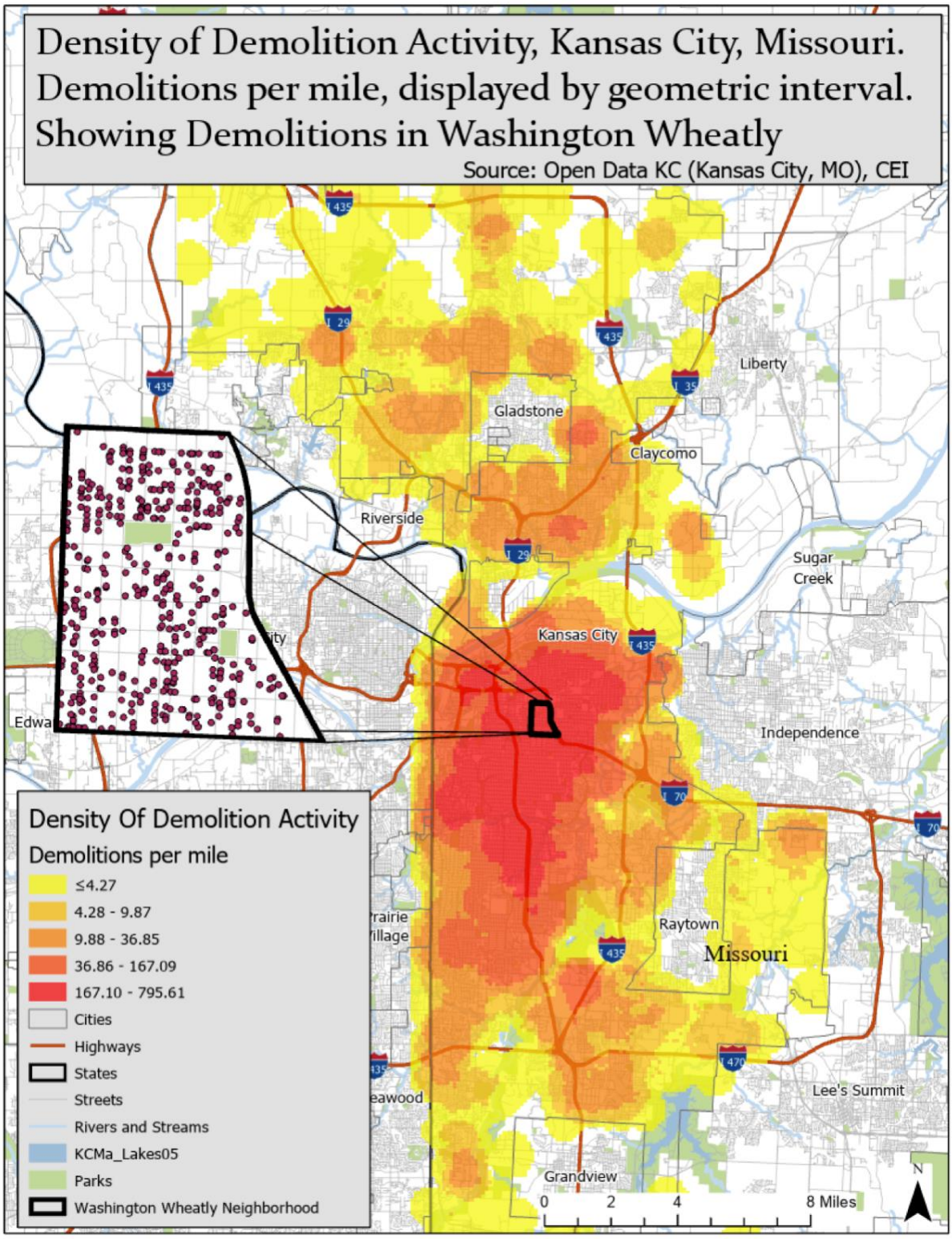
The built environment relevant to lead poisoning encompasses more than exterior housing conditions. The condition of a home's exterior paint becomes more indicative of BPb conditions if the paint on the home is likely to contain a high level of lead. It follows that the era of lead paint in which the home was built is important and that data has been gathered. As discussed in chapter 5 the soil-lead level is important for understanding BPb and so data associated with processes of depositing lead (demolition activity, location of gas stations from the era of lead poisoning, proximity to major roads and highways) are included in this analysis. It is important to note explicitly that some aspects of the built environment related to lead poisoning are not included in this analysis. The possibility of exposure to lead in the water supply is important (Hanna-Attisha et al. 2016b; Pulido 2016) but not controlled for directly[27] in my study. To the degree that Pb exposure from water shows up in the statistical analysis it may do so as an aspect the era of housing variable (older, corroded fixtures may be a source of lead exposure) and/or the community district variables (any localized issue with the water infrastructure will be present throughout observations within that geography). Airborne lead, though a much-reduced danger following the cessation of leaded gasoline is still generated as pollution through points of industrial activity (Moody et al. 2016) and in the exhaust from

[27] The KCMO Water Department releases one annual average vales for lead in the water flowing through the 2,800+ miles of water mains. The figure has not been incorporated into my analysis. A single figure can be useful if there is a catastrophic system wide failure as was the case in Flynt MI. There is nothing in the figure released by the Kansas City, MO, Water Department to indicate such a failure has taken place.("News – KC Water" n.d.).

some small engine planes (Miranda, Anthropolous, and Hastings 2011; Zahran et al. 2017). Community district and demolition data both may indirectly account for some of the airborne lead exposure but, as in the case with lead in water, they represent areas for future investigation and extension of the dataset and associated analysis.

In Kansas City and other rust belt cities housing demolitions reflect the geographies of race and class. Housing demolitions explicitly reshape the urban environment, yet the importance of that activity in reshaping the urban landscape is underrecognized. When demolitions are brought up in the peer-reviewed literature it is generally with regard to 'problem properties', buildings that are understood as a nuisance to the surrounding community. In that context housing demolitions are understood as a solution to a problem. But as we know there are no permanent solutions, only waystations on a road that does not end. Demolitions should be considered as important events in the life of a neighborhood and recognized for the environment which they create. In the larger context of a neighborhood demolitions are moments of transition where housing is erased from the landscape. In the context of this dissertation housing demolitions are considered as another active player in the construction of built environment. Rather than creating structures, vacant lots are created. In the vicinity where a home once stood a cloud is a temporary dust cloud. The character of the demolition dust is important. To the degree that lead was used in the construction of the home lead will be present in the dust cloud and the vacant lot where the home once stood. Map 6.7 illustrates where demolition activity that occurred in Kansas City, Missouri between 1990 and 2015. Comparing this map with the racial and class geographies illustrated in Map 6.4 shows that demolition activity is more pronounced in the Black and in the relatively poorer sections of the city. Recently a literature has emerged associating demolition activity with higher levels

Map 6.7: Density of Structure Demolitions



Map created by author.

of lead in the blood of children (Yin and Silverman 2015; DHD 2017).[28]

Engaging with and adding to these findings requires data. Records of demolition activity were provided by the City of Kansas City, MO, Kansas City, KS, and Gladstone, MO. Demolition records were not provided by the cities of Independence, Sugar Creek, North Kansas City or Raytown. Data availability thus narrowed the statistical analysis in this dissertation to those geographies cities for which data was provided. The data was provided in excel format as address level data. The data reported varies by municipality and over time. The precise date the demolition activity occurred was not always provided with the demolition records, however, the date the demolition permit was issued is always reported. It is this date that is used as a reference date for the demolition activity. The average number of demolitions per year in Kansas City Missouri (1990-2013) is 436, but this number hides significant variation. During the 1990's the average number of demolitions in Kansas City was 535, with the highest number of demolitions, 853, taking place in 1996. During the 2000's the average number of demolitions fell to 366 per year, bottoming out at 235 in 2005.

The assembled demolition records went through a version of the iterative geocoding process described in this chapter to associate the event with the parcel geography. Matching demolitions to the relevant parcel[29] in some cases required the manual placement where former parcels have been split or combined into new parcels. After the completion of the geocoding operation the demolition events are given a 100m buffer in GIS, in keeping with the literature reviewed in chapter 5. Demolition events are associated with every BPb encounter that falls

[28] A more extensive discussion of this literature is in chapter 5.

[29] In this case the target geography was all the parcels in Kansas City Missouri rather than the NHCS. Thus there is no shifting geography between years and the geocoding process is more streamlined than that developed for this dissertation.

within the 100m buffer. The associated records are then filtered to keep only those demolitions that fall between the birth of the child and the date of the test. This process created two demolition focused data fields, an indicator variable (1 indicating demolition(s) took place w/in 100m of the home during the child's lifetime) and a count variable (counting the number of demolitions taking place within 100m of the child's home during their lifetime). Only the indicator variable is used in my analysis.

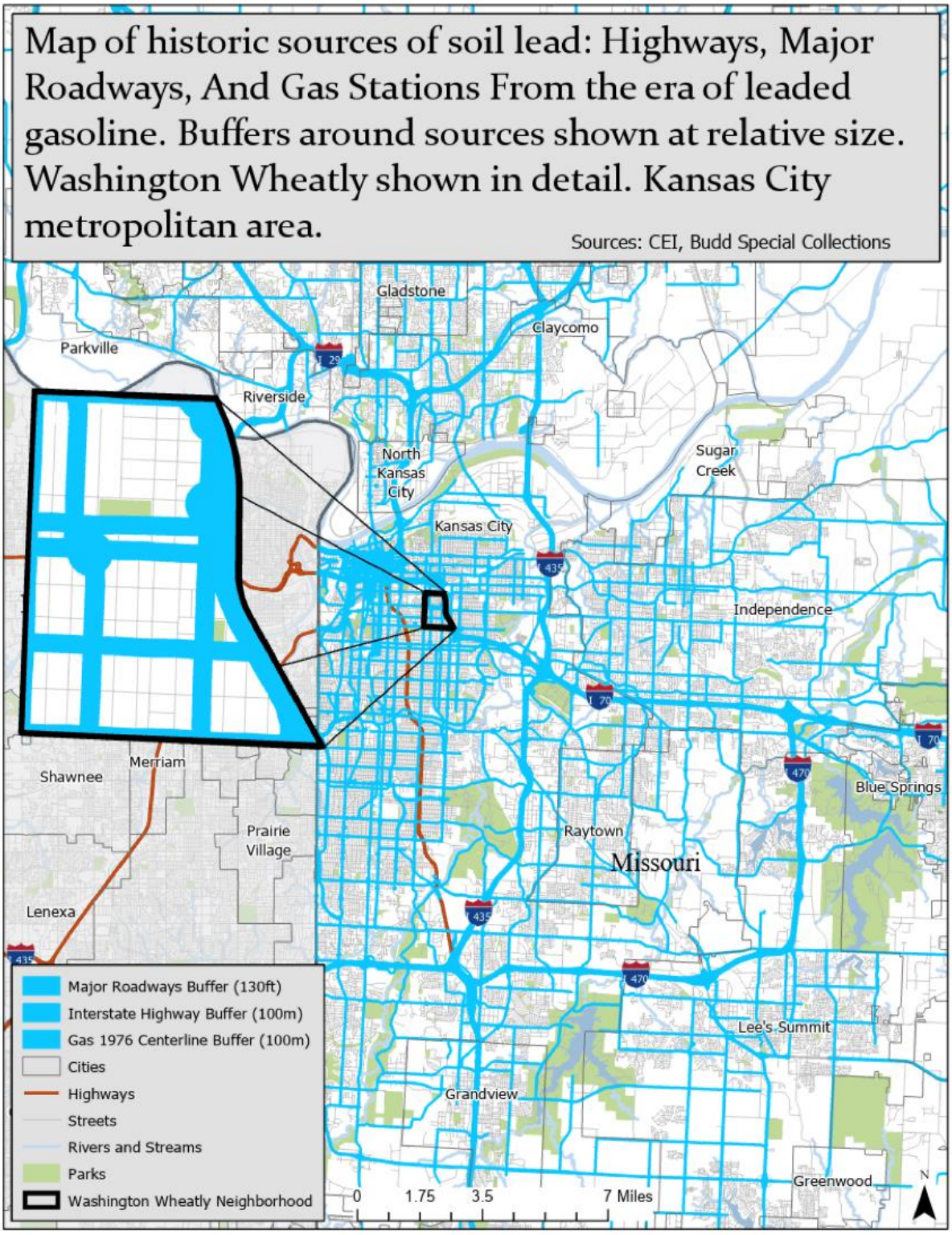
Though tetraethyl leaded gasoline is no longer being used in automobiles it is possible to observe its legacy in the soil where the emitted particles settled. The areas around highways and major roads that were active during the era of leaded gasoline receive the most attention but less apparent and possibly as important is the soil bourn legacy of the gas stations which suppled and maintained the cars and trucks. The CEI maintains separate GIS shapefiles of major roads and highways. These shapefiles were put into use through a GIS based buffering operation where a distance of 100m was added to each side of the highways system and 100 feet was added to each side of the major road system (for more information about the reasons for this buffering decision see chapter 5). Developing a database of legacy gas stations—those that were in operation during the era of leaded gasoline and may have contributed to the creation of lead loaded soil—is more complicated than the buffering of established shapefiles. The contemporary footprint of the legacy stations varies. Frequently there is no longer evidence that a gas station was ever present at a location. Archival resources must be relied on, even for the recent past. I made the simplifying assumption to focus on stations from the year 1976. That year is close enough to the high point of the use of leaded gasoline and to the end of the TEL era to be a suitable stand in for the era as a whole. Consulting the 1976 R. L. Polk business directory (*Kansas City (Jackson County, Mo.) City Directory, Including Gladstone*

and North Kansas City 1976) which lists the street address of every gas station in the city at the time for raw location data which was then transcribed into an excel database. There were 320 gas stations in Kansas City at the time. The assembled data are then geocoded using the same process as described with the demolitions data. As with the demolitions data a 100m buffer is added to capture the spatial extent of likely lead soil contamination. Map 6.8 shows the geography of likely lead soil contamination associated with these geography variables. In the assembly process an indicator variable is added to the BPb observation is the associated home falls within the likely lead soil buffer.

No magnitude or count variable was developed to account for homes fall within multiple buffers (i.e. no distinction is made between a home that is within 100m of a highway and 100m of a highway and 130ft of a major road, and a home that is within the relevant proximity to only one of these factors).

The era in which housing is built is an effective predictor of lead exposure due to the difference in the use of lead in paint and the use of that paint itself. Chapter three has gone into that history in some detail and chapter four has discussed some of the associated epidemiological literature. There are two principal methods for gaining access to era of housing records; the American Community Survey (ACS) estimates the average age of housing on the basis of occupant-reported recollections of age of housing; County property records are the other method for tracking the year in which the principal structure on a parcel was built. Either of these sources can be used to determine which era of lead paint the house was built. As noted in chapters three and four there are two critical dates dividing the eras–1952, when the paint industry in the US voluntarily stopped selling lead paint for use on the inside of buildings, and

Map 6.8: Geography Variables—Distance from Gas Stations, Major Roads and Highways



Map created by author.

1978 when the use of lead in exterior house paint was regulated to nearly zero. This era of housing periodization is forgiving of most discrepancies between the ACS and the county level records, however, when considering homes on the margin precision pays a premium. As a result, the county level records provided by MARC are used to determine the era of housing in this study with ACS data playing a subsidiary role.

The MARC age of housing record contains some missing values. The MARC data does not include any observations for two of the core counties in the Kansas City metropolitan area, Johnson County in Kansas and Platte county in Missouri. Missing values in Kansas are of no account to my Missouri focused dissertation. The missing values in Platte County have implications for the 24 NHCS Pb health encounters in this county (they are all from 2011). There is a more general issue with missing values among the data provided by MARC. Of the 499,108 parcels in the data 82,771 are listed as "0" and another 45,763 are listed as <NULL>, fully 25% of the year of construction data is missing. As with the absent Johnson County data, not all of these missing values are relevant to my analysis. Among the 9640 parcel level observations of blood lead levels 2189 were missing age of housing observations.

After investigating the parcels with missing data using online resources (google maps, Kansas City open records) three types of structure appear associated with a missing record: demolished buildings, commercial or industrially zoned buildings, and multi-family housing. Demolition activity, or lack of structure on the parcel, is logically associated with a <NULL> or ‘0’ value. However, if a child is identified as living at that address before a demolition took place the age of the parcel is necessary to ascertain. Buildings zoned for commercial or industrial use frequently are missing the age of structure attribution. Zoning does prevent clandestine residency in commercial buildings, and it is necessary to estimate age of

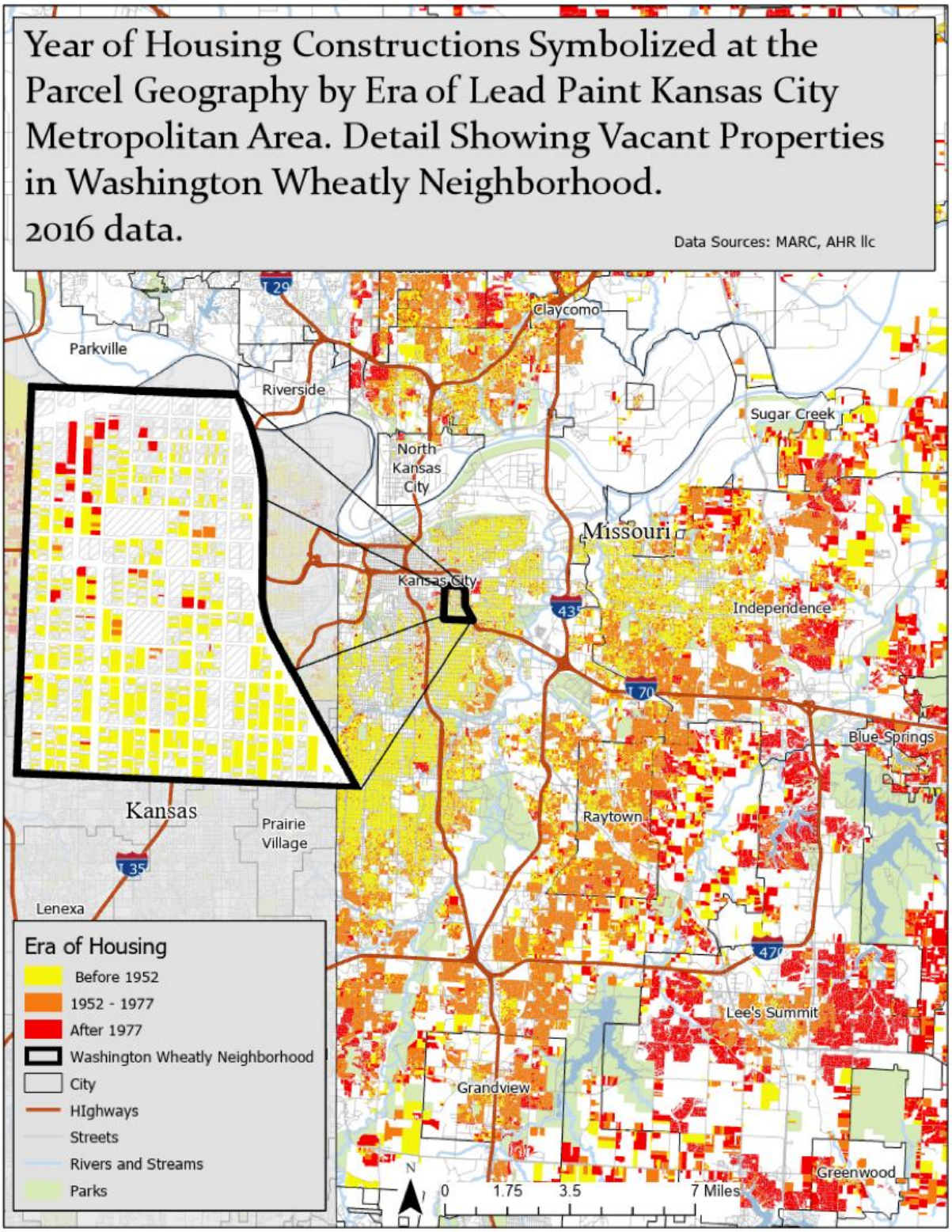
construction for many of these structures as well. The largest group of buildings lacking an associated date of construction are those intended for multi-family housing. The missing values range across all types of multi-family housing. The six-unit apartment buildings found throughout midtown and north east Kansas City, the high-rise apartment buildings on Armor Boulevard, the public housing complexes found along Brush Creek and in Pendleton Heights are all among the variety of multi-family housing without age of housing. It is not clear why there is a preponderance of missing data among this type of housing.

Resolving the problem of missing values requires a multi-part solution. The first step towards resolution involved consulting an expert on Kansas City housing. The architectural historian Cydney Millstein, at Architectural and Historical Research, LLC (AHR), provided age of construction for the homes of 646 health encounters and renovation dates for 428 of these encounters. The date of renovation for these multi-family homes is crucial.[30] If the renovations took place after the era of lead paint, as they did in every home in this sample, I can reasonably assume that the renovation activity removed the offending material and thus for considerations of lead exposure the relevant date for these homes is no longer the date of construction. After consulting AHR, 1543 observations of date of construction are still missing, (16.3% of the sample). To resolve these missing data an estimation procedure is developed which utilizes the growth pattern of the city which, illustrated in Map 6.9.[31]

[30] The importance of renovation activity as a source of possible contamination and as a reason to consider renovated homes as distinct from homes with the same original date of construction is discussed in the epidemiology literature (C. S. Clark et al. 1985; Bailey, Sargent, and Blake 1998; Mielke, Laidlaw, and Gonzales 2010).

[31] Multiple imputation, though it is not used here, is the standard method for estimating missing values in a data set. Methods based on the simple attribution of a mean value to missing data tends to underestimate the variation in the model. Multiple imputation is designed to reproduce the variance/covariance matrix that would be observed had the data not had any missing information (Schafer 1999). Additional investigation is necessary to quantify the difference between the multiple imputation method and the one used here.

Map 6.9: Year of Housing Construction



Map created by author.

The multi-stage estimation process requires several pre-analytical steps. First is the association of a unique id number with each of the several high-level geographies to be estimated with the parcel level KCHD lead observations. The geographies are census block, census block group, census tract, and neighborhood. This association is done using the ESRI ArcGIS pro software. The second pre-analytical step is the combination of the age of housing field supplied by MARC, with the updated age of housing and renovation information supplied by AHR. This merge is done in the R statistical computing language. Next calculate the mean age of housing value based on this combined age of housing field for each census block, census block group, census tract, and neighborhood in the survey area. Then tally the percent of missing observation for each of these geographies. Now the age of housing attribution algorithm can be applied. Beginning at the census block level apply the mean age of housing value from that geography to the missing age of housing value if more than 50% of the parcels in the individual census blocks have an identified value. If less than 50% of the parcels in the block geography have an identified age of housing value move on to the higher census block group value and apply the mean age of housing value from the block group geography to the missing parcel level value. If less than 50% of the parcels in the census block group geography have an identified age of housing value move on to the higher census track values. And so forth following this pattern through the neighborhood level. A final attribution of age of housing values is made to the parcel level encounters located in geographies that never achieve the 50% threshold of recorded values. These parcels receive an age of housing value from the

2016 ACS age of housing block group level estimates. Table 6.12 summarizes the Year of Construction variable by data source and the results of this estimation process.

Table 6.12: Year of Construction and Estimated Year of Construction

Year of Construction by Source						
	MARC	Research	Rehab	Total	Estimated Age	Total w/ Estimated Age
n	7271	218	428	7917	1543	9460
mean	1925.31	1966.665	1995.91	1930.267	1934.719	1930.993
sd	24.115	9.247	4.259	28.800	22.206	28.295
n ≤ 1951	5893	51	0	5944	1016	6960
1952 - 1977	1125	167	0	1292	480	1772
n ≥ 1978	253	0	428	681	47	728
Year of Construction Estimates by Geography						
	Block	Block Group	Tract	Neighborhood	ACS Estimate	Estimate Total
n	950	319	30	32	212	1543
mean	1928.87	1933.853	1931.93	1931.010	1963.184	1934.719
sd	23.752	23.705	18.798	17.696	14.181	22.206
n ≤ 1951	708	224	24	25	35	1016
1952 - 1977	227	88	6	7	152	480
n ≥ 1978	15	7	0	0	25	47

Data Sources: MARC, Architectural and Historical research, LLC. Table generated by author in estimation process.

Assembly Process, Filtration, New Variable Construction

The assembly of the data is as important as its collection. A poor assembly process can compromise the best collection of data. Appendix F provides a diagram of the assembly process. It describes the combination of shape files, the transformation of the shape files into .csv spreadsheets, the consolidation of multiple .csv into a single file, and the generation of new fields from the existing data discussed already. The assembly process begins with three separate shape files: the NHCS parcel file, the MARC date of housing construction file, and the KCMO Health blood lead observations geocoded to the parcel level (hereafter KCHD Pb).

These files are already connected in a preliminary way. The KCHD Pb file has a unique id field from the geocoding process linking encounters with NHCS parcels and the MARC file has a unique id field created from a spatial join associating it with the KCHD Pb file. There is no direct link between the MARC file and the NHCS file due to a discrepancy in the parcel boundaries.[32]

Each of the three assembly shape files are the subject of spatial joins. The NHCS file is joined with the three lead-soil geographic proximity buffers (highways, major roads, gas stations) to create indicator variables. The NHCS file is also joined with the geography layers for community districts, neighborhoods, census blocks, census block groups, and census tracts. The KCHD Pb layer receives a one-to-many spatial join from the buffered demolition layer. This allows each demolition record to be joined where appropriate with multiple health records. The MARC shapefile is joined with the year of housing construction file generated in consultation with AHR. When these joins are complete the shape files are exported as .csv files and imported into the R studio computing environment for further analysis. The KCHD_Pb.csv(1) file has multiple rows for each encounter due to the one to many join process. These multiple rows are filtered and consolidated in R to produce a file (KCHD_Pb.csv(2)) of unique health encounters with indicator and count variables for demolition activities as discussed in the section above. The NHCS .csv file is joined by unique id generating the file KCHD_Pb.csv(3). Meanwhile the missing date of construction values of MARC.csv file are estimated. When this estimation is complete the MARC file is joined to KCHD_Pb.csv(3) by unique id.

[32] This discrepancy is due to the activities of the NHCS survey process. One of the objectives of the survey is to develop a parcel file informed by direct observation of the city. An updated parcel map will by definition not be aligned with the official, inherited parcel boundary layer.

The fourth iteration of the KCHD_Pb.csv file is now filtered by several criteria to create a .csv without missing values for any of the critical files. Table 6.13 lists the filtering criteria and the number of encounters excluded with each criterion. The geocoding process is the first filtering process. The first filter applied in R is the removal of encounters associated with the anomalous single-family street address mentioned above. The next filter is by age of child, limiting the data to only those children between 6 and 72 months of age.[33] Filtering by age reduces some of the bias in the sample (children outside of that age range are only tested if they are suspected of having elevated BPb) and aligns the analysis with the epidemiological focus on children. The encounters associated with NHCS window and doors, porch, or exterior paint conditions rated '6' or 'not applicable' by the survey team are all filtered out. Then the encounters are filtered by city of residence due to the lack of complete demolition data for any municipality other than Kansas City MO. The last filter is to remove those observations without

Table 6.13: Filtering Incomplete Health Records

	Before Removal	After Removal	Total Removed	% removed
Geocoding	201,591	10,086	191,505	95.00%
Drury	10,086	9,460	626	6.21%
Age	9,460	8,566	894	9.45%
NHCS	8,566	8,315	251	2.93%
windoors	8,566	8,468	98	1.14%
porch	8,468	8,322	146	1.72%
exterior paint	8,322	8,315	7	0.08%
Kansas City	8,315	8,103	212	2.55%
Unknown Sex	8,103	8,077	26	0.32%

Source: Generated by author in assembly process.

[33] Reasons for choosing this age range are discussed in chapter 5.

a value for sex of child.[34] Thus, these observations have no easy explanation for their lack of definitive observation. At the conclusion of this filtration process there are 7944 encounters assembled for statistical analysis.

One step remains before statistical analysis, existing fields are reconstructed into a form suitable for statistical analysis. An indicator variable is created from the date the child was tested. If the BPb test took place during the winter (December - April) the value is 0 otherwise the value is 1. Three era of housing indicator variables are constructed from the date of housing construction variables. The era of housing corresponding to the three eras of lead paint usage (pre-1952, 1952 to 1977, post-1977). The NHCS variables are converted from a 5-point scale to three indicator variables. Housing conditions that previously scored a 1 or 2 ("severely deteriorated" or "seriously deteriorated") are represented as a 1 in the severely deteriorated (_det) variable, all other ratings receive a 0. Housing conditions that previously scored a 3 ("substandard") are represented with a 1 in the substandard (_sub) variable, all other ratings receive a 0. Housing conditions that were scored 4 or 5 ("good" or "excellent") receive a 1 in the good (_pass) variable, all other ratings receive a 0. Year of test is modeled as a square function, as discussed in chapter 4 (yr, yr^2). As discussed in chapter 4 the relationship between age of child and blood lead are observed to be related in a cubic function and so they are modeled as such here (months, months^2, months^3). Appendix G is a complete data dictionary for the fields consolidated and generated in the assembly process. The majority of these fields are not used in the statistical analysis an additional sub-setting of fields is implemented to

[34] The 26 observations (.32% of the total) have no value for sex of child, lower than the number of children expected to be intersex (1.7%).

create a file with only the necessary fields. The data dictionary for that file is found in table 6.14.

Table 6.14: Data Dictionary for Statistical Analysis

Field Name	Data Type	Description
TEST_RESUL	continuous	Measured BPb level, test result in micrograms per deciliter (µg/dL)
male	dichotomous	Sex of child, 1 – male, 0 – all others
female	dichotomous	Sex of child, 1 – female, 0 – all others
unk_sex	dichotomous	Sex of child, 1 – unknown, 0 – all others
capillary	dichotomous	Blood test type, 1– capillary exam (finger poke), 0 – all others
unk_sample	dichotomous	Blood test type, 1– unknown test type, 0 – known test type
venous	dichotomous	Blood test type, 1– venous blood draw, 0 – all others
months	continuous	Age of child in months (9-72)
wd_pass	dichotomous	Exterior condition of windows and doors, 1 – 'good' or 'excellent', 0 – all others
wd_sub	dichotomous	Exterior condition of windows and doors, 1 – 'substandard', 0 – all others
wd_det	dichotomous	Exterior condition of windows and doors, 1 – 'Severely deteriorated' or 'Seriously deteriorated', 0 – all others
porch_pass	dichotomous	Exterior condition of porch, 1 – 'good' or 'excellent', 0 – all others
porch_sub	dichotomous	Exterior condition of porch, 1 – 'substandard', 0 – all others
porch_det	dichotomous	Exterior condition of porch, 1 – 'Severely deteriorated' or 'Seriously deteriorated', 0 – all others
ep_pass	dichotomous	Exterior condition of exterior paint, 1 – 'good' or 'excellent', 0 – all others
ep_sub	dichotomous	Exterior condition of exterior paint, 1 – 'substandard', 0 – all others
ep_det	dichotomous	Exterior condition of exterior paint, 1 – 'Severely deteriorated' or 'Seriously deteriorated', 0 – all others
demo	dichotomous	Observed Demolitions, 1 - any demolition activity w/in 100m of home during lifetime of the child, 0 – no demolitions w/in 100m of home during lifetime of child

Table 6.14 (continued)

Field Name	Data Type	Description
in_prox	dichotomous	Soil Proxy, 1– Home w/in 100m of highway and/or 100m of old gas station, and/or 130ft of major roadway, 0 - home not in proximity of selected geography variables
not_winter	dichotomous	Season of test, 1 - BPb test took place between April and November, 0 - BPb test too place December to March
Y	continuous	Year of BPb test, 2000-2013
ERA	multinomial	Era of housing construction, 1 – pre-1952, 2 – 1952-1977, 3 – 1978 or later
CD_NAME	text	Community District, name of community district of residence, n = 24
nbr_Name	text	Neighborhood, Name of neighborhood of residence, n = 182
JURISDICTI	text	Jurisdiction of address: Health Department
test_yr	continuous	Year of BPb test, 2000 = 1, 2001 = 2, …, 2013=14

Source: Generated by author in data assembly process.

CHAPTER 7

STATISTICAL ANALYSIS

This dissertation is organized around the research question, ‘Do external measurements of parcel level housing conditions meaningfully add to our understanding of blood lead levels (BPb) in children’. That specific question is nested within a larger context. Blood lead levels are a subset of the field of health outcomes. Chapter 3 is focused on discussing the biological reasons pediatric lead exposure is toxic and the ways lead poisoning can manifest as latent sequelae. Parcel level housing conditions are a subset of the larger field of observations of the built environment, and externally measured housing conditions are a subset of parcel level housing conditions. Chapter 4 provides a history of the use and eventual regulation of lead in economic production. It is disingenuous to talk about housing conditions without also talking about who lives in the lead loaded homes being studied: predominantly Black, Latinx, immigrant, and poor families. Following the work in chapter 2, which places lead poisoning into a social surplus framework (understanding lead poisoning as the product of previous production decisions with socially mediated distributions), the discussion of biological effects in chapter 3 and the social history of lead in chapter 4, pediatric lead poisoning can be thought of as a spatially determined disinvestment activity which operates through the built environment. The issue then becomes how to effectively stop this disinvestment process.

Chapters 2-4 provide context for this dissertation's focus on making use of externally measured observations of parcel level housing conditions to address lead poisoning, relating the spatially involute built environment and health outcomes. Chapter 5 discusses the ways epidemiologists and other scholars have observed and modeled the spatial relationships of lead

poisoning using geospatial and statistical methods. The experience of race and class in the US is intimately tied to a lead poisoned geography, this is particularly true in hyper-segregated Kansas City, Missouri. The geographies in which Black, Latinx, immigrant, and poor families live are saturated with lead hazards in a way that the white and affluent areas of the city are not (Pulido 2000; McClintock 2015; Moody et al. 2016).

Chapter 6 discusses the data gathered for this dissertation, its characteristics as well as its shortcomings. Chapter 6 also discusses the first two contributions this dissertation makes to the joint stock of knowledge: a geocoding technique for address matching to a shifting geography and an assembly of previously isolated health related geospatial data. Together these contributions can lead to improvements in the understanding of the built environment's relationship with pediatric lead poisoning. The most critical shortcoming of the assembled data is the absence of detailed individual level demographic observations[1]. The collection of data about the built environment in the vicinity of the home compliments information about the home itself.

An answer to the crucial "why do this research" question is straightforward: to lower blood lead levels in kids. The intention of this peculiar act of looking is to see areas in which action can be taken. Aspects of chapter 2 place this dissertation in an expanded research agenda; discussing the importance of more accurate predictions of blood lead levels in terms of policy development, effective use of limited lead intervention resources, and how this environmental justice issue fits with the ethical imperative of democratic equality. This penultimate chapter develops a statistical model to begin answering that call. The final chapter

[1] In addition to demographic information consumption behavior, related to lead loaded products such as objects painted with lead pigments and cosmetics that contain lead, is needed.

briefly considers policy implications brought on by this statistical chapter and the research agenda upon which it is built.

Using a cross section of observational data, I estimate an ordinary least squares multiple regression model that relates child, housing, and geographic proximity focused explanatory variables to a dependent variable (a child's tested blood lead level). To understand more completely the relationships between the variables in the model, I estimate six versions of this model, restricting sets of explanatory variables, observing changes to regression coefficients and comparing the explanatory power of the various models via an F test of joint hypotheses. Finally, I discuss the results of the regression analysis in the context of expected coefficient values and the implications of the estimated values.

Model Specification

The omnibus equation 7.1 was introduced at the outset of chapter six before the discussion of data acquisition, geocoding, assembly, and subsetting procedures. It is important to note that I estimated and presented a different version of this model using the same data set for the EPA Region 7 Pediatric Lead Poisoning Prevention Summit in September 2019.[2] The model specification and regression results from that presentation are included in appendix H.[3] Equation 7.1 is designed to highlight the three groups of explanatory variables [child-focused

[2] There are many differences between the model in appendix H and the one discussed below are the non-linear modeling of year of test and age of child in months, the interaction of Era of Construction and Condition of Exterior Paint, the scaling of housing conditions on a five point scale rather than a three point scale, the exclusion of the roof and foundation and walls housing condition, the combination of 3 proximity variable into a single variable, the inclusion of a seasonality variable and the inclusion of indicator variables for community district.

[3] In CEI publication WP-1801-03 I report the regression estimates for a model relating pediatric lead poisoning and observations of the built environment in Kansas City Missouri (N. Wilson 2018a). The model and sample are substantially different from those presented here. Regression results will be supplied upon request to interested parties.

(C), housing (H), and geographic (G)] that I discuss in chapter 5 and below in chapter 7. Such groupings aid in narrative construction but are not necessary for mathematical clarity.

$$BPb_i = \alpha + \sum_{j=1}^{8} \beta_j C_{ji} + \sum_{j=9}^{20} \beta_j H_{ji} + \sum_{j=21}^{25} \beta_j G_{ji} + \epsilon_i \tag{7.1}$$

To introduce the model sufficiently it is necessary to discuss each term of the completely specified model. Specifying each explanatory variable begets equation 7.2.

$$\begin{aligned} BPb_i = {} & \beta_0 + \beta_1 \text{test\_yr}_i + \beta_2 test\_yr_i^2 + \beta_3 \text{months}_i + \beta_4 months_i^2 \\ & + \beta_5 months_i^3 + \beta_6 \text{male}_i + \beta_7 \text{venous}_i + \beta_8 \text{unk\_sample}_i \\ & + \beta_9 \text{ERA1}_i + \beta_{10} \text{ERA2}_i + \beta_{11} \text{wd\_sub}_i + \beta_{12} \text{wd\_det}_i \\ & + \beta_{13} \text{porch\_sub}_i + \beta_{14} \text{porch\_det}_i + \beta_{15} \text{ep\_sub}_i + \beta_{16} \text{ep\_det}_i \\ & + \beta_{17} \text{ERA1}_i * \text{ep\_sub}_i + \beta_{18} \text{ERA2}_i * \text{ep\_sub}_i \\ & + \beta_{19} \text{ERA1}_i * \text{ep\_det}_i + \beta_{20} \text{ERA2}_i * \text{ep\_det}_i + \beta_{21} \text{demo}_i \\ & + \beta_{22} \text{in\_prox}_i + \beta_{23} \text{not\_winter}_i + \beta_{24} \text{'East Side'}_i \\ & + \beta_{25} \text{Midtown}_i + \varepsilon_i \end{aligned} \tag{7.2}$$

Where a succinct description of each of the variables is as follows. A list of summary statistics is found in table 7.1 which follows the listing of variables.

BPb is the dependent variable: blood lead levels. It is a continuous variable measured in micrograms per deciliter that tells us how much lead is observed in the child's blood. The sample mean of the dependent variable is 3.08µg/dL (standard deviation 3.44). The distribution of blood lead levels is skewed to the right (maximum value is 63µg/dL) so it may be more

useful to note the 25th quantile of the distribution is 1µg/dL and the 75th quantile of the distribution is 4µg/dL.

Child Focused Variables

test_yr is the year of the blood lead test. It is a continuous variable where 2000=1 (the first year of BPb observations) through 2013=14 (the last year used in my analysis). The values are provided in the data from KCMO Health. The mean year of test was 7.17 (2006) with a standard deviation of 4.03. There are an unequal number of tests per year, the median year of a health observation was 9 (2008) and the modal year of 10 (2009).

months is the age of the child in months at the time of their test. The values are continuous and were provided by KCMO Health. The range has been limited to 6-72 months in accordance with discussion in chapters 4 and 5. The relationship between the age of the child and her observed blood lead level is specified as a cubic form that is discussed in the epidemiology literature and can be seen in the KCMO Health data. Biological and behavioral patterns explain the regularity of this relationship and to reflect this regularity the age of child variable is modeled through a cubic specification of three related variables. The mean age of a child in the study is 34.2 months (standard deviation 18), the median age is 33 months, and the modal age is 12 months.

male records the sex of the child. This dichotomous variable indicates the sex of the child where 1= male, 0 = any other sex. The data was provided by the KCMO Health. This variable is expected to indicate that male children are associated with slightly higher blood lead levels. 51.33% of the sample is identified as male.

venous is the type of blood lead test. This dichotomous variable indicates the blood test type where 1 = venous blood draw, 0 = any other sample type. The data was provided by KCMO Health. This variable is expected to indicate that venous samples are associated with higher blood lead levels. At least 62% of the sample were tested via a venous blood draw.

unk_sample is the type of blood lead test. This dichotomous variable indicates the blood test type where 1 = sample of unknown type, 0 = any other sample type. The data was provided by KCMO Health. This variable should tell us if there is a pattern between unknown sample type and the observed level of blood lead. 7.08% (572 observations) of the sample are of unknown blood test type.

Housing Focused Variables[4]

ERA1 indicates when the home of the child was built. ERA1 is a dichotomous variable where 1 = home built before 1952, 0 = home built in 1952 or later. Initial data was provided by the Mid-America Regional Council (MARC), missing values were computed according to a process described in chapter 5. Year of home construction is modeled as three dichotomous variables in keeping with changes in the usage of lead paint. The oldest homes are expected to be related to higher blood lead levels. 73.6% of the sample was built during the first era of home construction.

ERA2 indicates when the home of the child was built. ERA2 is a dichotomous variable where 1 = home was built between 1951 and 1978, 0 = home was built before or after this

[4] Not included among these variables are two of the NHCS housing conditions: roof, foundations and walls. Including these variables resulted in significant loss of information due to a preponderance of missing observations (see table 6.4 and associated discussion). Missing observations is a fundamental problem of the NHCS and other surveys of the type. Regression results for model specification including the omitted housing conditions are available upon request.

temporal range. Initial data was provided by MARC, missing values were computed according to a process described in chapter 5. Year of home construction is modeled as three dichotomous variables in keeping with changes in the usage of lead paint. The mid-Era homes are expected to be related to slightly higher blood lead levels than the newest homes, lower than the oldest homes. 18.4% of the sample (1485 observations) was built during the second era of home construction.

wd_sub[5] indicates observed condition of windows and doors. It is a dichotomous variable describing condition of the exterior windows and doors where 1 = substandard condition and 0 = any other condition. The variable is derived from CEI's Neighborhood Housing Conditions Survey (NHCS) data. Substandard conditions score a 3 in the NHCS ordinal ranking that runs 1-5. The wd_ variables are designed to show the decreasing quality of window and door conditions are associated with higher blood lead levels. 12.4% of the sample (1001 observations) exhibit this housing condition.

wd_det indicates observed condition of windows and doors. It is a dichotomous variable describing the exterior windows and doors where 1 = severely deteriorated or seriously deteriorated condition and 0 = any other condition. The variable is derived from CEI NHCS data. Severely deteriorated and seriously deteriorated are scored 1 and 2 in the NHCS ordinal ranking that runs 1-5. The wd_ variables are designed to show that decreasing quality of window and door conditions are associated with higher blood lead levels. 1.7% of the sample (138 observations) exhibit this housing condition.

[5] Equation 7.2 contains the assumption that there is no statistically significant difference between the NHCS rankings of 4&5 and similarly that rankings 1&2 can be combined without penalty. Small sample size motivates the second assumption. A test of the first assumption is available upon request.

porch_sub indicates observed condition of porch. It is a dichotomous variable describing the porch where 1 = substandard condition and 0 = any other condition. The variable is derived from CEI NHCS data. Substandard is a 3 on the NHCS ordinal ranking that runs 1-5. The porch_ variables are designed to show that decreasing quality of porch conditions are associated with higher blood lead levels.19.7% of the sample (1589 observations) exhibit this housing condition.

porch_det indicates observed condition of porch. It is a dichotomous variable describing the porch where 1 = severely deteriorated or seriously deteriorated condition and 0 = any other condition. The variable is derived from CEI NHCS data. Severely deteriorated and seriously deteriorated are scored 1 and 2 in the NHCS ordinal ranking that runs 1-5. The porch_ variables are designed to show that decreasing quality of porch conditions are associated with higher blood lead levels. 2.7% of the sample (223 observations) exhibit this housing condition.

ep_sub indicates observed condition of exterior paint. It is a dichotomous variable describing the exterior paint where 1 = substandard condition, 0 = any other condition. The variable is derived from CEI NHCS data. Substandard conditions score a 3 on the NHCS ordinal ranking that runs 1-5. The ep_ variables are designed to show that decreasing quality of exterior paint conditions are associated with higher blood lead levels. 26% of the sample (2101 observations) are associated with this housing condition.

ep_det indicates observed condition of exterior paint. It is a dichotomous variable describing the exterior paint where 1 = severely deteriorated or seriously deteriorated condition, 0 = any other condition. The variable is derived from CEI NHCS data. Severely deteriorated and seriously deteriorated are scored 1 and 2 in the NHCS ordinal ranking that runs 1-5. The ep_ variables are designed to show that decreasing quality of exterior paint

conditions are associated with higher blood lead levels. 5% of the sample (401 observations) are associated with this housing condition.

ERA1*ep_sub is the interacting ERA1 and ep_sub. This variable indicates if the child is living in a home from the earliest period of lead paint that has exterior paint in poor but not the worst condition. This interacted variable is designed to show the connection between blood lead levels and exterior paint in increasing states of disrepair is conditional on the era of construction with the expectation that paint in the worst condition from the earliest era of lead paint will be associated with the highest blood lead levels. 22.7% of the sample (1831 observations) are associated with this housing condition and era of construction.

ERA2*ep_sub is the interacting ERA2 and ep_sub. This variable indicates if the child is living in a home from the middle period of lead paint that has exterior paint in poor but not the worst condition. This interacted variable is designed to show the connection between blood lead levels and exterior paint in increasing states of disrepair is conditional on the era of construction with the expectation that paint in the worst condition from the earliest era of lead paint will be associated with the highest blood lead levels. 2.84% of the sample (229 observations) are associated with this housing condition and era of construction.

ERA1*ep_det is the interacting ERA1 and ep_det. This variable indicates if the child is living in a home from the earliest period of construction that has exterior paint in the worst condition. This interacted variable is designed to show the connection between blood lead levels and exterior paint in increasing states of disrepair is conditional on the era of construction with the expectation that paint in the worst condition from the earliest era of lead paint will be associated with the highest blood lead levels. 4.59% of the sample (371 observations) are associated with this housing condition and era of construction.

ERA2*ep_det is the interacting ERA2 and ep_det. This variable indicates if the child is living in a home from the middle period of construction that has exterior paint in the worst condition. This interacted variable is designed to show the connection between blood lead levels and exterior paint in increasing states of disrepair is conditional on the era of construction with the expectation that paint in the worst condition from the earliest era of lead paint will be associated with the highest blood lead levels. .36% of the sample (29 observations) are associated with this housing condition and era of construction.

Geographic Variables[6]

not_winter indicates the season in which the health encounter took place. This is a dichotomous variable where 1 = health encounter took place during the months April through November. 0 = health encounter took place during the months December through March. Variable derived from date of test data provided by KCMO Health. This variable indicates if the ground was likely to be unfrozen when the sample took place, thus the soil-lead content is available to be ingested by the child. A sample during the non-winter months is expected to be associated with higher blood lead levels, 58% of the sample (4740 observations) took place during the non-winter months.

Demo indicates if any structural demolition took place within 100m of the home of the child during the life of the child. This is a dichotomous variable where 1 = some demolition took place within 100m of the home address between birth and the BPb test date, 0 = no

[6] For this analysis two of the 24 assembled community district variables are included in the reported regression results and discussion. In the interest of clarity, the 22 Kansas City, MO community districts that did not achieve a meaningful level of statistical significance have been assumed into the intercept term. Regression results which specify each community district separately are available upon request.

demolition took place within 100m of the home address between birth and the BPb test date. Variable created through a spatial join and filtration of health data provided by KCMO Health and demolition data from KCMO open data. This variable is expected to indicate that living near building demolition activity increases blood lead levels. 66.4% of the sample (5363 observations) satisfy this condition.

in_prox indicates if the child's home is located in close proximity to known source of soil-lead. This is a dichotomous variable where 1 = home is within 100m of a highway and/or 100m of a gas station from the era of tetraethyl lead and/or 130ft of a major roadway. 0 = home in not within proximity of any of the relevant soil lead sources. These soil lead sources are selected and the distance from them designed with reference to peer reviewed literature. Variable constructed in GIS using CEI shapefiles, and archival data gleaned from UMKC special collections joined spatially with health data provided by KCMO Health. This variable is expected to indicate that living in proximity to likely sources of lead in soil is associated with higher blood lead levels. 28.7% of the sample (2314 observations) satisfies the identified criteria.

`East Side` indicates which community district the child's home is located in. This is a dichotomous variable where 1 = the child's home is located within the East Side Kansas City, Missouri community district and 0 = the child's home is not located within the selected community district. Variable is constructed in GIS using CEI shapefiles and geocoded KCMO Health Data. This variable is expected to indicate that living in this community district is associated with higher blood lead levels. 24.9% of the sample (2012 observations) are located within this community district.

Midtown indicates which community district the child's home is located in. This is a dichotomous variable where 1 = the child's home is located within the Midtown Kansas City, Missouri community district and 0 = the child's home is not located within the selected community district. Variable is constructed in GIS using CEI shapefiles and geocoded KCMO Health Data. This variable is expected to indicate that living in this community district is associated with higher blood lead levels. 2.4% of the sample (191 observations) are located within this community district.

$\boldsymbol{\varepsilon_i}$ is assumed to be a normally distributed error term with a mean of 0 and constant variance σ^2.

Table 7.1 lists summary statistics describing the variables in equation 7.2. The table also reports summary statistics for several variables which have been assumed into the intercept term (ERA3, e3_ep_sub, e3_ep_det, Other_ComDist) for reference when considering the implications of the regression analysis. With 21 dichotomous variables the intercept term describes the BPb level associated with the null case of all the listed dichotomous variables (a female child, who is tested via the capillary method, in the winter, lives in a home built during era three with windows and doors, porch, and exterior paint all in good condition, the child's home does not satisfy any of the parameters of the four interaction variables, is not located in proximity to any demolition activity nor any of the identified sources of soil-lead, nor is the child's home located in either of the two community districts which have independent explanatory variables, the year of the hypothetical intercept child's test is 1999). However, the intercept term contains little explanatory information on its own. The regression coefficients describe the average change in blood lead levels associated with a 1-unit change in the

Table 7.1: Summary Statistics

Variable Name	count	mean	std dev	min	max
BPb	8077	3.075379	3.438849	0	63
yr1	8077	7.1714	4.0293	1	14
months	8077	34.2340	18.3629	6	72
male	4147	0.5134	0.4999	-	-
venous	5035	0.6234	0.4846	-	-
unk_sample	572	0.0708	0.2565	-	-
ERA1	5946	0.7362	0.4407	-	-
ERA2	1486	0.1840	0.3875	-	-
ERA3	645	0.0799	0.2711	-	-
wd_sub	1001	0.1239	0.3295	-	-
wd_det	138	0.0171	0.1296	-	-
porch_sub	1589	0.1967	0.3976	-	-
porch_det	223	0.0276	0.1639	-	-
ep_sub	2101	0.2601	0.4387	-	-
ep_det	401	0.0496	0.2172	-	-
ERA1*ep_sub	1831	0.2267	0.4187	-	-
ERA2*ep_sub	229	0.0284	0.1660	-	-
ERA3*ep_sub	41	0.00508	0.0711	-	-
ERA1*ep_det	371	0.0459	0.2094	-	-
ERA2*ep_det	29	0.00359	0.0598	-	-
ERA3*ep_det	1	0.000124	0.0111	-	-
not_winter	4740	0.5869	0.4924	-	-
demo	5363	0.6640	0.4724	-	-
in_prox	2314	0.2865	0.4522	-	-
East Side	2012	0.2491	0.4325	-	-
Midtown	191	0.0236	0.1520	-	-
Other_ComDist	5874	0.7273	2.4506	-	-

explanatory dichotomous variables. Interpretation of the continuous nonlinear variables (year of test and age in months) is slightly more involved. The non-linear and interacted variable coefficients are not meaningful on their own and require multi-step processes to arrive at cogent interpretations of their relationship with BPb. The estimated coefficients and standard errors from equation 7.2 are presented in table 7.2.

Figure 7.2: Regression Estimates for Equation 7.2

	Equation 7.2	
Coefficient	Estimate	Std. Error
(Intercept)	2.848	0.338
Child Focused Variables		
test_yr	-0.701	0.0349
test_yr^2	0.0295	0.00250
months	0.1781	0.0298
months^2	-0.00472	0.000867
months^3	0.0000359	0.00000747
male	0.165	0.0702
venous	0.700	0.0826
unk_sample	-0.356	0.149
Housing Focused Variables		
ERA1	0.671	0.142
ERA2	0.329	0.158
wd_sub	-0.188	0.124
wd_det	0.316	0.289
porch_sub	0.274	0.103
porch_det	0.614	0.235
ep_sub	0.226	0.512
ep_det	-2.399	3.170
ERA1*ep_sub	0.0527	0.519
ERA2*ep_sub	-0.0755	0.560
ERA1*ep_det	3.066	3.172
ERA2*ep_det	2.026	3.225
Geographic Variables		
demo	0.257	0.0795
in_prox	-0.0804	0.0795
not_winter	0.184	0.0717
`East Side`	0.446	0.0860
Midtown	-0.784	0.241
Residual Standard Error:	3.148	

Interpreting the Omnibus Model

Child Focused Variables, Nonlinear and Dichotomous

The interpretation of the non-linear explanatory factors in equation 7.2 requires that we consider several related regression coefficients to understand the impact of year and age on pediatric blood lead levels (BPb). These factors, the year in which the test took place and the age of the child in months, are modeled respectively as quadratic and cubic functions (see chapters 5 and 6 for discussion of these modeling decisions). The signs of the regression estimates in table 7.2 are as expected for year of test (–, +), and age of child in months (+, –, +). Table 7.3 reports the estimated impact of year of test and age of child on BPb at their respective means and at other significant values. The rule of thumb for standard econometrics is a regression coefficient with a less than twice its standard error is statistically indistinguishable from zero. The rule of thumb is complicated by the presence of multicollinearity. Appendix I visually represents the coefficient estimates ±2 standard errors facilitating judgment of the estimates at a glance.

Table 7.3: Estimated BPb Impact at Meaningful Values of Nonlinear Factors

Year of Test	Impact on BPb from 1-unit change
at mean, 7.171 (2006)	-0.278
mean –1 sd, 3.141 (2002)	-0.516
mean +1 sd, 11.201 (2010)	-0.0401
Age of Child in Months	
mean, 34.23 months	-0.0188
12 months	0.0803
24 months	0.0136
60 months	-0.00058
Out of Sample Prediction	
test_yr, 16 (2015)	0.243
120 months	0.596

The impact of a change in test_yr on BPb depends on the value of the test_yr. At the mean of the test_yr variable (7.171) the estimated coefficient is -0.278,[7] meaning that in the year 2006 a 1 unit change in year of test is associated with a fall in BPb of .278µg/dL. Test_yr is modeled as non-linear variable in expecting the coefficient to change from year to year and that is what the coefficient estimates reflect. Computing the change in BPb at one standard deviation higher and one standard deviation lower than the mean test_yr (±4.03) demonstrates the quadratic relationship between blood lead levels and year of test. Tests from 2002 (one standard deviation before the mean year of test) are associated with a -0.516µg/dL change in blood lead level. The tests from 2010 (one standard deviation after the mean value) are associated with a -0.0401µg/dL change in BPb. As time passes blood lead levels are estimated to be falling at a consistently decreasing rate. Rather than asymptotically approaching zero (as observed in figure 5.4) the out of sample prediction in table 7.3 shows that estimated change in BPb associated with year of test turns positive. Such divergence from observed conditions suggests relationship between the observed year of test and BPb may be mis-specified in equation 7.2.

Interpreting the months (age of child at time of test in months) variable involves a similar process and yields more complicated results, reflecting the cubic specification of the of the relationship between BPb and mean age of child in months.[8] At the mean value of age in months the associated change in blood lead level with a 1-unit change is -.0188µg/dL. For a child at 34 months of age a 1 unit change in age is associated with a slight fall in BPb.

[7] $\frac{\Delta \text{BPb}}{\Delta test_{yr}} = \beta_1 + 2\beta_2 \overline{test_{yr}}^2$

[8] $\frac{\Delta \text{BPb}}{\Delta months} = \beta_3 + 2\beta_4 \overline{months} + 3\beta_5 \overline{months}^2$

Considering the coefficient values at other more significant moments in a child's life such as when they are likely to see a doctor for a routine checkup, the estimates produce the expected cubic shaped relationships. At 12 months, I estimate BPb changing at 0.08µg/dL per month, while at 24 months the rate of change has slowed to 0.014µg/dL per month. Note, at the mean of 34 months the expected change has already begun falling. As the child ages figure 5.2 shows that the change in BPb associated only with age falls toward zero. At 60 months the associated change in blood lead level is estimated to be approaching zero (-0.0006µg/dL per month) however in the out of sample prediction from table 7.3, at 120 months the associated change in BPb with a 1-unit change in months has risen to 0.596µg/dL. The out of sample estimate suggests that the relationship between age of child and BPb may be mis-specified in equation 7.2.

The coefficient estimates of male and venous variables yield unsurprising results. There are no warranted expectations regarding the unk_sample coefficient estimates. These are all categorical variables making them easier to interpret than the continuous non-linear variables already discussed. Mathematically their coefficient estimates describe the change in the dependent variable associated with a 1-unit change in the independent variable. More intuitively they describe changes BPb associated with a different categorical state than the ones associated with the variable (a male child, a venous exam, etc.).[9] For instance the male variable indicates whether or not a child is male, thus the estimated coefficient (.165) describes the average expected difference in BPb between the children that are and are not male. The estimated coefficient for the male variable is 2.3 times larger than its standard error. The

[9] n.b. this is not a causal relationship. For example, being identified as a male child doesn't cause blood lead levels to go up. There is something undescribed in the behavior, physiology, or treatment of male children that exposes them to higher amounts of blood lead.

venous variable is associated with an observed BPb .7µg/dL higher than those that are not derived from venous exams. The venous coefficient is 8.5 times larger than its standard error. This magnitude and sign of the coefficient is as expected, children with elevated initial capillary results are directed to be retested by way of the venous exams and is it these second tests which are recorded in the KCMO Health data. The magnitude of the unk_sample coefficient is about half that of the venous coefficient and of the opposite sign (-.356). How to interpret this result (not recording the result of the sample type is associated with lower blood lead levels) is not intuitively clear, and there is nothing in my literature review to assist in that task.

Dichotomous Geography Variables

The associated increase of .25µg/dL when any amount of demolition activity has taken place near a child's home corresponds with the literature emerging on this understudied vector of exposure. The coefficient estimate for demo is 3.2 times larger than its associated standard error.[10] The estimates for the in_prox variable is -.0804µg/dL. The sign of the estimate is surprising in that it diverges from an expected positive sign and magnitude as the variable is designed to indicate likely exposure to lead loaded soil suggesting these relationships are mis-specified.[11] As expected, children tested in the winter months (not_winter = 0) are estimated to have BPb levels .184µg/dL lower than if they are tested at any other time of year. This estimate is 2.564 times its associated standard error.

[10] The magnitude and standard error of the estimated coefficients is consistent across the results reported in tables 7.4 and 7.6

[11] Both the coefficient and the standard error of in_prox vary wildly for the models with restricted coefficients.

Interpreting the `East Side` and Midtown coefficients is straightforward. In this model children that live in the East Side community district are associated with BPb levels .446µg/dL higher than a child that lives in any other community district save Midtown. This result is not unexpected. This estimate is 5.2 times its standard error. Note, the result implies an additional lead burden associated with place that is distinct from the established sources (year of construction, condition of exterior paint, year of test, age of child, etc.) that are explicitly modeled in equation 7.2. The sign and the magnitude of the estimated Midtown coefficient should be conceptualized with respect to the location of all the other heath observations in the sample which are not evenly drawn from Kansas City. Residing in a home in Midtown is associated with an estimated decrease in BPb of .784µg.dL versus residing in a home that is not in Midtown. It makes some intuitional sense that unobserved factors exist in the East Side community district given the well-documented connection between environmental hazards generally and the make-up of that community district in terms of race and class.

Dichotomous and Interacted Housing Focused Variables

Turning to the estimates for the housing focused variables, the interpretations are by turns unsurprising and much more complicated than the dichotomous variables already discussed. The windows and doors, and porch variables are not interacted with the era of construction variables. The estimated coefficients of these variables warrant discussion. The estimated change in blood lead level for wd_sub is -.188µg/dL (se .124) and for wd_det .316 µg/dL (se .289). The sign of the wd_sub coefficient is unexpected and neither windows and doors coefficient is more than twice its associated standard error, these are ponderous results. Window replacement is a primary focus of lead hazard remediation due to the generation of

fine lead dust in their everyday operation when painted with lead. I expected the associated regression coefficients to be large associated with small standard errors, getting larger as their condition decayed but that is not what these estimates show. There is no intuitive explanation for wd_sub being associated with a fall in BPb. It may be the mixing of window condition and door condition into a single variable. An increase in magnitude for the wd_det coefficient is expected but the relatively large standard errors of the windows and doors variables cautions us from giving too much interpretive weight to either variable. The estimated change in BPb associated with porch_sub is .274 (se .103) and for porch_det .614 (se .235). The Importance of porch conditions is mentioned but not emphasized in the epidemiology literature reviewed in chapter 5 (J. Wilson et al. 2015). Nonetheless the estimated coefficients exhibit magnitude, sign, and standard error similar to what we see with the well-established era of housing construction variables. Behavioral possibilities that could explain the estimated value is the porch as portal through which everyone passes to enter and exit the home and/or the porch as a 'safe' place for infants to play, outside but confined away from the street and other threats and uncertainties that may abound.

The remaining housing focused variables are interacted with each other, the individual coefficients are not meaningful in themselves and the interpretation is more complicated than otherwise. To compute the associated change in BPb with a 1-unit change in ERA1 take the partial derivative of equation 7.2 with respect to ERA1. Rather than a single estimate relating changes in ERA1 and BPb there are three coefficient estimates to consider in the interpretation process.[12] This leaves the interacted exterior paint variables a roll in specifying the associated

[12] There is an analogous triad of estimates for each of the interacted terms (ERA1, ERA2, ep_sub, ep_det).

change in BPb with a 1-unit change in ERA1 when exterior paint is intact (0.671), when exterior paint is substandard (0.7237), and when exterior paint is severely deteriorated (3.737). These estimates conform to the expected stair-step pattern; the change in BPb associated with ERA1 increases as the home is observed to have paint in increasingly worse condition. Table 7.4 relays the estimates of each interacted term. The expected stair step pattern does not hold for the ERA2 variable. Though the estimated change in BPb in homes with the most deteriorated exterior paint associated with a 1-unit change in ERA2 is substantially larger (2.355) than the change similarly aged homes with intact exterior paint (0.329), the middle condition of homes with substandard exterior paint is associated with a smaller change in BPb (0.2535) than among the homes with intact exterior paint.

The estimated associated change in BPb with regard to era of housing, though it produces estimates for several associated exterior paint conditions, assumes there is no change in paint condition. Changes in BPb associated with a change in paint condition are estimated through the ep_sub and ep_det variables. The three estimates associated with a 1-unit change in the ep_sub variable are 0.226 for the newest homes, 0.1505 for the 1952-1977 homes, and 0.2787 for the oldest homes. The estimate for those homes in the middle range is unexpected,

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$$\frac{\Delta BPb}{\Delta ERA1} = \beta_9 + \beta_{17}(ep\_sub) + \beta_{19}(ep\_sevdet)$$

$$\frac{\Delta BPb}{\Delta ERA2} = \beta_{10} + \beta_{18}(ep\_sub) + \beta_{20}(ep\_sevdet)$$

$$\frac{\Delta BPb}{\Delta ep\_sub} = \beta_{15} + \beta_{18}(ERA1) + \beta_{20}(ERA2)$$

$$\frac{\Delta BPb}{\Delta ep\_sevdet} = \beta_{16} + \beta_{19}(ERA1) + \beta_{20}(ERA2)$$

theory predicts a stair step pattern which would produce an estimate between 0.226 and 0.2787. Estimated changes associated with a 1-unit change in ep_det reflect the expected stair step pattern but here the magnitude of the change is unexpected. The estimated change among the newest homes is -2.399; indicating an improvement in BPb levels in association with paint in worst condition. This estimate is an artifact of a single observation and should not be given interpretive weight. The estimated change among the homes built from 1952-1977 is -0.373, again a surprising magnitude which may also be a small sample artifact. Finally, the estimated

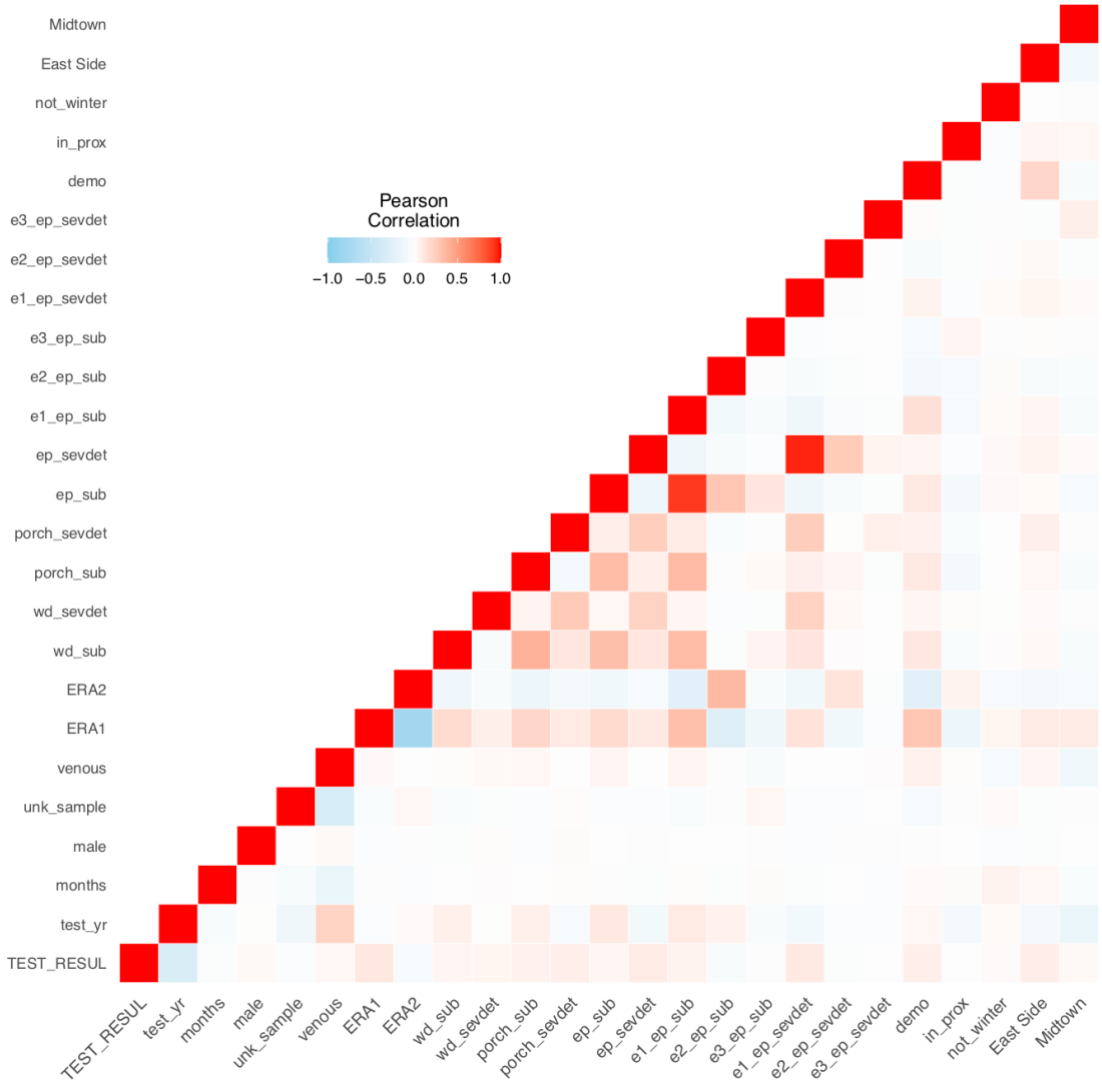
Table 7.4: Interpretation of Interacted Coefficients

Primary Variable	Value of Interaction terms	n	Interpretation	Magnitude of Change
ERA1	ep_sub =0, ep_det=0	3744	Pre-1952 Homes with intact exterior paint	0.671
ERA1	ep_sub=1, ep_det=0	1831	Pre-1952 Homes with substandard exterior paint	0.7237
ERA1	ep_sub=0, ep_det=1	371	Pre-1952 Homes with severely deteriorated exterior paint	3.737
ERA2	ep_sub=0, ep_det=0	1228	1952-1977 Homes with intact exterior paint	0.329
ERA2	ep_sub=1, ep_det=0	229	1952-1977 homes with substandard exterior paint	0.2535
ERA2	ep_sub=0, ep_det=1	29	1952-1977 homes with severely deteriorated exterior paint	2.355
ep_sub	ERA1=0, ERA2=0	269	Substandard exterior paint on post-1977 homes	0.226
ep_sub	ERA1=0, ERA2=1	229	Substandard exterior paint on 1952-1977 homes	0.1505
ep_sub	ERA1=1, ERA2=0	1831	Substandard exterior paint on pre-1952 homes	0.2787
ep_det	ERA1=0, ERA2=0	1	Deteriorated exterior paint on post-1977 homes	-2.399
ep_det	ERA1=0, ERA2=1	29	Deteriorated exterior paint on 1952-1977 homes	-0.373
ep_det	ERA1=1, ERA2=0	371	Deteriorated exterior paint on pre-1952 homes	0.667

change associated with ep_det among the homes built before 1952 is 0.677, a magnitude in keeping with theory.

When interpreting the estimates in table 7.2 note the presence if multicollinearity among the observations. Figure 7.1 visually represents a Pearson correlation matrix[13] which

Table 7.1: Correlation Diagram



[13] I use the following equation to compute each term of the Pearson correlation matrix of my model from Rodgers and Nicewander (1988).

$$r_{xy} = \frac{\sum_{i=1}^{n}(x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=1}^{n}(x_i - \bar{x})^2}\sqrt{\sum_{i=1}^{n}(y_i - \bar{y})^2}}$$

visually represents the degree of correlation between all the variables in my model. At a glance we observe a relatively intense correlation between ERA1 and ERA2 (-.7932).[14] This high degree of multicollinearity is not unexpected. ERA1 and ERA2 together describe over 92% of the sample. The restrictive assumption of the least squares model is a lack of *perfectly* correlated variables or any linear combination thereof. Variables are said to be highly multicollinear if their values (alone or in combination) are observed to move in consort—a rise (or fall) in one variable (or combination) is consistently echoed by a rise (or fall) in another. Multicollinearity is a universal problem and a matter of degree rather than a dichotomous presence/absence condition. As such there is no 'test for multicollinearity' but it is possible to measure its degree in any particular sample (Kmenta 1997, 431). The result of this high degree of multicollinearity is that the relevant regression estimates are likely to be imprecise For highly multicollinear variables the variance of the estimators will be high because of an inability of the least squares technique to isolate the effects of correlated variables. This manifests as regression estimates lacking the desired precision, which can be observed in larger standard errors.

Another effect of multicollinearity is an inability to separate changes in the dependent variable from changes in correlated independent variables. Much of the multicollinearity observed among the variables in equation 7.2 comes from the modeled interaction of era and exterior paint. The necessity of considering the coefficients exterior paint, era of construction, the interaction terms together, is reflected in several extreme values in the Pearson correlation

[14] A numerical version of the correlation matrix presented as figure 7.1 is reproduced as Appendix J.

matrix. The relative lack of precision in the regression estimates from the multicollinearity does not disqualify the results but care must be taken to represent the results accurately.

Understanding the Model Through Restricting of Coefficients

Through a series of F-tests I examine the statistical explanatory power of the variables included in equation 7.2. I group these restrictions into two series. The first series of restrictions are of the child focused and geography focused groups of variables. The second series of restrictions are of components of the housing focused variables.

Restricting Child and Geography Focused Variables

The first step in restricting the child and geography focused variables is to take the 8,077 observations that are selected through the geocoding and assembly processes described in Chapter 6 (a summary thereof is in table 6.14) and estimate equation 7.2. Then take the same 8,077 observations and estimate a model which restricts the Child Focused variables. Functionally this involves estimating equation 7.3.

$$BPb_i = \alpha + \sum_{j=9}^{20} \beta_j H_{ji} + \sum_{j=21}^{25} \beta_j G_{ji} + \epsilon_i \tag{7.3}$$

This restriction is equivalent to the assumption that β_1 through β_8 in equation 7.2 are all equal to zero. Repeat this estimation procedure while restricting the Geography focused variables as in equation 7.4.

$$BPb_i = \alpha + \sum_{j=1}^{8} \beta_j C_{ji} + \sum_{j=9}^{20} \beta_j H_{ji} + \epsilon_i \tag{7.4}$$

The restrictions producing model 7.4 are equivalent to the assumption that β_{21} through β_{25} in equation 7.2 are all equal to zero.

Table 7.5: Regression Estimates for Equations 7.3-7.4

	Equation 7.3		Equation 7.4		Equation 7.2	
Coefficients	Estimate	Std. Error	Estimate	Std. Error	Coefficients	Std. Error
(Intercept)	1.740	0.158	2.904	0.330	2.848	0.338
Child Focused Variables						
test_yr			-0.638	0.0338	-0.701	0.0349
test_yr2			0.02500	0.00242	0.0295	0.00250
months			0.181	0.0299	0.178	0.030
months2			-0.0047912	0.0008696	-0.0047239	0.0008666
months3			0.0000364	0.0000075	0.0000359	0.0000075
male			0.159	0.0704	0.165	0.0702
venous			0.719	0.0826	0.700	0.0826
unk_sample			-0.363	0.149	-0.356	0.149
Housing Focused Variables						
ERA1	0.950	0.152	0.750	0.140	0.671	0.142
ERA2	0.611	0.169	0.308	0.158	0.329	0.158
wd_sub	-0.118	0.132	-0.132	0.125	-0.188	0.124
wd_det	0.167	0.311	0.310	0.290	0.316	0.289
porch_sub	0.314	0.110	0.317	0.103	0.274	0.103
porch_det	0.978	0.253	0.707	0.235	0.614	0.235
ep_sub	1.193	0.550	0.234	0.513	0.226	0.512
ep_det	-1.534	3.413	-3.181	3.173	-2.399	3.170
ERA1*ep_sub	-1.030	0.557	0.082	0.520	0.0527	0.519
ERA2*ep_sub	-1.437	0.601	-0.076	0.561	-0.0755	0.560
ERA1*ep_det	2.694	3.416	3.890	3.175	3.066	3.172
ERA2*ep_det	1.351	3.472	2.861	3.228	2.026	3.225
Geography Variables						
demo	0.213	0.0855			0.257	0.0795
in_prox	0.118	0.0853			-0.0804	0.0795
not_winter	0.0824	0.0769			0.184	0.0716
`East Side`	0.482	0.0899			0.446	0.0860
Midtown	0.603	0.253			-0.783	0.241
Residual Standard Error		3.39		3.16		3.148

The results from these estimation procedures are presented in table 7.5. Because these estimates use the same sample to estimate associations between the built environment and BPb I use an F-test to examine if the assumptions involved in creating equations 7.3 and 7.4 result in a loss of explanatory statistical power. The hypothesis for the F-test restricting the Child-Focused variables is as follows:

$$\mathrm{H}_0: \beta_1 = \beta_2 = \beta_3 = \beta_4 = \beta_5 = \beta_6 = \beta_7 = \beta_8 = 0$$

$$\mathrm{H}_1: \text{At lead one } \beta_j \neq 0$$

The null hypothesis is that the regression coefficients for the variables test_yr, test_yr2, months, months2, months3, male, venous, and unk_sample are all equal to 0. The alternative hypothesis is that at least one of these coefficients is not equal to 0.[15] For this test the calculated statistic is 823.110 while the critical value is 2.513. We reject the null hypothesis that Child-Focused variables add no explanatory power to equation 7.2. This is as expected, the inclusion of the Child-Focused variables is warranted in keeping with the epidemiological literature discussed in chapter 5. Table 7.6 reports the results of each of the five f-tests I perform. The hypothesis of the F-test restricting the Geography-Focused variables is as follows:

$$\mathrm{H}_0: \beta_{21} = \beta_{22} = \beta_{23} = \beta_{24} = \beta_{25} = 0$$

$$\mathrm{H}_1: \text{At least one } \beta_j \neq 0$$

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$$F_{\alpha,q,n-k} = \frac{(SSE_r - SSE_u)/q}{(SSE_u)/(n-k)}$$

Where SSE_r is the sum of square errors for the restricted model. SSE_u is the sum of square errors for the unrestricted model. q is the numbers of restrictions required to make the restricted model. n is the number of observations in each model. k is the number of variables in the unrestricted model. The critical statistic indicating the upper bound of the confidence interval around the estimate of SSE_u is given by $F_{\alpha,q,n-k}$. Where F is a chi-squared distribution at the α level of confidence.

The null hypothesis is that the regression coefficients for the variables demo, in_prox, not_winter, 'East Side', Midtown are all equal to 0. The alternative hypothesis is that at least one of these coefficients is not equal to 0. For this test the calculated statistic is 67.115 while the critical value is 3.02. We reject the null hypothesis that Geography-Focused variables add no explanatory power to model 7.2. This is as expected, the inclusion of the Geography-Focused variables is warranted in keeping with the epidemiological literature discussed in chapter 5.

Table 7.6: Results of F-tests

Equation	Restrictions	Computed f-statistic	Critical Value	Result
7.3	Child Focused	823.110	2.513	Reject the Null
7.4	Geography Focused	67.115	3.020	Reject the Null
7.5	Housing Conditions and Interactions	32.231	2.804	Reject the Null
7.6	Era and Interactions	25.284	2.323	Reject the Null
7.7	Era and Housing Conditions	29.260	2.513	Reject the Null

Considering the changes in the estimated regression coefficients between the several regressions reinforces the importance of accurate modeling. The sign and magnitude of several the geographic variables changes with the relaxation of the restrictions on the child focused variables. The in_prox variable switches signs and the standard error gets relatively larger. The midtown coefficient switches signs while retaining a similar standard error. The not_winter coefficient more than doubles in magnitude while its standard error shrinks. All this underscores the findings of the F-test, that significant explanatory power is lost with the exclusion of the child-focused variables.

Restricting Housing Focused Variables

The Housing-Focused variables are critical to the second series of restrictions and though the restriction process follows the same form as the first series they carry a different set of implications. Investigating relationships between the built environment and pediatric BPb with a focus on parcel level housing conditions through statistical analysis is the third contribution of this dissertation. The restriction of the Housing-Focused variables, testing the explanatory power of the model without groups of the housing variables in three successive iterations, is an explicit step towards understanding the importance of era of construction and housing conditions separately and together. It may be that housing conditions, era of housing construction, and/or the interaction of terms do not significantly improve the fit of the model to the data. These restrictions explore that question and extend the discussion of my model in the presence of multicollinearity. If a group of housing focused variables does not enhance the explanatory power of the model, we can omit those variables, in the context of the estimates in table 7.2 addressing multicollinearity through restriction without significant penalty.

Work to understand this set of restrictions begins by estimating a series of models. Results of these estimations are presented in table 7.7. First, I restrict the housing conditions and the interaction terms and estimate equation 7.5.

$$BPb_i = \alpha + \sum_{j=1}^{8} \beta_j C_{ji} + \sum_{j=9}^{10} \beta_j H_{ji} + \sum_{j=21}^{25} \beta_j G_{ji} + \epsilon_i \quad (7.5)$$

Then, I restrict the era of construction variables and the interaction variables and estimate equation 7.6.

Table 7.7: Regression Estimates for Equations 7.5 - 7.7

	Equation 7.5		Equation 7.6		Equation 7.7		Equation 7.2	
Coefficient	Estimate	Std. Error	Estimate	Std. Error	Estimate	Std. Error	Estimate	Std. Error
(Intercept)	2.914	0.335	3.322	0.311	3.379	0.311	2.848	0.338
Child Focused Variables								
test_yr	-0.729	0.0339	-0.709	0.0348	-0.713	0.0341	-0.701	0.0349
test_yr2	0.0315	0.00241	0.0300	0.00249	0.0303	0.00243	0.0295	0.00250
months	0.178	0.0298	0.1785	0.0298	0.179	0.0298	0.1781	0.0298
months2	-0.00473	0.000868	-0.0047	0.000868	-0.00477	0.000868	-0.00472	0.000867
months3	0.0000359	0.00000749	0.0000361	0.00000749	0.0000365	0.00000749	0.0000359	0.00000747
male	0.164	0.0703	0.157	0.0703	0.160	0.0703	0.165	0.0702
venous	0.720	0.0827	0.714	0.0827	0.717	0.0827	0.700	0.0826
unk_sample	-0.349	0.149	-0.352	0.149	-0.342	0.149	-0.356	0.149
Housing Focused Variables								
ERA1	0.825	0.135					0.671	0.142
ERA2	0.335	0.150					0.329	0.158
wd_sub			-0.146	0.124			-0.188	0.124
wd_det			0.381	0.290			0.316	0.289
porch_sub			0.326	0.102			0.274	0.103
porch_det			0.676	0.234			0.614	0.235
rd$ep_sub			0.313	0.0906			0.226	0.512
rd$ep_det			0.637	0.176			-2.399	3.170
ERA1* ep_sub					0.485	0.0873	0.0527	0.519
ERA2*ep_sub					-0.0188	0.214	-0.0755	0.560
ERA1*ep_det					0.972	0.171	3.066	3.172
ERA2*ep_det					-0.422	0.589	2.026	3.225
Geography Variables								
demo	0.282	0.0794	0.367	0.0768	0.348	0.0773	0.257	0.0795
in_prox	-0.100	0.0792	-0.145	0.0785	-0.164	0.0784	-0.0804	0.0795
not_winter	0.187	0.0718	0.198	0.0716	0.193	0.0717	0.184	0.0717
`East Side`	0.482	0.0858	0.461	0.0861	0.478	0.0859	0.446	0.0860
Midtown	-0.884	0.239	-0.658	0.239	-0.673	0.238	-0.784	0.241
Residual Standard Error:	3.155		3.154		3.155		3.148	

$$BPb_i = \alpha + \sum_{j=1}^{8} \beta_j C_{ji} + \sum_{j=11}^{16} \beta_j H_{ji} + \sum_{j=21}^{25} \beta_j G_{ji} + \epsilon_i \quad (7.6)$$

Finally, I restrict the un-interacted era of construction and housing conditions variables and estimate equation 7.7.

$$BPb_i = \alpha + \sum_{j=1}^{8} \beta_j C_{ji} + \sum_{j=17}^{20} \beta_j H_{ji} + \sum_{j=21}^{25} \beta_j G_{ji} + \epsilon_i \quad (7.7)$$

To compute the second series of F-tests I again compare the estimated sum of squared errors for each model with restricted housing focused variables to those of the unrestricted model. The hypothesis for the F-test comparing equation 7.5 and 7.2 is:

$$\mathrm{H}_0: \beta_{11} = \beta_{12} = \beta_{13} = \beta_{14} = \beta_{15} = \beta_{16} = \beta_{17} = \beta_{18} = \beta_{19} = \beta_{20} = 0$$

$$\mathrm{H}_1: \text{At least one } \beta_j \neq 0$$

The hypothesis for the F-test comparing equation 7.6 to 7.2 is:

$$\mathrm{H}_0: \beta_{11} = \beta_{12} = \beta_{17} = \beta_{18} = \beta_{19} = \beta_{20} = 0$$

$$\mathrm{H}_1: \text{At least one } \beta_j \neq 0$$

The hypothesis for the F-test comparing equation 7.7 to 7.2 is:

$$\mathrm{H}_0: \beta_{21} = \beta_{22} = \beta_{23} = \beta_{24} = \beta_{25} = 0$$

$$\mathrm{H}_1: \text{At least one } \beta_j \neq 0$$

The contents of table 7.6 indicate that for each of these F-tests I can reject the null hypothesis that regression coefficients are statistically indistinguishable from zero. Though the computed values are only an order of magnitude larger than the critical values the test results are unambiguous, excising some of the housing-focused variables won't do. Multicollinearity is the price for increased explanatory power.

Given that the several groups of housing-focused variables have significantly more explanatory power in consort (as well as theoretical and methodological reasons for inclusion) we get a view into the effects of multicollinearity by tracking how the estimated coefficients change from the restricted models to the omnibus model. The estimated coefficients of the in_prox variable change appreciably from the restricted to the unrestricted models, though in_prox is not subject to a restriction assumption. The in_prox estimate also varied in the first series of restricted models (table 7.5) in a more extensive fashion. Among the variables subject to restriction, the era of housing construction coefficients change slightly between the restricted and unrestricted models with the ERA1 coefficient decreasing in magnitude by a little less than 20% while the standard error of both estimates are nearly unchanged. The estimates for the wd_sub, wd_det, porch_sub, and porch_det variables are nearly unchanged from the restricted to the unrestricted models.

The estimated coefficients and standard errors of the exterior paint related variables change wildly between the restricted and unrestricted models. Tracing these regression estimates from restricted to unrestricted models illustrates the impact of using highly multicollinear variables. The ep_sub and ep_det estimates in the restricted model describe changes in BPb of .313µg/dL and .637µg/dL both with a standard errors less than one quarter of the coefficient. In the unrestricted models the standard errors for both terms are larger than the coefficient estimates as the estimate for the ep_det variable changes sign and magnitude (-2.399, sd3.17). Wild changes in sign, magnitude, and standard error also seize upon the estimates of the interaction terms. Appendix I allows us to see at a glance the effects of multicollinearity. The effect of a high degree of multicollinearity on the regression estimates is observed in the wide error bars, a high degree of statistical uncertainty, around the ep_sub,

eb_det, and the interaction terms. The precision of the individual estimates falls while the explanatory power of the entire model increases.

The principal contribution of this dissertation is a statistical model of pediatric BPb focused on the individual in such a way as to incorporate observations of the built environment at the parcel level. A unique aspect of my model is the combination of era of housing construction and observed condition of exterior paint. On the basis of the regression estimate qualified policy recommendations are made. These recommendations are tempered by the limitations of a model that does not purport to engage in causal inference. Table 7.8 illustrates the limitation of my model (as is typical of regression models) to predict the long right tale of observed BPb values. The model is designed to describe statistical deviations from mean BPb rather than deviations from zero BPb. Such a focus accurately reflects the persistence of BPb

Table 7.8: Summary BPb Statistics for Sample Population and Fitted Values

	KCMO Health Observations	Equation 7.2 Fitted Values
Minimum	0	0.0134
1st Quartile	1	2.0569
Median	2	2.776
Mean	3.075	3.075
3rd Quartile	4	3.973
Standard Deviation	3.439	1.396
Maximum	63	7.981

among the sample population. The goal of lead policy is not to reduce the distribution of observed pediatric blood lead levels to the mean, rather to reduce the mean to zero, thus the regression technique employed here must be combined with a larger research agenda focused on the substantive relationships of lead poisoning to achieve the warranted goal.

The policy goal of eliminating pediatric lead poisoning, the limitations of my model and data, the statistical results discussed in this chapter, and the research agenda of this dissertation are all taken into account in the concluding chapter of this dissertation. There I discuss modeling and data developments necessary for the generation of a better statistical analysis, the future empirical investigations suggested by my research, and finally public policy actions which may effect the outcome which has motivated this entire research program.

CHAPTER 8

POLICY DISCUSSION

Inaction, with regard to lead in the built environment, is an unacceptably expensive policy position. Doing nothing to staunch the problem is literally a disinvestment process. Despite decades of attention pediatric lead poisoning remains widespread and an indicator of an unequal society. The most important contributions this dissertation can make are policy suggestions which effectively reduce the number of children exposed to a lead loaded environment. The *why* of my dissertation project has always been to address the implications of environmental inequality in Kansas City (and in the US more generally). This concluding chapter contains three main parts: first is an overview of research directions that follow from the statistical aspect of the dissertation, second, a review of the policy suggestions supported directly by the statistical analysis of the dissertation, third, a review of public policy which follows from the research agenda and modeling framework of the dissertation.

The data assembled for my statistical analysis complements a discussion of public policy. The data collected can be used to design, assess, and understand future lead hazard intervention policy in Kansas City. The catalog of assembled data is a template to be emulated in other cities. The problems created by lead in the built environment (particularly from legacy automotive emissions and lead paint) are common to all cities in the US. A standard set of observational data will facilitate comprehensive comparisons between cities and allow the judgment of the relative efficacy of intervention strategies employed in several cities. From mutual contributions to the joint stock of knowledge emerges a knowledge irreducible to its parts. Closing the interdisciplinarity circle entails sharing my research findings beyond the

economics community; returning insight and adding perspective to the public policy, epidemiology, and urban planning communities just as I have drawn insight and perspective from these fields in my research.

Corresponding to the second half of equation 2.3 (p. 28 above), my statistical research is focused on the relationship between the built environment and pediatric blood lead levels in Kansas City, MO between the years 2000 and 2013. Thus, my suggestions for future modeling, data acquisition, and lines of inquiry, as well as the policy implications of my statistical results are narrowly focused on these quantified relationships. The dissertation closes with a consideration of the broader policy implications of my research agenda following from the bundle of relationships implied by equations 2.1 - 2.3.

Future Modeling, Data Acquisition, and Lines of Inquiry

Scrutiny of my statistical investigation invites modeling and data acquisition developments to make it more comprehensive and statistically rigorous. The list that follows is necessarily incomplete, unknown datasets will be identified in the process of data acquisition and lines of inquiry launched from the advanced base I have prepared will call for modeling developments not listed here. My model will be improved by the inclusion of additional BPb observations. The analysis of blood lead tests since 2015 is warranted as is universal pediatric blood lead testing. The results presented in chapter 7 would be improved by increasing the proportion of the pediatric population tested for blood lead, regardless of race, class, or neighborhood. Developing information about race and class for the individual observations of BPb will enable the disaggregation of the sample to better describe the experience of lead poisoning along demographic lines. Parcel size varies systematically within and across Kansas

City neighborhoods such that it may be useful to use a size of parcel variable as a proxy for race and class, thus incorporating the city's unequal geography more comprehensively into the analysis.

There are several categories of data about the built environment that will usefully extend my model. Point sources of lead emissions along with volume of emissions data will facilitate the modeling of the continued creation of a lead loaded environment. Information about the water system such as information about the solvents used to treat the system and the presence of lead service lines used to connect individual houses to the system will enable the modeling of water as a vector of lead exposure. Adding demolitions data for communities outside of Kansas City, Missouri will improve the scope of the investigation to include more of the metropolitan area.

The spurious out of sample estimates for year of test and age of child variables suggest a different functional form should be used to model the non-linear relationship between those variables and BPb. There are packages in the computing software R designed to address issues arising from non-linear factors. The unexpected estimates associated with the in_prox variable and the considerable epidemiological research emphasizing the importance of soil as a vector of exposure recommend a comprehensive rethinking of the in_prox variable components. The development of detailed soil lead observations for the Kansas City metropolitan area is warranted. Short of a systematic survey of soil lead levels, more detailed observations and modeling of the historic sources of environmental lead are warranted.[16] The weather variable used in my research, not_winter, is crude and may be fruitfully expanded to reflect with more

[16] For instance, expanding the survey of gas stations from the era of leaded gasoline beyond the sample year 1976 and to include observations of heterogeneity of current conditions (leaking storage tanks vs. removed storage tanks, etc.).

nuance observed weather variations. Meteorological variables may be interacted with observed soil conditions for a model better informed by causal relationships.

Two additional time related issues: the first regards matching health encounters to housing conditions. My relation of BPb and the built environment relies on matching health encounters to housing observations that occur in the same year. An investigation into the rate at which housing conditions are observed to change in the NHCS will provide guidance for a wider temporal match window thus increasing the number of observations and improving the estimates of this model. The advantages of widening the match window will be tempered by a recognition that people, particularly the poor, change residencies[17] thus widening the match window will invite problems via the uncertain geographic context problem (UGCoP). The second temporal issue regards understanding the secular trend in BPb with regard to year of test. NHANES observations indicate the existence of a time trend (see figures 5.3 and 5.4) and the most impactful explanatory factor in my model is test_yr factor however it is losing explanatory power over time. Given that lead is a stable element and once introduced into the environment it is permanent, the observed relationship between time and BPb presents an important anomaly to be investigated.

The consistency of the coefficient estimates for the demo variable across the restricted and unrestricted models supports looking more closely at the relationship between demolition activity and BPb. My model does not account for variation in the number of demolitions, the size of the structure being demolished, when the demolished structure was built, nor when in the child's life demolition took place. Each of these factors, individually and in combination,

[17] The preponderance of evictions among the poor suggests the relationship between eviction activity and blood lead poisoning is an important research agenda to be investigated.

warrant closer attention. There is variation in the relationship between demolition activity and the lifecycle of its neighborhood that should be articulated clearly; a preponderance of demolitions in urban neighborhoods describe different social processes than a similar preponderance in suburban neighborhoods. Other spatial issues warrant investigation; BPb patterns at the neighborhood rather than the community district better reflects a common lived experience. Neighborhood level analysis allows the incorporation of census data on race, class, and education into the quantitative narrative, describing associations with lead poisoning at the geographic level of the lived experience in greater detail. The geography of food deserts warrants investigation, there is an established link between nutrition and a child's propensity to incorporate lead into their body.[18] The connection between food desert geography and blood lead levels is under investigated. Chapter 6 mentioned the existence of repeated observations for individual children in the KCMO Health data as indicated by a DCN variable. Avenues of investigation into the importance of movement between community districts, between neighborhoods, into and out of food deserts, and other life cycle events will be possible to the degree the DCN variable allows the repeated observation of individual children.

Policy Implications of Statistical Research

The NHCS data and KCMO Health data are the genesis of any insight this analysis contains yet both data sets (and their interaction) impose limits that warrant review. The NHCS data is not a complete survey of the metropolitan area. The Kansas City, MO urban core is

[18] The biological impact of small changes in blood lead is an important open question with significant potential impact on regulatory issues. Is the relationship between the magnitude of blood lead and health outcomes linear, and if not what is the shape of this relationship? Much work has been done to show that there is no safe level of blood lead.

repeatedly surveyed while suburban geographies are nearly absent from the NHCS. The KCMO Health lead data only contains observations for those children that are tested. The KCMO Health data is not a representative sample of the total pediatric population, though it is the complete population of test results. The geographies of the relatively affluent and white are underrepresented in the KCMO Health data. The limitations of the health set have been filtered through the limitations of the housing survey data before my statistical analysis took place. As mentioned in my review of neoclassical health economics the impact of endogeneity upon statistical estimates is ambiguous (Zivin and Neidell 2013). The policy recommendations that follow should be understood as conditional upon these limitations, some policy recommendations are designed to overcome these limitations.

Universal blood lead testing the first public policy suggested by my research. A program of universal blood lead testing will aid the research into the sources of lead exposure. Comprehensive BPb testing will enable the identification of worrisome clusters and variation where currently there is a paucity of information. To the degree they exist, null observations for blood lead will help statistical models relating exposure to the built environment be more precise. To the degree they do not exist, the absence of null results for blood lead and the indication of general exposure will galvanize public support for more aggressive and strident reductions in environmental lead. Universal lead testing will add precision to the geography of lead testing, allowing more robust investigation into the heterogeneous experience of space with the promise of producing knowledge which can precisely identify and prevent exposures from the built environment.

Strictly enforcing existing demolition regulations is required to hold the line while additional inquiries into the effects of demolition on health take place. My statistical results

support proximity to demolition activity as a factor associated with higher blood lead levels. We know from archival research that public infrastructure was identified by the lead industry as a target market and ideal use for interior lead paint (G. Markowitz and Rosner 2000) thus particular attention should be given to make sure that the demolition of large public buildings built before 1952 are done as safely as possible. Recognizing the existence of uncertainty regarding how to safely engage in demolition activity, the Detroit, MI Health Department[19] developed a comprehensive set of best practices to be followed before, during, and after demolition takes place (DHD 2017). Chapter 19 Article II Section 3303.6 of the Kansas City, Missouri Code of Ordinances regarding demolition states, "All material to be removed shall be wet *sufficiently* to lay the dust incidental to its removal" (Kansas City, MO n.d., itallics added) however no guidelines are in place for before or after demolition activity, nor is there guidance for the safe trucking of lead contaminated debris. First person experience indicates that Kansas City's existing demolition ordinance is not regularly enforced. While considering new regulations, enforcement of existing regulations is critical. Further investigation into the impact of demolition activity on BPb may indicate that new rules are needed. However well justified, rules are irrelevant when adherence and oversite is inadequate.

My statistical results recommend focusing remediation and abatement attention in Kansas City's East Side Community District. Era of housing construction data is inadequate to direct lead hazard interventions within a geography where the majority of housing is built during the era of most extreme lead paint usage. My statistical research demonstrates the importance of using age of housing in combination with condition of exterior paint to identify homes where lead hazard interventions can have the largest immediate impact is warranted.

[19] Similar work has been done in Baltimore, MD and Saint Louis, MO.

Age of housing and condition of paint observation send different signals depending on how they are combined. The series of f-tests I performed reinforce the importance of including the housing focused variables as modeled in equation 7.2. My research highlights the importance of exterior paint and porch condition being incorporated into a comprehensive process of identifying and reducing housing-based lead risks. The statistical results demonstrate the usefulness of the exterior observation of housing conditions at the parcel level for understanding blood lead levels in children, thus the implementation of additional neighborhood housing conditions surveys is warranted. Comprehensive housing condition surveys will provide up to date, flexible and low-cost data on the actual condition of the built environment.

Policy Implications of Research Agenda

Several base facts emerge from my review of the biology, epidemiology, and history of lead poisoning. Lead was built into our environment and there has been slow progress toward removing it from the built environment. Pediatric lead poisoning has a spatial footprint that is at first evidence of human activity and to the degree it persists evidence of human inactivity. This spatial footprint informs our understanding of what segregation on the basis of race and class mean experientially. To talk about the persistence of lead poisoning is to talk about systematic disinvestment on the basis of race and class. To address lead poisoning, removing it from the built environment and/or preventing additional accumulations is an investment in the equity of marginalized communities. Lead poisoned children grow up to be lead poisoned adults. Investing in prevention or removal of lead from the environment is an

investment in people that pays off intergenerationally. These base facts add weight to the policy suggestions that follow from the investigation presented in the preceding chapters.

In the abstract, the structural equations I put forward on p. 28 suggest solutions to the problems of lead poisoning: stop the production of a lead loaded environment through effective regulation (equation 2.1), actively remove lead from the built environment through remediation and abatement activity (equation 2.2), make it possible for all families to live in lead safe housing and neighborhoods without regard to race, class or ethnicity (equation 2.3). The generality of these solutions does not make them any less true. The problem is one of institutional adjustment. Housing in the United States is undergoing evolutionary drift; working through the institutional legacies of housing policy, industrial pollution, racism and class inequities, and the ameliorative policies adopted in the 1990's to fight lead poisoning (H. Needleman 1998). A consideration of mid-century urban renewal recognizes that massive, abrupt changes to housing policy are possible but do not necessarily create their intended outcomes. Minimal dislocation is preferable to the raising of entire neighborhoods and dislocating people en mass from their homes and community. Questions of the speed and comprehensiveness remain to be worked out through political means.

The lead-hazards presented by homes built before 1952 recommends focusing abatement and remediation efforts in those geographies where the preponderance of the homes are of that vintage. For the well-being of society as a whole, policy should be focused on helping those most impacted by lead poisoning. Despite the hundreds of millions of dollars already spent, the magnitude of the problem requires the dedication of additional resources. The data presented in figure 5.4 (p. 104) shows a stark contrast between the nationally representative NHANES sample and the KCMO Health Department data which draws heavily

racial and ethnic minority populations as well as the poor. More critically, figure 5.4 shows us that mean blood lead levels in the tested KCMO population held steady from 2008-2015 while the national trend continued downwards, a troubling and ponderous observation. The cause of the Kansas City trend is not apparent. Housing policy and industrial regulation have historically been the most important factors in combating lead poisoning and the health and social outcomes it produces. Housing and regulation with regard to lead are health and social policy, allowing BPb to plateau in some geographies is the tacit acceptance of the persistence of an unjust society (not merely an unequal one). Additional regulatory and abatement activities are called for.

Inspired by the possibilities of a Green New Deal and the comprehensive approach it imagines for combating environmental inequality I close with a list of policies to complement the lead-safe housing activities, demolition regulation, and universal childhood blood lead testing policies supported by my statistical research: restorative justice for the adult survivors of pediatric lead poisoning, enhanced educational assistance for children in areas with higher-than-average BPb, comprehensive lead service line replacement, Comprehensive soil testing and abatement, the development of lead-safe recycling techniques, enhanced oversite of/prohibition on lead in consumer products, the elimination of coal fired power plants, the development of alternative battery technologies. An ambitious list to be certain, but if it any of it were easy the list would be shorter. Individually these policy initiatives embody at minimum a jobs program. Together they embody a vision for an economy based on an ethics of democratic equality, justice, and care.

In the most ideal scenario, with the full funding and social dedication to the agenda I have articulated (as well as the unforeseen programs that are not yet enumerated), it will take

a decade or more to neutralize the threat of lead in the built environment. A program to bridge this temporal gap is necessary. Providing adequate nutrition for all children may not come to mind directly as a lead-fighting program, however using nutritional assistance to combat lead poisoning may be an effective bridge strategy. While the prophylactic effectiveness of nutritional interventions is not extensively studied, research suggests that deficiencies in iron, calcium, protein, and zinc are related to higher blood lead levels and potentially increase a child's vulnerability to the negative effects of lead (Schell et al. 2004). Similar effects have been observed with regard to expecting mothers transferring their lead burden to their children (Schell et al. 2003). Undernourished children are an afront to human dignity no less than lead poisoned children are. Action on this front is warranted. It may be possible to address both indignities simultaneously through programs like the Community Eligibility Provision (CEP). CEP is currently designed to allow schools serving a high-poverty population can enroll their entire student bodies in free lunch and breakfast programs. Altering the criteria for CEP to include high observed community BPb is a small change that could have overlapping benefits on health, education, and social outcomes by helping kids avoid the worst effects of lead poisoning.

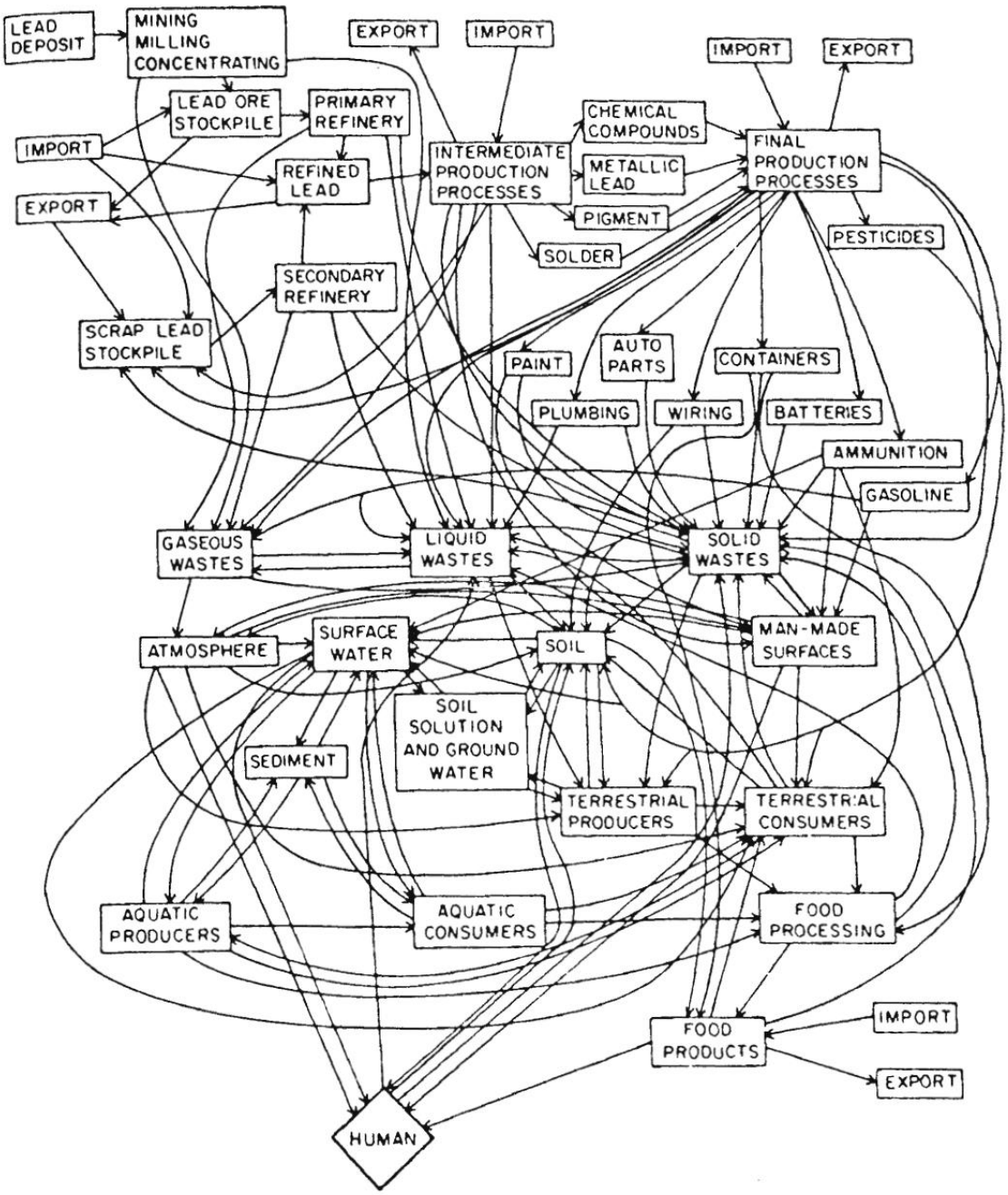
Writing for the Centers for Disease Control in 1991 Binder and Falk published the *Strategic Plan for the Elimination of Childhood lead poisoning*, an agenda for a comprehensive effort to stop the epidemic of childhood lead poisoning (Binder and Falk 1991). A strong national effort to eliminate the disease developed and federal policy shifted towards primary prevention. However, adoption of the most critical elements of the plan such as universal BPb testing were derailed. Thirty years later the scope of the work remaining to end lead poisoning could give us pause, but an appreciation of how far we have come should give us hope.

Together they remind us we must redouble our efforts if we are to end the scourge of childhood blood lead poisoning.

APPENDIX A

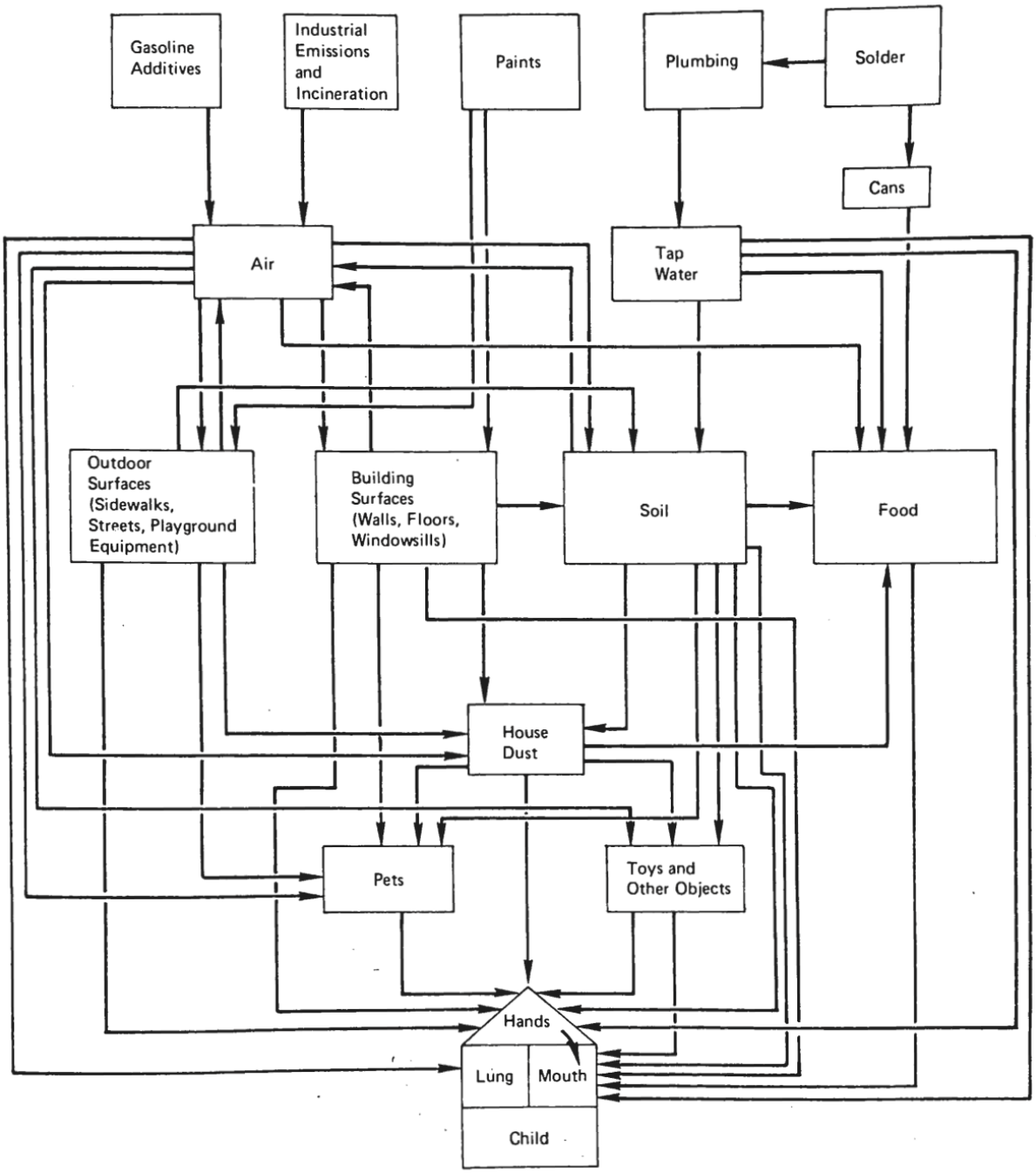
SCHEMAS OF SOURCES AND BIOLOGICAL PROCESSES OF PEDIATRIC LEAD POISONING

Figure A.1: Schema of Anthropogenic Sources of Lead



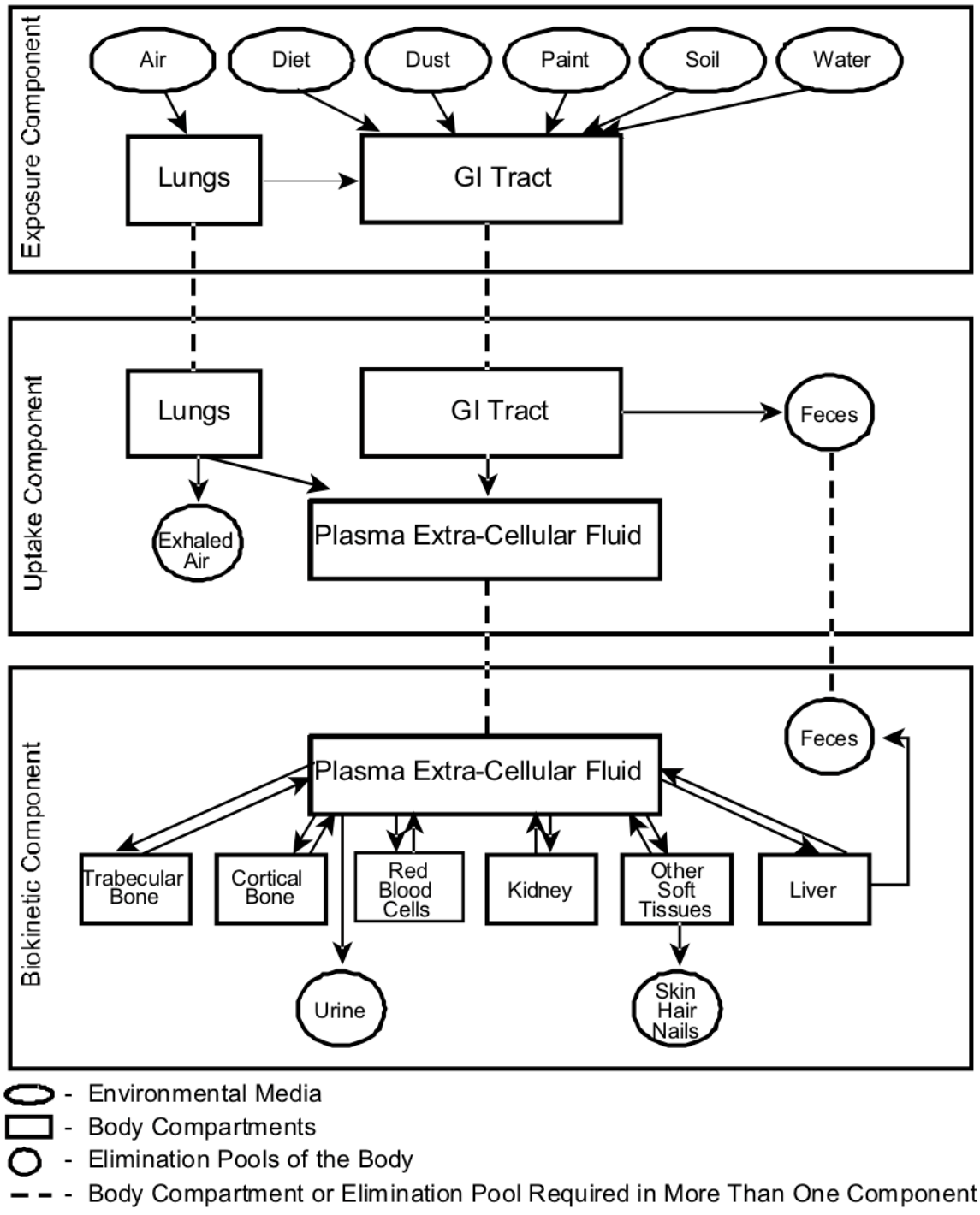
Source: (National Research Council (U.S.) 1980, 35)

Figure A.2: Lead Sources and Pathways in the Built Environment



Source: (National Research Council (U.S.) 1980, 41)

Figure A.3: Biological Schema of Lead Exposure



Source: (Syracuse Research Corporation 2007)

APPENDIX B

RECORD OF BRAINSTORMING SESSION TO DEVELOP A LIST OF RISK FACTORS RELATED TO PEDIATRIC LEAD POISONING

Modeling risk factors for lead poisoning when observations are individual patients

1. Should the variable we are modelling be binary, multinomial, count, other?
    a. Binary – the individual had lead poisoning on a specific date or did not have lead poisoning on that date. Values for variable are 0,1
    b. Multinomial – is the seriousness of the lead poisoning important? If so, do we have a convenient classifying scheme for seriousness? If we do and there are seriousness classifications, then values for variable are 0,1,2,…,g
    c. Continuous – the actual measurement of the lead content of blood test
    d. Count – should the variable be a count of the number of times within a time interval that the individual reports having lead poisoning. What is the appropriate time interval?
    e. Other?

2. What are the risk factors we should control for in the analysis of lead poisoning?
    a. Individual
        i. Age (date of birth)
        ii. Race/ethnicity (increased risk in AA, increased risk with some traditional practices, increased risk of renal involvement in some races?)
        iii. Gender
        iv. Obesity (maybe- related to toxic effects of lead, food anxiety which can increase the risk of lead poisoning)
        v. Asthma
        vi. Developmental Delay
        vii. Persistent hand to mouth behavior (thumbsucking etc.)
        viii. Pica (adult and child)
        ix. IQ
        x. Foreign born
    b. Family
        i. Family history of _____ (lead poisoning, thumbsucking, pica)
        ii. History of _____ traditional high risk practices (make up, jewelry, spices, dirt eating, gum sharing)
        iii. Marriage status (related to poverty which increases risk)
        iv. Income (related to poverty which increases risk of poor housing)
        v. insurance type (may see increased risk in private insurance)
        vi. race/ethnicity (increased risk AA, Latino infants, ethnic practices)
        vii. Maternal IQ
        viii. Family violence
        ix. socioeconomic status

x. Sibling with lead poisoning
xi. Family members with high risk occupation

c. Housing
    i. Date moved in

    ii. How many nights per week at this address?
    iii. Type (single, duplex, apt)
    iv. Age (decreased risk from residential lead paint if home is post 1978)
    v. Number of stories (stairs)
    vi. Flooring
    vii. Structural integrity(dilapidated housing increases risk)
    viii. Clutter inside
    ix. Yard condition(soil, grass, gravel, concrete)-(bare soil increases risk)
    x. Clutter outside
    xi. Registered rental
    xii. Basement?
    xiii. Pets- Pets may track contamination
    xiv. Mixed use housing-may be higher risk (auto repair shop below apt.)
    xv. Renovation/repair history
    xvi. History of code violations
    xvii. Open code violations
d. Environmental
    i. Street condition(road work increases risk esp curb blasting etc.)
    ii. Land use-previous or current high risk industry
    iii. Rural/urban-(Urban children have increased risk)
    iv. Air pollution PM 2.5
    v. Air pollution Lead
    vi. Nearby road work or demolition work or construction
e. Neighborhood
    i. Landscaping
    ii. Yards in neighborhood surface (soil, grass, gravel, concrete)
    iii. distance to schools
    iv. sidewalk condition
    v. yard condition-(bare soil)
    vi. proximity to busy streets-(road contamination can blow in)
    vii. crime data-(high crime linked to older housing, poverty and lead poisoning)
    viii. socioeconomic status of-
    ix. vacant lots contiguous to target residence

APPENDIX C

NEIGHBORHOOD HOUSING CONDITION SURVEY RATINGS GUIDE SUMMARY

Neighborhood Housing Conditions Survey
Ratings Guide Summary

Classification

1. Structure Type

1. **Residential.** The structure was built for residential use.
2. **Non-residential.** The structure was built for other than residential use.
3. **Vacant Lot.** There is no structure on the parcel.
4. **Parking Lot.** The parcel is used for parking.
5. **Park.** The parcel has a park
6. **Common Area.** A parcel common to townhome or condominium type properties.

2. Use Type

1. **Residential.** The structure is being used residentially.
2. **Non-residential.** The structure is being used other than residentially.
3. **Mixed.** The structure is being used both residentially and non-residentially.
4. **Un-ratable.** The parcel's current use cannot be determined.
5. **Not applicable.** Applies to parcels with no structure.

3. Residential Type

1. **Detached-1.** Single family dwelling
2. **Detached-2.** Duplex (designed and built as a duplex, not converted from single-family).
3. **Attached.** Structures such as row housing, sharing roofs and outside walls.
4. **Apartments.** All other (non-institutional) multi-family residential units.
5. **Non-residential.** Applies to all non-residential structure types.
6. **Not applicable.** Applies to parcels with no structure.

4. Structure Profile

1. **Single level**
2. **2-story**
3. **3-story**
4. **4-6 floors**
5. **Over 6 floors**
6. **Not applicable.** Applies to parcels with no structure.

5. Visible Address

1. **Yes**. The parcel has a visible address
2. **No**. The parcel has no visible address

Structure Conditions

1. Roof

1. **Severely Deteriorated.** There are holes visible through roof sheathing. Rafters are sagging or collapsed. Soffits and fascia boards are missing or display severe rot and deterioration.
2. **Seriously Deteriorated.** There are no holes present. The roof has sagging rafters, but sagging is not severe. Roofing shingles are extremely deteriorated. More than five shingles are currently missing on the front exposure of the roof. It appears some sheathing needs to be replaced. Soffits and fascia boards display moderate rot and deterioration.
3. **Substandard.** There are no holes or sagging. Roofing shingles are deteriorated and should be removed before new shingles are installed. Less than five shingles are missing on the entire roof. Soffits and fascia boards display slight rot deterioration.
4. **Good.** Roof shingles show slight wear. (discoloration can be seen from street, or faded color do to loss of rock). There are no holes or sagging rafters. Soffits and fascia boards may need painting, but there is no rot or deterioration.
5. **Excellent.** Roofing shingles show no wear. Soffits and fascia boards display no rot nor deterioration and are adequately installed.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).
7. **Un-ratable.** Characteristic applies to rated parcel, but rating could not be determined (e.g., roof rating for structures with flat roofs, or where line of sight to roof is obscured by trees).

2. Foundations and Walls

1. **Severely Deteriorated.** There are large holes, bulges, and/or leaning walls indicating a partial structural failure. More than 25% of the siding material displays rot or deterioration and needs to be replaced.
2. **Seriously Deteriorated.** There is slight leaning, but no sign of structural failure. More than 25% of the siding material displays rot or deterioration and needs to be replaced.
3. **Substandard.** There is no leaning. Some siding materials need replacing, but it is less than 25%.
4. **Good.** There is no leaning nor siding that needs to be replaced, and surfaces do need some painting.
5. **Excellent.** There is no leaning nor siding to be replaced. Surfaces are adequately painted.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

3. Windows and Doors

1. **Severely Deteriorated.** There are numerous windows or doors missing or boarded. Frames show signs of severe rot and deterioration. The building is open to entry.
2. **Seriously Deteriorated.** There are a couple of openings that are missing or boarded, but the building is not open to entry. Frames show signs of severe rot and deterioration.
3. **Substandard.** All windows and doors are in place, but there are some broken glass in one or more windows. Frames show signs of moderate rot and deterioration, but mostly only need to be painted.
4. **Good.** There is no broken glass present and doors are secure. Frames on windows or doors need paint, but nothing needs replacing.
5. **Excellent.** There is not broken glass present and all frames are adequately painted.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

4. Porches

1. **Severely Deteriorated.** There is leaning of vertical support members or sagging of beams and joists. Rot and deterioration are extensive. Parts of the porch are missing. The porch does not appear safe.
2. **Seriously Deteriorated.** There is slight leaning or sagging, but moderate to extensive rot and deterioration. All parts of the porch are present, and it appears safe to use.
3. **Substandard.** There is slight leaning or sagging that needs to be corrected, but no rot or deterioration. Some painting is needed.
4. **Good.** There is no leaning or sagging, but some painting is needed.
5. **Excellent.** There is no leaning or sagging. All components are adequately painted or protected against weathering.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

5. Exterior Paint.

1. **Severely Deteriorated.** Over 50 % of the exterior walls are peeling. Rot and deterioration are extensive. Parts of the exterior walls are missing. Extensive work to prepare for painting (more than two weeks).
2. **Seriously Deteriorated.** Between 10 and 50% of the exterior walls are peeling. There is a moderate to extensive amount of rot and deterioration. Moderate to extensive work will be needed to prepare the walls for painting (less than two weeks).
3. **Substandard.** Less than 10% of the exterior walls are peeling or faded in color. There is no rot or deterioration present. Some painting is needed.

4. **Good.** There is no peeling paint, but some fading is present, some fresh paint is going to be needed.
5. **Excellent.** All components are adequately painted or protected against weathering.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

Grounds Conditions

1. Private Sidewalks and Driveways

1. **Severely Deteriorated.** The sidewalk is broken and settled with more than one tripping hazard present and/or has sections missing. Has severely deteriorated pavement and does not prevent the tracking of mud into the street. If the driveway was gravel in the first place, will have severe weeds within the exposure.
2. **Seriously Deteriorated.** The sidewalk displays numerous tracks over 1/2 inch wide and breaks there is no tripping hazard present. AND/OR the driveway was originally paved but has severe scaling, cracking, or other signs of deterioration. The full surface needs to be re-paved.
3. **Substandard.** The sidewalk and driveway contain numerous cracks over 1/2 inch wide and over 50% of the surface needs to be repaved.
4. **Good.** The sidewalk and driveway display only a few cracks over 1/2 inch wide, but some patching or sealing of cracks is all that is needed.
5. **Excellent.** There are no cracks wider than 1/2 inch present in either the sidewalk or driveway.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

2. Lawn & Shrubs

1. **Severely Deteriorated.** The vegetation (grass) has grown over 3 feet high. Shrubs appear to have not been trimmed in several years (windows, doors covered).
2. **Seriously Deteriorated.** The vegetation (grass) is between 1 and 3 feet high. Shrubs appear to have not been trimmed within the last year (overgrowing home).
3. **Substandard.** Vegetation (grass) is about 1 foot high. Shrubs need trimming, but appear to have been trimmed within the last year (shrubs still have some shape).
4. **Good.** Vegetation (grass) is under 1 foot and shrubs do not need trimming. There are lawn weeds, like dandelions, present. It appears the lawn doe not receive supplemental fertilizer, but yard is cut regularly.
5. **Excellent.** Vegetation (grass) is under 6 inches high and there are few or no lawn weeds, like dandelions, present. It appears the lawn regularly receives fertilizer and yard is cut regularly.

3. Vehicles

1. **Severe Problem.** There are over 3 vehicles parked in the yard and several appear to be disabled or unlicensed.
2. **Serious Problem.** There are 1 to 3 vehicles parked in the yard. At least one appears to be disabled or unlicensed.
3. **Substandard.** There is one vehicle parked in the yard but it appears to be operable and licensed. Or, there is one or more vehicles in the driveway that appears to be disabled or unlicensed.
4. **Good.** There are no vehicles parked in the yard, but there may be one vehicle on a driveway that is unlicensed.
5. **Excellent.** There are no vehicles parked in the yard. No disabled or unlicensed vehicles are present.

4. Litter

1. **Severe Problem.** There are piles of trash, which may include brush, present on the property. Due to the volume and size of trash items, it will take a dump truck to haul it all off in one load.
2. **Serious Problem.** There are piles of trash, which may include brush, present. It will take a full size pick up to haul it off in one load. It is not practical to attempt to place the trash in plastic bags.
3. **Substandard.** There is trash scattered across the property. It will not fill a pick up. There trash can be placed in trash bags and it will fill between one and five 30 gallon trash bags.
4. **Good.** There is some litter scattered across the property. It can be placed in plastic bags and it will not fill one 30 gallon bag.
5. **Excellent.** There is no litter present.

5. Open Storage

1. **Severe Problem.** There are numerous items stored in the yard that should be stored inside. The items are so numerous, they would more than fill an average 2 car garage.
2. **Serious Problem.** There are numerous items stored in the yard that should be stored inside. The would fill a one car garage.
3. **Substandard.** The items stored outside would fit inside a small (up to 9 by 12 feet) storage shed.
4. **Good.** There are no unacceptable items stored outside, but there are numerous acceptable items that still present a clutter appearance.
5. **Excellent.** There are no unacceptable items present. Acceptable items, if present, are few in number and do not present a cluttered appearance.

Public Infrastructure Conditions

1. Public Sidewalk

1. **Severely Deteriorated.** The sidewalk has sections missing, broken, or heaved. There is more than one tripping hazard present due to displacement of sections or missing sections. More than half the sections need replaced.
2. **Seriously Deteriorated.** There are tipping hazards present due to displaced cracks, settling and heaving. 1/4 to 1/2 of the sections need to be replaced.
3. **Substandard.** There are cracks over 1/2 wide present, but no tripping hazards. Less than 1/4 of the section need to be replaced.
4. **Good.** There is only a few cracks present, however does not present a hazard. Some patching of cracks is needed, but no sections need replacement.
5. **Excellent.** There are no cracks present. There is no settling or heaving creating tripping hazards. They are in great shape and will be there for a long time.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

2. Curbs

1. **Severely Deteriorated.** There are no curbs present, with or without open ditch drainage.
2. **Seriously Deteriorated.** Curbs are present and display severe deterioration. There are sections missing. More than 1/2 of the curb would have to be replaced in order to fill in gaps.
3. **Substandard.** Curbs show deterioration. Up to 1/2 the curb would have to be replaced to fill gaps.
4. **Good.** There is some wear or deterioration but there are no sections missing.
5. **Excellent.** There is no wear and are benefit to water control within the neighborhood.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

3. Streetlights

1. **Severe Problem.** There are no streetlights on the block.
2. **Serious Problem.** Streetlights are more than 8 houses apart. Lights present appear to be broken, or tree limbs block illumination.
3. **Substandard.** Streetlights are more than 6 houses apart. Lights work, but tree limbs block illumination.
4. **Good.** Streetlights are 5 houses apart. Some tree limbs are near the lights, but not blocking illumination.
5. **Excellent.** Streetlights are less than 5 houses apart. No tree limbs growing near lights.

4. Catch Basins

1. **Severely Deteriorated.** The catch basin is severely deteriorated. It is broken or collapsed and creating a danger to pedestrians or traffic (leaves water standing for days, or would be dangerous to evening walking).
2. **Seriously Deteriorated.** The catch basin is severely deteriorated and needs replacing, but is not creating a dangerous situation (i.e. doesn't create traffic hazards during rains, etc.)
3. **Substandard.** The catch basin is not deteriorated but it is substantially blocked with leaves and litter (just needs to be cleaned out).
4. **Good.** There are leaves and litter in the catch basin, but it still functions adequately.
5. **Excellent.** There are no defects or leaves and litter present. Catch basin is in perfect operational condition.
6. **Not applicable.** Characteristic does not apply to rated parcel (e.g., roof rating for parcels with no structure, public sidewalk rating for parcels with no sidewalks, etc.).

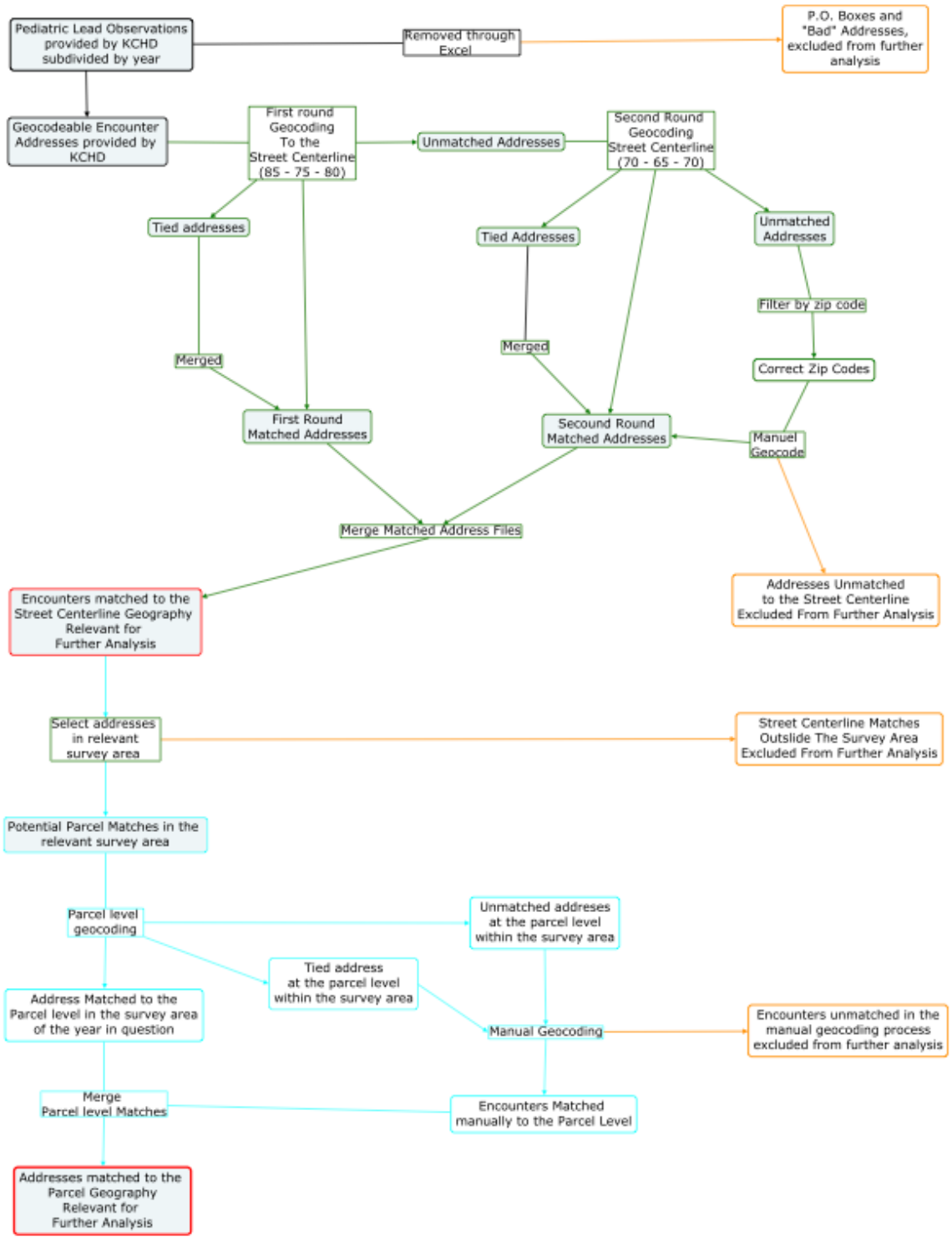
5. Street Condition

1. **Severely Deteriorated.** The pavement is severely deteriorated. There are more than 7 potholes present. Vehicles cannot safely exceed 15 miles per hour due to the uneven surface. Resurfacing is needed on the whole block.
2. **Seriously Deteriorated.** The pavement is deteriorated. There are 4-6 potholes present, but traffic flow is not substantially affected. Resurfacing is needed.
3. **Substandard.** There are 3 or less potholes, mostly cracks in the surface. Patching, not resurfacing, is needed.
4. **Good.** There are no potholes. There are some cracks, but none wider than 2 inches.
5. **Excellent.** There are no cracks and no potholes present.

APPENDIX D

GEOCODING SCHEMA

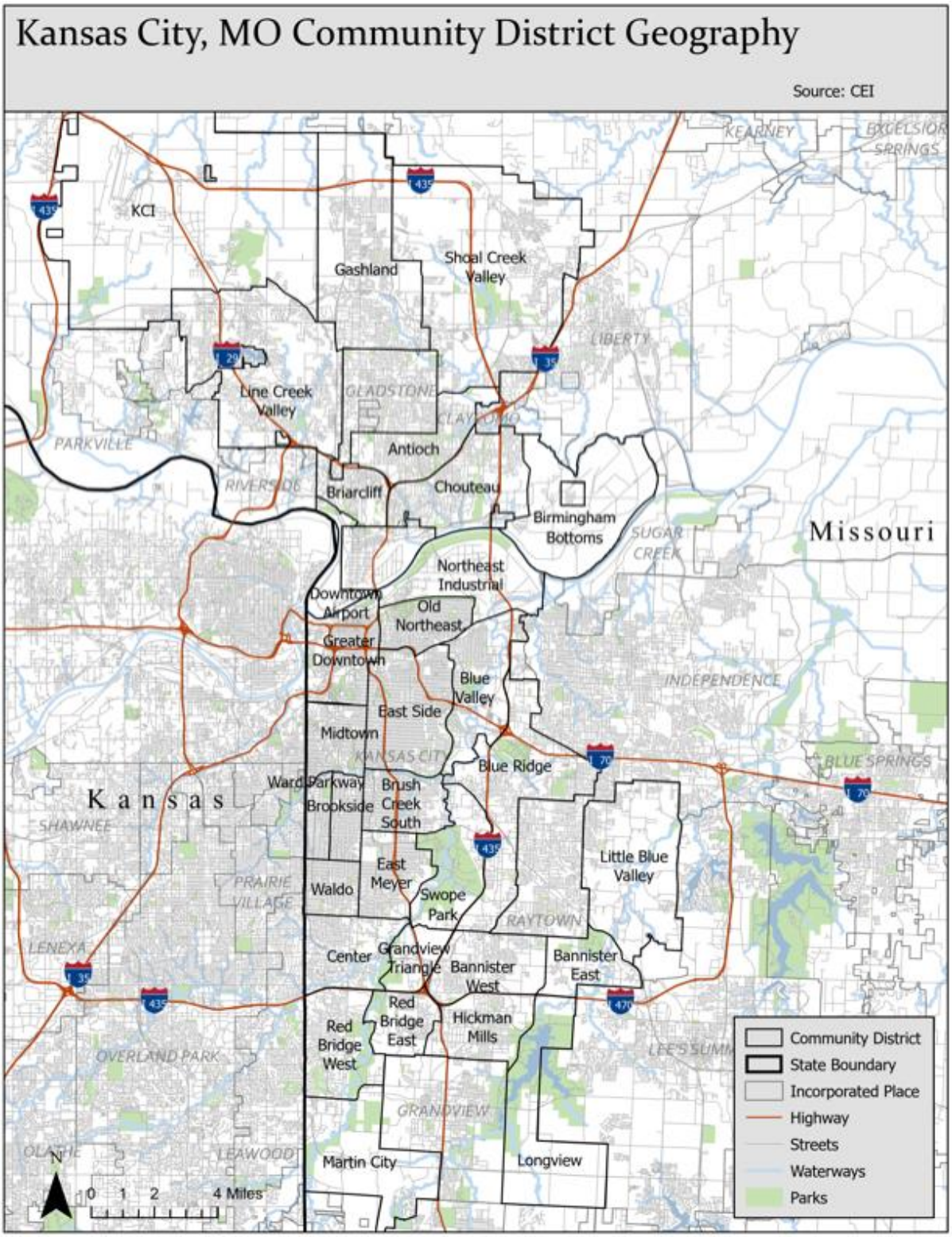
Figure D.1: Geocoding Schema



APPENDIX E

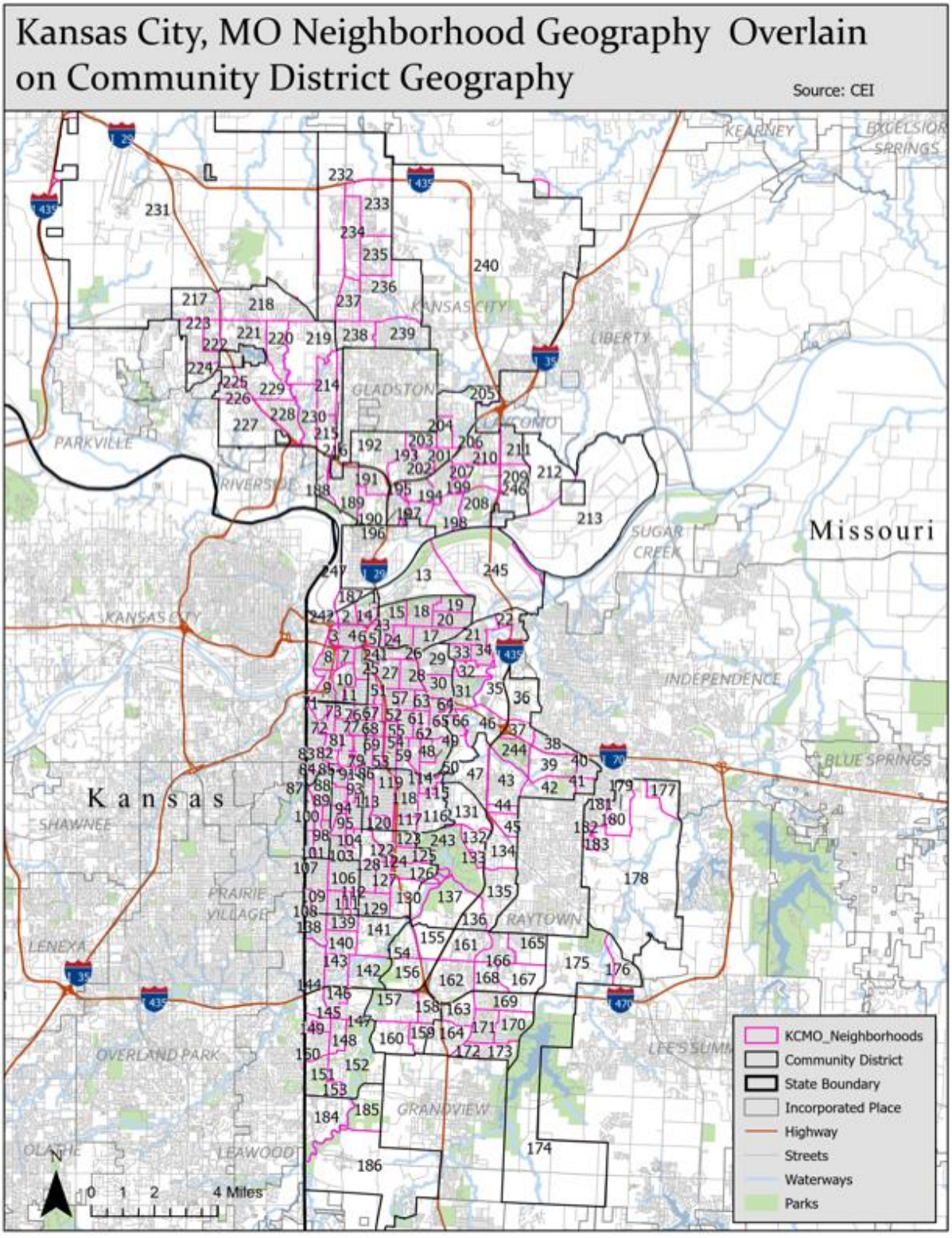
COMMUNITY DISTRICT AND NEIGHBORHOOD GEOGRAPHY

Map E.1: Kansas City, MO Community Districts



Map generated by author.

Map E.2: Neighborhood Geography Overlain on Community District Geography



Map generated by author.

Table E.3: Listing of KCMO Neighborhoods and Corresponding Community Districts

ID Number	Neighborhood Name	Community District
1	Columbus Park Industrial	Greater Downtown
2	River Market	Greater Downtown
3	Quality Hill	Greater Downtown
4	CBD Downtown	Greater Downtown
5	Paseo West	Greater Downtown
6	Hospital Hill	Greater Downtown
7	Crossroads	Greater Downtown
8	Westside North	Greater Downtown
9	Westside South	Greater Downtown
10	Crown Center	Greater Downtown
11	Union Hill	Greater Downtown
12	Longfellow	Greater Downtown
13	Northeast Industrial District	Northeast Industrial
14	Columbus Park	Greater Downtown
15	Pendleton Heights	Old Northeast
16	Independence Plaza	Old Northeast
17	Lykins	Old Northeast
18	Scarritt Point	Old Northeast
19	North Indian Mound	Old Northeast
20	South Indian Mound	Old Northeast
21	Sheffield	Blue Valley
22	North Blue Ridge	Blue Valley
23	Parkview	Old Northeast
24	Forgotten Homes	Old Northeast
25	Beacon Hills	East Side
26	18th & Vine	East Side
27	Wendell Phillips	East Side
28	Washington Wheatley	East Side
29	East 23rd Street PAC North	East Side
30	East 23rd Street PAC South	East Side
31	South Blue Valley	Blue Valley
32	Central Blue Valley	Blue Valley
33	West Blue Valley	Blue Valley
34	East Blue Valley	Blue Valley
35	Blue Valley Industrial	Blue Valley
36	Western Blue Township	Blue Ridge

ID Number	Neighborhood Name	Community District
37	Cunningham Ridge	Blue Ridge
38	Ashland Ridge	Blue Ridge
39	Riss Lake	Blue Ridge
40	Sterling Acres	Blue Ridge
41	Sterling Gardens	Blue Ridge
42	Stayton Meadows	Blue Ridge
43	Eastwood Hills East	Blue Ridge
44	Coachlight Square	Blue Ridge
45	Lewis Heights	Blue Ridge
46	Leeds	Blue Valley
47	Eastwood Hills West	Blue Ridge
48	Vineyard Northwest	East Side
49	Vineyard	East Side
50	Vineyard Estates	Blue Valley
51	Mount Hope	East Side
52	Ivanhoe Northwest	East Side
53	Ivanhoe Southwest	East Side
54	Ivanhoe Southeast	East Side
55	Ivanhoe Northeast	East Side
56	Key Coalition	East Side
57	Santa Fe	East Side
58	Oak Park Northwest	East Side
59	Oak Park Southwest	East Side
60	Oak Park Southeast	East Side
61	Palestine West	East Side
62	Palestine East	East Side
63	South Roundtop	East Side
64	Seven Oaks	East Side
65	Boulevard Village	East Side
66	Dunbar	Blue Valley
67	Center City	East Side
68	Squier Park	East Side
69	Manheim Park	East Side
70	Volker	Midtown
71	Coleman Highlands	Midtown
72	Roanoke	Midtown
73	Valentine	Midtown
74	Hanover Place	Midtown

ID Number	Neighborhood Name	Community District
75	Broadway Gillham	Midtown
76	North Hyde Park	Midtown
77	Central Hyde Park	Midtown
78	South Hyde Park	Midtown
79	Rockhill	Midtown
80	Southmoreland	Midtown
81	Westport	Midtown
82	Plaza Westport	Midtown
83	West Plaza	Midtown
84	Westwood	Midtown
85	Country Club Plaza	Midtown
86	Volker Park	Midtown
87	Sunset Hill West	Ward Parkway
88	Sunset Hill	Ward Parkway
89	Ward Estates	Ward Parkway
90	Countryside	Brookside
91	South Plaza	Brookside
92	Western 49-63	Brookside
93	Crestwood	Brookside
94	Brookside Park	Brookside
95	Morningside	Brookside
96	Wornall Homestead	Brookside
97	Country Club	Ward Parkway
98	Greenway Fields	Ward Parkway
99	Country Club District	Ward Parkway
100	Stratford Gardens	Ward Parkway
101	Romanelli West	Ward Parkway
102	Armour Fields	Ward Parkway
103	Armour Hills	Brookside
104	Oak Meyer Gardens	Brookside
105	Holmes Park	Brookside
106	Tower Homes	Waldo
107	Ward Parkway	Waldo
108	Ward Parkway Plaza	Waldo
109	West Waldo	Waldo
110	Waldo Homes	Waldo
111	Rolling Meadows	Waldo
112	Rockhill Manor	Waldo

ID Number	Neighborhood Name	Community District
113	Eastern 49-63	Brush Creek South
114	Mount Cleveland	Brush Creek South
115	Sheraton Estates	Brush Creek South
116	Swope Parkway-Elmwood	Brush Creek South
117	South Town Fork Creek	Brush Creek South
118	North Town Fork Creek	Brush Creek South
119	Blue Hills	Brush Creek South
120	Citadel	Brush Creek South
121	Neighbors United For Action	East Meyer
122	Blenheim Square Research Hospital	East Meyer
123	Swope Park Campus	East Meyer
124	Self Help Neighborhood Council	East Meyer
125	Foxtown East	East Meyer
126	Noble & Gregory Ridge	East Meyer
127	East Meyer 7	East Meyer
128	East Meyer 6	East Meyer
129	Marlborough Heights/Marlborough Pride	East Meyer
130	Marlborough East	East Meyer
131	Brown Estates	Swope Park
132	Swope Park Ridge-Winchester	Swope Park
133	Strupwood	Swope Park
134	East Swope Highlands	Blue Ridge
135	Park Farms	Blue Ridge
136	Oldham Farms	Blue Ridge
137	Hillcrest	Swope Park
138	Western Hills	Center
139	Santa Fe Hills	Center
140	Boone Hills	Center
141	Legacy East	Center
142	Linden Hills & Indian Heights	Center
143	Willow Creek	Center
144	Lea Manor	Center
145	Country Lane Estates	Red Bridge West
146	Bridlespur	Red Bridge West
147	Red Bridge North	Red Bridge West
148	Red Bridge South	Red Bridge West
149	Foxcroft & Glen Arbor	Red Bridge West
150	Verona Hills	Red Bridge West

ID Number	Neighborhood Name	Community District
151	Blue Hills Estates	Red Bridge West
152	Mission Lake	Red Bridge West
153	Woodbridge	Red Bridge West
154	Hidden Valley	Grandview Triangle
155	Sechrest	Grandview Triangle
156	Oakwood	Grandview Triangle
157	St. Catherines Gardens	Red Bridge East
158	Royal Oaks	Red Bridge East
159	Terrace Lake Gardens	Red Bridge East
160	Calico Farms	Red Bridge East
161	Loma Vista	Bannister West
162	Fairlane	Bannister West
163	Stratford Estates	Hickman Mills
164	Hickman Mills	Hickman Mills
165	White Oak	Bannister West
166	Fairwood & Robandee	Bannister West
167	Robandee South	Bannister West
168	Bannister Acres	Bannister West
169	Hickman Mills South	Hickman Mills
170	Ruskin Hills	Hickman Mills
171	Ruskin Heights	Hickman Mills
172	Crossgates	Hickman Mills
173	Kirkside	Hickman Mills
174	Longview	Longview
175	Highview Estates	Bannister East
176	Unity Ridge	Bannister East
177	Country Valley-Hawthorn Square	Little Blue Valley
178	Little Blue	Little Blue Valley
179	Blue Vue Hills	Little Blue Valley
180	Glen Lake	Little Blue Valley
181	Fairway Hills	Little Blue Valley
182	Woodson Estates	Little Blue Valley
183	Timber Valley	Little Blue Valley
184	Martin City	Martin City
185	Blue Ridge Farms	Martin City
186	Richards Gebaur	Martin City
187	Harlem	Downtown Airport
188	Briarcliff West	Briarcliff

ID Number	Neighborhood Name	Community District
189	Briarcliff & Claymont	Briarcliff
190	River View	Briarcliff
191	Crestview	Briarcliff
192	Davidson	Antioch
193	Antioch Acres	Antioch
194	Chaumiere	Chouteau
195	Colonial Square	Chouteau
196	River Forest	Chouteau
197	Cooley Highlands	Chouteau
198	Chouteau Estates	Chouteau
199	Holiday Hills	Chouteau
200	Winnwood	Chouteau
201	Glenhaven	Antioch
202	Sherwood Estates	Antioch
203	Country Club Estates & Big Shoal	Antioch
204	Foxwoods-Carriage Hills	Antioch
205	Ravenwood-Somerset	Antioch
206	Maple Park West	Chouteau
207	Winnwood Gardens	Chouteau
208	Hill Haven	Chouteau
209	Winnetonka	Chouteau
210	Maple Park	Chouteau
211	Gracemor-Randolph Corners	Chouteau
212	Minneville	Birmingham Bottoms
213	Birmingham Bottoms	Birmingham Bottoms
214	Clayton	Line Creek Valley
215	Lakeview Terrace	Line Creek Valley
216	Tanglewood & Regency North	Line Creek Valley
217	Prairie Point-Wildberry	Line Creek Valley
218	Coves North	Line Creek Valley
219	Barry Harbour	Line Creek Valley
220	Platte Brook North	Line Creek Valley
221	The Coves	Line Creek Valley
222	Platte Ridge	Line Creek Valley
223	Park Forest	Line Creek Valley
224	Royal Oaks North	Line Creek Valley
225	Linden Park	Line Creek Valley
226	Hawthorne & Picture Hills	Line Creek Valley

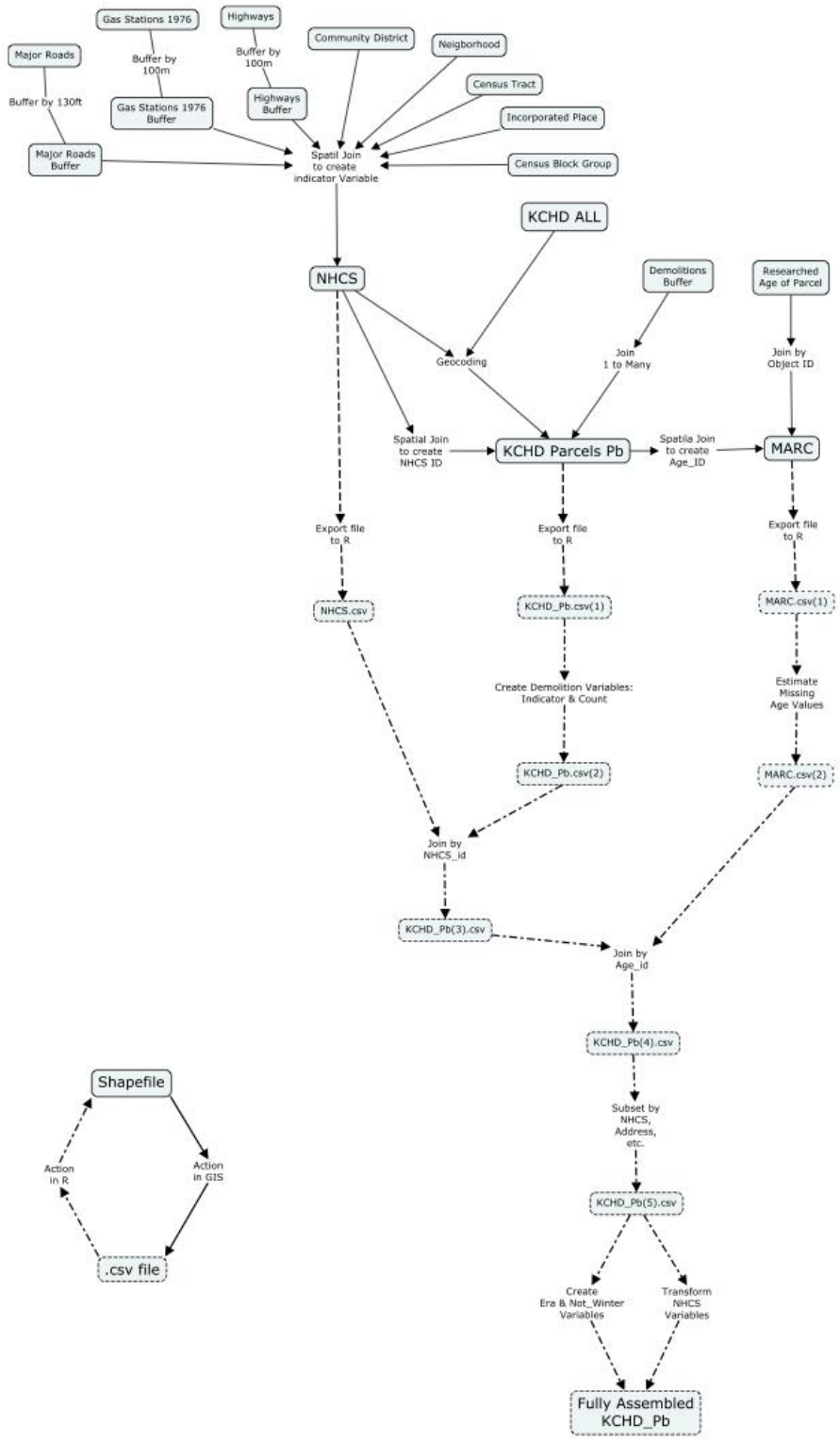
ID Number	Neighborhood Name	Community District
227	Parkdale & Walden	Line Creek Valley
228	Breen Hills	Line Creek Valley
229	Park Plaza	Line Creek Valley
230	Line Creek & Northern Heights	Line Creek Valley
231	KCI & 2nd Creek	KCI
232	Outer Gashland-Nashua	Gashland
233	Nashua	Gashland
234	Meadowbrook Heights	Gashland
235	New Mark	Gashland
236	Gashland	Gashland
237	Sherrydale	Gashland
238	Jefferson Highlands	Gashland
239	Ridgefield	Gashland
240	Shoal Creek	Shoal Creek Valley
241	Area 241	Greater Downtown
242	West Bottoms/CID	Greater Downtown
243	Swope Park	Swope Park
244	Truman Sports Complex	Blue Ridge
245	Area 245	Northeast Industrial
246	Area 246	Chouteau
247	Downtown Airport	Downtown Airport

source: Center for Economic Information

APPENDIX F

DATA ASSEMBLY SCHEMATIC

Figure F.1: Diagram of Data Assembly Process



APPENDIX G

COMPLETE DATA DICTIONARY

Field Name	Filed Type	Description
Status	text	Status of geocoding operation, m – matched, u – unmatched
Match_type	text	Type of geocoding match, Automatic or manual match
Collect_Da	date	Date on which BPb sample was taken m/d/y
DOB	date	Done on which child was born m/d/y
DCN	numeric	Department Client Number. Unique Identifier: 8-digits
SEX	text	Sex of child
STREET_ADD	text	Street address, single field
CITY	text	City of address
STATE	text	State of address
ZIP_PLUS_4	numeric	Zip plus 4 of address
JURISDICTI	text	Jurisdiction of address: Health Department
COUNTY	text	County of Address: Clay, Jackson, Platte
COUNTY_FIP	numeric	Numeric code of County: 47, 95, 165
TEST_RESUL	continuous	Measured BPb level, test result in micrograms per deciliter (µg/dL)
AGE_AT_TES	continuous	Age of child in months
SAMPLE_TYP	text	Type of blood lead test: Venous, Capillary, or Unknown
SC_UOM	text	Unit of measure of blood test: micrograms per deciliter (µg/dL)
Year	numeric	year of test result
Program	numeric	NHCS program of related to housing
NHCS_id	numeric	Primary Key for all NHCS parcels
Age_id.x	numeric	Primary Key for all Date of Housing construction parcels
Residentia	text	Indicate if demolished home was residential or non-residential
Housing_Un	numeric	Number of housing units destroyed in demolition activity
Single_Fam	text	Indicates type of structure demolished
Permit_Dat	date	Date demolition permit granted
Year_1	numeric	Year of BPb test, 0-12
DEMO	indicator	Observed Demolitions, 1 - any demolition activity w/in 100m of home during lifetime of the child, 0 – no demolitions w/in 100m of home during lifetime of child
N_DEMO	continuous	Observed Demolitions, count of demolition activity w/in 100m of home during lifetime of the child
MONTH	date	Month of demolition activity
PID	numeric	Foreign Key from City/County (like kiva PIN, not unique)
PIDSRC	numeric	Unclear from Support Materials
STRNO	numeric	Address Field: Street Number

Field Name	Filed Type	Description
STRDIR	text	Address Field: Street direction
STRTYPE	text	Address Field: Street Type
LANDUSE	text	Description of Land Use from Government Agency
PRGM	numeric	Unclear from Support Materials
YEAR.y	numeric	Year of Housing Survey
INSPDATE	date	Date of Housing Condition Survey for which results are below
VISADDR	Indicator	Indicates if parcel has a visible address, 1 – yes, 2 – n0
STYPE	numeric	Index variable describing type of structure on parcels, n =6
UTYPE	numeric	Index variable describing use type of structure on parcel, n=5
RESTYPE	numeric	Index variable describing number and type of residencies, n=6
SPROFILE	numeric	Index variable describing the number of floors of structure on parcel
ROOF	ordered	Roof Condition
FOUNDATN	ordered	Condition of Foundation and Walls
WINDOORS	ordered	Condition of Windows and Doors
PORCH	ordered	Condition of Porches
EXTPAINT	ordered	Condition of Exterior Paint
PVTWALKS	ordered	Condition of Private Sidewalks and Driveways
LAWN	ordered	Condition of the Lawn and Shrubs
VEHICLES	ordered	Status of Vehicles Parked on the Property
LITTER	ordered	Assessment of Litter on the Property
OPENSTRG	ordered	Assessment of Open Storage on the Property
ACCSTRUC	ordered	Condition of Accessory Structures
PUBWALKS	ordered	Condition of Public Sidewalks
CURB	ordered	Condition of Curbs Adjacent to the Property
STRLITES	ordered	Condition of the Street lights Adjacent to the Property
CATCHBSN	ordered	Condition of the Catch basins Adjacent to the Property
STRTCON	ordered	Condition of the Street Adjacent to the Property
PID_updt	numeric	updated PID field
PRGM_PID	numeric	Primary Key for all NHCS parcels
Gas_prox	indicator	Soil Proxy, 1– Home w/in 100m of old gas station, 0 – home not w/in 100m of old gas station
Major_rd_p	indicator	Soil Proxy, 1– Home w/in 130ft of major roadway, 0 - home not in proximity of selected proximity variables

Field Name	Filed Type	Description
Highway_pr	indicator	Soil Proxy, 1– Home w/in 100m of highway a, 0 - home not in proximity of selected proximity variables
Place_NAME	text	Incorporated place, Name of Incorporated place of residence, n = 5
nbr_Name	text	Neighborhood, Name of neighborhood of residence, n = 182
nbr_id	numeric	Unique Id for neighborhood of residence, n =182
CD_ID	numeric	Unique id for community district of residence, n=25
CD_NAME	text	Community District, name of community district of residence, n = 25
TRACTCE	numeric	Unique id for US census tract of residence, n = 131
BG_GEOID	numeric	Unique ID for US census block group of residence, n = 330
Shape_Leng	numeric	GIS Field: Length of Parcel Perimeter on which Home is Located
Shape_Area	numeric	GIS Field: Area of the Parcel on which House is Located
Age_id.y	numeric	Primary Key for all Date of Housing construction parcels
PCL_BLT	numeric	Year of construction for parcel on structure, MARC record
P_age	numeric	Year of construction for structure on parcel, MARC, research and estimate combined
P_B_R	numeric	Era of housing construction for PCL_BLT, 1 – pre-1952, 2 – 1952-1977, 3 – 1978 or later
P_age_1	numeric	Era of housing construction for P_age field, 1 – pre-1952, 2 – 1952-1977, 3 – 1978 or later
Y_T	numeric	?
YEAR	numeric	Year of BPb test, 2000-2013
ZIP	numeric	Zip code of residence
Y	text	Year of BPb test, 2000-2013
SEASON	text	Season of BPb test, ‘winter’ or ‘not winter’
months	continuous	Age of child in months (9-72)
months2	continuous	Age of child in months squared (81-5184)
months3	continuous	Age of child in months (729-373248)
prox	continuous	Count of soil proxies close to residence. w/in 100m of highway, 100m of old gas station, 130ft of major roadway
P	dichotomous	Soil Proximity, 1– Home w/in 100m of highway and/or 100m of old gas station, and/or 130ft of major roadway, 0 - home not in proximity of selected proximity variables

Field Name	Filed Type	Description
Roofpass	dichotomous	Exterior condition of roof, 1 – ‘good’ or ‘excellent’, 0 – all others
RoofSub	dichotomous	Exterior condition of roof, 1 – ‘substandard’, 0 – all others
RoofSevDet	dichotomous	Exterior condition of roof, 1 – ‘Severely deteriorated’ or ‘Seriously deteriorated’, 0 – all others
FoundationPass	dichotomous	Exterior condition of foundation and walls, 1 – ‘good’ or ‘excellent’, 0 – all others
FoundationSub	dichotomous	Exterior condition of foundation and walls, 1 – ‘substandard’, 0 – all others
FoundationSevDet	dichotomous	Exterior condition of foundation and walls, 1 – ‘Severely deteriorated’ or ‘Seriously deteriorated’, 0 – all others
wd_pass	dichotomous	Exterior condition of windows and doors, 1 – ‘good’ or ‘excellent’, 0 – all others
wd_sub	dichotomous	Exterior condition of windows and doors, 1 – ‘substandard’, 0 – all others
wd_sevdet	dichotomous	Exterior condition of windows and doors, 1 – ‘Severely deteriorated’ or ‘Seriously deteriorated’, 0 – all others
porch_pass	dichotomous	Exterior condition of porch, 1 – ‘good’ or ‘excellent’, 0 – all others
porch_sub	dichotomous	Exterior condition of porch, 1 – ‘substandard’, 0 – all others
porch_sevdet	dichotomous	Exterior condition of porch, 1 – ‘Severely deteriorated’ or ‘Seriously deteriorated’, 0 – all others
ep_passs	dichotomous	Exterior condition of exterior paint, 1 – ‘good’ or ‘excellent’, 0 – all others
ep_sub	dichotomous	Exterior condition of exterior paint, 1 – ‘substandard’, 0 – all others
ep_sevdet	dichotomous	Exterior condition of exterior paint, 1 – ‘Severely deteriorated’ or ‘Seriously deteriorated’, 0 – all others
male	dichotomous	Sex of child, 1 – male, 0 – all others
female	dichotomous	Sex of child, 1 – female, 0 – all others
unk_sex	dichotomous	Sex of child, 1 – unknown, 0 – all others
capillary	dichotomous	Blood test type, 1– capillary exam (finger poke), 0 – all others
unk_sample	dichotomous	Blood test type, 1– unknown test type, 0 – known test type
venous	dichotomous	Blood test type, 1– venous blood draw, 0 – all others
not_winter	dichotomous	Season of test, 1 - BPb test took place between April and November, 0 - BPb test too place December to March

Field Name	Filed Type	Description
in_prox	dichotomous	Soil Proxy, 1– Home w/in 100m of highway and/or 100m of old gas station, and/or 130ft of major roadway, 0 - home not in proximity of selected proximity variables
ERA	continuous	Era of housing construction, 1 – pre-1952, 2 – 1952-1977, 3 – 1978 or later
demo	dichotomous	Observed Demolitions, 1 - any demolition activity w/in 100m of home during lifetime of the child, 0 – no demolitions w/in 100m of home during lifetime of child

source: Generated by author in data assembly and geocoding processes.

APPENDIX H

MODEL PRESENTED TO EPA REGION 7 PEDIATRIC LEAD POISONING PREVENTION SUMMIT, SEPTEMBER 5, 2019

$$BPb_i = \alpha + \sum_{h=1}^{n_h} \beta_h C_{hi} + \sum_{k=9}^{n_k} \beta_k H_{ji} + \sum_{l=21}^{n_l} \beta_l P_{li} + \epsilon_i$$

- BPb_i is a *n* x 1 vector of blood lead levels.
- C_{hi} is a *n* x *h* matrix of child focused variables.
- H_{ji} is a *n* x *k* matrix of housing focused variables.
- P_{li} is a *n* x *l* matric of proximity focused variables.
- β_h, β_k, β_l are *h* x 1, *k* x 1, and *l* x 1 vectors of child focused, housing focused, and proximity focused regression coefficients.
- ϵ_i is a normally distributed random disturbance with mean=0 and var=σ^2.

1. Child Focused Variables: Year of Test, Sex, Sample Type
2. Housing Focused Variables: Age of Variables, Condition of Parcels
3. Proximity Focused Variables: Street Type, 1976 Gas Stations, Demolitions

Table H.1: Preliminary Model Without Child Focused or Proximity Variables

Part	Coefficient	Estimate	Std. Error	t value	Pr(>|t|)	
	(Intercept)	6.67297	1.36696	4.882	1.08E-06	***
1	TEST after 2005					
1	FEMALE					
1	SAMPLE TYPE UNKNOWN					
1	SAMPLE TYPE VENOUS 2					
2	Built 1951-1977					
2	Built After 1977					
2	ROOF (2)	1.21257	0.55656	2.179	0.029391	*
2	ROOF (3)	0.12133	0.54341	0.223	0.823329	
2	ROOF (4)	0.1982	0.54486	0.364	0.716043	
2	ROOF (5)	0.51411	0.55949	0.919	0.358188	
2	FOUNDATION (2)	-0.36781	0.89541	-0.411	0.681253	
2	FOUNDATION (3)	0.14515	0.85024	0.171	0.864451	
2	FOUNDATION (4)	0.03905	0.84586	0.046	0.963184	
2	FOUNDATION (5)	0.31535	0.85085	0.371	0.710921	
2	WINDOWS & DOORS (2)	-0.26859	0.83503	-0.322	0.747722	
2	WINDOWS & DOORS (3)	0.17547	0.77352	0.227	0.820554	
2	WINDOWS & DOORS (4)	0.3566	0.77025	0.463	0.643406	
2	WINDOWS & DOORS (5)	0.01432	0.77727	0.018	0.985298	
2	PORCH(2)	-1.89128	1.04199	-1.815	0.069559	.
2	PORCH(3)	-2.20275	1.0256	-2.148	0.031767	*
2	PORCH(4)	-2.50998	1.02535	-2.448	0.014393	*
2	PORCH(5)	-2.54877	1.0308	-2.473	0.013437	*
2	EXTERIOR PAINT (2)	-1.00988	0.48287	-2.091	0.036528	*
2	EXTERIOR PAINT (3)	-1.731	0.4555	-3.8	0.000146	***
2	EXTERIOR PAINT (4)	-1.85453	0.45647	-4.063	4.90E-05	***
2	EXTERIOR PAINT (5)	-1.97649	0.47089	-4.197	2.74E-05	***
3	DEMO					
3	Gas Station proximity					
3	Major Road proximity					
3	Highway proximity					

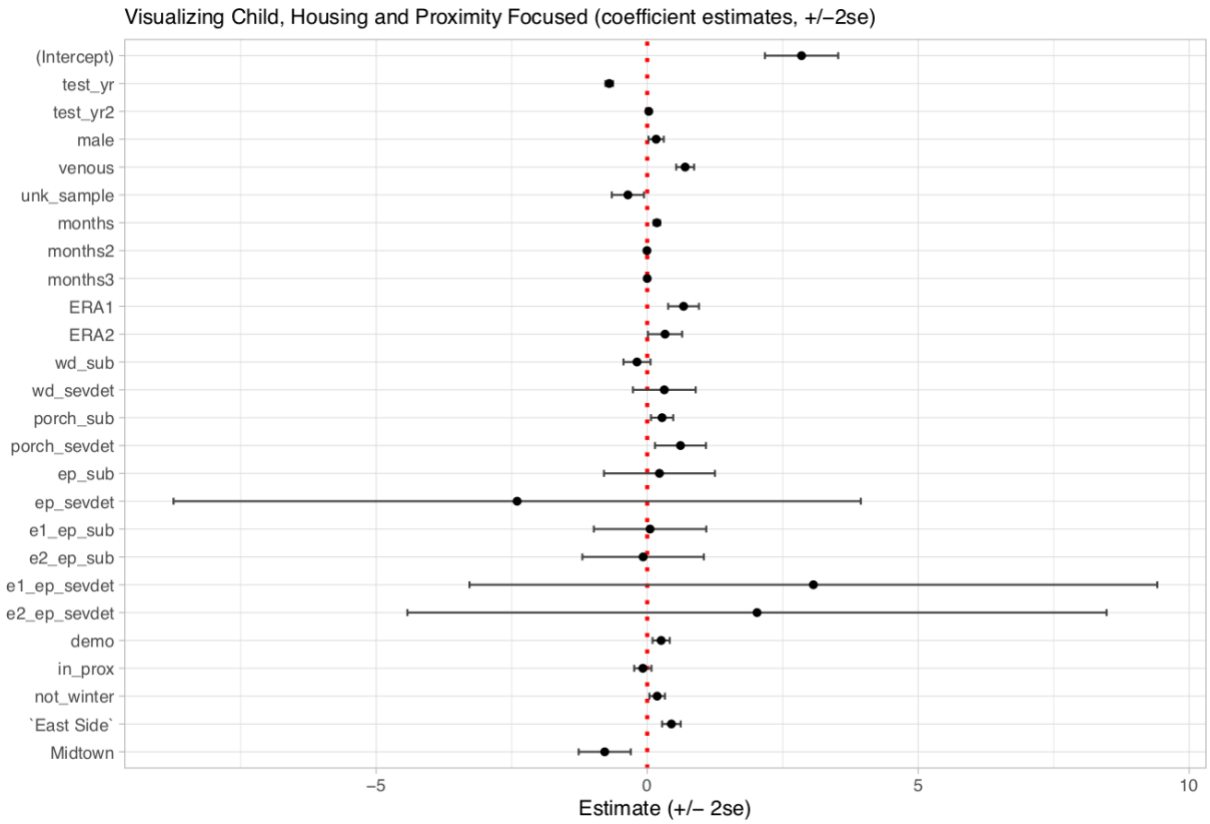
Table H.2: Completely Specified Preliminary Model

Part	Coefficient	Estimate	Std. Error	t value	Pr(>|t|)	
	(Intercept)	9.95023	1.56771	6.347	2.35E-10	***
1	TEST after 2005	-2.32656	0.09515	-24.452	<2e-16	***
1	FEMALE	-0.13998	0.08057	-1.737	0.082369	.
1	SAMPLE TYPE UNKNOWN	-0.19547	0.1681	-1.163	0.244949	
1	SAMPLE TYPE VENOUS	0.64474	0.0918	7.024	2.38E-12	***
2	Built 1951-1977	-0.72165	0.11402	-6.329	2.64E-10	***
2	Built After 1977	-0.73662	0.16286	-4.523	6.20E-06	***
2	ROOF (2)	0.3326	0.59093	0.563	0.573566	
2	ROOF (3)	-0.17569	0.579	-0.303	0.761562	
2	ROOF (4)	-0.07961	0.58003	-0.137	0.890831	
2	ROOF (5)	-0.02722	0.5917	-0.046	0.963305	
2	FOUNDATION (2)	-1.32694	0.96099	-1.381	0.167387	
2	FOUNDATION (3)	-1.17316	0.92231	-1.272	0.203424	
2	FOUNDATION (4)	-1.40941	0.91758	-1.536	0.124587	
2	FOUNDATION (5)	-1.17725	0.92206	-1.277	0.20173	
2	WINDOWS & DOORS (2)	-0.3531	0.82926	-0.426	0.670268	
2	WINDOWS & DOORS (3)	-0.33879	0.76298	-0.444	0.657028	
2	WINDOWS & DOORS (4)	-0.01058	0.75919	-0.014	0.988885	
2	WINDOWS & DOORS (5)	-0.30742	0.76597	-0.401	0.688176	
2	PORCH(2)	-2.85759	1.18128	-2.419	0.015588	*
2	PORCH(3)	-3.15662	1.166	-2.707	0.006803	**
2	PORCH(4)	-3.2977	1.16522	-2.83	0.004668	**
2	PORCH(5)	-3.30402	1.16972	-2.825	0.004748	**
2	EXTERIOR PAINT (2)	-0.13209	0.47037	-0.281	0.778857	
2	EXTERIOR PAINT (3)	-0.48987	0.443	-1.106	0.268856	
2	EXTERIOR PAINT (4)	-0.72929	0.44345	-1.645	0.100108	
2	EXTERIOR PAINT (5)	-0.91828	0.4577	-2.006	0.044864	*
3	DEMO	0.36507	0.10421	3.503	0.000463	***
3	Gas Station proximity	0.32957	0.21311	1.546	0.122039	
3	Major Road proximity	0.01654	0.10718	0.154	0.877368	
3	Highway proximity	-0.32445	0.25265	-1.284	0.199111	

APPENDIX I

VISUALIZING COEFFICIENT ESTIMATES AND STANDARD ERRORS

Figure I.1: Coefficient Estimates



APPENDIX J

PEARSON CORRELATION MATRIX FOR EQUATION 7.2

Figure J.1: Pearson Correlation Matrix for Equation 7.2

	TEST_RESUL	test_yr	months	male	unk_sample	venous	ERA1	ERA2	wd_sub	wd_sevdet	porch_sub	porch_sevdet
TEST_RESUL	1											
test_yr	-0.32907	1										
months	-0.00482	-0.05115	1									
male	0.02274	0.00267	0.00446	1								
unk_sample	-0.02135	-0.13093	-0.045	-0.00452	1							
venous	0.03112	0.22058	-0.15893	0.026	-0.35518	1						
ERA1	0.10825	-0.01777	0.00391	-0.01454	-0.02966	0.01995	1					
ERA2	-0.06728	0.01534	-0.02006	0.00322	0.0296	-0.00286	-0.79315	1				
wd_sub	0.03944	0.06467	-0.00099	-0.00522	-0.02474	0.00698	0.17316	-0.13398	1			
wd_sevdet	0.04503	0.00222	0.00815	0.00601	-0.0066	0.02164	0.06809	-0.05027	-0.04959	1		
porch_sub	0.05746	0.06677	0.00136	-0.01424	-0.01886	0.02986	0.20016	-0.14576	0.38949	0.04771	1	
porch_sevdet	0.07521	-0.05536	0.00765	0.01286	0.0065	-0.00314	0.09402	-0.07416	0.11091	0.26933	-0.08339	1
ep_sub	0.03691	0.10381	0.00485	0.00129	-0.01627	0.03511	0.18207	-0.11475	0.32856	0.03289	0.34053	0.07233
ep_sevdet	0.0906	-0.09319	-0.00549	-0.01356	-0.0231	-0.00115	0.09803	-0.06587	0.11815	0.22497	0.07615	0.25022
e1_ep_sub	0.05153	0.09411	0.00614	0.00113	-0.02498	0.04309	0.32413	-0.25709	0.3393	0.04271	0.35168	0.08924
e2_ep_sub	-0.0345	0.05716	-0.00701	-0.00086	0.00518	0.00346	-0.28534	0.35975	-0.00765	-0.01677	0.00366	-0.02878
e3_ep_sub	0.00482	-0.04714	0.0101	0.00331	0.03461	-0.04515	-0.11931	-0.03392	0.04716	-0.00942	0.02162	-0.01204
e1_ep_sevdet	0.09618	-0.08918	-0.00476	-0.01477	-0.01908	-0.00033	0.13136	-0.10419	0.1203	0.22664	0.06697	0.2554
e2_ep_sevdet	-0.00694	-0.0231	-0.00065	0.0046	-0.01657	-0.0046	-0.10027	0.12642	0.00883	0.02403	0.04319	0.00252
e3_ep_sevdet	-0.00348	-0.01704	-0.01408	-0.01143	-0.00307	0.00865	-0.01859	-0.00528	-0.00419	-0.00147	-0.00551	0.06604
demo	0.06979	0.03032	0.01413	0.00286	-0.06008	0.05725	0.28843	-0.25076	0.10608	0.03513	0.10281	0.05748
in_prox	0.00137	-0.08587	0.01191	-0.00114	0.00334	0.00764	-0.13576	0.05461	-0.02641	0.00732	-0.07456	-0.0299
not_winter	0.01366	0.0179	0.05453	-0.02247	0.01698	-0.0632	0.04483	-0.05455	0.00959	0.00197	-0.00095	-0.00287
East Side	0.07841	-0.07572	0.03092	-0.00861	-0.00947	0.04004	0.08823	-0.08509	0.02923	0.01684	0.03397	0.06718
Midtown	0.02514	-0.15951	-0.02834	-0.00174	0.00468	-0.10269	0.08392	-0.06759	-0.0437	0.00463	-0.04422	0.00361

	ep_sub	ep_sevdet	e1_ep_sub	e2_ep_sub	e3_ep_sub	e1_ep_sevdet	e2_ep_sevdet	e3_ep_sevdet	demo	in_prox	not_winter	East Side	Midtown
ep_sub	1												
ep_sevdet	-0.13552	1											
e1_ep_sub	0.91314	-0.12375	1										
e2_ep_sub	0.28809	-0.03904	-0.09249	1									
e3_ep_sub	0.12047	-0.01633	-0.03867	-0.0122	1								
e1_ep_sevdet	-0.1301	0.95999	-0.1188	-0.03748	-0.01567	1							
e2_ep_sevdet	-0.03559	0.26263	-0.0325	-0.01025	-0.00429	-0.01317	1						
e3_ep_sevdet	-0.0066	0.04869	-0.00602	-0.0019	-0.00079	-0.00244	-0.00067	1					
demo	0.10275	0.0371	0.14977	-0.08062	-0.05984	0.05342	-0.05371	0.00792	1				
in_prox	-0.06986	-0.02002	-0.05727	-0.05875	0.04336	-0.01869	-0.00599	-0.00705	-0.0107	1			
not_winter	0.02122	0.0193	0.0177	0.01002	0.00332	0.01835	0.00833	-0.01326	-0.02038	-0.01945	1		
East Side	0.01803	0.04891	0.03685	-0.04837	0.0072	0.04456	0.02286	-0.00641	0.20125	0.04342	-0.00043	1	
Midtown	-0.05885	0.01695	-0.04923	-0.02658	-0.01112	0.01645	-0.00934	0.0715	-0.03419	0.03294	-0.01173	-0.08964	1

REFERENCES

"10 Policies to Prevent and Respond to Childhood Lead Exposure An Assessment of the Risks Communities Face and Key Federal, State, and Local Solutions." 2017. Health Impact Project. Robert Wood Johnson Foundation, The PEW charitable trusts. heathimpactproject.org.

Advisory Committee on Childhood Lead Poisoning Prevention. 2012. "Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention." Report. Centers for Disease Control and Prevention.

AECF. 2011. "Responsible Demolition: A Baltimore Case Study with National Implications." East Baltimore Redevelopment Initiative: Responsible Development. Baltimore, Md.: Annie E. Casey Foundations. www.aecf.org.

Agee, Mark D., and Thomas D Crocker. 1996a. "Parental Altruism and Child Lead Exposure." *Journal of Human Resources* 31 (3): 677–91.

———. 1996b. "Parents' Discount Rates for Child Quality." *Southern Economic Journal* 63 (1): 36–50.

Aizer, Anna, Janet Currie, Peter Simon, and Patrick Vivier. 2016. "Do Low Levels of Blood Lead Reduce Children's Future Test Scores?" National Bureau of Economic Research. http://www.nber.org/papers/w22558.

Akkus, Cem, and Esra Ozdenerol. 2014. "Exploring Childhood Lead Exposure through GIS: A Review of the Recent Literature." *International Journal of Environmental Research and Public Health* 11 (6): 6314–34. https://doi.org/10.3390/ijerph110606314.

American Academy of Pediatrics. 2016. "Lead Exposure in Children." 2016. https://www.aap.org/en-us/advocacy-and-policy/aap-health-initiatives/lead-exposure/Pages/Lead-Exposure-in-Children.aspx.

Anderson, Elizabeth S. 1999. "What Is the Point of Equality?" *Ethics* 109 (2): 287–337. https://doi.org/10.1086/233897.

Anselin, Luc. 1999. "Spatial Econometrics." University of Texas at Dallas. http:www.csiss.ncgia.ucsb.edu/aboutus/presentations/files/baltchap.pdf.

Armbruster, David A., and Terry Pry. 2008. "Limit of Blank, Limit of Detection and Limit of Quantitation." *The Clinical Biochemist. Reviews* 29 Suppl 1 (August): S49-52.

Arrow, Kenneth. 1963. "Uncertainty and the Welfare Economics of Medical Care." *The American Economic Review* 53 (5): 941–73.

ATSDR. 2017. "Lead Toxicity." Case Studies in Environmental Medicine. Agency for Toxic Substances and Disease Registry. https://www.atsdr.cdc.gov/csem/lead/docs/CSEM-Lead_toxicity_508.pdf.

Ayres, C.E. 1962. "Introduction." In *Theory of Economic Progress,* 2nd ed., v–xxv. New York: Schocken Books.

Baggetta, Matthew, and David M. Bredenkamp. 2019. "Systematic Social Observation in the Study of Civil Society Organizations." *Sociological Methods & Research*, April, 004912411982614. https://doi.org/10.1177/0049124119826148.

Bailey, Adrian J., James D. Sargent, and Megan K. Blake. 1998. “A Tale of Two Counties: Childhood Lead Poisoning, Industrialization, and Abatement in New England.” *Economic Geography*, 96–111.

Banzhaf, Spencer, Lala Ma, and Christopher Timmins. 2019. “Environmental Justice: The Economics of Race, Place, and Pollution.” *Journal of Economic Perspectives* 33 (1): 185–208. https://doi.org/10.1257/jep.33.1.185.

Bashir, Samiya A. 2002. “Home Is Where the Harm Is: Inadequate Housing as a Public Health Crisis.” *American Journal of Public Health* 92 (5): 733–38. https://doi.org/10.2105/AJPH.92.5.733.

Baylis, F., N. P. Kenny, and S. Sherwin. 2008. “A Relational Account of Public Health Ethics.” *Public Health Ethics* 1 (3): 196–209. https://doi.org/10.1093/phe/phn025.

Beale, Linda, Juan Jose Abellan, Susan Hodgson, and Lars Jarup. 2008. “Methodologic Issues and Approaches to Spatial Epidemiology.” *Environmental Health Perspectives* 116 (8): 1105–10. https://doi.org/10.1289/ehp.10816.

Bellinger, David C. 2008. “Lead Neurotoxicity and Socioeconomic Status: Conceptual and Analytical Issues.” *NeuroToxicology* 29 (5): 828–32. https://doi.org/10.1016/j.neuro.2008.04.005.

Bellinger, David, Herbert L. Needleman, Richard Bromfield, and Maureen Mintz. 1984. “A Followup Study of the Academic Attainment and Classroom Behavior of Children with Elevated Dentine Lead Levels.” *Biological Trace Element Research* 6 (3): 207–23.

Berney, Barbara. 1993. “Round and Round It Goes: The Epidemiology of Childhood Lead Poisoning, 1950-1990.” *The Milbank Quarterly* 71 (1): 3. https://doi.org/10.2307/3350273.

Beyer, Kirsten. 2016. “The Lessons of Dr. John Snow: A Call for Translational Health Geography.” In *Research Advances in Behavioral, Economic and Health Geography Inspired by Gerard Rushton*, edited by Alan Phipps, 85–97. University of Windsor.

Billick, I H, A S Curran, and D R Shier. 1979. “Analysis of Pediatric Blood Lead Levels in New York City for 1970-1976.” *Environmental Health Perspectives* 31 (August): 183–90. https://doi.org/10.1289/ehp.7931183.

Billick, I. H., A. S. Curran, and D. R. Shier. 1980. “Relation of Pediatric Blood Lead Levels to Lead in Gasoline.” *Environmental Health Perspectives* 34 (February): 213–17.

Billings, Stephen B., and Kevin T. Schnepel. 2017a. “Life After Lead: Effects of Early Interventions for Children Exposed to Lead.” Working Paper. https://dl.dropboxusercontent.com/content_link/dtDxEJhPZsiwWtV3tCqpXK6yukvN0tj1sthqXeFdwMsOjREFKwrrUlJACXAGwVN9/file?dl=1.

———. 2017b. “The Value of a Healthy Home: Lead Paint Remediation and Housing Values.” *Journal of Public Economics* 153: 69–81.

Binder, Sue, and Henry Falk. 1991. “Strategic Plan for the Elimination of Childhood Lead Poisoning.” Reports-Descriptive ED 344 700 PS 020 537. Atlanta, Georgia: Center For Disease Control.

Binns, H. J., C. Campbell, M. J. Brown, and for the Advisory Committee on Childhood Lead Poisoning Prevention. 2007. “Interpreting and Managing Blood Lead Levels of Less Than 10 g/DL in Children and Reducing Childhood Exposure to Lead: Recommendations of the Centers for Disease Control and Prevention Advisory

Committee on Childhood Lead Poisoning Prevention." *PEDIATRICS* 120 (5): e1285–98. https://doi.org/10.1542/peds.2005-1770.

Black, Harvey. 2005. "Getting the Lead out of Electronics." *Environmental Health Perspectives* 113 (10): A682.

Boreland, Frances, David Lyle, Anthony Brown, and David Perkins. 2015. "Effectiveness of Introducing Point of Care Capillary Testing and Linking Screening with Routine Appointments for Increasing Blood Lead Screening Rates of Young Children: A before-after Study." *Archives of Public Health* 73 (1). https://doi.org/10.1186/s13690-015-0111-y.

Boulding, Kenneth E. 1969. "Economics as a Moral Science." *The American Economic Review* 59 (1): 1–12.

Bowles, Doug. 2000. "Mapping Community Development in Kasnas City-Summary Report." 9902. MSCDC Economic Report. Kansas City, MO: Center For Economic Information, University of Missouri-Kansas City.

———. 2005. "Bowles, Douglas Henri. Geographic Information Systems-Based Analysis of Metropolitan Development, Decline, and Recovery." Kansas City, MO: Univeristy of Missouri-Kansas City.

Bowles, Doug, and Peter Eaton. 2010. "New Partner Presentation." PDF slideshow presented at the NNIP Partnership Meeting, Washington D.C., May.

Braveman, Paula, and Laura Gottlieb. 2014. "The Social Determinants of Health: It's Time to Consider the Causes of the Causes." *Public Health Reports* 129 (1_suppl2): 19–31. https://doi.org/10.1177/00333549141291S206.

Bress, W.C., and J.H. Bidanset. 1991. "Percutaneous in Vivo and in Vitro Absorption of Lead." *Veterinary and Human Toxicology* 33 (3): 212–14.

Breysse, Jill, Sherry Dixon, Jonathan Wilson, Carol Kawecki, Rodney Green, Janet Phoenix, Warren Galke*, and Scott Clark. 2008. "Selecting a Lead Hazard Control Strategy Based on Dust Lead Loading and Housing Condition: II. Application of Housing Assessment Tool (HAT) Modeling Results." *Journal of Occupational and Environmental Hygiene* 5 (8): 540–45. https://doi.org/10.1080/15459620802222587.

Briant, A, P. P. Combes, and M. Lafourcade. 2010. "Dots to Boxes: Do the Size and Shape of Spatial Units Jeopardize Economic Geography Estimations?" *Journal of Urban Economics* 67: 287–302.

Briggs, Vernon M. 1999. "Economics, Ethics, and Public Policy." *Journal of Economic Issues* 33 (1): 210–11. https://doi.org/10.1080/00213624.1999.11506150.

Brubaker, Christopher J., Vincent J. Schmithorst, Erin N. Haynes, Kim N. Dietrich, John C. Egelhoff, Diana M. Lindquist, Bruce P. Lanphear, and Kim M. Cecil. 2009. "Altered Myelination and Axonal Integrity in Adults with Childhood Lead Exposure: A Diffusion Tensor Imaging Study." *NeuroToxicology* 30 (6): 867–75. https://doi.org/10.1016/j.neuro.2009.07.007.

Buchanan, James M. 1987. "What Should Economists Do." In *Economics: Between Predictive Science and Moral Philosophy*, 1st ed, 21–34. Texas A & M University Economics Series, no. 7. College Station, [TX]: Texas A & M University Press.

Buchanan, James M., and Warren J. Samuels. 1975. "On Some Fundamental Issues in Political Economy: An Exchange of Correspondence." *Journal of Economic Issues* 9 (1): 15–38. https://doi.org/10.1080/00213624.1975.11503251.

"Building and Operating Neighborhood Indicator Systems: A Guidebook." 1999. Washington D.C.: The Urban Institute.

Burdé, Brigitte de la, and McLin S. Choate. 1972. "Does Asymptomatic Lead Exposure in Children Have Latent Sequelae?" *The Journal of Pediatrics* 88 (6): 1088–91.

Byers, Randolph K., and Elizabeth E. Lord. 1943. "Late Effects of Lead Poisoning on Mental Development." *American Journal of Diseases of Children* 66 (5): 471–94.

Caldwell, Kathleen L., Po-Yung Cheng, Jeffery M. Jarrett, Amir Makhmudov, Kathryn Vance, Cynthia D. Ward, Robert L. Jones, and Mary E. Mortensen. 2017. "Measurement Challenges at Low Blood Lead Levels." *Pediatrics* 140 (2): e20170272. https://doi.org/10.1542/peds.2017-0272.

Canfield, Richard L., Charles R. Henderson Jr, Deborah A. Cory-Slechta, Christopher Cox, Todd A. Jusko, and Bruce P. Lanphear. 2003. "Intellectual Impairment in Children with Blood Lead Concentrations below 10 Mg per Deciliter." *New England Journal of Medicine* 348 (16): 1517–26.

Carpenter, David O., and Rick Nevin. 2010. "Environmental Causes of Violence." *Physiology & Behavior* 99 (2): 260–68. https://doi.org/10.1016/j.physbeh.2009.09.001.

Caughy, Margaret O, Patricia J O'Campo, and Jacqueline Patterson. 2001. "A Brief Observational Measure for Urban Neighborhoods." *Health & Place* 7 (3): 225–36. https://doi.org/10.1016/S1353-8292(01)00012-0.

Cecil, Kim M, Christopher J Brubaker, Caleb M Adler, Kim N Dietrich, Mekibib Altaye, John C Egelhoff, Stephanie Wessel, et al. 2008. "Decreased Brain Volume in Adults with Childhood Lead Exposure." Edited by John Balmes. *PLoS Medicine* 5 (5): e112. https://doi.org/10.1371/journal.pmed.0050112.

Center for Disease Control. 2020. "Blood Lead Levels in Children | Lead | CDC." April 28, 2020. https://www.cdc.gov/nceh/lead/prevention/blood-lead-levels.htm.

———. n.d. "Blood Lead Levels in Children." National Center for Environmental Health. Accessed September 5, 2017. https://www.cdc.gov/nceh/lead/acclpp/lead_levels_in_children_fact_sheet.pdf.

Centers for Disease Control and Prevention (CDC), and National Center for Health Statistics (NCHS). 2001. "National Health and Nutrition Examination Survey Laboratory Procedure Manual. Blood Cadmium and Lead. NHANES 2000-2001." Hyattsville, MD: Nutritional Biochemistry Branch Division of Laboratory Sciences National Center for Environmental Health, CDC. https://wwwn.cdc.gov/nchs/data/nhanes/1999-2000/labmethods/lab06_met_lead_and_cadmium.pdf.

———. 2018. "National Health and Nutrition Examination Survey 2015-2016 Data Documentation, Codebook, and Frequencies." Hyattsville, MD: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/PBCD_I.htm.

Chester, Lynne, and Joy Paton. 2013. "The Economic-Environment Relation: Can Post-Keynesians, Régulationists and Polanyians Offer Insights?" *European Journal of Economics and Economic Policies: Intervention* 10 (1): 106–21.

Chetty, Raj, N Hendren, F. Lin, J. Mejerovitz, and B. Scuderi. 2016. “Childhood Environment and Gender Gaps in Adulthood.” *The American Economic Review* 106 (5): 282–88.

Chetty, Raj, Nathaniel Hendren, Patrick Kline, and Emmanuel Saez. 2014. “Where Is the Land of Opportunity? Geography of Intergenerational Mobility in the United States.” *Quarterly Journal of Economics* 129 (4): 1553–1623.

Chisolm, J. J. Jr., E.D. Mellits, and S.A. Quaskey. 1985. “The Relationship between the Level of Lead Absorption in Children and the Age, Type, and Condition of Housing.” *Environmental Research* 38: 31–45.

Clark, C.S., R.L. Bornschein, P. Succop, S.S. Que Hee, P.B. Hammond, and B. Peace. 1985. “Condition and Type of Housing as an Indicator of Potential Environmental Lead Exposure and Pediatric Blood Lead Levels.” *Environmental Research* 38: 46–53.

Clark, Scott, Warren Galke, Paul Succop, JoAnn Grote, Pat McLaine, Jonathan Wilson, Sherry Dixon, et al. 2011. “Effects of HUD-Supported Lead Hazard Control Interventions in Housing on Children’s Blood Lead.” *Environmental Research* 111 (2): 301–11. https://doi.org/10.1016/j.envres.2010.11.003.

Clark, Scott, JoAnn Grote, Jonathan Wilson, Paul Succop, Mei Chen, Warren Galke, and Pat McLaine. 2004. “Occurrence and Determinants of Increases in Blood Lead Levels in Children Shortly after Lead Hazard Control Activities.” *Environmental Research* 96 (2): 196–205. https://doi.org/10.1016/j.envres.2003.11.006.

Clean Air Act. 1967. *42 U.S. Code*. Vol. §§7401-7671q.

Coase, Ronald H. 1960. “The Problem of Social Cost.” *Journal of Law and Economics* 3: 1–44.

Control, Centers for Disease, Prevention, and others. 2005. *Preventing Lead Poisoning in Young Children: A Statement*. US Department of Health and Human Services, Centers for Disease Control and Prevention.

Council on Environmental Health. 2016. “Prevention of Childhood Lead Toxicity.” *Pediatrics* 138 (1): e20161493–e20161493. https://doi.org/10.1542/peds.2016-1493.

Cowen, Tyler, ed. 1999. *Public Goods and Market Failures: A Critical Examination*. 2. print. New Brunswick: Transaction Publishers.

Dapul, Heda, and Danielle Laraque. 2014. “Lead Poisoning in Children.” *Advances in Pediatrics* 61 (1): 313–33. https://doi.org/10.1016/j.yapd.2014.04.004.

Davies, D. J. A., J. M. Watt, and I. Thornton. 1987. “Lead Levels in Birmingham Dusts and Soils.” *The Science of the Total Environment* 67: 177–85.

Davison, Elizabeth L., and William R. Smith. 2001. “INFORMING COMMUNITY POLICING INITIATIVES WITH GIS ASSISTED MULTI-SOURCE DATA AND MICRO-LEVEL ANALYSIS.” *Journal of Crime and Justice* 24 (1): 85–108. https://doi.org/10.1080/0735648X.2001.9721618.

Dealer, Rachel Dissell, Brie Zeltner, The Plain. 2017. “Idea of Social Investment for Lead Clean up in Cleveland Advances: 10,000 Homes in 10 Years Is Target.” Cleveland.Com. December 17, 2017. http://www.cleveland.com/metro/index.ssf/2017/12/eliminating_lead_hazards_from_10000_cleveland_area_homes_can_it_be_done.html.

Dewey, John. 1927. *The Public and Its Problems*. Denver: Allan Swallow.

———. 2004. *Reconstruction in Philosophy*. Enl. ed. Mineola, N.Y: Dover Publications.

———. 2008. *The Collected Works of John Dewey. [...] Vol. 14: The Middle Works, 1899 - 1924 1922: [Human Nature and Conduct]*. Edited by Jo Ann Boydston and Murray G. Murphey. Carbondale: Southern Illinois Univ. Press.

DHD. 2017. "Task Force Recommendations for Improving Demolition Safety and Health Standards." Version 1. Detroit, MI: City of Detroit Health Department. https://www.bridgemi.com/sites/default/files/task_force_recommendations.pdf.

Dignam, Timothy A., Jose Lojo, Pamela A. Meyer, Ed Norman, Amy Sayre, and W. Dana Flanders. 2008. "Reduction of Elevated Blood Lead Levels in Children in North Carolina and Vermont, 1996–1999." *Environmental Health Perspectives* 116 (7): 981–85. https://doi.org/10.1289/ehp.10548.

Dignam, Timothy, Rachel B. Kaufmann, Lauren LeStourgeon, and Mary Jean Brown. 2019. "Control of Lead Sources in the United States, 1970-2017: Public Health Progress and Current Challenges to Eliminating Lead Exposure." *Journal of Public Health Management and Practice* 25: S13–22. https://doi.org/10.1097/PHH.0000000000000889.

Diorio, Joseph J. 1999. "Fugitive Lead Dust from the Demolition of Residential Housing." *Journal of Environmentla Health* 61 (6): 36.

Dixon, Sherry, Jonathan Wilson, Carol Kawecki, Rodney Green, Janet Phoenix, Warren Galke*, Scott Clark, and Jill Breysse. 2008. "Selecting a Lead Hazard Control Strategy Based on Dust Lead Loading and Housing Condition: I. Methods and Results." *Journal of Occupational and Environmental Hygiene* 5 (8): 530–39. https://doi.org/10.1080/15459620802219799.

Eaton, Peter, Colleen Hernandez, and Phillip Olson. n.d. "KCMo Neighborhood Housing Survey." Kansas CIty, MO: Center For Economic Information, University of Missouri-Kansas City.

Edwards, S.E., M.L. Miranda, and B. Strauss. 2014. "Geocoding Large Population-Level Administrative Datasets at Highly Resolved Spatial Scales." *Transactions in GIS* 18 (4): 586–603.

English, Peter C. 2001. *Old Paint: A Medical History of Childhood Lead-Paint Poisoning in the United States to 1980*. New Brunswick, NJ: Rutgers University Press.

Epstein, Helen. 2013. "Lead Poisoning: The Ignored Scandal." *The New York Review of Books* 60 (5).

Epstein, Roy J. 1998. "Lead Paint, Toxic Torts, and the Housing Stock: A Case Study in Risk Assessment." *The Journal of Real Estate Finance and Economics* 17 (2): 127–38.

Evans, Gary W., Heidi Saltzman, and Jana L. Cooperman. 2001. "Housing Quality and Children's Socioemotional Health." *Environment and Behavior* 33 (3): 389–99. https://doi.org/10.1177/00139160121973043.

Farfel, Mark R., Anna O. Orlova, Peter S.J. Lees, Charles Rohde, Peter J. Ashley, and J. Julian Chisolm. 2003. "A Study of Urban Housing Demolitions as Sources of Lead in Ambient Dust: Demolition Practices and Exterior Dust Fall." *Environmental Health Perspectives* 111 (9): 1228–34. https://doi.org/10.1289/ehp.5861.

Farfel, Mark R., Anna O. Orlova, Peter S.J. Lees, Charles Rohde, Peter J. Ashley, and J. Julian Chisolm. 2005. "A Study of Urban Housing Demolition as a Source of Lead in Ambient Dust on Sidewalks, Streets, and Alleys." *Environmental Research* 99 (2): 204–13. https://doi.org/10.1016/j.envres.2004.10.005.

Fatima, Rida, Uniba Tariq, Masooma Mehmood, and Ghazala Yaqub. 2016. "Childhood Lead Poisoning and Associated Health Impacts- A Brief Review." *International Journal of Environment, Agriculture and Biotechnology* 1 (3): 422–25. https://doi.org/10.22161/ijeab/1.3.18.

Faure, Elodie, Aurélie M.N. Danjou, Françoise Clavel-Chapelon, Marie-Christine Boutron-Ruault, Laure Dossus, and Béatrice Fervers. 2017. "Accuracy of Two Geocoding Methods for Geographic Information System-Based Exposure Assessment in Epidemiological Studies." *Environmental Health* 16 (1). https://doi.org/10.1186/s12940-017-0217-5.

Fergusson, David M., L. John Horwood, and Michael T. Lynskey. 1997. "Early Dentine Lead Levels and Educational Outcomes at 18 Years." *Journal of Child Psychology and Psychiatry* 38 (4): 471–78.

Filippelli, Gabriel M., and Mark A. S. Laidlaw. 2010. "The Elephant in the Playground: Confronting Lead-Contaminated Soils as an Important Source of Lead Burdens to Urban Populations." *Perspectives in Biology and Medicine* 53 (1): 31–45. https://doi.org/10.1353/pbm.0.0136.

Filippelli, Gabriel M., Mark A.S. Laidlaw, Jennifer C. Latimer, and Robyn Raftis. 2005. "Urban Lead Poisoning and Medical Geology: An Unfinished Story." *GSA Today* 15 (1): 4. https://doi.org/10.1130/1052-5173(2005)015<4:ULPAMG>2.0.CO;2.

Flegal, A. Russell, and Donald R. Smith. 1992. "Lead Levels in Preindustrial Humans." *New England Journal of Medicine* 326 (19): 1293–94. https://doi.org/10.1056/NEJM199205073261916.

Forstater, Mathew. 2003. "Must Spontaneous Order Be Unintended? Exploring the Possibilities for Consciously Enhancing Creative Discovery and Imaginative Problem Solving." In *The Evolution of Scientific Knowledge*, edited by Hans Siggaard Jensen, Lykke Margot Richter, and Morten Thanning Vandelø, 189–2008. New Horizons in Institutional and Evolutionary Economics. Northhampton, MA: Edward Elgar.

———. 2004a. "Cumulative Causation a La Lowe: Radical Endogeneity, Methodology, and Human Intervention." In *Growth, Distribution, and Effective Demand: Alternatives to Economic Orthodoxy: Essays in Honor of Edward J. Nell*, edited by Edward J. Nell, George Argyrous, and Gary Mongiovi, 309–16. Armonk, N.Y: M.E. Sharpe.

———. 2004b. "Envisioning Provisioning: Adolph Lowe and Heilbroner's Worldly Philosophy." *Social Research* 71 (2).

Fortson, Jane G., and Lisa Sanbonmatsu. 2010. "Child Health and Neighborhood Conditions: Results from a Randomized Housing Voucher Experiment." *Journal of Human Resources* 45 (4): 840–64. https://doi.org/10.1353/jhr.2010.0025.

Franklin, Benjamin. To Benjamin Vaughan. 1786. "Letter on Lead Poisoning," July 31, 1786.

Giannakouros, Panayotis, and Lihua Chen. 2018. "A Problem-Solving Approach to Data Analysis for Economics." *Forum for Social Economics* 47 (1): 87–114. https://doi.org/10.1080/07360932.2015.1078737.

Gleick, Peter H. 2010. *Bottled and Sold: The Story behind Our Obsession with Bottled Water*. Washington, DC: Island Press.

Goldberg, Daniel W, John P Wilson, Craig A Knoblock, Beate Ritz, and Myles G Cockburn. 2008. "An Effective and Efficient Approach for Manually Improving Geocoded

Data." *International Journal of Health Geographics* 7 (1): 60. https://doi.org/10.1186/1476-072X-7-60.
Gotham, Kevin Fox. 2014. *Race, Real Estate, and Uneven Development: The Kansas City Experience, 1900-2010*. Second edition. Albany: SUNY Press.
Gould, Elise. 2009. "Childhood Lead Poisoning: Conservative Estimates of the Social and Economic Benefits of Lead Hazard Control." *Environmental Health Perspectives* 117 (7): 1162–67. https://doi.org/10.1289/ehp.0800408.
Gould, Stephen Jay. 1982. "A Nation of Morons." *New Scientist*, May 6, 1982.
Grandjean, Philippe, and Philip J Landrigan. 2014. "Neurobehavioural Effects of Developmental Toxicity." *The Lancet Neurology* 13 (3): 330–38. https://doi.org/10.1016/S1474-4422(13)70278-3.
Griffith, Daniel A., Philip G. Doyle, David C. Wheeler, and David L. Johnson. 1998. "A Tale of Two Swaths: Urban Childhood Blood-Lead Levels across Syracuse, New York." *Annals of the American Association of Geographers* 88 (4): 640–65.
Grossman, Michael. 1972. "On the Concept of Health Capital and the Demand for Health." *Journal of Political Economy* 80 (2): 223–55.
Gruchy, Allan G. 1977. "Institutional Economics: Its Development and Prospects." In *Economics in Institutional Perspective: Memorial Essays in Honor of K. William Kapp*, edited by K. William Kapp, Rolf Steppacher, Brigitte Zogg-Walz, and Hermann Hatzfeldt, 11–27. Lexington, Mass: Lexington Books.
Gulson, Brian, and Alan Taylor. 2017. "A Simple Lead Dust Fall Method Predicts Children's Blood Lead Level: New Evidence from Australia." *Environmental Research* 159 (November): 76–81. https://doi.org/10.1016/j.envres.2017.07.047.
Haggerty, Mark, and Colleen Johnson. 1996. "The Social Construction of the Distribution of Income and Health." *Journal of Economic Issues* 30 (2): 525–32. https://doi.org/10.1080/00213624.1996.11505816.
Haley, Valerie B., and Thomas O. Talbot. 2004. "Geographic Analysis of Blood Lead Levels in New York State Children Born 1994-1997." *Environmental Health Perspectives* 112 (15): 1577–82. https://doi.org/10.1289/ehp.7053.
Hanna-Attisha, Mona, Jenny LaChance, Richard Casey Sadler, and Allison Champney Schnepp. 2016a. "Elevated Blood Lead Levels in Children Associated With the Flint Drinking Water Crisis: A Spatial Analysis of Risk and Public Health Response." *American Journal of Public Health* 106 (2): 283–90. https://doi.org/10.2105/AJPH.2015.303003.
———. 2016b. "Elevated Blood Lead Levels in Children Associated With the Flint Drinking Water Crisis: A Spatial Analysis of Risk and Public Health Response." *American Journal of Public Health* 106 (2): 283–90. https://doi.org/10.2105/AJPH.2015.303003.
Harris, Jeffrey E. 2020. "The Subways Seeded the Massive Coronavirus Epidemic in New York City." Working Paper 27021. National Bureau of Economic Research.
Hastings, Douglas, and Marie Lynn Miranda. 2012. "Using GIS-Based Models to Protect Children from Lead Exposure." In *Community-Based Operations Research*, edited by Michael P. Johnson, 167:173–87. International Series in Operations Research & Management Science. New York, NY: Springer New York. https://doi.org/10.1007/978-1-4614-0806-2_7.

Hayden, F. Gregory. 1983. “Integration of Social Indicators Into Holistic Geobased Models.” *Journal of Economic Issues* 17 (2): 325–34.
Hayek, F. A. 1945. “The Use of Knowledge in Society.” *The American Economic Review* 35 (4): 519–30.
“Healthy Children and Youth Lead Risk Assessment Guide.” 2009. Missouri Department of Social Services, Missouri Department of Health and Senior Services. https://health.mo.gov/living/environment/lead/pdf/HCYLeadRiskAssessmentGuide.pdf.
Heilbroner, Robert L. 1988. *Behind the Veil of Economics: Essays in the Worldly Philosophy*. New York; London: W.W. Norton & Co.
Hirsch, Jana, and Amy Hillier. 2013. “Exploring the Role of the Food Environment on Food Shopping Patterns in Philadelphia, PA, USA: A Semiquantitative Comparison of Two Matched Neighborhood Groups.” *International Journal of Environmental Research and Public Health* 10 (1): 295–313. https://doi.org/10.3390/ijerph10010295.
Holt, Richard P.F., Steven Pressman, and Clive L. Spash, eds. 2009. *Post Keynesian and Ecological Economics: Confronting Environmental Issues*. Northhampton, MA: Edward Elgar.
Holton, W. Conard. 2002. “Holton - Locating Lead_Mapping Leads to Intervention (2002).Pdf.” *Environmental Health Perspectives* 110 (0): A533.
Hu, Howard, Antonio Aro, Marinelle Payton, Susan Korrick, David Sparrow, Scott T. Weiss, and Andrea Rotnitzky. 1996. “The Relationship of Bone and Blood Lead to Hypertension: The Normative Aging Study.” *Jama* 275 (15): 1171–76.
Jackson, Shannon. 2008. “The City from Thirty Thousand Feet: Embodyment, Creativity, and the Use of Geographic Information Systems as Urban Planning Tools.” *Technology and Culture* 49 (2): 325–46.
Jacobs, Salvatore Cali, Alison Welch, Sherry L. Dixon, Anne Evens, Amy Pelka Mucha, Nicole Vahl, and John Bartlett. 2012. “Lead and Other Heavy Metals in Dust Fall from Single Family Housing Demolition.” *Public Health Reports* 128 (6): 145–62.
Jacobs, David E. 1995. “Lead-Based Paint as a Major Source of Childhood Lead Poisoning: A Review of the Evidence.” In *Lead In Paint, Soil, and Dust: Health Risks, Exposures Studies, Control Measures, and Quality Assurance, ASTM STP 1226*, edited by Michael E. Beard and S. D. Allen Iske, 175–87. Philadelphia, PA: American Society for Testing and Materials.
Jacobs, David E., Mary Jean Brown, Andrea Baeder, Marissa Scalia Sucosky, Stephen Margolis, Jerry Hershovitz, Laura Kolb, and Rebecca L. Morley. 2010. “A Systematic Review of Housing Interventions and Health: Introduction, Methods, and Summary Findings.” *Journal of Public Health Management and Practice* 16: S5–10. https://doi.org/10.1097/PHH.0b013e3181e31d09.
Jacobs, David E., Robert P. Clickner, Joey Y. Zhou, Susan M. Viet, David A. Marker, John W. Rogers, Darryl C. Zeldin, Pamela Broene, and Warren Friedman. 2002. “The Prevalence of Lead-Based Paint Hazards in U.S. Housing.” *Environmental Health Perspectives* 110 (10): A599–606.
Jacobs, David E., and Rick Nevin. 2006. “Validation of a 20-Year Forecast of US Childhood Lead Poisoning: Updated Prospects for 2010.” *Environmental Research* 102 (3): 352–64. https://doi.org/10.1016/j.envres.2005.12.015.

Jaquez, G. 2012. “A Research Agenda: Does Geocoding Positional Error Matter in Health.” *Spatial and Spatio-Temporal Epidemiology* 3: 7–16.

Jean Brown, Mary, Jaime Raymond, David Homa, Chinaro Kennedy, and Thomas Sinks. 2011. “Association between Children’s Blood Lead Levels, Lead Service Lines, and Water Disinfection, Washington, DC, 1998–2006.” *Environmental Research* 111 (1): 67–74. https://doi.org/10.1016/j.envres.2010.10.003.

Jones, David J. 2012. “Primary Prevention and Health Outcomes: Treatment of Residential Lead-Based Paint Hazards and the Prevalence of Childhood Lead Poisoning.” *Journal of Urban Economics* 71 (1): 151–64. https://doi.org/10.1016/j.jue.2011.06.002.

Jusko, Todd A., Charles R. Henderson, Bruce P. Lanphear, Deborah A. Cory-Slechta, Patrick J. Parsons, and Richard L. Canfield. 2007. “Blood Lead Concentrations < 10 Mg/DL and Child Intelligence at 6 Years of Age.” *Environmental Health Perspectives* 116 (2): 243–48. https://doi.org/10.1289/ehp.10424.

Kaiser, R., A. K. Henderson, W. R. Daley, M. Naughton, M. H. Khan, M. Rahman, S. Kieszak, and C. H. Rubin. 2001. “Blood Lead Levels of Primary School Children in Dhaka, Bangladesh.” *Environmental Health Perspectives* 109 (6): 563–66.

Kane, Natalie. 2020. “An Interdisciplinary Health Disparities Research and Intervention Strategy Applied to the Problem of Pediatric Asthma in Kansas City.” Dissertation, Kansas CIty, MO: Univeristy of Missouri-Kansas City.

Kane, Natalie, and Peter Eaton. 2018. “Modeling Address Level Asthma Conditions.” CEI Working Paper 1602–02. Kansas City, MO: Center For Economic Information.

Kansas City (Jackson County, Mo.) City Directory, Including Gladstone and North Kansas City. 1976. Kansas CIty, MO: R.L. Polke & co.

Kansas City, MO. n.d. *Demoltion*. *Code of Ordinances*. Vol. Part 1 Chapter 19. Accessed November 30, 2020. https://library.municode.com/mo/kansas_city/codes/code_of_ordinances?nodeId=PTIICOOR_CH18BURECO_ARTIIBUCO_S18-40ADINBUCO2018AM.

Kapp, K. William. 1970. “Environmental Disruption and Social Costs: A Challange to Economists.” *Kyklos* 23 (4): 833–48.

———. 1978. *The Social Costs of Business Enterprise*. Revised and Extended ed. Spokesman University Paperback ; No. 16. Nottingham: Spokesman Books.

Kennedy, Chinaro, Robert Lordo, Marissa Scalia Sucosky, Rona Boehm, and Mary Jean Brown. 2016. “Evaluating the Effectiveness of State Specific Lead-Based Paint Hazard Risk Reduction Laws in Preventing Recurring Incidences of Lead Poisoning in Children.” *International Journal of Hygiene and Environmental Health* 219 (1): 110–17. https://doi.org/10.1016/j.ijheh.2015.09.009.

Kennedy, Kevin. 2011. “Housing and Health: Making the Connection Using Geographic Information Systems.” Practicum Report, Kansas CIty, MO: University of Missouri-Kansas City.

Kerr, Suzi, and Richard G. Newell. 2003. “Policy Induced Technology Adoption: Evidence from the U.S. Lead Phasedown.” *The Journal of Industrial Economics* 51 (3): 317–43.

Keynes, John Maynard. 1964. *The General Theory of Employment, Interest, and Money*. 1st Harvest/HBJ ed. San Diego: Harcourt, Brace, Jovanovich.

Kim, Dohyeong, M. Alicia Overstreet Galeano, Andrew Hull, and Marie Lynn Miranda. 2008. “A Framework for Widespread Replication of a Highly Spatially Resolved Childhood Lead Exposure Risk Model.” *Environmental Health Perspectives* 116 (12): 1735–39. https://doi.org/10.1289/ehp.11540.

Kim, Rokho, Andrea Rotnitzky, David Sparrow, Scott T. Weiss, Carrie Wagner, and Howard Hu. 1996. “A Longitudinal Study of Low-Level Lead Exposure and Impairment of Renal Function: The Normative Aging Study.” *JAMA* 275 (15): 1177–81.

King, Katherine E. 2015. “A Comparison of Two Methods for Measuring Land Use in Public Health Research: Systematic Social Observation Versus Remote Sensing–Based Coded Aerial Photography.” *SAGE Open* 5 (2): 215824401558943. https://doi.org/10.1177/2158244015589438.

Kizer, Kenneth W. 1985. “Lead.” *JAMA* 254 (9): 1226–27.

Kmenta, Jan. 1997. *Elements of Econometrics*. 2nd ed. Ann Arbor: University of Michigan Press.

Krieger, James, and Donna L. Higgins. 2002. “Housing and Health: Time Again for Public Health Action.” *American Journal of Public Health* 92 (5): 758–68. https://doi.org/10.2105/AJPH.92.5.758.

Kwan, Mei-Po. 2012. “The Uncertain Geographic Context Problem.” *Annals of the American Association of Geographers* 102 (5): 958–68.

Lagerwerff, John V., and A. W. Specht. 1970. “Contamination of Roadside Soil and Vegetation with Cadmium, Nickel, Lead, and Zinc.” *Environmental Science & Technology* 4 (7): 583–86. https://doi.org/10.1021/es60042a001.

Laidlaw, Mark A.S., and Gabriel M. Filippelli. 2008. “Resuspension of Urban Soils as a Persistent Source of Lead Poisoning in Children: A Review and New Directions.” *Applied Geochemistry* 23 (8): 2021–39. https://doi.org/10.1016/j.apgeochem.2008.05.009.

Laidlaw, Mark A.S., Howard W. Mielke, Gabriel M. Filippelli, David L. Johnson, and Christopher R. Gonzales. 2005. “Seasonality and Children’s Blood Lead Levels: Developing a Predictive Model Using Climatic Variables and Blood Lead Data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA).” *Environmental Health Perspectives* 113 (6): 793–800. https://doi.org/10.1289/ehp.7759.

Lanphear, Bruce P. 2017. “Low-Level Toxicity of Chemicals: No Acceptable Levels?” Edited by Linda S. Birnbaum. *PLOS Biology* 15 (12): e2003066. https://doi.org/10.1371/journal.pbio.2003066.

Lanphear, Bruce P., David A. Burgoon, Steven W. Rust, Shirley Eberly, and Warren Galke. 1998. “Environmental Exposures to Lead and Urban Children’s Blood Lead Levels.” *Environmental Research Section A* 76: 120–30.

Lanphear, Bruce P., Richard Hornung, and Mona Ho. 2005. “Screening Housing to Prevent Lead Toxicity in Children.” *Public Health Reports* 120 (3): 305–10.

Lanphear, Bruce P., Richard Hornung, Mona Ho, Cynthia R. Howard, Shirley Eberly, and Karen Knauf. 2002. “Environmental Lead Exposure during Early Childhood.” *The Journal of Pediatrics* 140 (1): 40–47. https://doi.org/10.1067/mpd.2002.120513.

Lanphear, Bruce P., Richard Hornung, Jane Khoury, Kimberly Yolton, Peter Baghurst, David C. Bellinger, Richard L. Canfield, et al. 2005. “Low-Level Environmental Lead

Exposure and Children's Intellectual Function: An International Pooled Analysis." *Environmental Health Perspectives* 113 (7): 894–99. https://doi.org/10.1289/ehp.7688.

Lanphear, Bruce P., Thomas D. Matte, John Rogers, Robert P. Clickner, Brian Dietz, Robert L. Bornschein, Paul Succop, et al. 1998. "The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels: A Pooled Analysis of 12 Epidemiologic Studies." *Environmental Research* 79 (1): 51–68.

Lanphear, Bruce P, Stephen Rauch, Peggy Auinger, Ryan W Allen, and Richard W Hornung. 2018. "Low-Level Lead Exposure and Mortality in US Adults: A Population-Based Cohort Study." *The Lancet Public Health* 3 (4): e177–84. https://doi.org/10.1016/S2468-2667(18)30025-2.

Lanphear, Bruce P., Michael Weitzman, Nancy L. Winter, Shirley Eberly, Benjamin Yakir, Martin Tanner, Mary Emond, and Thomas D. Matte. 1996. "Lead-Contaminated House Dust and Urban Children's Blood Lead Levels." *American Journal of Public Health* 86 (10): 1416–21.

Lash, Donald A. 1997. "Cost Allocation of Lead Paint Abatement in Distressed Buildings in New York City." *Journal of Affordable Housing & Community Development Law*, 299–308.

Laudan, Larry. 1981. *Science and Hypothesis: Historical Essays on Scientific Methodology*. The University of Western Ontario Series in Philosophy of Science, v. 19. Dordrecht, Holland ; Boston, U.S.A. : Hingham, MA: D. Reidel ; Sold and distributed in the U.S.A. and Canada by Kluwer Boston Inc.

Lee, Frederic S. 2014. "Modeling as a Research Method in Heterodox Economics." Book Chapter. Kansas City, MO.

———. 2018. *Microeconomic Theory: A Heterodox Approach*. Edited by Tae-Hee Jo. Routledge Advances in Heterodox Economics 37. London New York: Routledge.

Lee, Lisa M. 2012. "Public Health Ethics Theory: Review and Path to Convergence." *Journal of Law, Medicine & Ethics* 40 (1): 85–98. https://doi.org/10.1111/j.1748-720X.2012.00648.x.

Leggett, R W. 1993. "An Age-Specific Kinetic Model of Lead Metabolism in Humans." *Environmental Health Perspectives* 101 (7): 598–616. https://doi.org/10.1289/ehp.93101598.

Lejano, Raul P, and Jonathon E Ericson. 2005. "Tragedy of the Temporal Commons: Soil-Bound Lead and the Anachronicity of Risk." *Journal of Environmental Planning and Management* 48 (2): 301–20. https://doi.org/10.1080/0964056042000338190.

Levin, Ronnie, Mary Jean Brown, Michael E. Kashtock, David E. Jacobs, Elizabeth A. Whelan, Joanne Rodman, Michael R. Schock, Alma Padilla, and Thomas Sinks. 2008. "Lead Exposures in U.S. Children, 2008: Implications for Prevention." *Environmental Health Perspectives* 116 (10): 1285–93. https://doi.org/10.1289/ehp.11241.

Lidsky, T. I., and J. S. Schneider. 2003. "Lead Neurotoxicity in Children: Basic Mechanisms and Clinical Correlates." *Brain* 126 (1): 5–19. https://doi.org/10.1093/brain/awg014.

Lin-Fu, Jane. 1980. "Lead Poisoning and Undue Exposure in Children: History and Current Status." In *Low Level Lead Exposure: The Clinical Implications of Current Research*, edited by Needleman, Herbert, 5–16. New York: Raven Press.

———. n.d. “Modern History of Lead Poisoning.” In *Human Lead Exposure*, 23–43.
Loeb, Alan. 1999. “Paradigms Lost: A Case Study Analysis of Models of Corporate Responsibility for the Environment.” *Business and Economic History* 28 (2): 95–108.
“Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention.” 2012. Report of the Advisory Committee on Childhood Lead Poisoning Prevention. Centers for Disease Control and Prevention.
Lyngbye, Troels, Ole N. Hansen, and Philippe Grandjean. 1990. “Predictors of Tooth-Lead Level with Special Reference to Traffic: A Study of Lead-Exposure in Children.” *International Archives of Occupational and Environmental Health* 62 (6): 417–22. https://doi.org/10.1007/BF00379057.
Mack, Ronald B. 1973. “Lead in History.” *Clinical Toxicology Bulliten* 3 (2): 37–44.
Maddaloni, Mark, Nancy Lolacono, William Manton, Conrad Blum, John Drexler, and Joseph Graziano. 1998. “Bioavailability of Soilborne Lead in Adults, by Stable Isotope Dilution.” *Environmental Health Perspectives* 106 (supplement 6): 1589–95.
Manson, Steven M., Heather A. Sander, Debarchana Ghosh, J. Michael Oakes, Myron W. Orfield, Jr, William J. Craig, Thomas F. Luce, Jr, Eric Myott, and Shipeng Sun. 2009. “Parcel Data for Research and Policy.” *Geography Compass* 3 (2): 698–726. https://doi.org/10.1111/j.1749-8198.2008.00209.x.
Markowitz, Gerald E, and David Rosner. 2014. *Lead Wars: The Politics of Science and the Fate of America’s Children*. Berkeley; New York: University of California Press ; Milbank Memorial Fund.
Markowitz, Gerald, and David Rosner. 2000. “‘ Cater to the Children’: The Role of the Lead Industry in a Public Health Tragedy, 1900-1955.” *American Journal of Public Health* 90 (1): 36.
Markowitz, M E, P E Bijur, H A Ruff, K Balbi, and J F Rosen. 1996. “Moderate Lead Poisoning: Trends in Blood Lead Levels in Unchelated Children.” *Environmental Health Perspectives* 104 (9): 968–72. https://doi.org/10.1289/ehp.96104968.
Markus, Julie, and Alex B. McBratney. 2001. “A Review of the Contamination of Soil with Lead II. Spatial Distribution and Risk Assessment of Soil Lead.” *Environment International* 27: 399–411.
Mazzucato, Mariana. 2011. *The Entrepreneurial State*. London: Demos.
McClintock, Nathan. 2015. “A Critical Physical Geography of Urban Soil Contamination.” *Geoforum* 65 (October): 69–85. https://doi.org/10.1016/j.geoforum.2015.07.010.
McClure, Leland F., Justin K. Niles, and Harvey W. Kaufman. 2016. “Blood Lead Levels in Young Children: US, 2009-2015.” *The Journal of Pediatrics* 175 (August): 173–81. https://doi.org/10.1016/j.jpeds.2016.05.005.
McElroy, Jane A., Patric L. Remington, Amy Trenthaam-Dietz, Stephanie A. Robert, and Polly A. Newcomb. 2003. “Geocoding Addresses from a Large Population-Based Study: Lessons Learned.” *Epidemiology* 14 (4): 399–407.
Medley, Sara Sullivan. 1982. “Childhood Lead Toxicity: A Paradox of Modern Technology.” *Annals of the American Academy of Political and Social Science* 461 (May): 63–73.
Melody, William H. 1987. “Information: An Emerging Dimension of Institutional Analysis.” *Journal of Economic Issues* 21 (3): 1313–39.
Mielke, Howard W., Dianne Dugas, Paul W. Mielke Jr, Kabrina S. Smith, and C. R. Gonzales. 1997. “Associations between Soil Lead and Childhood Blood Lead in

Urban New Orleans and Rural Lafourche Parish of Louisiana." *Environmental Health Perspectives* 105 (9): 950.
Mielke, Howard W., Christopher R. Gonzales, Eric T. Powell, and Paul W. Mielke. 2013. "Environmental and Health Disparities in Residential Communities of New Orleans: The Need for Soil Lead Intervention to Advance Primary Prevention." *Environment International* 51 (January): 73–81. https://doi.org/10.1016/j.envint.2012.10.013.
Mielke, Howard W., Mark A.S. Laidlaw, and Chris Gonzales. 2010. "Lead (Pb) Legacy from Vehicle Traffic in Eight California Urbanized Areas: Continuing Influence of Lead Dust on Children's Health." *Science of The Total Environment* 408 (19): 3965–75. https://doi.org/10.1016/j.scitotenv.2010.05.017.
Mielke, Howard W., and Patrick L. Reagan. 1998. "Soil Is an Important Pathway of Human Lead Exposure." *Environmental Health Perspectives* 106 (February): 217. https://doi.org/10.2307/3433922.
Mielke, Howard W., Patrick L. Reagan, J.L. Adams, and Paul W. Jr. Mielke. 1989. "Soil-Dust Lead and Childhood Lead Exposure as a Function of City Size and Community Traffic Flow: The Case for Lead Abatement in Minnesota." *Environmental Geochemistry and Health* 9: 253–71.
Mielke, Howard W., and Sammy Zahran. 2012. "The Urban Rise and Fall of Air Lead (Pb) and the Latent Surge and Retreat of Societal Violence." *Environment International* 43 (August): 48–55. https://doi.org/10.1016/j.envint.2012.03.005.
Milstien, Cydney. 2017. "Age of Housing Reports." Housing Research Kansas City.
Minsky, Hyman P. 1986. *Stabilizing an Unstable Economy*. New Haven: Yale University Press.
Miranda, Marie Lynn, Rebecca Anthropolous, and Douglas Hastings. 2011. "A Geospatial Analysis of the Effects of Aviation Gasoline on Childhood Blood Lead Levels." *Environmental Health Perspectives* 119 (19): 1513–16.
Miranda, Marie Lynn, Dana C. Dolinoy, and M. Alicia Overstreet. 2002. "Mapping for Prevention: GIS Models for Directing Childhood Lead Poisoning Prevention Programs." *Environmental Health Perspectives* 110 (9): 947–53.
"Missouri Childhood Lead Poisoning Prevention Program." 2018. Annual Report for Fiscal Year 2018. Missouri Department of Health and Senior Services. https://health.mo.gov/living/environment/lead/.
Missouri Revisor of Statutes. 2001. *Title XLI Codes and Standards*. Vol. 701.326. https://revisor.mo.gov/main/OneSection.aspx?section=701.326&bid=31658.
MO Department of Health and Senior Services. 2008. "Childhood Blood Lead Testing and Follow-Up Guidelines For Children Six (6) Months through Seventy Two (72) Months of Age."
MO DSS. 2013. "Acronyms." Acronyms. 2013. https://dss.mo.gov/mhd/oversight/pdf/acronyms.pdf.
"MO HealthNet for Kids | Mydss.Mo.Gov." n.d. Accessed June 19, 2020. https://mydss.mo.gov/healthcare/mohealthnet-for-kids.
Moody, Heather A., Joe T. Darden, Bruce Wm. Pigozzi, and Sue C. Grady. 2016. "The Relationship of Neighborhood Socioeconomic Differences and Racial Residential Segregation to Childhood Blood Lead Levels in Metropolitan Detroit." *Journal of Urban Health* 93 (5): 820–39. https://doi.org/10.1007/s11524-016-0071-8.

Moore, Terry. 1978. “Why Allow Planners to Do What They Do? A Justification from Economic Theory.” *Journal of the American Institute of Planners* 44 (4): 387–98. https://doi.org/10.1080/01944367808976917.

Morales, Leo S., Peter Gutierrez, and Jose J. Escarce. 2005. “Demogrgaphic and Socioeconomic Factors Associated with Blood Lead Levels Among Mexican-American Children.” *Public Health Reports* 120 (August): 448–54.

More, Alexander F., Nicole E. Spaulding, Pascal Bohleber, Michael J. Handley, Helene Hoffmann, Elena V. Korotkikh, Andrei V. Kurbatov, et al. 2017. “Next-generation Ice Core Technology Reveals True Minimum Natural Levels of Lead (Pb) in the Atmosphere: Insights from the Black Death.” *GeoHealth* 1 (4): 211–19. https://doi.org/10.1002/2017GH000064.

Morello-Frosch, Rachel, Manuel Pastor Jr, and James Sadd. 2002. “Integrating Environmental Justice and the Precautionary Principle in Research and Policy Making: The Case of Ambient Air Toxics Exposures and Health Risks among Schoolchildren in Los Angeles.” *The Annals of the American Academy of Political and Social Science* 584 (1): 47–68.

Morgan, Mary S. 2012. *The World in the Model: How Economists Work and Think*. Cambridge ; New York: Cambridge University Press.

Motto, Harry L., Robert H. Daines, Daniel M. Chilko, and Carlotta K. Motto. 1970. “Lead in Soils and Plants: Its Relation to Traffic Volume and Proximity to Highways.” *Environmental Science & Technology* 4 (3): 231–37. https://doi.org/10.1021/es60038a009.

Mucha, Amy Pelka, Nicole Stites, Anne Evens, Patrick M. MacRoy, Victoria W. Persky, and David E. Jacobs. 2009. “Lead Dustfall from Demolition of Scattered Site Family Housing: Developing a Sampling Methodology.” *Environmental Research* 109 (2): 143–48. https://doi.org/10.1016/j.envres.2008.10.010.

Muller, Christopher, Robert J. Sampson, and Alix S. Winter. 2018. “Environmental Inequality: The Social Causes and Consequences of Lead Exposure.” *Annual Review of Sociology* 44 (1): 263–82. https://doi.org/10.1146/annurev-soc-073117-041222.

Naidu, Sirisha C., Panayiotis T. Manolakos, and Thomas E. Hopkins. 2013. “Environmental Justice in Ohio.” *Review of Radical Political Economics* 45 (3): 384–99. https://doi.org/10.1177/0486613412470094.

National Academies of Sciences, Engineering, and Medicine. 2019. *Integrating Social Care into the Delivery of Health Care: Moving Upstream to Improve the Nation’s Health*. Washington, D.C.: National Academies Press. https://doi.org/10.17226/25467.

National Research Council (U.S.), ed. 1980. *Lead in the Human Environment: A Report*. Washington, D.C: National Academy of Sciences.

Needleman, Herbert. 1998. “Childhood Lead Poisoning: The Promise and Abandonment of Primary Prevention.” *American Journal of Public Health* 88 (12): 1871–77. https://doi.org/10.2105/AJPH.88.12.1871.

Needleman, Herbert L., Charles Gunnoe, Alan Leviton, Robert Reed, Henry Peresie, Cornelius Maher, and Peter Barret. 1979. “Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels.” *THe New England Journal of Medicine* 300 (13): 689–95.

Needleman, Herbert L., Julie A. Riess, Michael J. Tobin, Gretchen E. Biesecker, and Joel B. Greenhouse. 1996. “Bone Lead Levels and Delinquent Behavior.” *Jama* 275 (5): 363–69.

Needleman, Herbert L., Allan Schell, David Bellinger, Alan Leviton, and Elizabeth N. Allred. 1990. “The Long-Term Effects of Low Doses of Lead in Childhood: An 11-Year Follow-up Report.” *The New England Journal of Medicine* 322 (2): 83–88.

Needleman, Herbert L., Orhan Tuncay, and Irving Shapiro. 1972. “Lead Levels in Deciduous Teeth of Urban and Suburban American Children.” *Nature* 235 (January): 111–12.

Nevin, Rick. 2000. “How Lead Exposure Relates to Temporal Changes in IQ, Violent Crime, and Unwed Pregnancy.” *Environmental Research* 83 (1): 1–22. https://doi.org/10.1006/enrs.1999.4045.

———. 2007. “Understanding International Crime Trends: The Legacy of Preschool Lead Exposure.” *Environmental Research* 104 (3): 315–36. https://doi.org/10.1016/j.envres.2007.02.008.

———. 2009. “Trends in Preschool Lead Exposure, Mental Retardation, and Scholastic Achievement: Association or Causation?” *Environmental Research* 109 (3): 301–10. https://doi.org/10.1016/j.envres.2008.12.003.

———. 2012. “Lead Poisoning and The Bell Curve.” MPRA paper. https://mpra.ub.uni-muenchen.de/36569/.

———. 2016. *Lucifer Curves The Legacy of Lead Poisoning.* Cork: BookBaby. http://public.eblib.com/choice/PublicFullRecord.aspx?p=4593620.

Nevin, Rick, David E. Jacobs, Michael Berg, and Jonathan Cohen. 2008. “Monetary Benefits of Preventing Childhood Lead Poisoning with Lead-Safe Window Replacement.” *Environmental Research* 106 (3): 410–19. https://doi.org/10.1016/j.envres.2007.09.003.

“News – KC Water.” n.d. Accessed August 16, 2020. https://www.kcwater.us/news/.

Nigg, Joel T., Molly Nikolas, G. Mark Knottnerus, Kevin Cavanagh, and Karen Friderici. 2010. “Confirmation and Extension of Association of Blood Lead with Attention-Deficit/Hyperactivity Disorder (ADHD) and ADHD Symptom Domains at Population-Typical Exposure Levels: ADHD and Blood Lead.” *Journal of Child Psychology and Psychiatry* 51 (1): 58–65. https://doi.org/10.1111/j.1469-7610.2009.02135.x.

Nriagu, Jerome O. 1990. “The Rise and Fall of Leaded Gasoline.” *The Science of the Total Environment* 92: 13–28.

O’Campo, P. 2003. “Invited Commentary: Advancing Theory and Methods for Multilevel Models of Residential Neighborhoods and Health.” *American Journal of Epidemiology* 157 (1): 9–13. https://doi.org/10.1093/aje/kwf171.

Office for Civil Rights (OCR). 2008. “Summary of the HIPAA Privacy Rule.” Text. HHS.Gov. May 7, 2008. https://www.hhs.gov/hipaa/for-professionals/privacy/laws-regulations/index.html.

Olen, Michael E. 2019. “Deconstruction: Reducing the Costs to Deconstruct Blighted Buildings.” MS Thesis in Construction Management, University of Wisconsin-Stout. http://www2.uwstout.edu/content/lib/thesis/2019/2019olenm.pdf.

“Open Data | City of Kansas City, Missouri Mayor’s Office.” n.d. Accessed May 2, 2020. http://kcmayor.org/mayors-agenda-items/open-data.

Openshaw, S. 1983. *Classification Using Information Statistics*. Concepts and Techniques in Modern Geography, no. 38. Norwich [Norfolk]: Geo Books.

OSHA, and US Department of Labor. n.d. "Demolition - Overview | Occupational Safety and Health Administration." Safety and Health Topics / Demolition. Accessed October 6, 2020. https://www.osha.gov/demolition.

Oyana, Tonny J., and Florence M. Margai. 2010. "Spatial Patterns and Health Disparities in Pediatric Lead Exposure in Chicago: Characteristics and Profiles of High-Risk Neighborhoods." *The Professional Geographer* 62 (1): 46–65. https://doi.org/10.1080/00330120903375894.

Paquet, Catherine, Margaret Cargo, Yan Kestens, and Mark Daniel. 2010. "Reliability of an Instrument for Direct Observation of Urban Neighbourhoods." *Landscape and Urban Planning* 97 (3): 194–201. https://doi.org/10.1016/j.landurbplan.2010.06.001.

Parsons, Patrick J, Andrew A Reilly, and Debra Esernio-Jenssen. 1997. "Screening Children Exposed to Lead: An Assessment of the Capillary Blood Lead Fingerstick Test." *Clinical Chemistry* 43 (2): 302–11. https://doi.org/10.1093/clinchem/43.2.302.

Patridge, Jeff, Robert H. Schneider, Pat Phillips, Felicia Rabito, and Chuck Shorter. 2006. "Using Spatial Analyst to Determine Effects of Demolition on Blood Lead Levels in St. Louis City Children A Preliminary Look." Missouri Department of Health and Senior Services. http://www.quasipm.com/uploads/2/6/2/3/2623302/moarticle.pdf.

Patterson, Clair C. 1965. "Contaminated and Natural Lead Environments of Man." *Archives of Environmental Health* 2 (September): 344–71.

———. 1998. "Scientific Meaning of Meanings: Quests for Discoveries Concerning Our Cultural Ills." *Environmental Research Section A* 78: 177–84.

Pearce, J.M.S. 2007. "Burton's Line in Lead Poisoning." *European Neurology* 57 (2): 118–19. https://doi.org/10.1159/000098100.

Peirce, Charlse Sanders. 2006. "How to Make Our Ideas Clear." In *Pragmatism Old & New: Selected Writings*, edited by Susan Haack and Robert Lane, 127–50. Amherst, NY: Prometheus Books.

Power, Marilyn. 2004. "Social Provisioning as a Starting Point for Feminist Economics." *Feminist Economics* 10 (3): 3–19. https://doi.org/10.1080/1354570042000267608.

"Prior HHS Poverty Guidelines and Federal Register References." 2015. ASPE. November 23, 2015. https://aspe.hhs.gov/prior-hhs-poverty-guidelines-and-federal-register-references.

Proost, Stef, and Jacques-François Thisse. 2019. "What Can Be Learned from Spatial Economics?" *Journal of Economic Literature* 57 (3): 575–643. https://doi.org/10.1257/jel.20181414.

Pulido, Laura. 2000. "Rethinking Environmental Racism: White Privilege and Urban Development in Southern California." *Annals of the Association of American Geographers* 90 (1): 12–40. https://doi.org/10.1111/0004-5608.00182.

———. 2016. "Flint, Environmental Racism, and Racial Capitalism." *Capitalism Nature Socialism* 27 (3): 1–16. https://doi.org/10.1080/10455752.2016.1213013.

Rabito, F.A., S. Iqbal, C.F. Shorter, P. Osman, P.E. Philips, E. Langlois, and L.E. White. 2007. "The Association between Demolition Activity and Children's Blood Lead Levels." *Environmental Research* 103 (3): 345–51. https://doi.org/10.1016/j.envres.2006.09.011.

Ravikrishna, Raghunathan, Han-Woong Lee, Stephen Mbuligwe, K. T. Valsaraj, and John H. Pardue. 2010. “Air Quality during Demolition and Recovery Activities in Post-Katrina New Orleans.” *Environmental Toxicology and Chemistry* 29 (7): 1438–44. https://doi.org/10.1002/etc.210.

Reddy, Aravind, and Charles L. Braun. 2010. “Lead and the Romans.” *Journal of Chemical Education* 87 (10): 1052–55. https://doi.org/10.1021/ed100631y.

Reiss, Albert J. jr. 1971. “Systematic Observation of Natural Social Phenomina.” *Sociological Methodology* 3: 3–33.

Reissman, Dori B., Forrest Staley, Gerald B. Curtis, and Rachel B. Kaufmann. 2001. “Use of Geographic Information System Technology to Aid Health Department Decision Making about Childhood Lead Poisoning Prevention Activities.” *Environmental Health Perspectives* 109 (1): 89.

Renner, Rebecca. 2009. “Out of Plumb: When Water Treatment Causes Lead Contamination.” *Environmental Health Perspectives* 117 (12): A542.

Residential Lead-Based Paint Hazard Reduction Act of 1992 –- Title X. 1992. https://www.epa.gov/lead/residential-lead-based-paint-hazard-reduction-act-1992-title-x.

Reuben, Aaron, Avshalom Caspi, Daniel W. Belsky, Jonathan Broadbent, Honalee Harrington, Karen Sugden, Renate M. Houts, Sandhya Ramrakha, Richie Poulton, and Terrie E. Moffitt. 2017. “Association of Childhood Blood Lead Levels With Cognitive Function and Socioeconomic Status at Age 38 Years and With IQ Change and Socioeconomic Mobility Between Childhood and Adulthood.” *JAMA* 317 (12): 1244. https://doi.org/10.1001/jama.2017.1712.

Reyes, Jessica Wolpaw. 2015. “LEAD EXPOSURE AND BEHAVIOR: EFFECTS ON ANTISOCIAL AND RISKY BEHAVIOR AMONG CHILDREN AND ADOLESCENTS.” *Economic Inquiry* 53 (3): 1580–1605. https://doi.org/10.1111/ecin.12202.

Richmond-Bryant, Jennifer, Qingyu Meng, J. Allen Davis, Jonathan Cohen, David Svendsgaard, James S. Brown, Lauren Tuttle, et al. 2013. “A Multi-Level Model of Blood Lead as a Function of Air Lead.” *Science of The Total Environment* 461–462 (September): 207–13. https://doi.org/10.1016/j.scitotenv.2013.05.008.

Ringquist, Evan J. 2005. “Assessing Evidence of Environmental Inequities: A Meta-Analysis.” *Journal of Policy Analysis and Management* 24 (2): 223–47. https://doi.org/10.1002/pam.20088.

Rinsky, Jessica L., Sheila Higgins, Kim Angelon-Gaetz, Doris Hogan, Pierre Lauffer, Megan Davies, Aaron Fleischauer, et al. 2018. “Occupational and Take-Home Lead Exposure Among Lead Oxide Manufacturing Employees, North Carolina, 2016.” *Public Health Reports* 133 (6): 700–706. https://doi.org/10.1177/0033354918795442.

Robbins, Lionel. 1945. *An Essay into the Nature & Significance of Economic Science*. 2nd ed. London: Macmillan and Co.

Roberts, James R., Thomas C. Hulsey, Gerald B. Curtis, and J. Routt Reigart. 2003. “Using Geographic Information Systems to Assess Risk for Elevated Blood Lead Levels in Children.” *Public Health Reports* 118 (June): 221–30.

Robinson, Ws. 2009. “Ecological Correlations and the Behavior of Individuals*.” *International Journal of Epidemiology* 38 (2): 337–41. https://doi.org/10.1093/ije/dyn357.

Rodgers, Joseph Lee, and W. Alan Nicewander. 1988. “Thirteen Ways to Look at the Correlation Coefficient.” *The American Statistician* 42 (1): 59. https://doi.org/10.2307/2685263.

Rogers, Michelle L., James A. Lucht, Alyssa J. Sylvaria, Jessica Cigna, Robert Vanderslice, and Patrick M. Vivier. 2014. “Primary Prevention of Lead Poisoning: Protecting Children from Unsafe Housing.” *American Journal of Public Health* 104 (8): e119–24.

Romieu, Isabelle, Eduardo Palazuelos, Fernando Meneses, and Mauricio Hernandez-Avila. 1992. “Vehicular Traffic as a Determinant of Blood-Lead Levels in Children: A Pilot Study in Mexico City.” *Archives of Environmental Health: An International Journal* 47 (4): 246–49. https://doi.org/10.1080/00039896.1992.9938356.

Ronis, Martin J. J., Jay Gandy, and Thomas Badger. 1998. “ENDOCRINE MECHANISMS UNDERLYING REPRODUCTIVE TOXICITY IN THE DEVELOPING RAT CHRONICALLY EXPOSED TO DIETARY LEAD.” *Journal of Toxicology and Environmental Health, Part A* 54 (2): 77–99. https://doi.org/10.1080/009841098158935.

Rosen, G. 1958. *A History of Public Health*. New York, NY: MD publications.

Rosner, David. 2015. “There Was a Time When If We Worried about the Dangers Posed by Toxins Such as Lead, Cadmium, Mercury, or Asbestos.” *The Milbank Quarterly* 93 (1): 8–11.

Rosner, David, and Gerald Markowitz. 1985. “A Gift of God’?: The Public Health Controversy over Leaded Gasoline during the 1920s.” *American Journal of Public Health* 75 (4): 344–52.

———. 2016. “Building the World That Kills Us: The Politics of Lead, Science, and Polluted Homes, 1970 to 2000.” *Journal of Urban History* 42 (2): 323–45. https://doi.org/10.1177/0096144215623954.

Rothstein, Richard. 2017. *The Color of Law: A Forgotten History of How Our Government Segregated America*. First edition. New York London: Liveright Publishing Corporation, a division of W. W. Norton & Company.

Ruddock, John C. 1924. “Lead Poisoning in Children: With Special Reference to Pica.” *Journal of the American Medical Association* 82 (21): 1682–84.

Rushton, Gerard. 2003. “Public Health, GIS, and Spatial Analytic Tools.” *Annual Review of Public Health* 24: 43–56.

Rushton, Gerard, Marc P. Armstrong, Josephine Gittler, Barry R. Greene, Claire E. Pavlik, Michele M. West, and Dale L. Zimmerman. 2006. “Geocoding in Cancer Research.” *American Journal of Preventive Medicine* 30 (2): S16–24. https://doi.org/10.1016/j.amepre.2005.09.011.

Rutherford, Malcolm. 1994. *Institutions in Economics: The Old and the New Institutionalism*. Historical Perspectives on Modern Economics. Cambridge [England] ; New York, NY, USA: Cambridge University Press.

Sampson, Robert J., and Stephen W. Raudenbush. 1999. “Systematic Social Observation of Public Spaces: A New Look at Disorder in Urban Neighborhoods.” *American Journal of Sociology* 105 (3): 603–51. https://doi.org/10.1086/210356.

Sandel, Megan, Andrea Baeder, Asa Bradman, Jack Hughes, Clifford Mitchell, Richard Shaughnessy, Tim K. Takaro, and David E. Jacobs. 2010. “Housing Interventions and Control of Health-Related Chemical Agents: A Review of the Evidence.” *Journal of Public Health Management and Practice* 16: S24–33. https://doi.org/10.1097/PHH.0b013e3181e3cc2a.

Sanders, Talia, Yiming Liu, Virginia Buchner, and Paul B. Tchounwou. 2009. “Neurotoxic Effects and Biomarkers of Lead Exposure: A Review.” *Review of Environmental Health* 24 (1): 15–45.

Sargent, James D., Mary Jean Brown, Jean L. Freeman, Adrian Bailey, David Goodman, and Daniel H. Freemand. 1995. “Childhood Lead Poisoning in Massachusetts Communities: Its Association with Sociodemographic and Housing Characteristics.” *American Journal of Public Health* 85 (4): 528–34.

Savage, David G. 2018. “Supreme Court Lets Stand California Ruling Holding Lead Paint Makers Liable for $400-Million Cleanup Cost.” *Los Angeles Times*, October 15, 2018, sec. Politics. https://www.latimes.com/politics/la-na-pol-supreme-court-grants-20181015-story.html.

Schafer, Joseph L. 1999. “Multiple Imputation: A Primer.” *Statistical Methods in Medical Research* 8 (1): 3–15. https://doi.org/10.1177/096228029900800102.

Schell, Lawrence M, Melinda Denham, Alice D Stark, Marta Gomez, Julia Ravenscroft, Patrick J Parsons, Aida Aydermir, and Renee Samelson. 2003. “Maternal Blood Lead Concentration, Diet during Pregnancy, and Anthropometry Predict Neonatal Blood Lead in a Socioeconomically Disadvantaged Population.” *Environmental Health Perspectives* 111 (2): 195–200.

Schell, Lawrence M, Melinda Denham, Alice D Stark, Julia Ravenscroft, Patrick Parsons, and Elaine Schulte. 2004. “Relationship between Blood Lead Concentration and Dietary Intakes of Infants from 3 to 12 Months of Age.” *Environmental Research* 96: 264–73. https://doi.org/10.1016/j.envres.2004.02.008.

Schlenker, Thomas L. 1994. “Screening for Pediatric Lead Poisoning: Comparability of Simultaneously Drawn Capillary and Venous Blood Samples.” *JAMA* 271 (17): 1346. https://doi.org/10.1001/jama.1994.03510410058033.

Schuch, Laura, Andrew Curtis, and Joel Davidson. 2017. “Reducing Lead Exposure Risk to Vulnerable Populations: A Proactive Geographic Solution.” *Annals of the American Association of Geographers* 107 (3): 606–24. https://doi.org/10.1080/24694452.2016.1261689.

Schulman, Steven. 1996. “Racial Inequality and Radical Institutionalism: A Research Agenda.” In *Inequality: Radical Institutionalist Views on Race, Gender, Class, and Nation*, edited by William Dugger, 251–73. Contributions in Economics and Economic History 178. Westport Connectecut: Greenwood Press.

Schwarz, K., Steward T.A. Pickett, Richard G. Lathrop, Kathleen C. Weathers, Richard V. Pouyat, and Mary L. Cadenasso. 2012. “The Effects of the Urban Built Environment on the Spatial Distribution of Lead in Residential Soils.” *Environmental Pollution* 163: 32–39.

Sen, Amartya. 1984. “Rights and Capabilities.” In *Resources, Values, and Development*, 307–24. Cambridge, Mass: Harvard University Press.
Settle, Dorothy M., and Clair C. Patterson. 1980. “Lead in Albacore: Guide to Lead Pollution in America.” *Science* 207 (4436): 1167–76.
Shackle, G. L. S. 1967. *Time in Economics*. Professor Dr. F. De Vries Lectures. Amsterdam: North-Holland Publishing Company.
Shao, Liyang, Lianjun Zhang, and Zhen Zhen. 2017. “Exploring Spatially Varying Relationships between Children’s Lead Poisoning and Environmental Factors: Children, Lead Poisoning, and Environmental Factors.” *Annals of the New York Academy of Sciences* 1404 (1): 49–60. https://doi.org/10.1111/nyas.13453.
Silbergeld, Ellen K. 1995. “Annotation: Protection of the Public Interest, Allegations of Scientific Misconduct, and the Needleman Case.” *American Journal of Public Health* 85 (2): 165–66.
———. 2003. “The Unbearable Heaviness of Lead.” *Bulletin of the History of Medicine* 77 (1): 164–71. https://doi.org/10.1353/bhm.2003.0038.
Sonderman, Jennifer S., Michael T. Mumma, Sarah S. Cohen, Elizabeth L. Cope, William J. Blot, and Lisa B. Signorello. 2013. “A Multi-Stage Approach to Maximizing Geocoding Success in a Large Population-Based Cohort Study through Automated and Interactive Processes.” *Geospatial Health* 6 (2): 273–84.
Stefanek, Matthew, Joe Diorio, and Larry Frisch. 2005. “Cost of Child Lead Poisoning to Taxpayers in Mahoning County, Ohio.” *Public Health Reports* 120 (June): 311–16.
Sturgeon, James I. n.d. “Institutions, Ceremonial Valuation and Institutional Theory.” Manuscript. University of Missouri Kansas-City. Accessed July 14, 2016. http://cas.umkc.edu/econ/_resources/OIE/readings/Sturgeon/CereVal.html.
Sui, Daniel. 2014. “Opportunities and Impediments for Open GIS: Opportunities and Impediments for Open GIS.” *Transactions in GIS* 18 (1): 1–24. https://doi.org/10.1111/tgis.12075.
Sutton, P.M., M. Athanasoulis, P. Flessel, G. Guirguis, M. Haan, R. Schlag, and L.R. Goldman. 1995. “Lead Levels in the Household Environment of Children in 3 High-Risk Communities in California.” *Environmental Research* 68 (1): 45–57. https://doi.org/10.1006/enrs.1995.1007.
Swope, Carolyn B., and Diana Hernández. 2019. “Housing as a Determinant of Health Equity: A Conceptual Model.” *Social Science & Medicine* 243 (December): 112571. https://doi.org/10.1016/j.socscimed.2019.112571.
Syracuse Research Corporation. 2007. “User’s Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows.” EPA 9285.7-42. United States Environmental Protection Agency, Office of Superfund Remediation and Technology Innovation.
Teichman, Janis, Dan Coltrin, Karen Prouty, and William A. Bir. 1993. “A Survey of Lead Contamination in Soil Along Interstate 880 Alameda County California.” *American Industrial Hygiene Association Journal* 54 (9): 557–60.
Tepper, Lloyd B. 2007. “Industrial Plumbism: Antiquity to Modern Times.” *IA. The Journal of the Society for Industrial Archeology* 33 (2): 53–66.
Tesh, Sylvia Noble. 1988. *Hidden Arguments: Political Ideology and Disease Prevention Policy*. New Brunswick, N.J: Rutgers University Press.

"The Dust of Buildings in Process of Demolition and Its Prevention." 1903. *The Lancet* 162 (4168): 175.
Thomas Reibling, Ellen, Gregg P. Macey, Xee Her, and Jonathon Ericson. 2001. "An Investigation of Environmental Racism Claims: Testing Environmental Management Approaches with a Geographic Information System." *Environmental Management* 27 (6): 893–907. https://doi.org/10.1007/s002670010197.
Thomson, Hilary, Sian Thomas, Eva Sellstrom, and Mark Petticrew. 2009. "The Health Impacts of Housing Improvement: A Systematic Review of Intervention Studies From 1887 to 2007." *American Journal of Public Health* 99 (S3): S681–92. https://doi.org/10.2105/AJPH.2008.143909.
Tilton, George R. 1998. "Clair Cameron Patterson 1922 - 1995." Biographical Memoir. Washington D.C. http://www.nasonline.org/publications/biographical-memoirs/memoir-pdfs/patterson-clair-c.pdf.
Todorova, Zdravka. 2014. "Consumption as a Social Process." *Journal of Economic Issues* 48 (3): 663–78. https://doi.org/10.2753/JEI0021-3624480304.
———. 2015. "Social Provisioning within a Culture-Nature Life Process." *Review of Political Economy* 27 (3): 390–409. https://doi.org/10.1080/09538259.2015.1058090.
Troesken, Werner, and Patricia Beeson. 2003. "The Significance of Lead Water Mains in American Cities. Some Historical Evidence." In *Health and Labor Force Participation over the Life Cycle: Evidence from the Past*, edited by Dora L. Costa, 181–201. A National Bureau of Economic Research Conference Report. Chicago: University of Chicago Press.
Tullock, Gordon. 1972. "Economic Imperialism." In *Theory of Public Choice*, edited by James M Buchanan and Robert Tollison, first. Ann Arbor: University of Michigan Press.
U.S. Department of Health and Human Services. 2007. "Toxicilogical Profile for Lead." Atlanta, Georgia: Agency for Toxic Substances and Disease Registry.
US EPA, OCSPP. 2013. "Lead Renovation, Repair and Painting Program." Collections and Lists. US EPA. February 12, 2013. https://www.epa.gov/lead/lead-renovation-repair-and-painting-program.
Vaidyanathan, Ambarish, Forrest Staley, Jeffrey Shire, Subrahmanyam Muthukumar, Chinaro Kennedy, Pamela A. Meyer, and Mary Jean Brown. 2009. "Screening for Lead Poisoning: A Geospatial Approach to Determine Testing of Children in At-Risk Neighborhoods." *The Journal of Pediatrics* 154 (3): 409–14. https://doi.org/10.1016/j.jpeds.2008.09.027.
Van Staveren, Irene. 2007. "Beyond Utilitarianism and Deontology: Ethics in Economics." *Review of Political Economy* 19 (1): 21–35. https://doi.org/10.1080/09538250601080776.
Varian, Hal, and Allan Gibbard. 1978. "Economic Models." *The Journal of Philosophy* 75 (11): 664–77.
Vaziri, Nosratola D. 2008. "Mechanisms of Lead-Induced Hypertension and Cardiovascular Disease." *American Journal of Physiology-Heart and Circulatory Physiology* 295 (2): H454–65. https://doi.org/10.1152/ajpheart.00158.2008.

Veblen, Thorstein. 1919. “Why Is Economics Not an Evolutionary Science?” In *The Place of Science in Modern Civilization and Other Essays*, 4th ed., 56–81. New York City: The Viking Press.

Viscusi, W. Kip. 1996. “Economic Foundations of the Current Regulatory Reform Efforts.” *Journal of Economic Perspectives* 10 (3): 119–34.

———. 2005. “The Value of Life.” Discussion Paper. Harvard, John M. Olin Center for Law, Econoimcs, and Business. http://www.law.harvard.edu/programs/olin_center/.

Vivier, Patrick M., Marissa Hauptman, Sherry H. Weitzen, Scott Bell, Daniela N. Quilliam, and John R. Logan. 2011. “The Important Health Impact of Where a Child Lives: Neighborhood Characteristics and the Burden of Lead Poisoning.” *Maternal and Child Health Journal* 15 (8): 1195–1202. https://doi.org/10.1007/s10995-010-0692-6.

Waldman, Irwin D., and Ian R. Gizer. 2006. “The Genetics of Attention Deficit Hyperactivity Disorder.” *Clinical Psychology Review* 26 (4): 396–432. https://doi.org/10.1016/j.cpr.2006.01.007.

Waldron, H.A. 1973. “Lead Poisoning in the Ancient World.” *Medical History* 17 (4): 391–99.

Waller, William T. 1992. “Economic Security and the State.” In *The Stratified State: Radical Institutionalist Theories of Participation and Duality*, edited by William M. Dugger, 153–72. Studies in Institutional Economics. Armonk, NY: Sharpe.

Warren, Christian. 2000. *Brush with Death: A Social History of Lead Poisoning*. Baltimore, Md.: Johns Hopkins University Press.

Wei, Evelyn, Alison Hipwell, Dustin Pardini, Jennifer M Beyers, and Rolf Loeber. 2005. “Block Observations of Neighbourhood Physical Disorder Are Associated with Neighbourhood Crime, Firearm Injuries and Deaths, and Teen Births.” *Journal of Epidemiology & Community Health* 59 (10): 904–8. https://doi.org/10.1136/jech.2004.027060.

Whelan, Elizabeth A., Greg M. Piacitelli, Barbara Gerwel, Teresa Schnorr, Charles Muller, Janie Gittleman, and Thomas Matte. 1997. “Elevated Blood Lead Levels in Children of Construction Workers.” *American Journal of Public Health* 87 (8): 1352–55.

Whitsel, E. A. 2004. “Accuracy and Repeatability of Commercial Geocoding.” *American Journal of Epidemiology* 160 (10): 1023–29. https://doi.org/10.1093/aje/kwh310.

Wickham, Hadley, and Garrett Grolemund. 2016. *R for Data Science: Import, Tidy, Transform, Visualize, and Model Data*. First edition. Sebastopol, CA: O’Reilly.

Wilson, Benjamin, Natalie Kane, Neal Wilson, Peter Eaton, and Doug Bowles. 2018. “Housing, Health and History: Interdisciplinary Spatial Analysis in Pursuit of Equity for Future Generations.” In *Intergenerational Responsibility in the 21st Century*, edited by Julia M. Puaschunder, 77–103. Series in Economic Development. Wilmington, Delaware: Vernon Press.

Wilson, Benjamin, Neal Wilson, and Sierra Martin. 2019. “Using GIS to Advance Social Economics Research: Geocoding, Aggregation, and Spatial Thinking.” *Forum for Social Economics*, January, 1–25. https://doi.org/10.1080/07360932.2018.1509798.

Wilson, Ian Harold, and Simon Barton Wilson. 2016. “Confounding and Causation in the Epidemiology of Lead.” *International Journal of Environmental Health Research* 26 (5–6): 467–82. https://doi.org/10.1080/09603123.2016.1161179.

Wilson, Jonathan, Sherry L. Dixon, David E. Jacobs, Judith Akoto, Katrina S. Korfmacher, and Jill Breysse. 2015. “An Investigation into Porch Dust Lead Levels.” *Environmental Research* 137 (February): 129–35. https://doi.org/10.1016/j.envres.2014.11.013.

Wilson, Neal. 2018a. “Modeling Childhood Blood Lead Levels with Detailed Housing Characteristics.” CEI Working Paper 1801–03. Kansas City, MO: Center For Economic Information.

———. 2018b. “The Useful Metal, the Aping Disease, and the Institution of Childhood Lead Poisoning.” In *Annual Meeting of the Association For Institutional Thought*. San Antonio Texas.

Yeoh, B, S Woolfenden, D Wheeler, G Alperstein, and B Lanphear. 2006. “Household Interventions for Prevention of Domestic Lead Exposure in Children.” In *Cochrane Database of Systematic Reviews*, edited by The Cochrane Collaboration. Chichester, UK: John Wiley & Sons, Ltd. https://doi.org/10.1002/14651858.CD006047.

Yin, Li, and Robert Silverman. 2015. “Housing Abandonment and Demolition: Exploring the Use of Micro-Level and Multi-Year Models.” *ISPRS International Journal of Geo-Information* 4 (3): 1184–1200. https://doi.org/10.3390/ijgi4031184.

Zahran, Sammy, Terrence Iverson, Shawn P. McElmurry, and Stephan Weiler. 2017. “The Effect of Leaded Aviation Gasoline on Blood Lead in Children.” *Journal of the Association of Environmental and Resource Economists* 4 (2): 575–610. https://doi.org/10.1086/691686.

Zahran, Sammy, Mark A. S. Laidlaw, Shawn P. McElmurry, Gabriel M. Filippelli, and Mark Taylor. 2013. “Linking Source and Effect: Resuspended Soil Lead, Air Lead, and Children’s Blood Lead Levels in Detroit, Michigan.” *Environmental Science & Technology* 47 (6): 2839–45. https://doi.org/10.1021/es303854c.

Zandbergen, P. 2009. “Geocoding Quality and Implications for Spatial Analysis.” *Geography Compass* 3 (2): 547–68.

Zein-Elabdin, E. O. 2009. “Economics, Postcolonial Theory and the Problem of Culture: Institutional Analysis and Hybridity.” *Cambridge Journal of Economics* 33 (6): 1153–67. https://doi.org/10.1093/cje/bep040.

Zenk, Shannon N., Amy J. Schulz, Graciela Mentz, James S. House, Clarence C. Gravlee, Patricia Y. Miranda, Patricia Miller, and Srimathi Kannan. 2007. “Inter-Rater and Test–Retest Reliability: Methods and Results for the Neighborhood Observational Checklist.” *Health & Place* 13 (2): 452–65. https://doi.org/10.1016/j.healthplace.2006.05.003.

Zhang, Chaosheng, Lin Luo, Weilin Xu, and Valerie Ledwith. 2008. “Use of Local Moran’s I and GIS to Identify Pollution Hotspots of Pb in Urban Soils of Galway, Ireland.” *Science of The Total Environment* 398 (1–3): 212–21. https://doi.org/10.1016/j.scitotenv.2008.03.011.

Zhou, Ying, Tursynbek Nurmagambetov, Matthew McCord, and Wan-Hsiang Hsu. 2017. “Economic Valuation of Selected Illnesses in Environmental Public Health Tracking:” *Journal of Public Health Management and Practice* 23: S18–27. https://doi.org/10.1097/PHH.0000000000000641.

Ziliak, Stephen Thomas, and Deirdre N. McCloskey. 2008. *The Cult of Statistical Significance: How the Standard Error Costs Us Jobs, Justice, and Lives*. Economics, Cognition, and Society. Ann Arbor: University of Michigan Press.
Zivin, Joshua Graff, and Matthew Neidell. 2013. "Environment, Health, and Human Capital." *Journal of Economic Literature* 51 (3): 689–730.

VITA

Neal James Wilson was born November 9, 1976 in Chicago, Illinois. He was educated in public schools graduating from high school in Ithaca, New York. He enrolled at the University of Missouri–Kansas City as a non-traditional student, graduating with a Bachelor of Science in Economics in 2005.

While enrolled as an undergraduate Mr. Wilson began to work as a craft baker, a profession he would remain in for over a decade. During this time he co-founded the Museum of Bottled Water. In the wake of the slow recovery from the 2008 global financial crisis Mr. Wilson enrolled in the masters program at University of Missouri-Kansas City and was awarded the Master of Arts degree in Economics in May 2014.

Since 2014 Mr. Wilson has presented at over a dozen academic conferences, organized a conference of interdisciplinary research, co-authored one book chapter and another peer reviewed article. Starting in 2015 he has been a research consultant with the Center for Economic Information, participating three grant funded projects investigating the social determinants of health. He is currently project manager of the HUD funded Impact Lead-Kansas City grant which extends a line of inquiry suggested by his dissertation research. He plans to keep this position until the close of the grant in December 2023. Mr. Wilson has taught Economics at the college level since 2016. Starting in 2019 he has been special faculty in the School of Critical Studies teaching economics at The California Institute of the Arts.