NBER WORKING PAPER SERIES

LEAD EXPOSURE AND BEHAVIOR: EFFECTS ON ANTISOCIAL AND RISKY BEHAVIOR AMONG CHILDREN AND ADOLESCENTS

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Working Paper 20366
http://www.nber.org/papers/w20366

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
August 2014

I would like to thank Claudia Goldin, Jun Ishii, Lawrence Katz, Ronnie Levin, Erzo Luttmer, René Reyes, Steven Rivkin, Katharine Sims, and seminar participants at Amherst College, ClarkUniversity, Harvard University, RAND, the University of Massachusetts, the University of Delaware, APPAM, the University of Rochester, and the Childhood Lead Poisoning Prevention Program in Rochester, New York for valuable advice and comments. Many individuals at government agencies and petroleum industry companies generously provided information on lead in gasoline, and Jenny Ying shared data on teen pregnancy policies. Steve Trask provided excellent research assistance. Any remaining errors are my own. This research was supported by Amherst College and the National Bureau of Economic Research. The views expressed herein are those of the author and do not necessarily reflect the views of the National Bureau of Economic Research.

Lead Exposure and Behavior: Effects on Antisocial and Risky Behavior among Children and Adolescents

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NBER Working Paper No. 20366
August 2014
JEL No. I18,J13,K49,Q53,Q58

ABSTRACT

It is well known that exposure to lead has numerous adverse effects on behavior and development. Using data on two cohorts of children from the NLSY, this paper investigates the effect of early childhood lead exposure on behavior problems from childhood through early adulthood. I find large negative consequences of early childhood lead exposure, in the form of an unfolding series of adverse behavioral outcomes: behavior problems as a child, pregnancy and aggression as a teen, and criminal behavior as a young adult. At the levels of lead that were the norm in United States until the late 1980s, estimated elasticities of these behaviors with respect to lead range between 0.1 and 1.0.

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I. Introduction

A growing body of literature argues that social scientists should cast a wider lens by considering early life experiences and environments as potentially important determinants of later life outcomes. Almond and Currie (2010), for example, cite abundant results showing that events in early childhood can have long-lasting impacts on adult outcomes, largely through effects on development of cognitive and non-cognitive skills.[1] Characteristics such as the ability to learn well, to listen, to cooperate, and to be respectful of others are established early in life and are foundational elements of human capital. On the other hand, characteristics such as easy distractability, aggression, and a short temper are counterproductive. There is an emerging consensus that formative early life influences may substantially shape adult behavior and outcomes.

This paper investigates the effect of early childhood lead exposure on a variety of social and behavioral outcomes from childhood through young adulthood. The central idea is that children exposed to even moderate amounts of lead, a toxic metal, will be more likely to exhibit behavior problems in childhood, to engage in risky behavior in the teenage years, or engage in violent or criminal behavior in young adulthood. More broadly, I argue that environmental toxicants and social problems may be closely linked, and that elucidating these links may provide insight both into the understanding of social problems and also the potential of public health and environmental policy to improve social health.

Why should lead be considered as a possible factor affecting social behavior at the societal level? Lead is a hazardous neurotoxicant with a wide range of adverse effects on human health and behavior, such as decreasing IQ, increasing learning disabilities, behavior problems, and aggressive behavior, as well as adversely affecting cardiovascular functioning and other physiological processes. It is reasonably well established that lead has particularly strong behavioral effects on young children, and that some of these effects persist as individuals age. It is also the case that leaded gasoline provided the major source of lead exposure for the U.S. population through the early 1980s, that individual lead levels were high enough to expect broad societal effects on health and behavior, and that these lead levels declined drastically as lead was

[1] See also: Case and Paxson (2010), Lochner and Moretti (2004), Heckman et al. (2006), Katz et al. (2001), Graff Zivin and Neidell (2013).

phased out of gasoline. Together, these facts indicate that one might expect lead to have had substantial effects on behavior at the societal level, and substantial effects on temporal changes in such behavior. Moreover, they suggest that one might be able to discern the effects of lead on behavior by studying cohorts of children born during the phaseout of lead from gasoline.

During the late 1970s in the United States, lead was removed from gasoline under the Clean Air Act. This phaseout of lead from gasoline (primarily between 1975 and 1985) did not occur in a uniform fashion throughout the country, providing a useful natural experiment. To measure childhood lead exposure, I employ detailed data on the lead content of gasoline (from a variety of government sources) as well as individual-level data on children's blood lead levels from the second National Health and Nutrition Examination Survey (NHANES II). To measure behavioral outcomes, I use National Longitudinal Survey of Youth (NLSY) data on two cohorts born during the phaseout of lead from gasoline: a cohort of 3,833 children of the NLSY 1979 sample women (born between 1979 and 1985) and a cohort of 7,889 children from the NLSY 1997 (born between 1980 and 1984). The primary analysis includes these individuals, for whom gasoline lead content is used as an instrument to predict childhood blood lead in a split-sample instrumental variables strategy. Outcomes considered include childhood behavior problems, teen sexual behavior and pregnancy, teen substance abuse, aggression, and crime.

This investigation of a broad range of adverse effects of lead is distinguished by its scale, scope, and identification. In scale: I use observations on more than 10,000 individuals, drawn from nationally representative samples. In scope: I investigate effects on a wide range of behavioral outcomes, at multiple ages, and with a range of social implications. In identification: I employ a plausibly exogenous change in childhood lead exposure in order to identify a causal impact of lead on social behavior. Thus, while many earlier investigations employed smaller samples or investigated a narrower range of effects, this paper contributes to a growing literature that uses novel non-experimental methods to investigate broad health and social effects of environmental exposures. The specific aim of this paper is to employ careful individual-level analysis to understand lead's broad impact on social behavior in the United States.

The paper proceeds as follows. Section II provides background on social behavior, lead, and the relationship between the two. Section III sets out a simple theory by which lead may affect behavior. Section IV outlines the data employed, and Section V describes the empirical approach. Sections VI and VII discuss the results. Section VIII provides interpretation, and

Section IX concludes.

II. Behavior and Lead

Behavior

Problems such as attention-deficit and hyperactivity disorder (ADHD), teenage pregnancy, substance abuse, and crime impose large costs on society and pose challenges to policymakers. Estimates indicate that approximately 20% of children and adolescents in the U.S. display symptoms of behavioral and mental disorders, 8% of children have learning disabilities, and 7% of children have ADHD.[2] Other common disruptions to behavior include aggressivity, disinhibition, and impulsivity. Moreover, these childhood problems often serve as precursors to a variety of mental health problems, risky behaviors, and antisocial behaviors in later life.

The types of adolescent behaviors that may be implicated include early sexual activity, teenage pregnancy, substance abuse, and juvenile delinquency. Impulsivity has been linked to teen sex, teen pregnancy, and substance abuse.[3] Loeber (1990) argues that decreased levels of impulse control by American children are largely responsible for the increasing prevalence of antisocial and delinquent behavior among juveniles. Richardson (2000) writes that "the ADHD brain has problems putting on the brakes and controlling actions" and that "rage and violence are often life-long problems for people with untreated ADHD."[4] ADHD, currently the most common chronic mental health problem among children, has been shown to reduce test scores and to increase the probability of delinquency.[5] Recent evidence indicates that more than two-thirds of juvenile delinquents meet diagnostic criteria for one or more psychiatric disorders,[6] and three independent studies find that children with ADHD are five times more likely to be delinquent than children without ADHD.[7] More generally, behavior issues in childhood can substantially impair accumulation of human capital and social capital, leading to a broad range of

[2] U.S. Department of Health and Human Services (1999). National Institutes of Mental Health (1996).
[3] Gruber (2001), Hoffman and Maynard (2008), Maynard (1996), Sweeney et al. (2000).
[4] Fishbein (2000).
[5] Currie and Stabile (2006); Visser et al. (2014).
[6] Teplin et al. (2002).
[7] Studying a group of 678 thirteen-year-olds, Moffitt and Silva (1988) report that, controlling for other factors, 58% of ADHD children became delinquent, compared with only 10% of non-ADHD children. Satterfield (1987) finds that ADHD children were six times more likely to be arrested for at least one serious offense as a teenager. Dalsgaard (2003) finds that children with ADHD are five times more likely to be convicted of any crime by age 30 and twelve times more likely to be convicted of a violent crime.

effects.

While the prevalence of many of these behaviors prompts concern, it is their substantial trends that have particularly puzzled analysts. Child behavior problems appear to have risen in prevalence and severity in the last twenty or thirty years, although changing attitudes and definitions make it difficult to track trends accurately or directly. Substance abuse rates show a different trend, having peaked in the late 1970s, declined until the early 1990s, and risen mildly since then.[8] Teenage pregnancy rates rose until the early 1990s and declined substantially thereafter, but trends vary across states and racial groups.[9] Juvenile delinquency, criminal behavior, and violence show similar trends to that of teen pregnancy, peaking in the early 1990s and exhibiting sharp declines thereafter. While a number of plausible explanations for these trends have been posited, understanding these behaviors, their trends, and the role of policy remain active areas of inquiry.[10]

Lead

Lead is an extremely useful metal but unfortunately has also proved to be a dangerous toxin, particularly for young children who are at a sensitive stage of their neurobehavioral development.[11] It is now widely accepted that lead easily crosses the blood-brain barrier[12], that lead exposure can be detrimental even at extremely low levels[13], and that the adverse neurological effects of lead persist into older ages.[14] Historically, the main environmental sources of lead exposure for the average child have been leaded gasoline, lead-based paint, and lead water pipes.

While paint is a major source of environmental lead exposure, it is not as readily absorbed as lead from gasoline nor did it experience drastic changes in the time period under consideration. The possible danger from lead paint hazards in older housing and in central cities

[8] Johnston et al. (2009); Markowitz et al. (2005).
[9] Hoffman and Maynard (2008).
[10] See, e.g., Levitt and Donohue (2001), Levitt (2004).
[11] Children also absorb lead more efficiently from their environment. Hammond (1988) reports that children absorb up to 50% of lead they ingest, compared with 8% for adults. Bellinger (2004) provides a good review of the differences in lead absorption between children and adults.
[12] Costa et al. (2004).
[13] It is important to distinguish between lead exposure and lead poisoning. Lead exposure is simply exposure to some level of lead. Lead poisoning encompasses a certain set of symptoms and occurs at particularly high levels of exposure (usually blood lead levels in excess of 25 μg/dL). This paper is primarily concerned with lead exposure, not lead poisoning.
[14] Bellinger (2008).

should not be downplayed; it is just not possible to assess their effects with the empirical strategy employed in this paper. [15] Likewise, the importance of water mains and pipes as a source of lead exposure is becoming more evident, but there are few easily-observed drastic changes and consequently diminished opportunities for identification of these effects.[16]

The current paper will focus on exposure to lead from gasoline sources, taking advantage of a unique opportunity provided by U.S. environmental history and policy: the phaseout of lead from gasoline in the 1970s and early 1980s. Lead was first added to gasoline in the late 1920s to boost engine power, and the lead content of gasoline rose throughout the middle part of the century, remaining high until the 1970s. As Devra Davis writes, "[f]ormulating gasoline with liquid lead had indeed created a massive delivery system for spreading an insidious poison into the lungs, hearts, and brains of millions."[17] Lead from gasoline can be absorbed into an individual's body directly from breathing in gasoline exhaust from the air or indirectly via contact with lead deposits that have accumulated in soil. During the years when gasoline contained high levels of lead, gasoline represented "the most ubiquitous source of lead found in the air, dust, and dirt in urban areas"[18] and blood lead levels were highly correlated with recent gasoline lead consumption.[19]

In December 1973, under the authorization of the Clean Air Act, the EPA mandated a timetable for the reduction of lead in gasoline, requiring petroleum companies to meet certain targets of maximum grams of lead per gallon of gasoline. The average lead content of the gasoline produced by each refinery was to be reduced from 2.0 grams per total gallon to a maximum of 0.5 grams per total gallon by 1979. Over the next decade, as scientific, political, and legal battles played out, the timetable was delayed slightly and further reductions were implemented. [20] The reduction was aided by the advent of catalytic converters, originally introduced in the early 1970s to limit sulfur emissions, but which coincidentally limited lead

[15] The lead content of paint declined relatively smoothly from 1920 on, with breaks in 1950 when lead-based paint was banned for interior use and in 1978 when it was banned for all residential uses. The primary danger since 1970 stems from older housing with deteriorating paint; children absorb lead from paint sources directly when they eat paint chips or indirectly when deteriorating paint creates lead dust.
[16] See Troesken (2006) on the economic history of lead in water pipes.
[17] Davis (2002), p.67.
[18] U.S. Environmental Protection Agency (1973).
[19] Schwartz and Pitcher (1989).
[20] There are several excellent detailed accounts of the history of lead regulation in the United States: Warren (2000), Markowitz and Rosner (2013).

emissions because they were fouled by leaded gasoline. Ultimately, the phaseout was a success: gasoline lead dropped by 99% between 1975 and 1990.[21] The National Health and Nutrition Examination Survey (NHANES) confirmed that the reduction in lead emissions was closely associated with corresponding large reductions in the blood lead levels of Americans in all demographic groups.[22] The entire distribution shifted downward, and the national mean dropped from 16 μg/dL in 1976 to 3 μg/dL in 1991.[23] This decline in lead exposure from gasoline sources between 1975 and 1985, resulting from the Clean Air Act, will be the main source of identification for the current analysis.

Lead and Behavior

The association between lead exposure during early development and subsequent deficits in cognitive development and behavior is widely accepted. Neurological research indicates that exposure to lead impairs brain development (particularly the prefrontal corex, hippocampus, basal ganglia, and cerebellum) and damages neurotransmitter systems (particularly the dopaminergic, glutamatergic, and cholinergic pathways) in ways that impair cognition, attention, and impulse control.[24] Early childhood exposure (before age 6) appears to be most harmful to psychological and cognitive development, and there is evidence that these effects persist to a great degree.[25]

Increased lead levels are significantly associated with decreased mental skills, including reduced IQ, reduced verbal competence, increased reading disabilities, and reduced academic

[21] U.S. Environmental Protection Agency (1998). Between 1975 and 1990, all measures of lead exposure (on-road vehicle emissions, other emissions, air lead, gasoline lead, and blood lead) declined drastically and in concert with one another. Between 1975 and 1990, total lead emissions declined by 97% and gasoline lead went from the dominant source of lead (80% of emissions) to a minor source (8% of emissions).
[22] U.S. Environmental Protection Agency (1991), Brody et al. (1994).
[23] Bodily lead levels can be measured in blood, teeth, or bone. A blood lead level is the concentration of lead in blood, and is measured in micrograms per deciliter (μg/dL) or micromoles per liter (μmol/L). Blood lead is a good measure of recent exposure, while dentine and bone lead better reflect cumulative lifetime exposure.
[24] Goodlad et al. (2013), Costa et al. (2004), Cory-Slechta (1995). The EPA's 2013 *Integrated Science Assessment for Lead* provides the following explanation for the biological mechanisms for lead's effects: "A number of mechanisms, including changes in neurogenesis, synaptogenesis and synaptic pruning, long term potentiation, and neurotransmitter function have been identified that provide biological plausibility for epidemiologic and toxicological findings of persistent cognitive and behavioral effects that result from Pb [lead] exposures during prenatal and early childhood periods. Furthermore, the normal dynamic and rapid rate of development that occurs early in life in the CNS [central nervous system] makes insults early in life especially problematic in that they can permanently change the trajectory of brain development such that there are little or no compensatory pathways to replace the lost potential for proper brain development (Bayer, 1989)." (EPA (2013) pg. 1-77.)
[25] Banks et al. (1997), Bellinger (2008), Needleman et al. (1996), Environmental Protection Agency (2013).

performance.[26] Canfield, Henderson et al. (2003) report an IQ decrement of 0.74 of a point per 1 μg/dL of blood lead for blood lead below 10 μg/dL, and a smaller decrement of 0.25 above 10 μg/dL.[27] To put this in context, this means that the decrease in average blood lead for young children in the 1980s (from 15 to 4 μg/dL)[28] would produce about a 6 point increase in IQ, or about one-third of a standard deviation. Moreover, the EPA (2013) review of the literature finds that deficits persist even after blood lead levels decline. Coscia, Ris et al. (2003) argue that by contributing to weak verbal, reading, and other abilities, lead exposure "deflects such youth's development in an antisocial direction."

While much research has focused on IQ effects, many studies have also found strong associations between higher lead levels and aggressive behavior, impulsivity, hyperactivity, and attention impairment.[29] Early in this literature, Needleman and Bellinger (1981) reported that children with above-average (but still moderate) lead levels are more than three times as likely to be distractible, hyperactive, impulsive, and to have low overall functioning. More recently, Goodlad et al. (2013) perform a meta-analysis of 33 studies showing that lead is significantly associated with ADHD symptoms (inattention and hyperactivity/impulsivity), with correlations of 0.16 and 0.13 respectively. Similarly, Marcus, Fulton and Clarke (2010) review the literature on lead and conduct problems, reporting a significant correlation of 0.19 for conduct problems such as conduct disorder, oppositional defiant disorder, and delinquency from their meta-analysis. In addition, a number of studies have found higher lead levels among children who are hyperactive or who have other behavior problems, and among juvenile delinquents and criminals.[30] Research in psychology and neurotoxicology has also established direct links between higher childhood lead exposure and a greater likelihood of adult criminal behavior.[31] These studies and the meta-analyses thereof make substantial efforts to control for confounding factors, but of course are limited in their ability to definitely establish causality. They do, however, consistently find statistically significant effects in widely varying contexts and at a

[26] Bryce-Smith (1983).
[27] An early meta-analysis by Needleman and Gatsonis (1990) reports a lead-IQ correlation of -0.15, similar to the correlations reported for ADHD and conduct problems.
[28] U.S. Centers for Disease Control and Prevention (CDC) (1994).
[29] Wilson and Petersilia (1995), Needleman et al. (1990), Needleman and Gatsonis (1991), Banks et al. (1997).
[30] Denno (1990); Needleman (1985). Needleman et al. (2002) show that adjudicated delinquents were four times as likely to have high lead levels than non-delinquents. Masters et al. (1998) and Bryce-Smith (1983) show that violent criminals exhibit higher levels of lead in their bodies than non-violent criminals or the general population.
[31] Denno (1990); Needleman et al. (1996); Dietrich et al. (2001); Wright et al. (2008).

wide range of lead levels.

In 2013, the Environmental Protection Agency conducted an exhaustive *Integrated Science Assessment for Lead*, drawing on thousands of scientific studies to detail lead's causal effects on a range of health outcomes. With regard to the outcomes of interest in this paper, they conclude the following:

> Whether prospective or cross-sectional or previously reviewed or recently published, most of the epidemiologic studies found associations between childhood blood or tooth Pb levels and attention decrements, impulsivity, and hyperactivity (Figure 4-9 and Table 4-11). *EPA (2013), p. 4-187*

> Collectively, the evidence from prospective studies indicated associations of teacher and parental ratings of aggressive, antisocial, and delinquent behavior with biomarkers of cumulative Pb exposure, i.e., age 0-6 year average blood, lifetime (to ages 4-5 or 11-13 years) average blood, tooth, and bone Pb level. *EPA (2013), p. 4-183*

The EPA report also confirms a "common finding" that lead's cognitive and behavioral effects are non-linear, with marginal effects that are larger at lower lead levels. However, the report finds no clear explanation for this particular dose-response shape.

Recently, a growing literature has employed non-experimental data to establish lead's adverse effects. Investigating IQ and cognitive impairment, Ferrie, Rolf and Troesken (2012) link lead in water to lower intelligence test scores for American army enlistees during World War II; Rau, Reyes and Urzua (2012) study effects on academic performance in Chile; Nilsson (2009) studies the cognitive and labor market effects of the removal of lead from gasoline in Sweden; and McLaine, Navas-Acien et al. (2013) and Reyes (2011) show effects on school test scores in Rhode Island and Massachusetts respectively. Investigating lead and crime, social scientists have contributed to the discussion with a variety of identification strategies: Mielke and Zahran (2012) use a small panel of U.S. cities, Reyes (2007) uses a full panel of U.S. states, Nevin (2000) uses a single U.S. time series, Nevin (2007) uses a panel of developed countries, and Masters et al. (1998) use a cross section of U.S. states. (Nevin (2000) also investigates effects on teen pregnancy.) Overall, the literature suggests that there may be a substantial elasticity of crime with respect to lead, likely between 0.2 and 0.8.

In sum, a diverse multi-disciplinary literature reaches the consensus that early childhood lead exposure negatively affects cognitive development and behavior in ways that increase the

likelihood of aggressive and antisocial acts. These links to behavior pertain even at the moderate lead levels (5-15 μg/dL) that were common in the U.S. through the early 1980s. I note that, while the medical or biological science literature makes compelling broad arguments for how and why lead should affect behavior, a good portion of the work investigating individual outcomes uses smaller, selected, populations or focuses on relatively narrow sets of outcomes. This paper aims to add to the literature by using a large nationally-representative dataset to investigate a broad set of outcomes over a range of ages. The goal is to form a coherent picture of lead's full effects on individuals and thereby understand the extent and nature of lead's broad consequences for society. Several authors, including Gould (2009), have endeavored to calculate the full costs of lead by extrapolating from individual scientific papers. The current paper aims to advance our understanding by establishing lead's societal effects *directly* (rather than via extrapolation). In a sense, this is analogous to moving from *in vitro* analysis to *in vivo* analysis, if the society is the living organism under consideration.

III. Theory

This paper aims to study a causal link between childhood lead exposure and various aspects of social behavior. While the details of the underlying physiological mechanism by which lead impairs neurodevelopment and behavior are beyond the scope of this paper, the broad outlines of that mechanism can be expressed within the framework of the standard economic model of individual behavior.

First, I write the performance outcome P as a function of ability, characteristics, and environment:

$$P = p(A,B,X,F,C) \; . \qquad (1)$$

The variables A and B represent cognitive and behavioral (non-cognitive) ability, respectively. The variables X, F, *and* C, respectively represent vectors of individual, family, and community characteristics. It is assumed that these characteristics will have direct, indirect, and interactive effects on P. For example, family income would enter directly as a family characteristic but might also influence other characteristics or ability.

Lead exposure is modeled as a function of family and community characteristics, and is also influenced by government policy G and any protective behavior H undertaken by the family:

$$L = l(F,C,G,H) \; . \qquad (2)$$

Lead exposure would, in turn, affect both types of ability directly:

$$A = a(L,X,F,C) \qquad \text{and} \qquad B = b(L,X,F,C). \qquad (3)$$

The inclusion of the various characteristics indicates that those characteristics can modify both the lead level resulting from a given exposure and the effect of that lead level on ability. Ultimately, performance is a complex function of characteristics, behaviors, and policies:

$$\begin{aligned} P &= p\,(A\,,B\,,X\,,F\,,C\,) \qquad (4) \\ &= p\,(a(L,X,F,C)\,,b(L,X,F,C)\,,X\,,F\,,C\,) \\ &= p\,(a(l(F,C,G,H),X,F,C),\, b(l(F,C,G,H),X,F,C)\,,X\,,F\,,C\,)\,. \end{aligned}$$

It is apparent that the precise structure of these relationships is too intricate to be determined by means of a simplified reduced-form model of this type. However, several simple qualitative hypotheses can be stated. If the performance outcome P is a positive outcome (an economic good), then:

$\partial P/\partial B > 0$	Higher ability causes higher performance
$\partial B/\partial L < 0$	Higher lead level causes lower ability
$\partial P/\partial L < 0$	Higher lead level causes lower performance (follows via the chain rule from $\partial B/\partial L < 0$ and $\partial P/\partial B < 0$).
$\partial P/\partial G > 0$	Government policy that reduces lead improves performance (follows via the chain rule from $\partial L/\partial G < 0$ and $\partial P/\partial L < 0$).
$\partial^2 P / \partial L^2 > 0$	Marginal effect of lead declines with lead

The middle three are the main hypotheses that will be tested by this paper. The last is suggested by the results of Canfield et al. (2003) and others, but is theoretically more ambiguous. Lastly, we note that the foregoing theory describes the effects of an individual child's lead on that individual child's outcome. It is also possible that there may be spillover effects of lead through peer effects as group behavior deteriorates.

IV. Data

The National Longitudinal Surveys of Youth (NLSY) provide a rich set of longitudinal data on two cohorts of individuals born during the phaseout of lead from gasoline. These will provide the primary data for this analysis. Data from various sources on blood lead and gasoline

lead will provide measures of lead exposure. Lastly, a variety of other data sources will provide control variables relevant to specific outcomes.

NLSY 79 Child and Young Adult

The NLSY 79 surveyed a nationally-representative sample of 12,686 individuals who were between 14 and 21 years of age in 1978.[32] Individuals in the NLSY79 sample were surveyed annually or biannually since 1979. Beginning in 1986, the children of the women of the NLSY79 were interviewed biannually in the NLSY79 Child and Young Adult Surveys (NLSY79 CHYA).[33] It is estimated that the NLSY79 CHYA surveys include 90% of the children born to the NLSY79 women. It is important to note that, while the NLSY79 CHYA sample can be a nationally representative sample of the children of these women, it is not itself a nationally representative sample of children more generally. The most obvious potential bias, widely noted in the literature, is that children born in earlier years were born to younger mothers, many of them teens. Analysis in this paper will include the 3,833 children born between 1979 and 1985, and will address this possible bias with controls for mother's age at birth and child's birth cohort. Together, the Child and Young Adult Surveys provide a rich set of longitudinal data. Individual observations can be linked to the maternal and family demographics collected in the main NLSY79.

The NLSY79 Child Supplement includes additional information on child behavior, school performance, test scores, sexual activity, and substance abuse. Child behavior is measured using the Child Behavior Problems Index (BPI), which evaluates "the frequency, range, and type of childhood behavior problems for children age four and over."[34] It is constructed from a set of 32 questions answered by a parent or teacher about child behavior, including items such as whether a child argues too much, acts impulsively, bullies others, or is depressed. The answer is given qualitatively, as "never," "sometimes," or "often." Sub-scales of the BPI can be created by summing groups of items to represent certain aspects of behavior; the

[32] U.S. Bureau of Labor Statistics (2005), Baker et al. (1993).
[33] *NLSY79 Child & Young Adult Data User's Guide*, Center for Human Research of The Ohio State University (2004).
[34] Ibid, p.78, Zill (1985), Zill (1990). See also Achenbach and Edelbrock (1979) for a discussion of the BPI's predecessor, the more extensive Child Behavior Checklist.

Oppositional, Antisocial, Headstrong, and Hyperactive Subscales are employed in this paper.[35] A list of BPI items and subscales is shown in Appendix Table 1. The NLSY79 Child Supplements also contain additional data on risky behavior in the pre-teen years. These include the use of licit or illicit substances, such as alcohol, cigarettes, marijuana, or hard drugs. The NLSY79 Child supplements also contain limited data on aggressive behavior, including whether the individual hurt someone else badly enough that that person needed medical attention.

The NLSY79 Young Adult Surveys overlap with and extend the NLSY79 Child Surveys. The Young Adult Surveys start at age 15 or 16. They provide more comprehensive self-reported data on sexual activity, substance abuse, and criminal activity. The sexual activity data includes the dates of any pregnancies, but does not include substantial other information about sexual activity. The substance use data is similar to that in the Child Surveys. The additional data that is most relevant to the present analysis is the self-reported data on aggressive or criminal behavior, including whether the teenager hit or threatened to hit someone, hurt someone badly enough to need a doctor, or was convicted of a crime.[36]

The main strengths of the NLSY 79 CHYA are the detail of the child behavior variables and the availability of at least some data on teen risky and criminal behavior. The main weaknesses are twofold. First, the sample may not be nationally representative of each birth cohort, particularly because earlier births are, by construction, to younger mothers. Second, the data is collected only every two years, and some of the self-reported retrospective teen data may not be as reliable as we would like. The NLSY97, which I discuss now, addresses some of these deficiencies but brings others of its own.

NLSY 97

The National Longitudinal Survey of Youth 1997 (NLSY97) surveyed a nationally-representative sample of 8,984 individuals who were born between 1980 and 1984.[37] This

[35] Each behavior problems index or subscale is calculated by assigning, for each behavior problem item, a value of 0 to "never," 1 to "sometimes," and 2 to "often", and summing those values over the relevant set of behaviors.
[36] The NLSY79 Young Adult Surveys contain the following categories for crime convictions: assault, robbery, theft, fencing stolen goods, destruction of property, other property offenses, possession of marijuana, selling of marijuana, possession of illicit drugs, sale and manufacture of drugs, major traffic offenses, drinking under age, and miscellaneous other offenses.
[37] Two subsamples comprise the NLSY97 cohort: a nationally-representative cross-section of 6,748 individuals and a supplemental oversample of 2,236 Hispanic or Latino and black individuals. See the *NLSY97 User's Guide*, Center for Human Research of The Ohio State University (2005).

analysis employs the 7,889 individuals who were born in the United States. Individuals in the NLSY97 were first surveyed in 1997 (at ages 12 through 17), and were surveyed annually thereafter. Attrition after 3 rounds was below 10%, and after 7 rounds was below 15%. Like the NLSY79 CHYA, the NLSY97 provides detailed data on demographics, sexual activity, substance abuse, and criminal activity. The NLSY97 also provides some limited retrospective data about childhood behavior, via a scaled ranking of the individual's behavioral or emotional problems as a child. Where the NLSY97 adds value is in measuring teen risky behavior, aggressive behavior, and criminal behavior. There are detailed questions about the age at first sexual activity, pregnancies, and the use of alcohol, cigarettes, and marijuana. There are also questions about when and whether the individual attacked someone intentionally with the goal of hurting or fighting. The crime data include arrests, charges, and convictions with some detail about particular categories of offenses.[38]

The NLSY97 offers several advantages over the NLSY79 CHYA. First, it provides a larger and nationally-representative sample. Second, it contains more thorough and annual data on activity in the teen years. However it also has some disadvantages: it contains only limited information on the childhood years, some of the teen data is based on recall of events several years prior, the sample does not include those born in the earliest part of the phaseout of lead from gasoline, and selective attrition could potentially bias some results.[39]

Thus, used together, these two primary NLS datasets should be able to provide a reasonably complete, if imperfect, picture of behavior in the childhood and teen years. The variables from both the NLSY79 CHYA and the NLSY97 are summarized in Table 1. The demographic characteristics confirm that the NLSY97 sample is closer to nationally-representative than the NLSY79 CHYA sample, with, for example, a sample that is 71% white and has an average family income of $51,000. We also get a sense of the prevalence of behavior problems, with the main index showing that the average child exhibits 9 to 10 problem behaviors, while the subscales show the average severity of particular aspects of behavior. The

[38] The NLSY97 contains the following categories for crime charges and convictions: assault, burglary, theft, robbery, destruction of property, possession of drugs, sale of drugs, public order offense, major traffic offense, other property offense, other offense.

[39] A preliminary investigation reveals that the sample that does drop out appears to have had more behavior problems in childhood, possibly indicating that those with worse outcomes are more likely to drop out. More detailed analysis comparing those who do not drop out of the survey with those who do may be warranted, and could shed light on these issues.

data on teen risky behavior is as expected, with rates of teen pregnancy around 15 to 25% and rates of substance use that range from one-quarter to one-half. The data on aggressive behavior is perhaps most surprising, showing that the likelihood of hitting, attacking, or hurting someone reaches almost one-third by age 17. Lastly, behavior that rises to the level of officially criminal is less likely, with a 23% arrest rate, a 13% rate of being charged, and conviction rates around 10% for any type of crime.

Lead

Data on individual blood lead concentrations comes from the second National Health and Nutrition Examination Survey (NHANES II). The NHANES II measured blood lead concentrations of a nationally representative sample of 9,372 individuals, including 2,322 children under the age of 6, in the years 1976 to 1980. The blood lead concentrations were obtained through direct physical examination, and measure the actual concentration of lead in an individual's blood (in micrograms per deciliter of blood, denoted μg/dL). The NHANES II data include geographic location, individual demographics (such as age, race, and family income) and the exact date on which the blood sample was taken. Table 2 shows the distribution of blood lead over the period 1976 to 1980. It is clear that the entire distribution of blood lead shifted downward during this time period, with the mean and median dropping by half, or 9 μg/dL. The NHANES III data, which is not used directly in this analysis, confirms that this swift downward movement continued in the 1980s.

Data on gasoline lead concentration at the state-month level is constructed from a number of government and industry sources. In the period 1975 to 1985, four grades of gasoline were consumed (regular unleaded, premium unleaded, regular leaded, and premium leaded). The shares consumed and the lead concentration of the grades differed from state to state. I calculate the average grams of lead per gallon of gasoline in a given state in a given month by summing over grades the product of the share of that grade and the grams per gallon of that grade. *The Yearly Reports of Gasoline Sales by States* provide data on the shares of the grades of gasoline, and *Petroleum Products Surveys* provide data on the lead concentrations.[40]

Table 2 shows the distribution of gasoline lead concentrations over the period under

[40]Ethyl Corporation (1976-1984). National Institute for Petroleum Energy Research and BETC (1947-1989). The measurement of gasoline lead is discussed at length in Reyes (2007).

consideration. Gasoline lead dropped swiftly between 1976 and 1980, and continued to decline in the early 1980s. The maps in Figure 1 show the geographic variation of gasoline lead in 1979, 1981, and 1983. In each year, a state is colored based on the deviation of its lead concentration from the national average lead concentration in that year. The figure therefore nets out the national trend, displaying the remaining substantial variation of gasoline lead across states and over time. We see that states move at different rates, some leading, some lagging, and some starting out ahead and then falling behind. It is this variation across states in the timing of the lead phaseout that will be used to identify lead's effects.

These data on lead in blood and lead in gasoline will be used together. The next section outlines the strategy employed to estimate early childhood blood lead for individual children born during the phaseout of lead from gasoline.

Other Data

Control variables are gathered from a variety of other sources, primarily the U.S. Bureau of the Census and other government agencies.[41] For teen pregnancy, an additional vector of state-level control variables includes the minimum wage, maximum welfare benefit, and indicator variables for welfare reform (either via a waiver under AFDC or a TANF reform), a parental living arrangement requirement in welfare, and parental involvement requirement for teenage abortion. For crime, an additional vector of state-level covariates includes the state per-capita income, the poverty rate, the unemployment rate, a concealed weapons law, lagged police per capita, and lagged prisoners per capita.

[41] Sources for these variables are as follows. Income, Poverty, Minimum Wage, Unemployment, and Population are from the Statistical Abstract of the United States, published annually by the U.S. Bureau of Census. Income is converted into 2000 dollars. The unemployment rate represents the percent unemployed among civilian non-institutional population 16 years of age and older. The poverty rate is the percentage of the state population below the federal poverty line as defined in that year. Data on gun laws is from Lott and Mustard (1997) and has been updated appropriately. The variable indicates whether the state had a non-discretionary concealed handgun law in that year. Such a law requires local law enforcement authorities to grant concealed weapons permits to anyone meeting certain pre-established criteria. Data on police is from the Uniform Crime Reports for the United States, published annually by the Federal Bureau of Investigation. It is measured as police per 1000 population and is lagged by one year. Data on prisoners is from Correctional Populations in the United States, published annually by the Bureau of Justice Statistics. It is measured as prisoners per 1000 population and is lagged by one year. Data on Waiver and TANF implementation dates and minor parent living arrangement requirements are from The State Policy Documentation Project (SPDP). and Crouse (1999) "State Implementation of Major Changes to Welfare Policies, 1992-1998." Data on parental involvement requirements for teenage abortion are from the Alan Guttmacher Institute.

V. Empirical Approach

The goal of this paper is to identify a causal effect of childhood lead exposure on behavior. I model the propensity to exhibit a certain behavioral trait as a function of childhood blood lead. The basic regression equation is thus:

$$\text{Prob (outcome)}_i = \alpha_1 \text{BloodLead}_i + \boldsymbol{X}_i \boldsymbol{\beta} + \varepsilon_i \ . \quad (5)$$

where $\boldsymbol{X}_i$ is a vector of individual demographics. While the NLSY provides a rich set of behavioral outcomes for a nationally representative sample of individuals born during the phaseout of lead from gasoline, it does not include direct measures of the childhood blood lead of those individuals. However, this gap can be filled: by employing a straightforward split-sample instrumental variables strategy, the childhood blood lead of the NLSY individuals can be estimated using the NHANES II blood lead data.

I use the gasoline lead data and the NHANES blood lead data to estimate the following equation on the sample of young children in the NHANES in the period 1976 to 1980:

$$\text{BloodLead}_i = \gamma \text{GasLead}_i + \boldsymbol{Y}_i \boldsymbol{\theta} + \varepsilon_i \ . \quad (6)$$

where $\boldsymbol{Y}_i$ is a vector of individual demographics during childhood: age, gender, race, and income.[42] I then use the data on the relevant gasoline lead concentration for each individual in the NLSY samples (matched to that individual by age, year, and state), together with the coefficients estimated from Equation 6, to predict the childhood blood lead concentration for each of the NLSY individuals:

$$\widehat{\text{BloodLead}}_i = \hat{\gamma} \text{GasLead}_i + \boldsymbol{Y}_i \hat{\boldsymbol{\theta}} \ . \quad (7)$$

This yields a predicted childhood blood lead concentration for each individual in the NLSY samples.[43] I can then investigate how this (predicted) measure of childhood blood lead affects the propensity to exhibit certain behavioral traits later in life, by putting it into Equation 5 in place of the (unobservable) actual childhood blood lead:

$$\text{Prob (outcome)}_i = \alpha_1 \widehat{\text{BloodLead}}_i + X_i \boldsymbol{\beta} + \varepsilon_i \ . \quad (8)$$

This strategy merits some discussion. First I address the choice of the measure of

[42] Income is broken into categories based on the federal poverty line (FPL): less than twice the FPL (low), two to three times the FPL (middle), and more than three times the FPL (high). This was done in response to the need to find a common set of demographic variables in the NHANES and NLSY.

[43] Note that the first-stage regression of blood lead on gasoline lead can be varied in a number of ways: it can be done on the full sample of young children, or on subsets by smaller aging groupings; the variables included as controls can be changed; fixed effects for state and year can be included or excluded.

gasoline lead: grams per gallon. Gasoline was a major source of lead exposure circa 1980, and changes in gasoline lead concentrations were induced by the Clean Air Act. While one could consider using the share of unleaded gasoline or several other measures, grams per gallon is well-suited to serve as a measure of gasoline lead exposure in this analysis for several reasons. First, EPA policy specifically targeted grams per gallon. Second, this policy was imposed on petroleum companies, not states. Therefore, the changes in grams per gallon at the state level were induced by national environmental policy but – because they were mediated through company decisions – were not endogenous to state environmental policy. Consequently, the cross-state variation in the phaseout is most evident in the cross-state variation in grams per gallon. Moreover, the variation resulted not from state policy or state-specific EPA policy, but rather from the interaction between the broad policy application and a variety of features of the petroleum and auto industries, such as the network of pipelines, setup of gas stations, or age of the stock of cars.[44] Through the 1970s and 1980s, the lead industry fought aggressively to halt or slow regulations on lead, and historical accounts depict a fight that took place at the national level. Markowitz and Rosner (2013) write that "[w]hat had been considered a narrow concern of local public health officials by the 1980s had become of national interest to more than sixteen federal agencies…" (p.122). Historians make almost no mention of state- or region-specific lobbying that might have influenced state or regional trends in the phaseout of lead. For these reasons, grams of lead per gallon appears to have experienced substantial and largely random reductions in the period 1975 to 1985, reductions that varied significantly from state to state and that were induced by EPA policy.[45] The substantial variation in gasoline lead across states and over time is shown in Figure 1.

Gasoline lead concentration would not matter, however, unless that lead from gasoline somehow found its way into children's blood. Existing work verifies a strong relationship between gasoline lead and blood lead during the time period in question.[46] Blood lead

[44] The network of petroleum pipelines delivered gasoline with different lead contents to different regions of the country. Even within a region, the lead content of different grades of gasoline (regular, midgrade, premium, superpremium) differed significantly (by as much as 50%). Demand for the different grades of gasoline also varied with consumer preference and with the age of the stock of cars (which also varied with climate). Even the number of gasoline pumps available at gas stations affected the path of the introduction of unleaded gasoline, and particularly the phaseout of high-lead premium gasoline between 1979 and 1980. Sources: Gibbs (1990), Gibbs (1993), Gibbs (1996) and Gibbs personal communication.

[45] See Reyes (2007) for a detailed discussion of the merits of different ways of measuring lead.

[46] Schwartz and Pitcher (1989), Sweeney et al. (2000), Billick et al. (1980).

concentration is the best measure of the lead that is present in a child's body at a young age, and therefore the best measure of the lead that has the potential to affect an individual's development or behavior later in life. By modeling the relationship between gasoline lead and blood lead, taking account of other possible factors, the methodology just outlined produces a reliable estimate of a child's blood lead, incorporating the variation that arises from exposure to lead from gasoline sources in different contexts and demographic circumstances.

Several factors make this strategy feasible in the present circumstance. First, the NHANES II provides blood lead measures in the 1976-1980 time period, which also happens to be the first half of the phaseout of lead from gasoline. Second, the NLSY79 CHYA and NLSY97 provide two cohorts of individuals born between 1979 and 1985, which happens to be the second half of the phaseout. Third, various sources provide data on gasoline lead concentrations for the 1976 to 1985 time period, the entire phaseout. Thus, the proximity and overlap of these time periods, combined with the availability of detailed geographic codes in all of the data sets, renders the split-sample instrumental variables strategy a feasible means by which the NHANES blood lead data and the gasoline lead data can be used to estimate childhood blood lead for the NLSY individuals.

The present paper applies this methodology to the NLSY79 CHYA and the NLSY97. I investigate effects of lead on child behavior problems, teen risky behavior, aggressive behavior, and criminal behavior. The basic specification is as follows:

$$\text{Prob(outcome)}_{ist} = \alpha\, \text{PredictedBloodLead}_i + \boldsymbol{X}_i\boldsymbol{\beta} + \mu_i + \gamma_i + \mu_i \times \gamma_i + \rho_{ist} + \varepsilon_{ist} \quad (9)$$

The outcomes are of various kinds and are measured at various ages – from child behavior problems at age 8 through to pregnancy at age 18, etc. In contrast, the predicted blood lead is estimated at a single period in time – the individual's lead-sensitive early childhood years, age 0 to 3. To be perfectly clear: α, the coefficient of interest, measures the effect of an individual's blood lead in early childhood on that individual's behavior later in life (be it 5 years later at age 8, or 15 years later at age 18.) An α significantly different from zero indicates a significant effect of early childhood lead exposure on later life behavior.

The indices identify an individual i, state s, and year t. The vector $\boldsymbol{X}$ includes dummies for the child's age, gender, race or ethnicity (black, Hispanic, or other), mother having graduated

from high school, mother being a teen mother, and a continuous variable for real family income.[47] The μ_i are categories for the mother's age at the child's birth and the γ_i are categories for the child's birth cohort.[48] The inclusion of these dummy variables accounts for persistent differences that might arise between different groups of mothers or different birth cohorts. This is especially important for the NLSY79 CHYA sample, which has a disproportionate share of young mothers. The ρ_{ist} are dummies for census region of residence at the time of the outcome being considered.[49]

For teen pregnancy, an additional vector of state-level control variables includes the minimum wage, maximum welfare benefit, and indicator variables for welfare reform, a parental living arrangement requirement in welfare, and parental involvement requirement for teenage abortion. For crime, similarly, an additional vector of state-level covariates includes per-capita income, the poverty rate, the unemployment rate, a concealed weapons law, lagged police per capita, and lagged prisoners per capita.

The specification for Equation 9 is chosen to match the outcome under consideration: ordinary least squares for a continuous variable, probit for a binary variable, ordered probit for an ordered categorical variable, or Tobit for a censored continuous variable. Because of the variety of outcomes and these specifications, I will discuss most results as the elasticity of the outcome under consideration with respect to lead.[50] Standard errors are Huber-White robust and

[47] Family income is measured in year 2000 dollars. For individuals with multiple observations, it is calculated as the family's median income during the observed portion of childhood.

[48] Categories for mother's age at child's birth are: under 17 years old, 17 to 19, 20 to 22, and 23 or above. Categories for child's birth cohort are in two-year groupings for the NLSY79 CHYA and single years for the NLSY97.

[49] Inclusion of state fixed effects would not leave enough variation for reliable identification. About one-quarter of the states have fewer than 30 observations, which means they have fewer than 5 observations in a given year.

[50] The ordered categorical model merits particular attention. Individual behavior items are rated in three categories: "never," "sometimes," or "often." For example: the child never acts impulsively, sometimes acts impulsively, or often acts impulsively. Linear regression is inappropriate in this situation because the distances between the categories are unknown. An ordered probit model is appropriate:

$y_i^* = \mathbf{x}_i \boldsymbol{\beta} + \varepsilon_i$ (y^* is the latent variable)

$y_i = m$ if $\tau_{m-1} \leq y_i^* < \tau_m$ for $m = 1$ to J

$Prob(y = m \mid \mathbf{x}) = \Phi(\tau_m - \mathbf{x}\boldsymbol{\beta}) - \Phi(\tau_{m-1} - \mathbf{x}\boldsymbol{\beta})$

Such a model requires careful interpretation: the coefficients are not meaningful in isolation. If an independent variable increases the latent dependent variable, shifting the probability distribution rightward across the outcome categories, it may shift probability out of the lowest cell (or leave the cell unchanged) and shift probability into the right-most cell (or leave the cell unchanged). However, its effects on the middle cells are ambiguous, since mass will shift in and out across the multiple interior cutpoints. Consequently, analysis should examine elasticities across particular boundaries and changes in probability of different outcomes in response to changes in the independent variable. For more detail, see Greene (2008) or Long and Freese (2006).

clustered on region or state of residence, as appropriate. Standard errors are also adjusted via bootstrap for the fact that the main independent variable of interest, blood lead, was itself predicted in the first stage.[51] In addition, to investigate possible variation in the functional form of lead's effects on outcomes, I estimated results using the level of blood lead, the natural log of blood lead, and a spline of blood lead with cutpoints at 5 μg/dL and 10 μg/dL. Because preliminary analysis suggested that the marginal effect of lead was declining in many cases, and the spline generally did not provide enough improvement to justify its additional complexity, the log specification will be employed as the primary specification. Results with other specifications will be discussed as appropriate.

VI. Results: Predicting Blood Lead

I perform OLS regressions, following Equation 6, of blood lead on gasoline lead on the sample of children under the age of 6 with blood lead measures in the NHANES II in the 1976-1980 period. Recall that blood lead and gasoline lead can be linked by state, year, and month. Table 3 shows these results, where the vector of controls includes age, indicator variables for gender, race, and income category (income up to two times the federal poverty line, income between two and three times the federal poverty line, and income above three times the federal poverty line), as well as state fixed effects. Gasoline lead is a strong predictor of blood lead: one gram of lead per gallon of gasoline increases blood lead by 4.13 μg/dL. Subsequent columns show variations on this base specification. The effect of gasoline lead almost doubles when state fixed effects are included and drops somewhat upon the subsequent addition of year fixed effects. The coefficient changes slightly when race or income categories are excluded (columns 4 and 5). Supplementary investigations indicate that, while the effect of lead exposure on blood lead is evident for all age groups, it is strongest for young children. This can be seen in the final column of the table, showing a coefficient of 7.69 (standard error of 1.93) when the sample is restricted to children aged 2 to 4 years. Further results also show little sensitivity to functional

[51] Because predicted blood lead is derived using estimated coefficients for equation (6), the traditional standard error estimated for predicted blood lead in equation (9) would be a lower bound for the true standard error. To correct for this, I bootstrap the first stage, deriving 100 different estimates for predicted blood lead using 100 different subsamples of the NHANES, and running the equation (9) regression separately for each of those 100 estimates. I then integrate the 100 resulting coefficient distributions, and calculate the point estimate and standard error from the resulting distribution.

form, for example using the log of grams per gallon instead of the level. Overall, gasoline lead concentration appears to be a robust predictor of children's blood lead concentration in the years 1976 to 1980.

I employ the coefficients obtained via this approach, running Equation 6 on an NHANES sample matched within a centered 3-year age window, to predict childhood blood lead at each year of age for the individuals in the NLSY samples. (The specification employed is the one shown in column 3 of Table 3.) From this, I calculate early childhood blood lead as the average of predicted childhood blood lead over ages 0 to 3 for each child. For the NLSY79 CHYA sample, the mean predicted early childhood blood lead is 7.7 μg/dL; the 25th percentile is 4.8, the median is 7.0, and the 75th percentile is 9.8. For the NLSY97 sample, the mean predicted early childhood blood lead is 7.7; the 25th percentile is 5.4, the median is 7.0, and the 75th percentile is 9.3. These values can be seen in Table 2. The predictive power of gasoline lead in explaining blood lead is substantial: gasoline lead is able to explain a decline of 3.4 μg/dL in the average blood lead between 1976 and 1980, or half of the national change over this time period (even though year dummies were included in the regression).[52]

VII. Results: Behavior

I now investigate how childhood blood lead is related to childhood behavior problems, teen risky behavior, and aggressive and criminal behavior. This section discusses the results of regressing various outcomes on an individual's predicted early childhood blood lead (age 0 to 3). Most results will be discussed as elasticities, measured at a blood lead of 7.5 μg/dL, which is close to the mean and median of predicted blood lead in the sample. This enables easy comparison of the magnitude of effects across different outcomes as well as different econometric specifications. Results using both the NLSY 79 and 97 will be discussed together, but it will be noted from which sample specific results derive.

Preliminary Results

Before discussing the primary regression analysis, it is worthwhile to take a preliminary look at the relationship between lead and the various outcomes to be considered. Figure 2 graphs

[52] The actual reduction in blood lead between 1976 and 1980 was 6.5 μg/dL. Changes in gasoline lead (from 1.8 gpg to 0.7 gpg) predict a reduction of 3.4 μg/dL over this time period.

three composite outcomes for behavior, teen pregnancy, and violence against two measures of lead (blood lead and gasoline lead). Each datapoint represents a cohort defined by state of birth and birth year, for the years 1979 to 1985. The graphs show significant positive associations between lead and these outcomes. While the regression analysis below will control for a wide variety of fixed effects and other factors influencing these outcomes, it is still helpful to see that these raw relationships support the hypothesis that lead affects behavior.

Childhood Behavior Problems

Table 4 shows the full regression results for the total behavior problems index, for children age 4 to 12 in the NLSY 79. In the linear specification, the coefficient on blood lead is 0.13, highly significant with a standard error of 0.06. This corresponds to an elasticity of 0.07 at a blood lead of 5, an elasticity of 0.12 at a blood lead of 7.5, and an elasticity 0.15 at a blood lead of 11. The estimated elasticities are similar for the log and spline specifications, and there is evidence that the effect of lead is indeed non-linear. Higher lead does appear to cause more behavior problems in childhood.

Several of the other covariates in these specifications show the expected effects. Girls exhibit fewer behavior problems, with approximately 10% better behavior than boys. Having a mother who finished high school (rather than one who did not finish high school) is associated with almost 20% fewer behavior problems, *ceteris paribus*. Higher income groups show slightly fewer behavior problems as well. These effects are all right-signed and of reasonable magnitude. To get a sense of the size of the lead effect, I note that an increase of 1 μg/dl of blood lead has approximately the same effect on behavior as a decrease of approximately $5,000 of family income. This is substantial.

However, the effects of black race and teen mother also appear to decrease behavior problems, which is the opposite sign of what we would expect. The teen mother effect is explained by the fact that the regression also includes a set of dummies for mother's age at child's birth, child's birth cohort, and their interactions. I will instead focus on the unexpected black race result, for which I see two possible interpretations. First, when blood lead is appropriately controlled, black race may, in fact, be associated with decreased rather than increased behavior problems. In this scenario, the apparent "effect" of black race that is expected would, in reality, be due largely to higher lead levels among blacks. This seems

unlikely, or at least not a conclusion to be drawn lightly. Second, it could be that blacks happen to have higher lead levels and also happen to have more behavior problems, though it may not be causally linked. Since black race is included as an independent variable in the first stage prediction of blood lead, this odd result could arise due to this confounding. One way to distinguish between these two possibilities, and to come closer to determining the true effect of lead and the true effect of black race, is to omit black race from the first-stage prediction of blood lead but still include it in the second stage. In a sense, this will yield a conservative estimate of the effect of lead *per se*, purging the lead coefficient of any racially-mediated effects (even if those effects are indeed real). When these regressions are run, the coefficient on lead is reduced by approximately one-third and the coefficient on black race has a modest right-signed effect on behavior problems. These results indicate that the effect of lead *per se* on behavior problems is indeed real: lead is not merely serving as a proxy for race. However, the moderate drop in the coefficient on lead lends some support to the possibility that lead is responsible for some portion of the higher levels of behavior problems observed among black children.

Table 5a shows the elasticities with respect to blood lead of a number of additional behavior problems subscales. The first row shows the total behavior problems index discussed above. The second row shows results for a fifteen-item scale of "oppositional action," as defined by Cooksey, Menaghan and Jekielek (1997) for use with the NLSY79 Child Sample. The next three rows show three of the classic BPI subscales for antisocial, hyperactive, and headstrong behavior. Each includes 5 or 6 behaviors, and they are mutually exclusive. They include behaviors such as bullying others, breaking things, arguing too much, having a strong temper, or having difficulty concentrating. Details are shown in Appendix Table 1. The last row is the only one showing results from the NLSY 97 data; it includes the retrospective assessment of behavioral or emotional problems in childhood (the single child behavior variable available in the NLSY 97).

Overall, these results are consistent with those for total behavior problems. For children age 4 to 12, the elasticities are significant and hover around 0.1. For children age 6 to 7 – between Kindergarten and second grade – the elasticities are insignificant.[53] It is possible that

[53] Because the NLSY 79 CHYA are done every other year, children are surveyed either when they are 6 or when they are 7, but not both. Similarly, they are observed either when they are 11 or 12, but not both. I have therefore grouped children into two-year age groupings in order to focus on particular ages.

many of these behaviors – such as cheating, being easily confused, bullying, or obsessive thinking – are either not yet developed or are less apparent in children so young. In contrast, for children age 8 to 9 all of the indices show substantial elasticities, and for children age 11 to 12 all of the indices show right-signed elasticities, with some significant. The estimated elasticities for children age 8 to 9 – between the 3rd and 4th grades – are generally just over 0.2, with the significant ones ranging from 0.22 to 0.27.[54] Running these specifications separately for boys and girls, and I find somewhat stronger effects for boys, who exhibit significant and larger elasticities in both the 8 to 9 and 11 to 12 age groups, while girls show effects only at age 8 to 9. Overall, these results imply that a 10% increase in blood lead is associated with a 1% reduction in child behavior problems. Accordingly, the 50% decline in blood lead that occurred in this time period would be associated with a 5% decline in behavior problems.

Beyond investigating general behavior problems, or even specific categories of behavior, I can try to look for effects on particular behaviors that might be especially influenced by lead. This is ambitious, placing a large burden on subjective reports of detailed aspects of behavior. One of the challenges in investigating lead's effects on behavior is that the scientific literature does not provide a clear-cut "fingerprint" of lead; there does not appear to be a consensus on what behaviors lead should or should not affect. Table 5b shows results for three behaviors that the literature suggests *might* be particularly affected by lead: being impulsive or acting without thinking, bullying or being cruel and/or mean to others, and having a very strong temper or losing one's temper easily. Note that separate elasticities are shown not only for each behavior but also for *sometimes* exhibiting the behavior (rather than never) and for *often* exhibiting the behavior (rather than sometimes).

The results are mixed, but they suggest that lead increases the likelihood of these behaviors, particularly in later childhood. The estimated elasticities in the log specification are insignificant and right-signed for "impulsive" and "bully" in the full age sample. On the other hand, strong temper shows significant and large effects in the full age sample: the elasticity for moving from "never" to "sometimes" is 0.10, and for moving from "sometimes" to "often" is 0.25. In the spline specification, shown in Appendix Table 2b, the elasticities are significant and

[54] Elasticities from the spline specification are shown in Appendix Table 2a. They confirm effects of lead on child behavior problems, although the results do differ: estimated elasticities for all ages are about twice as large, those for ages 6 to 7 are larger (but still insignificant), those for ages 8 to 9 are now insignificant, and those for ages 11 to 12 are much larger and consistently significant.

large for "impulsive" (0.59 for "often") and "strong temper" (0.75 for "often"), and larger but still insignificant for "bully".

The next portion of Table 5b breaks this down further into separate age groups, showing some significant effects for bullying and strong temper among the 8 to 9 age group, and generally right signed and insignificant effects for the others. (Impulsive behavior among the youngest age group (ages 6 to 7) shows wrong-signed significant effects, the only such estimates in the table). For children 8 and up, the elasticity of sometimes bullying other children (rather than never doing so) is estimated to be between 0.0 and 0.5, while the elasticity for often bullying other children (rather than sometimes) is estimated to be approximately twice as large, between 0.2 and 1.0. For strong temper at ages 8 to 9, the estimated elasticities are between 0.0 and 0.4 for "sometimes," and 0.1 and 1.0 for "often."

In the spline specification, shown in Appendix Table 2b, the elasticities are significant and large in the 11 to 12 age group for all three behaviors on both margins. The estimated elasticities for the spline range between 0.3 and 1.9; they are highest for "often" exhibiting these behaviors and for bullying. Further investigation separating boys and girls reveals that for the most part these individual item results are driven by the boys, who exhibit significant elasticities for all three behaviors – 0.1 to 0.4 for sometimes, and 0.5 to 1.0 for often – while the girls show no significant results.

These results are robust to the use of alternate methods of predicting blood lead. Overall, they provide supportive evidence that higher lead increases the prevalence of these outwardly aggressive and antisocial behaviors, particularly as children get older.

Teen Risky Behavior

Moving on to the teen years, Table 6 shows results for teen risky behavior: sexual activity, pregnancy, and substance use. In most cases, these variables are available in both the NLSY79 CHYA and the NLSY97. We see a large and significant elasticity of 4.3 for having sex by age 13; the estimated elasticity is very similar for both boys and girls. There are also substantial effects for girls on being pregnant as a teenager, either by age 17 or age 19; the estimated elasticities are between 0.6 and 1.1, and are reasonably consistent between the two samples. The last measure of sexual activity is whether males report having gotten a partner pregnant as a teen, and this shows an elasticity of 1.7. It is important to note that this variable

probably suffers from mis-reporting by males; note the gender gap between these variables in Table 1.[55] Further sensitivity testing of two different functional forms and the inclusion or exclusion of additional covariates in the first or second stage indicates that these results are quite robust. The elasticities are still in the range of 0.5 to 1.0 or more, and almost all remain highly significant.

The practical magnitude of the effects on early sex and teen pregnancy can be assessed by estimating changes in the probability of these outcomes that would be induced by certain changes in blood lead. To be specific, we can consider the change in probability associated with a change in blood lead from 15 μg/dl to 5 μg/dl, a change that approximates the population-wide reduction that resulted from the phaseout of lead from gasoline. This calculation yields a predicted 12 percentage point decrease in the likelihood of pregnancy by age 17, and a 24 percentage point decrease in the likelihood of pregnancy by age 19 (from a 40% chance to a 16% chance). This is undoubtedly large: the lead decrease reduces the likelihood of teen pregnancy by more than half. It is also sizeable when compared to the prevalence of teen pregnancy in the sample. This suggests that changes in lead could potentially be responsible for some of the decline in teen pregnancy in the 1990s, a decline which has proved difficult to explain.

The final rows of Table 6 show results for substance use as a teen: use of alcohol by age 13, cigarettes by age 13, and marijuana by age 17. These are less consistent, but do provide support for an effect of lead on these behaviors. In particular, early consumption of alcohol shows an elasticity of 0.2 to 0.6. These results are somewhat stronger for boys, who exhibit significant elasticities of approximately one-third for early drinking and smoking in both samples. Given the large and growing literature on the adverse and "gateway" effects of early alcohol use, these results merit further investigation in future work.

[55] Most of the teen behavior variables in the NLSY are constructed from self-reports. In the absence of other corroborative reports, one cannot make detailed statements about potential bias from using such self-reports. However, the gap between male and female reports of teen pregnancy is certainly substantial and worth noting; it is plausible that it results not only from age gaps but from the greater ability of males, for obvious reasons, to answer "no" when the true answer may be "yes." In addition, many of the teen risky behavior results could potentially be refined by accounting for who is present at the interview – whether the individual youth was alone, or whether he or she was accompanied by a parent or guardian, sibling, or peer. It has been suggested elsewhere that the presence of these other individuals influences reporting of teen risky behaviors.

Aggression, Violence, and Criminal Behavior

I now turn to consider impacts on aggression, violence, and criminal behavior. Table 7 shows these elasticities, combining results that draw on slightly different questions available in the two NLSY samples. I first consider effects on aggression and violence: hitting, hurting, or attacking others. The elasticity of hitting or threatening to hit someone, either by age 15 or by age 17, is estimated to be 0.4 to 0.5. This appears to translate, though not completely, into actually hurting someone badly enough that they require medical attention: the elasticity for that outcome is 0.31. Similarly, the estimated elasticities for attacking someone by ages 13 or 17 are, respectively, 0.50 and 0.36. Results for other ages are similar but are not shown in the table. Interestingly, girls show stronger effects for hitting (elasticities of 0.8), while boys exhibit more consistent and larger effects for the more serious behaviors of hurting and attacking (elasticities as high as 1.0).

Next we turn to considering lead's effects on criminal arrests, charges, and convictions. It is important to keep two issues in mind when interpreting these results. First, measures of criminal activity in the NLSY are from individual self-reports which may be subject to underreporting or misreporting. Second, there is a great deal of leakage in the criminal justice system from the commission of a criminal act to arrest, charge, and conviction: only a small percentage of individuals who commit a particular criminal act are actually convicted of having done so, and that places a heavy burden on a sample that is just shy of 8,000 individuals. (For any given birth state, there are probably fewer than ten individuals in the sample who have been convicted of a crime.)

In Table 7, being arrested or charged with a crime show elasticities of 0.37 and 0.51, respectively, while being convicted of a crime has an elasticity of 0.64 in the NLSY97 and 0.60 in the NLSY79. These are sizable elasticities, suggesting a substantial effect of early childhood blood lead on criminal behavior as a teenager. To assess effects on more specific crime categories, I construct two (non-comprehensive) sub-categories: violent crime, comprised of assault and robbery, and property crime, comprised of theft, burglary, destruction of property, and other property offenses.[56] For violent crime, the results are insignificant. For property crime, the elasticity is significant in the NLSY79 sample but not in the NLSY97 sample.

[56] These two categories do not include drug crimes, traffic offenses, and other offenses to public order.

Alternate specifications, linear or a piecewise linear spline, yield similar results: while the elasticities are reduced by a third or half, the notable differences are lower standard errors for violent crime and now statistically significant elasticities for property crime in the NLSY97 in addition to the NLSY79. Given the questions raised by these sub-category results, further work will focus more specifically on criminal behavior in order to test the robustness of these crime results in greater detail.

Robustness

The above analysis and discussion incorporates a fair number of alternate outcomes, and also addresses a number of common potential concerns. In this section, I would like to address several additional queries. First, as a validity check for the methodology of using predicted blood lead, I test whether the parent's outcomes are affected by her child's predicted blood lead. Using analogous specifications to those above and looking at maternal outcomes available in the data prior to 1980 (and therefore prior to the child's birth), I find no significant impact of the child's blood lead on: the number of times the mother attacked someone else, the mother having been stopped by the police for something other than a minor traffic offense, and the mother's age of first consumption of alcohol. The point estimates are small and the p-values all exceed 0.50. This falsification test helps confirm that the child's blood lead is not merely proxying for something else about the mother or family. It thereby provides indirect evidence for the split-sample IV approach employed above.

Second, to further test the robustness of the IV approach, I try using gasoline lead directly as the lead measure. As explained above, when a child is exposed to lead from gasoline sources, that exposure is mediated through a range of environmental and demographic factors. By accounting for both the initial gasoline lead and this process of exposure, the paper's main strategy of using a first stage to predict blood lead should produce a more useful and appropriate measure of the relevant biological lead level of each child. Consequently, gasoline lead, while a valuable measure, is also an incomplete one; but it can be useful for testing robustness. Doing this, I find the results to be mostly robust for child behavior and teen risky behavior, and mostly not robust for aggression and crime. The results for behavior are similar (though smaller) for boys; there are fewer results for girls. Results for teen risky behavior are still statistically significant and robust, though slightly smaller. Results for aggression and crime are smaller and

many become insignificant, with the significance fading as one moves to older ages and more severe activities. Taken together, while these results show some sensitivity, for the most part they provide further support for the paper's main hypothesis.

Third, we might wonder whether lead contributes to socioeconomic differences in behavioral outcomes, or whether lead's effects may differ for different demographic groups. In the intellectual history of lead, these are fascinating and charged questions. Herbert Needleman received an especially belligerent response from the lead industry when his 1979 article in the *New England Journal of Medicine* suggested that lead was an "equal-opportunity pollutant, the effects of which were not limited to poor African-American children."[57]

To investigate these factors in the NLSY data, I perform the above analyses separated by parental education (less than high school vs. high school vs. college or more), income (less than twice the poverty line vs. more than thrice the poverty line), and race/ethnicity (black or Hispanic vs. white). I find that, while *all* children are harmed by lead, advantaged groups are harmed *more* by lead. The estimated effects of lead are larger and more consistently significant for children whose parents are more highly educated, whose families have higher income, or who are white. For the education and income breakdowns, this divergence between advantaged groups and disadvantaged groups is particularly apparent when looking at lead's effects on child behavior problems.

In order to understand this result, recall that lead from gasoline was ubiquitous in the 1980s: it was in the very air children breathed, and everyone was affected regardless of income, education, or race. While children in more advantaged families might have been protected from many of the adverse environmental or social influences that children in disadvantaged families had to contend with, they were *not* protected from gasoline lead. Thus, whereas for the disadvantaged children lead may have been just one more adverse influence (on top of numerous others), for many of the advantaged children it was perhaps the only or the primary adverse influence. In a way, the advantaged children had more to lose. Consequently, gasoline lead may have been an equalizer of sorts. Note that the story for paint lead may be substantially different, since paint exposures are likely to follow the familiar pattern whereby the disadvantaged suffer greater exposure and the advantaged are largely insulated.

[57] Needleman (1979). The quote is from p. 127 of *When Smoke Ran Like Water* by Devra Davis.

VIII. Interpretation

The above analysis indicates that higher childhood lead exposure is associated with substantial adverse behavioral consequences from childhood through young adulthood. Very approximately, the key elasticities with respect to blood lead are 0.1 for child behavior problems, 0.8 for teenage pregnancy, 0.4 for teenage aggressive behavior, and 0.5 for teenage criminal behavior. These estimates are generally robust: to different functional forms and measurements of lead, as well as to the inclusion of individual characteristics, family characteristics, additional controls that might affect specific outcomes, and an extensive set of dummy variables to control for maternal cohort, child cohort, and regional effects. Together, they paint a picture that exposure to lead in early childhood triggers an unfolding series of adverse behavioral outcomes. This evidence suggests that, by increasing aggression and other behavior problems, even moderate exposure to lead in early childhood can have substantial and persistent adverse effects on individual behavior.

Why is this relevant, either practically or historically? If lead exposure were rare, this might be interesting but not particularly germane to our understanding of environmental history or social behavior. However, moderate exposure at precisely these levels was the norm for all residents of the United States born between the 1950s and the early 1980s. Consequently, this is not just an interesting but irrelevant story; rather, it is the reality of U.S. history. These effects were not estimated on a carefully selected sample of exposed individuals, but rather on nationally representative (or almost representative) samples of individuals born in the United States circa 1980. Thus, any societal-level effects on social behavior that are implied by the above results may have actually taken place in recent decades.

What then can we make of the behavioral effects of the Clean Air Act's phaseout of lead from gasoline? Consider what we know about larger trends in the outcomes under consideration. Given the difficulties inherent in measuring behavior problems objectively and consistently over time, I will set aside childhood behavior problems for now. In the 1990s, teen births declined by one-fifth, teen crime declined by one-third, smoking rose by a third, and marijuana use doubled. Commenting on these trends, Jonathan Gruber writes that "[f]or smoking and marijuana, the correlation is perhaps not surprising and provides some credence to the notion of

complementarities between the use of these substances." On the other hand, Gruber argues that the co-movement of the first two – teen pregnancy and crime – is harder to explain:

> "… [T]he parallel movements in crime and teen pregnancy are more surprising. There is no direct link between these behaviors, one of which is almost exclusively the purview of males and the other by definition exclusively the purview of females. But there is an implicit link as these are the two most "deviant" activities that males and females can pursue as teens."
>
> Gruber, *Risky Behavior Among Youths*, p. 16

Gruber goes on to argue that the fact that the two "move so closely together suggests that there are clear taste shifts among teens regarding the pursuit of very risky activities." The current results may present a slightly different yet closely related answer to the puzzle of this co-movement. Crime and teen pregnancy are indeed "deviant" activities, but they are also impulsive. Moreover, crime is often aggressive. Considered in light of a behavioral economics model of youth risk-taking, it is possible that lead exposure leads teens, whether male or female, to take these impulsive, aggressive, or risky actions. It is possible that the "taste shift" hypothesized by Gruber is a "taste shift" or a "propensity shift" induced by a lead shift in early childhood.

Earlier work used the FBI Uniform Crime Report data to investigate the effect of lead on crime from this macro perspective,[58] and future work will use the Vital Statistics Natality data to investigate the effect of lead on teen pregnancy from the same macro perspective. Given the possibility that lead may affect such a wide range of social behaviors, it will require several separate analyses, done from different perspectives, to effectively piece together the full picture of lead's effects. For now, the current paper suggests that these effects could be substantial.

IX. Conclusion

This project aims to join and strengthen three different threads from medicine, social science, and public policy. First, the medical, toxicology, epidemiology, and psychology literatures show that environmental toxicants can impair neurobehavioral and mental functioning, in ways that lower IQ and increase the impulsive and aggressive behavior. Numerous studies investigate a range of exposure levels to many different toxicants, but they

[58] Reyes (2007)

encounter difficulty in studying the possibly sub-clinical effects of low-level long-term exposures to multiple toxicants among the general population. Second, social scientists struggle to explain trends in mental health and social behaviors, including learning disabilities, adolescent violence, teen pregnancy, and substance abuse. The complex variety of factors influencing these behaviors and conditions presents a challenge to researchers, and they are increasingly looking more closely at early life influences. Third, policy-makers and public health professionals strive to understand the role of environmental and public health policy in improving health and welfare and to design policies that will maximize social welfare. This paper, and the larger research agenda of which it is a part, aims to join and strengthen these threads.

The foregoing results suggest that lead – and other environmental toxicants that impair behavior – may be missing links in social scientists' explanations of social behavior. Social problems may be, to some degree, rooted in environmental problems. As a consequence, environmental or public health policy aimed at reducing exposure to environmental toxicants may be effective in reducing the social and economic costs associated with child behavior problems, teen pregnancy, aggression, and crime.

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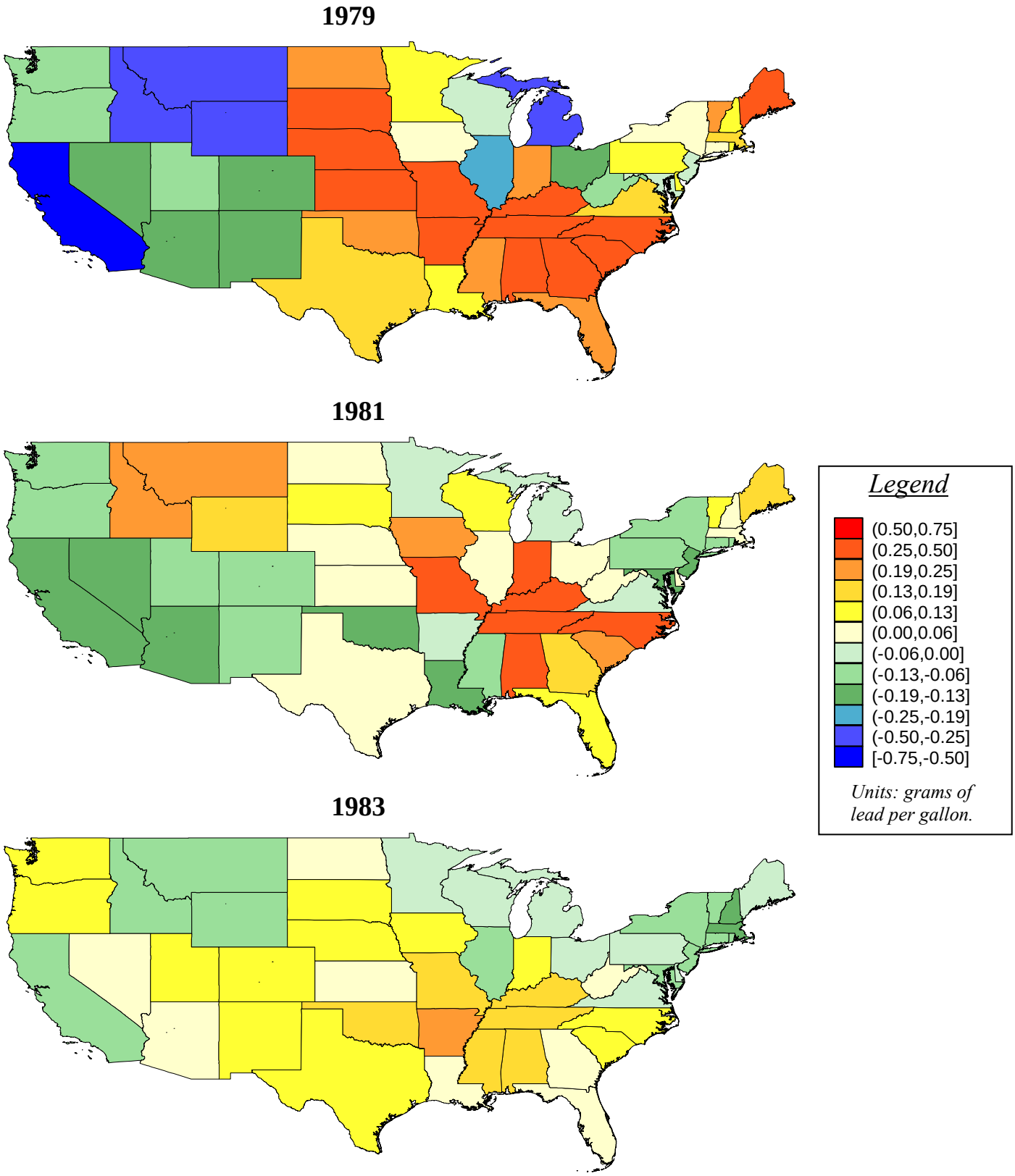
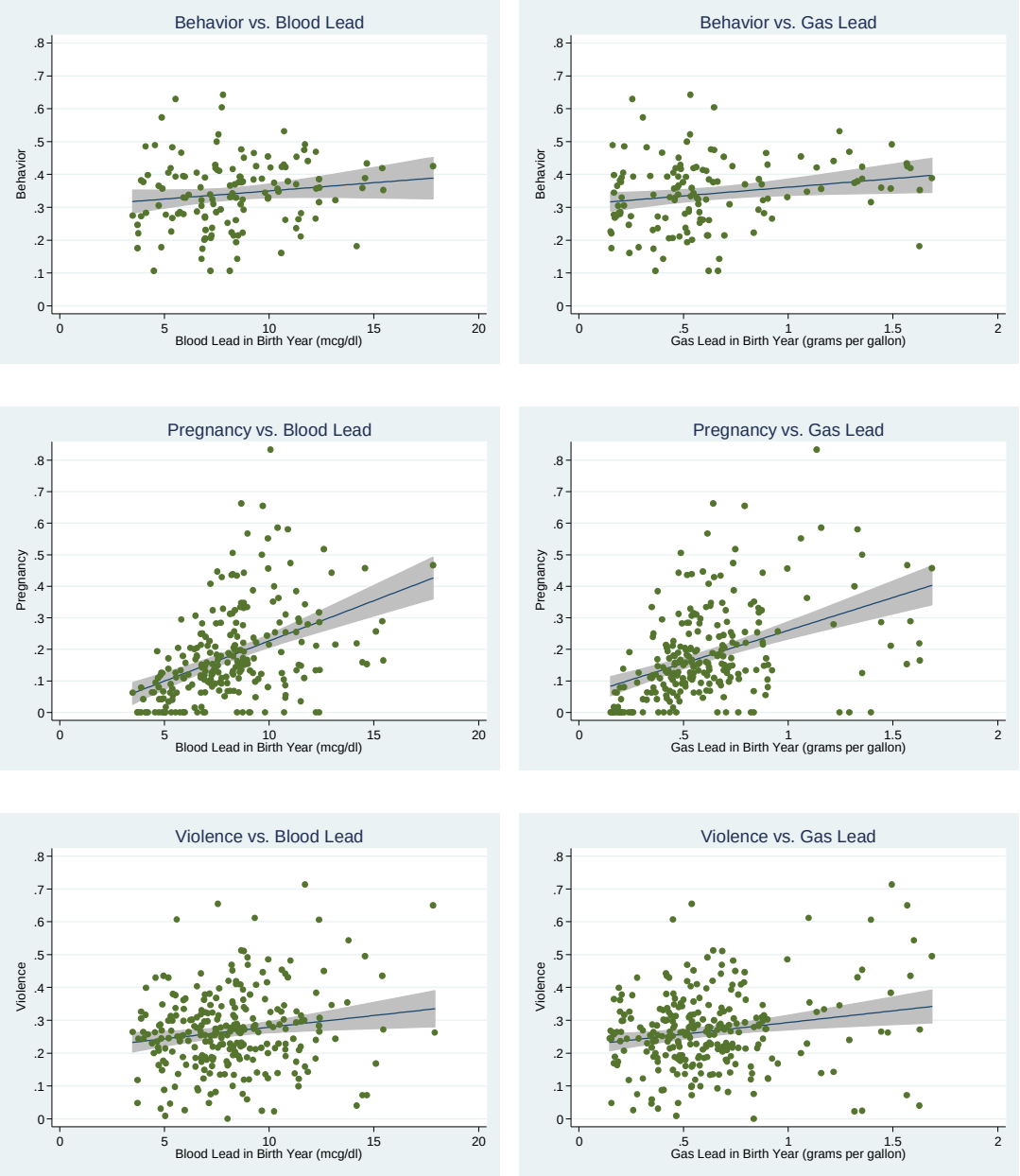
Figure 1. Gasoline Lead by State and Year.

(Deviation of state's annual average lead from national annual average.)



Notes. In each year, the map shows the deviation of each state's annual average grams of lead per gallon of gasoline (gpg) from the national annual average gpg in that year. The coloring is consistent over the years, ranging from red (indicating 0.50 to 0.75 gpg above the national average) to blue (indicating 0.50 to 0.75 gpg below the national average). The national annual average gpgs were 1.30 in 1979, 0.64 in 1981, and 0.38 in 1983.

Figure 2. Outcomes vs. Lead.



Notes. Each point represents a particular state in a particular birth year. The sample includes all 50 states plus the District of Columbia and the birth years from 1979 to 1985 are shown. The outcomes are composite behavior outcomes for that birth cohort, and they range from 0 to 1. Behavior represents the total behavior problems index. Pregnancy is the average of available teen pregnancy outcomes (pregnancy by age 19 in both the NLSY79 and NLSY97, and having gotten a partner pregnant by age 19 from the NLSY97). Violence is the average of available violent action outcomes (hit, hurt, and attack outcomes in both the NLSY79 and NLSY97). The line and shading show the OLS regression line with a 95% confidence interval.

Table 1. Summary of Variables.

	NLSY 1979 CHYA		NLSY 1997	
	Mean	*Std. Dev.*	*Mean*	*Std. Dev.*
Number of Observations	3452		7889	
Panel A. Characteristics				
Child Characteristics				
Year of birth	1981.7	(2.41)	1982.0	(1.42)
Female	49%		49%	
Race: white	58%		71%	
Race: black	19%		15%	
Race: hispanic	8%		10%	
Race: other	15%		4%	
Maternal and Family Characteristics				
Teen mother	49%		21%	
Mother high school graduate	87%		78%	
Family income ($, year 2000)	38.39	(31.45)	51.17	(37.20)
Lead				
Predicted childhood blood lead (μg/dl)	7.66	(3.70)	7.67	(3.14)
Panel B. Child Behavior				
Behavior Problem Indices (ages 4 to 12)				
Total (0 to 28)	9.59	(6.14)		
Behavioral / Emotional Scale (0 to 8)			1.53	(1.55)
Behavior Problem Subscales (ages 4 to 12)				
Oppositional (0 to 30)	6.32	(4.87)		
Antisocial (0 to 12)	1.65	(1.83)		
Hyperactive (0 to 10)	2.32	(2.09)		
Headstrong (0 to 10)	2.94	(2.22)		
Behavior Problem Items (at age 10-11)				
Impulsive - Sometimes	47%			
Impulsive - Often	7%			
Bully - Sometimes	25%			
Bully - Often	4%			
Temper - Sometimes	32%			
Temper - Often	9%			

Table 1. Summary of Variables (continued).

	NLSY 1979 CHYA		NLSY 1997	
	Mean	*Std. Dev.*	*Mean*	*Std. Dev.*
Panel C. Teen Risky Behavior				
Sex and Pregnancy				
Sex (by age 13)			5%	
Pregnant (by age 17)	12%		15%	
Pregnant (by age 19)	21%		28%	
Got partner pregnant (by age 19)			5%	
Substance Use				
Alcohol (by age 13)	32.1%		27.1%	
Cigarettes (by age 13)	34.8%		30.2%	
Marijuana (by age 17)	49.9%		53.3%	
Age first drank alcohol	14.5	(2.5)	12.6	(2.57)
Age first smoked cigarettes	14.0	(3.0)	13.6	(2.6)
Panel D. Aggressive and Criminal Behavior				
Aggressive Behavior				
Hit someone (by age 15)	22.5%			
Hit someone (by age 17)	31.8%			
Hurt someone badly (by age 17)	31.1%			
Attacked someone (by age 13)			12.6%	
Attacked someone (by age 17)			31.9%	
Criminal Behavior (by age 17)				
Arrested			22.7%	
Charged			13.2%	
Convicted	12.4%		8.5%	
Convicted of violent crime	2.4%		1.9%	
Convicted of property crime	4.2%		3.5%	

Notes. Means are shown, with standard deviations in parentheses. Data sources are as described in text. Behavioral outcomes are from the National Longitudinal Survey of Youth (NLSY) data on two cohorts born during the phaseout of lead from gasoline: "NLSY 1979 CHYA" is a cohort of 3,833 children of the NLSY 1979 sample women (born between 1979 and 1985); "NLSY 1997" is a cohort of 7,889 children from the NLSY 1997 sample (born between 1980 and 1984). Predicted childhood blood lead is calculated as described in the text. Lead content of gasoline is from a variety of government sources; individual blood lead measurements are from the second National Health and Nutrition Examination Survey (NHANES II).

Table 2. Summary of Lead Variables.

	Mean	*Std. Dev.*	*25th pctile*	*Median*	*75th pctile*
Blood Lead					
Blood lead in NHANES II, 1976 to 1980	16.5	6.7	12.0	15.0	20.0
Blood lead in NHANES II, 1976	19.3	7.2	14.0	18.0	23.0
Blood lead in NHANES II, 1980	9.9	3.2	8.0	9.0	11.0
Gasoline Lead					
Grams of lead per gallon of gasoline, 1976 to 1980	1.48	0.30	1.30	1.50	1.69
Grams of lead per gallon of gasoline, 1980 to 1984	0.58	0.16	0.46	0.55	0.70
Grams of lead per gallon of gasoline, 1976	1.72	0.24	1.59	1.75	1.87
Grams of lead per gallon of gasoline, 1980	0.75	0.10	0.69	0.75	0.84
Grams of lead per gallon of gasoline, 1984	0.42	0.07	0.37	0.42	0.46
Predicted Blood Lead					
Predicted blood lead for NLSY79 CHYA sample, born 1979 to 1985	7.7	4.0	4.8	7.0	9.8
Predicted blood lead for NLSY97 sample, born 1980 to 1984	7.7	3.6	5.4	7.0	9.3

Notes. Lead content of gasoline is from a variety of government sources, as described in the text. Predicted childhood blood lead is calculated as described in the text; the individual blood lead measurements used in that calculation are from the NHANES II.

Table 3. Regression of Blood Lead on Gasoline Lead.

	(1)	(2)	(3)	(4)	(5)	(6)
Gasoline Lead	4.13 **	7.52 **	5.95 **	6.19 **	5.81 **	7.69 **
	(0.42)	(0.60)	(1.14)	(1.15)	(1.20)	(1.93)
Age	-0.18 *	-0.17 **	-0.18 **	-0.23 **	-0.21	-0.28
	(0.10)	(0.08)	(0.08)	(0.09)	(0.09)	(0.35)
Female	-0.43	-0.30	-0.31	-0.33	-0.38	-0.24
	(0.28)	(0.25)	(0.25)	(0.25)	(0.25)	(0.41)
Race - Black	5.19 **	5.37 **	5.26 **		6.08 **	4.97 **
	(0.45)	(0.42)	(0.42)		(0.42)	(0.68)
Low Income (< 2x poverty)	2.94 **	2.74 **	2.70 **	3.68 **		3.65 **
	(0.38)	(0.33)	(0.33)	(0.33)		(0.56)
Middle Income (2x to 3x poverty)	0.78 **	0.52 *	0.50	0.45		0.88 *
	(0.35)	(0.30)	(0.30)	(0.31)		(0.52)
Constant	9.10 **	4.44 **	3.80 **	3.64 **	4.59 **	1.77 **
	(0.73)	(0.89)	(1.65)	(1.74)	(1.67)	(2.83)
Ages included	0 to 6 yrs	0 to 6 yrs	0 to 6 yrs	0 to 6 yrs	0 to 6 yrs	2 to 4 yrs
State dummies	No	Yes	Yes	Yes	Yes	Yes
Year dummies	No	No	Yes	Yes	Yes	Yes
R-squared	0.21	0.32	0.33	0.26	0.30	0.36
Number of observations	2322	2322	2322	2322	2322	856

Notes. Regression on the sample of 2,322 children age 0 to 6 with blood lead measurements in the NHANES II. Covariates and state and year fixed effects are included as shown. Standard errors are Huber-White robust and clustered by state. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.

Table 4. The Effect of Early Childhood Lead on the Total Behavior Problems Index.

	Linear	*Log*	*Spline*
Blood Lead	0.134 **		
	(0.060)		
Log Blood Lead		1.122 **	
		(0.367)	
Blood Lead 0 to 5 mcg/dL			0.358 *
			(0.190)
Blood Lead 5 to 10 mcg/dL			0.133
			(0.084)
Blood Lead 10+ mcg/dL			0.011
			(0.073)
Age	-0.058 *	-0.057 *	-0.058 *
	(0.029)	(0.030)	(0.029)
Female	-0.974 **	-0.964 **	-0.970 **
	(0.211)	(0.209)	(0.208)
Race - Black	-1.065 *	-0.999 **	-0.751
	(0.540)	(0.439)	(0.559)
Race - Hispanic	-0.470	-0.462	-0.466
	(0.421)	(0.421)	(0.422)
Race - Other	-0.177	-0.179	-0.173
	(0.418)	(0.418)	(0.420)
Mother - high school graduate	-1.750 **	-1.754 **	-1.760 **
	(0.418)	(0.418)	(0.418)
Mother - teen mother	-0.302	-0.378	-0.410
	(0.364)	(0.363)	(0.369)
Income ($k)	-0.057 **	-0.055 **	-0.056 **
	(0.008)	(0.008)	(0.008)
Elasticity BPI wrt lead, at lead = 5	0.073 **	0.122 **	0.192 *
	(0.034)	(0.042)	(0.100)
Elasticity BPI wrt lead, at lead = 7.5	0.114 **	0.117 **	0.034
	(0.037)	(0.038)	(0.218)
Elasticity BPI wrt lead, at lead = 11	0.148 **	0.112 **	0.001
	(0.063)	(0.035)	(0.007)

Notes. Analysis on NLSY sample as described in text. Regression is pooled OLS on the sample of children, born between 1979 and 1985, ages 4 to 12, and observed during the years 1986 to 1998. Data weights provided by the NLSY are employed. Control variables include: child age, gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, teen mother, and a continuous variable for real family income in year 2000 dollars. Dummy variables are included for the mother's age at the child's birth, the child's birth cohort, and a set of interactions between the two sets of dummies; for a set of interactions between teen mother and child's birth cohort; and for the census region of residence. Weights provided by the NLSY are employed. Results are adjusted via bootstrap to account for the fact that blood lead was predicted in a first stage. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.

Table 5a. Elasticities of Child Behavior Problems Indices & Subscales with respect to Blood Lead.

	Age 4-12	*Age 6-7*	*Age 8-9*	*Age 11-12*
Total	0.117 **	0.000	0.215 **	0.141 *
	(0.038)	(0.046)	(0.063)	(0.079)
Oppositional	0.132 **	0.030	0.225 **	0.131
	(0.052)	(0.066)	(0.079)	(0.106)
Antisocial	0.130 *	-0.009	0.272 **	0.124
	(0.072)	(0.093)	(0.107)	(0.142)
Hyperactive	0.109 **	-0.049	0.098	0.117
	(0.050)	(0.080)	(0.079)	(0.127)
Headstrong	0.128 **	0.102	0.227 **	0.177 **
	(0.051)	(0.080)	(0.085)	(0.091)
Behavioral/Emotional Problems	0.259 **			
	(0.128)			

Table 5b. Elasticities of Individual Child Behavior Problems with respect to Blood Lead.

	Age 4-12	
	Sometimes	**Often**
Impulsive	0.020	0.071
	(0.032)	(0.116)
Bullies	0.073	0.158
	(0.090)	(0.195)
Strong Temper	0.095 **	0.252 **
	(0.047)	(0.126)

	Age 6-7		*Age 8-9*		*Age 11-12*	
	Sometimes	**Often**	**Sometimes**	**Often**	**Sometimes**	**Often**
Impulsive	-0.116 *	-0.432 *	0.027	0.098	0.092	0.313
	(0.066)	(0.242)	(0.044)	(0.160)	(0.082)	(0.282)
Bullies	0.015	0.032	0.262 **	0.568 **	0.210	0.459
	(0.143)	(0.302)	(0.127)	(0.280)	(0.149)	(0.332)
Strong Temper	0.054	0.140	0.184 **	0.479 **	0.117	0.311
	(0.084)	(0.218)	(0.080)	(0.214)	(0.107)	(0.287)

Notes. Analysis on NLSY sample as described in text, using log specification. All regressions were run on the NLSY79 CHYA sample of children, born between 1979 and 1985, ages 4 to 12, and observed during the years 1986 to 1998, except for the "Behavioral/Emotional Problems" row which is from the full NLSY97 sample. The table shows elasticities of each behavioral outcome with respect to the blood lead, evaluated at the sample mean. In Table 5a, regression is ordinary least squares on the total behavior problems index, the behavior subscale indicated, or the behavioral/emotional problems scale. In Table 5b, regression is ordered probit on the individual outcome indicated, where the categories are "never," "sometimes," and "often." Elasticities shown in the "sometimes" column are for moving from "never" to "sometimes;" elasticities shown in the "often column are for moving from "sometimes" to "often." Control variables include: child gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, and a continuous variable for real family income in year 2000 dollars. Dummy variables are included for the mother's age at the child's birth, the child's birth cohort, and a set of interactions between the two sets of dummies. Dummy variables are included for the census region of residence. Weights provided by the NLSY are employed. Results are adjusted via bootstrap to account for the fact that blood lead was predicted in a first stage. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.

Table 6. Elasticity of Teen Risky Behavior Indices with respect to Blood Lead

	Elasticity (NLSY79)	Elasticity (NLSY97)
Sex and Pregnancy		
Had sex by age 13		4.276 **
		(0.414)
Pregnant by age 17	1.041 **	0.635 **
	(0.559)	(0.243)
Pregnant by age 19	1.142 **	0.600 **
	(0.440)	(0.151)
Got partner pregnant by age 19		1.736 **
		(0.552)
Substance Use		
Alcohol by age 13	0.556 **	0.221 **
	(0.190)	(0.088)
Cigarettes by age 13	0.257	0.259 **
	(0.195)	(0.082)
Marijuana by age 17	0.568 **	0.050
	(0.161)	(0.051)

Notes. Analysis on NLSY samples as described in text and indicated at the top of the columns. Regressions are probit, and the table shows elasticities of each outcome with respect to blood lead, evaluated at the sample mean. Control variables include: child gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, and a continuous variable for real family income in year 2000 dollars. An additional vector of state-level control variables includes: the minimum wage, maximum welfare benefit, and indicator variables for welfare reform (either via a waiver under AFDC or a TANF reform), a parental living arrangement requirement in welfare, and parental involvement requirement for teenage abortion. Dummy variables are included for the mother's age at the child's birth, the child's birth cohort, and a set of interactions between the two sets of dummies. Dummy variables are included for the census region of residence. Weights provided by the NLSY are employed. Results are adjusted via bootstrap to account for the fact that blood lead was predicted in a first stage. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.

Table 7. Elasticity of Attacks and Criminal Activity with respect to Lead.

	Elasticity (NLSY79)	Elasticity (NLSY97)
Hit, Hurt, or Attack		
Hit someone by age 15	0.441 ** (0.174)	
Hit someone by age 17	0.503 ** (0.132)	
Hurt someone badly by age 17	0.308 ** (0.112)	
Attacked someone by age 13		0.500 ** (0.227)
Attacked someone by age 17		0.359 ** (0.107)
Crime (by age 17)		
Arrested		0.374 ** (0.168)
Charged		0.511 ** (0.247)
Convicted	0.595 ** (0.265)	0.642 ** (0.358)
Convicted of a violent crime	0.736 (0.767)	0.208 (0.765)
Convicted of a property crime	2.391 ** (0.644)	0.691 (0.600)

Notes. Analysis on NLSY samples as described in text and indicated at the top of the columns. Regressions are probit, and the table shows elasticities of each outcome with respect to blood lead, evaluated at the sample mean. Control variables include: child gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, and a continuous variable for real family income in year 2000 dollars. An additional vector of state-level covariates includes: state per-capita income, the poverty rate, the unemployment rate, a concealed weapons law, lagged police per capita, and lagged prisoners per capita Dummy variables are included for the mother's age at the child's birth, the child's birth cohort, and a set of interactions between the two sets of dummies. Dummy variables are included for the census region of residence. Weights provided by the NLSY are employed. Results are adjusted via bootstrap to account for the fact that blood lead was predicted in a first stage. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.

Appendix Table 1. Child Behavior Problems Index.

		Subscales			
Item	**Description**	**Oppositional**	**Antisocial**	**Headstrong**	**Hyperactive**
1	Sudden changes of mood or feeling				
2	Complains no one loves him or her				
3	High strung, tense, nervous			x	
4	Cheats or tells lies	x	x		
5	Too fearful or anxious				
6	Argues too much	x		x	
7	Difficulty concentrating	x			x
8	Easily confused, in a fog				x
9	Bullies or is cruel/mean to others	x	x		
10	Disobedient at home	x		x	
11	Not sorry after misbehaving	x	x		
12	Trouble getting along with other children	x			
13	Impulsive, acts without thinking	x			x
14	Feels worthless or inferior				
15	Not liked by other chidlren	x			
16	Obsessive, difficulty getting mind off thoughts				x
17	Restless or overly active	x			x
18	Stubborn, sullen, or irritable	x		x	
19	Very strong temper or loses it easily	x		x	
20	Unhappy, sad, depressed				
21	Withdrawn				
22	Breaks things deliberately (<12 years)	x	x		
23	Clings to adults				
24	Cries too much				
25	Demands lots of attention				
26	Too dependent on others				
27	Feels others are out to get him or her				
28	Hangs around kids who get into trouble				
29	Secretive				
30	Worries too much				
31	Disobedient at school (> 5 years)	x	x		
32	Trouble getting along with teachers (> 5 years)	x	x		

Appendix Table 2a. Elasticities of Child Behavior Problems Indices & Subscales with respect to Blood Lead, Spline Specification.

	Age 4-12	*Age 6-7*	*Age 8-9*	*Age 11-12*
Total	0.173 **	0.111	0.047	0.382 **
	(0.094)	(0.109)	(0.185)	(0.144)
Oppositional	0.242 **	0.166	0.065	0.432 **
	(0.093)	(0.136)	(0.184)	(0.159)
Antisocial	0.257 **	0.143	0.254	0.530 **
	(0.137)	(0.176)	(0.259)	(0.199)
Hyperactive	0.279 **	0.119	-0.010	0.397 **
	(0.095)	(0.186)	(0.185)	(0.198)
Headstrong	0.183 *	0.237	-0.003	0.317 *
	(0.110)	(0.173)	(0.207)	(0.182)
Behavioral/Emotional Problems	0.390			
	(0.284)			

Appendix Table 2b. Elasticities of Individual Child Behavior Problems with respect to Blood Lead, Spline Specification.

	Age 4-12	
	Sometimes	**Often**
Impulsive	0.152 **	0.587 **
	(0.069)	(0.276)
Bullies	0.195	0.418
	(0.186)	(0.401)
Strong Temper	0.278 **	0.750 **
	(0.113)	(0.313)

	Age 6-7		*Age 8-9*		*Age 11-12*	
	Sometimes	**Often**	**Sometimes**	**Often**	**Sometimes**	**Often**
Impulsive	-0.060	-0.254	0.077	0.279	0.306 *	1.088 *
	(0.109)	(0.463)	(0.128)	(0.475)	(0.169)	(0.610)
Bullies	-0.187	-0.386	0.224	0.454	0.861 **	1.943 **
	(0.359)	(0.740)	(0.334)	(0.687)	(0.268)	(0.634)
Strong Temper	0.153	0.404	0.039	0.090	0.392 *	1.098 *
	(0.198)	(0.529)	(0.240)	(0.562)	(0.225)	(0.624)

Notes. Analysis on NLSY sample as described in text, using spline for blood lead with cutpoints at 5 and 10 μg/dl. All regressions were run on the NLSY79 CHYA sample of children, born between 1979 and 1985, ages 4 to 12, and observed during the years 1986 to 1998, except for the "Behavioral/Emotional Problems" row which is from the full NLSY97 sample. The table shows elasticities of each behavioral outcome with respect to blood lead, evaluated at the sample mean. In Table 5a, regression is ordinary least squares on the total behavior problems index, the behavior subscale indicated, or the behavioral/emotional problems scale. In Table 5b, regression is ordered probit on the individual outcome indicated, where the categories are "never," "sometimes," and "often." Elasticities shown in the "sometimes" column are for moving from "never" to "sometimes;" elasticities shown in the "often column are for moving from "sometimes" to "often." Control variables include: child gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, and a continuous variable for real family income in year 2000 dollars. Dummy variables are included for the mother's age at the child's birth, the child's birth cohort, and a set of interactions between the two sets of dummies. Dummy variables are included for the census region of residence. Weights provided by the NLSY are employed. Results are adjusted via bootstrap to account for the fact that blood lead was predicted in a first stage. Significance is indicated by ** for $p<0.05$ and * for $p<0.10$.