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

JESSICA WOLPAW REYES

Amherst College and NBER

Abstract

It is well known that exposure to lead has numerous adverse effects on behavior and development, but little work to date has examined the cumulative lifetime effect of such exposure on aspects of behavior. In this paper, I use data on two cohorts of children from the NLSY to investigate 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 ranging from behavior problems as a child, to pregnancy and aggression as a teen, and to criminal behavior as a young adult. Estimated elasticities of these behaviors with respect to lead range between 0.2 and 0.6. This evidence suggests that, by increasing impulsivity and aggression, even moderate exposure to lead in early childhood can have substantial and persistent adverse effects on individual behavior. Moreover, such moderate exposure was the norm for residents of the United States born between the 1950s and the early 1980s.

Department of Economics, Amherst College, jwreyes@amherst.edu or jwreyes@post.harvard.edu. I would like to thank Claudia Goldin, Jun Ishii, Lawrence Katz, Erzo Luttmer, Linda Reyes, René Reyes, Steve Rivkin, and seminar participants at Amherst College, Clark University, RAND, the University of Massachusetts, 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 data on lead in gasoline. Steve Trask provided excellent research assistance. Any remaining errors are my own. This research was supported by the National Bureau of Economic Research.

I. Introduction

Individual people make up a society, and the way those people behave can substantially influence how a society functions. A large literature in economics seeks to explain trends in mental health and social behaviors, including learning disabilities, adolescent violence, teen pregnancy, and crime. The complex variety of factors influencing these behaviors and conditions presents a significant challenge to researchers. At the same time, a vast literature in neurotoxicology, epidemiology, and psychology shows that exposure to environmental toxicants such as lead can substantially impair neurobehavioral and mental functioning and lead to more impulsive and aggressive behavior. In this paper, I bring these insights from the scientific literature to bear in an economic context. I propose that environmental toxicants and social problems may be closely linked, and that elucidating these links may provide insight not only into the understanding of social problems but also the potential of public health policy and environmental policy to improve social health. The central idea is that children exposed to even small or 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.

Why should we care about behavior? At the most fundamental level, individual behavior provides the basis for productive interaction in society. 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 and can lead to negative externalities. Moreover, poor behavior that develops early in life may have a compounding effect by interfering with the development of traditional human and social capital. The negative

behaviors considered in this paper – aggression, teen pregnancy, violence – are of concern to policymakers and economists. Many, too, have exhibited time trends in recent decades that have been difficult to explain.

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 increasing learning disabilities, behavior problems, and aggressive behavior, as well as affecting cardiovascular functioning and other physiological processes. It is relatively 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 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 through 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 7,889 children from the NLSY 1997 cohort (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.

The paper proceeds as follows. Section II provides background on social behavior, lead, and the relationship between the two. Section III outlines the data employed, and Section IV describes the empirical approach. Sections V and VI discuss the results. Section VII provides interpretation, and Section VIII concludes.

II. Background

Behavior

Problems like 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. [1] 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 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

[1] U.S. Surgeon General's Report, US DHHS, 1999. National Institutes of Mental Health [1996]

teen sex, teen pregnancy, and substance abuse.[2] 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."[3] 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.[4] Recent evidence indicates that more than two-thirds of juvenile delinquents meet diagnostic criteria for one or more psychiatric disorders,[5] and three independent studies find that children with ADHD are five times more likely to be delinquent than children without ADHD.[6] More generally, behavior issues in childhood can substantially impair accumulation of human capital and social capital, leading to a broad range of effects.

While the prevalence of many of these behaviors prompts concern, it is their substantial trends that have 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.[7] Teenage pregnancy rates rose until the early 1990s and declined substantially thereafter, but

[2] Sweeney (2000); Maynard (1997); Gruber [2000].
[3] Fishbein [2000].
[4] Currie and Stabile [2006]; CDC (2002).
[5] Teplin et al (2002).
[6] 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.
[7] Monitoring the Future, 2002.

trends vary across states and racial groups.[8] Juvenile delinquency, criminal behavior, and violence, show similar trends to that of teen pregnancy, peaking in the early 1990s and exhibiting sharp declines thereafter.

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.[9] It is now widely accepted that lead exposure can be detrimental even at extremely low levels and that the adverse neurological effects of lead persist into older ages.[10] 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 should not be downplayed; it is just not possible to assess their effects with the empirical strategy employed in this paper. 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.[11]

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[8] CDC (2001).

[9] 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.

[10] 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.

[11] 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. For information on dangers from lead in water pipes and mains, see Troesken (2006).

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. 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"[12] and blood lead levels were highly correlated with recent gasoline lead consumption.[13]

In 1974, 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 few years, the time-table was delayed slightly and further reductions were implemented. Ultimately, the phase-out was a success: gasoline lead dropped by 99% between 1975 and 1990.[14] 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.[15] The entire distribution shifted downward, and the national mean dropped from 16

[12] U.S. Environmental Protection Agency [1973].
[13] Schwartz and Pitcher [1989].
[14] 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).
[15] U.S. Environmental Protection Agency [1991]; Brody, et al. [1994].

μg/dL in 1976 to 3 μg/dL in 1991.[16] 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 and disrupts neurotransmitter function in ways that impair cognition and reduce impulse control. 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.[17]

Many studies have found an association between higher lead levels and aggressive behavior, impulsivity, hyperactivity, and attention impairment.[18] Increased lead levels are also associated with decreased mental skills, including reduced IQ, reduced verbal competence, increased reading disabilities, and reduced academic performance.[19] Needleman, H and Bellinger [1981] report 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

[16] Bodily lead levels can be measured in a variety of ways. The most common are blood lead levels, dentine lead levels, and bone lead levels. 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). A dentine lead level is the concentration of lead in teeth, and is measured in parts per million. Bone lead levels are measured similarly. Blood lead is a good measure of recent exposure, while dentine and bone lead are regarded as good indicators of cumulative lifetime exposure.

[17] Banks, et al. [1997]. Bellinger (2004); Needleman, H., et al. [1996]. Numerous studies find significant effects on teen and adult behavior using either early childhood blood lead or current bone lead (which indicates cumulative lifetime exposure.)

[18] Wilson and Petersilia [1995]; Needleman, H. , et al. [1990]; Needleman, H and Gatsonis [1991]; Banks, et al. [1997].

[19] Bryce-Smith [1983]. The present paper will not investigate mental skills and academic performance directly; that is being investigated in a separate companion analysis.

functioning.[20] With regard to IQ, an increase in blood lead level of 1 μg/dL appears to produce a decrease in IQ of approximately one-half of a point.[21] Coscia *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."

Moreover, researchers in psychology and neurotoxicology have established direct links between higher childhood lead exposure and a greater likelihood of adult criminal behavior.[22] Economists have also contributed to the discussion; Reyes [2007] uses a panel of U.S. states to show a strong association between lead exposure and violent crime rates at the national level.[23] Overall, the literature suggests that there may be a substantial elasticity of crime with respect to lead, possibly as high as 0.80.

Finally, numerous studies have found higher lead levels among children who are hyperactive or who have other behavior problems,[24] and among juvenile delinquents and criminals.[25] In sum, a large, multi-disciplinary, and diverse 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

[20] The study uses dentine lead levels, which are a good indicator of lifetime exposure. While the dentine lead levels cannot be directly compared to blood lead levels, the lead levels even for the high lead group in this study were relatively unremarkable in the 1970s.

[21] Schwartz [1994] reports that an increase in blood lead level of 1 μg/dL produces a decrease of one-quarter of an IQ point. This estimate controls for socioeconomic and other factors that could affect IQ. Canfield, et al. [2003] report an effect of 0.8 of an IQ point per 1 μg/dL of blood lead in lower ranges, and an average effect of 0.46. Furthermore, Liu *et al* (2002) report evidence that IQ deficits persist even after blood lead levels decline.

[22] Denno [1990] finds that lead poisoning is the most significant predictor of disciplinary problems and one of the most significant predictors of delinquency, adult criminality, and the number and severity of offenses. Needleman *et al.* [1996] find a significant relationship between the amount of lead in bone (a good measure of past exposure) and antisocial, delinquent, and aggressive behaviors. Dietrich, et al. [2001] followed a cohort of 195 inner-city youths from birth through adolescence, and found a clear linear relationship between childhood blood lead levels and the number of delinquent acts. Most recently, Cecil *et al.* (2008) report a strong relationship between early childhood blood lead and arrest rates.

[23] See also Nevin [2000], who uses a national time series, and Masters, et al. [1998], who employs a single cross-section.

[24] Denno [1990]; Needleman, Herbert L. [1985].

[25] 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.

even at the moderate lead levels (5-20 μg/dL) that were common in the U.S. through the early 1980s.

III. 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. 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). 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.[26] Analysis in this paper will include the 3,833 children born between 1979 and 1985, and will try to address this possible bias. Together, the Child and Young Adult Surveys provide a rich set of longitudinal data. Individual observations can be linked to the maternal and

[26] Controls for mother's age birth, child birth cohort, and interactions between the two will be employed in an attempt to alleviate bias from this selection.

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). The BPI was created to measure "the frequency, range, and type of childhood behavior problems for children age four and over."[27] 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 Oppositional, Antisocial, Headstrong, and Hyperactive Subscales are employed in this paper.[28] 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

[27] Zill [1985], Zill [1990], and the NLSY79 Child and Young Adult Users' Guide, p.78. See also Achenbach and Edelbrock [1979] for a discussion of the BPI's predecessor, the more extensive Child Behavior Checklist.
[28] Each behavior problems index or subscale is calculated by assigning, for each behavior problem items, a value of 0 to "never," 1 to "sometimes," and 2 to "often", and summing those values over the relevant set of behaviors.

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.[29]

[Table 1: discuss summary of vars, sample means etc here]

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 as well.

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.[30] This 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,

[29] 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.

[30] Two subsamples comprise the NLSY97 cohort: a nationally-representative cross-section of 6,748 individuals and a supplemental oversample sample of 2,236 Hispanic or Latino and black individuals. See the NLSY97 User's Guide (2005).

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.[31]

[Table 1: discuss summary of vars, sample means etc here]

The NLSY97 provides 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.[32] Thus, used together, these two primary NLS datasets should be able to provide a reasonably complete, but far from perfect, picture of behavior in the childhood and teen years.

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 concentration of a nationally representative sample of 9,372 individuals, including 2,322 children

[31] 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 offenses, other property offense, other offense.

[32] 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.

under the age of 6, in the years 1976 to 1980.[33] 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 μg/dL).[34] 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.

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.[35]

These data, on lead in blood and lead in gasoline, will be used together. In Section IV below, I will discuss the split-sample instrumental variables strategy I apply to the blood lead and gasoline lead data to estimate early childhood blood lead for individual children born during the phaseout of lead from gasoline.

[Table 2: discuss time patterns here, possibly add a figure]

Other Data

[Describe teen pregnancy and crime controls here, as well as anything else.]

[33] While there was a 27% non-response rate at the examination phase, Forthofer (1983) uses a comparison to the 1976 National Health Interview Survey to show that "the nonresponse and poststratiftcation adjustments performed by the National Center for Health Statistics" have effectively eliminated non-response bias.
[34] See the above footnote re different methods of measuring lead in the human body.
[35] Details of these sources and the construction of the grams of lead per gallon are described in the Data Appendix. Alternate measures of gasoline lead exposure can be considered, and are discussed at length in Reyes (2007).

IV. Empirical Approach

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

$$\text{Prob (outcome)}_i = \alpha_1 \text{BloodLead}_i + \boldsymbol{X}_i \boldsymbol{\beta} + \varepsilon_i \ . \quad (1)$$

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 easily 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:

$$\text{BloodLead}_i = \gamma \text{GasLead}_i + \boldsymbol{Y}_i \boldsymbol{\theta} + \varepsilon_i \ . \quad (2)$$

where $\boldsymbol{Y}_i$ is a vector of individual demographics during childhood. 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 2, to predict the childhood blood lead concentration for each of the NLSY individuals:

$$\widehat{\text{BloodLead}}_i = \hat{\gamma} \text{GasLead}_i + \boldsymbol{Y}_i \hat{\boldsymbol{\theta}} \quad (3)$$

This yields a predicted childhood blood lead concentration for each individual in the NLSY

samples.[36] I can then investigate how this (predicted) measure of childhood blood lead affects the propensity to exhibit certain behavioral traits later in life, but putting it in to Equation 1 in place of (the unobservable) actual childhood blood lead:

$$\text{Prob}(\text{outcome})_i = \alpha_1 \text{PredictedBloodLead}_i + \boldsymbol{X}_i \boldsymbol{\beta} + \varepsilon \ . \qquad (4)$$

It is worth highlighting why this is a desirable strategy. Gasoline was a major source of lead exposure circa 1980, and changes in gasoline lead concentrations were induced by the Clean Air Act. Grams per gallon is well-suited to serve as a measure of gasoline lead exposure in this analysis for several reasons: i) the changes in grams per gallon were indirectly induced by EPA policy; ii) EPA policy *specifically* targeted grams per gallon; iii) EPA policy was imposed on petroleum companies, not states. As a consequence, 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 government policy or state-specific EPA policy, but rather from the interaction between the broad policy application and a variety of features of the petroleum industry.[37] 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 indirectly induced by EPA policy.[38]

Gasoline lead concentration would not matter, however, unless that lead from gasoline somehow found its way into children's blood. Blood lead concentration is the best measure of

[36] 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.

[37] 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 phase-out of high-lead premium gasoline between 1979 and 1980. Sources: Gibbs [1990;1993;1996] and personal communication.

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

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, this methodology 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. Lastly, 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.[39]

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:

$$\text{Prob(outcome)}_{ist} = \alpha\, \text{PredictedBloodLead}_i + \boldsymbol{X}_i\boldsymbol{\beta} + \mu_i + \gamma_i + \mu_i \times \gamma_i + \rho_{ist} + \varepsilon_{ist} \quad (5)$$

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

[39] More generally, as long as an individual's geographic location and demographic information are known, blood lead circa 1980 can be predicted for almost any individual. This methodology therefore opens the door to many important datasets, none of them intended to look at effects of lead.

estimated at a single period in time – the individual's early childhood years, age 0 to 3. The goal is to understand how early childhood blood lead affects later behavior. 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.) If α is significantly different from zero, then we will have identified a significant effect of childhood lead exposure on later life behavior.

The vector $\boldsymbol{X}$ includes dummies for the child's gender, race or ethnicity (black, Hispanic, or other), mother having graduated from high school, and a continuous variable for real family income.[40] 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.[41] The inclusion of these dummy variables accounts for persistent differences that might arise between different groups of mothers or different birth cohorts. This is particularly important for the NLSY79 CHYA sample, which has a disproportionate share of young mothers. The ρ_i are dummies for census region of residence at the time of the outcome being considered.

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, similarly, an additional vector of state-level covariates includes variables for per-capita income, the poverty rate, the unemployment rate, a concealed weapons law, lagged police per capita, and lagged prisoners per capita.

[40] 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.
[41] 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.

The specification for Equation 5 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. In addition, to investigate possible variation in the functional form of lead's effects on outcomes, I estimate results using the level of blood lead, the natural log of blood lead, and a spline of blood lead with cutpoints at 5 mcg/dL and 10 mcg/dL. Because preliminary analysis suggested that the marginal effect of lead was declining in many cases, and the spline did not appear to provide enough improvement to justify its additional complexity, the log specification will be employed as the primary specification.

[Discuss ordered probit and appropriate interpretation of those results.]

V. Results: Predicting Blood Lead

I perform OLS regressions, following Equation 2, 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 3 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 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 2 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. It is worth noting that the predictive power of gasoline lead in explaining blood lead is substantial here: 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).[42]

[42] 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.

VI. Results: Behavior

We can 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 (and also comfortably below the *current* CDC acceptable level of 10). This enables easy comparison of the magnitude of effects across different outcomes as well as different econometric specifications (OLS, probit, ordered probit, and Tobit). Results using both the NLSY 79 and 97 will be discussed together, but it will be noted from which sample specific results derive.

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 baseline linear specification, the coefficient on blood lead is 0.230, highly significant with a standard error of 0.029. This corresponds to an elasticity of 0.09 at a blood lead of 4, an elasticity of 0.18 at a blood lead of 7.5, and an elasticity 0.21 at a blood lead of 11.

Several of the other covariates in these specifications show the expected effects. Girls show fewer behavior problems, with approximately 10% better behavior than boys. Having a mother who finished high school (rather than not finishing) is associated with almost 20% fewer behavior problems, ceteris paribus. Higher income groups show fewer behavior problems as well, with an elasticity of behavior with respect to income of approximately 0.06. Thus, these effects are all right-signed and of reasonable magnitude.

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. I will 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 broadly assumed 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). This is shown in Appendix Table 2. In each specification, the coefficient on lead is reduced by approximately one-third, and the coefficient on black race now 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 5 shows the elasticities with respect to blood lead of a number of additional behavior problems indices and sub-indices. 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 0.2. Looking specifically at children age 6 to 7[43] – between Kindergarten and second grade – the only significant elasticity is that for the headstrong subscale (0.20); the others are right-signed and insignificant. It is possible that many of these behaviors – such as cheating, being easily confused, bullying, or obsessive thinking – either do not manifest themselves so early or are not readily apparent in children so young. In contrast, for children age 11 to 12 – between fifth and seventh grades – all of the indices show substantial elasticities. The estimated elasticities are generally in the range of 0.17 to 0.21, except for the headstrong subscale which shows an elasticity of 0.29. Thus, the results in Table 5 imply that a 10% increase in blood lead is associated with a 2% reduction in child behavior problems.

[43] 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 keep the whole sample yet observe them at close to the same age.

Beyond investigating general behavior problems, or even specific categories of behavior, I can try to look specifically 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. Table 5b shows results for three such behaviors: 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. The estimated elasticities are insignificant for implusive and bully, but are generally right-signed and bully approaches significance for ages 10 and 11 (the t-statistics are approximately 1.50). On the other hand, strong temper shows consistent, significant, and large effects. The elasticity for moving from "never" to "sometimes" is 0.15, and for moving from "sometimes" to "often" is 0.42. The next portion of the table, breaking this down further into separate age groups, show some significant effects for bullying and strong temper among the 8-9 and 10-11 age groups, and generally right signed and insignificant effects for the others. Overall, this provides mild supportive evidence that lead is increasing these outwardly aggressive behaviors.[44]

Teen Risky Behavior

Moving to older behaviors, Table 6 shows results for teen risky behavior: sex, 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 1.9 for having sex by age 13. There are also substantial effects for girls on being pregnant as a teenager, either by age 17 or age 19. The

[44] One of the challenges to any study that aims to investigate lead's effects on behavior is that the scientific literature does not provide clear-cut guidelines on what lead's "fingerprint" would look like, or what behaviors lead should or should not affect. These behaviors were chosen because their association with lead exposure is more widely accepted.

estimated elasticities are in between 0.9 and 1.1, and are reasonably consistent between the two samples. Further sensitivity testing two different functional forms and the inclusion or exclusion of additional covariates indicates that these results are generally robust.

The last measure of sexual activity is whether males report having gotten a partner pregnant as a teen, and no significant effect is found. (This could plausibly result from under-reporting by these males.)[45]

The magnitude of the effects on early sex and teen pregnancy can be assessed more clearly 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 that change in blood lead from 5 to 15, a change that approximates the population-wide change that resulted from the phaseout of lead from gasoline. This calculation yields a predicted 14 percentage point increase in the likelihood of pregnancy by age 17, and a 27 percentage point increase in the likelihood of pregnancy by age 19 (from a 16% chance to a 42% chance). This is undoubtedly large: the lead increase more than doubles the likelihood of teen pregnancy. It is also sizeable when compared to an 8.7% prevalence of teen pregnancy in the sample. Moreover, it is also worth noting that a move of blood lead from 15 *down* to 5 mcg/dl is reasonably close to what occurred during the phaseout of lead from gasoline. 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. These results for sexual activity and pregnancy are generally robust to the use of alternate methods of predicting blood lead.

[45] Most of the teen behavior variables in the NLSY are constructed from self-reports. In the absence of other corroborative reports, it is not possible to 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 from the greater ability of males, for obvious reasons, to answer "no" when the true answer may be "yes."

The final rows of Table 6 show results for substance use by age 13. These are less consistent, but suggest some effect of lead on these behaviors. In particular, early consumption of alcohol shows an elasticity of 0.2 to 0.6. Given the large and growing literature on the adverse and "gateway" effects of early alcohol use, these results merit further investigation. Future work will investigate effects on these behaviors in more detail.[46]

Aggression, Violence, and Criminal Behavior

We now turn to consider impacts on aggression, violence, and criminal behavior. Table 7 shows these elasticities, combining results that draw on a slightly different questions available in the two NLSY samples. First we 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 0.5. This appears to translate, though not completely, into actually hurting someone badly enough that they require medical attention: the elasticity for that outcome is 0.35. Similarly, the estimated elasticities for attacking someone by ages 13 or 17 are, respectively, 0.50 and 0.36. Results for other ages similar but are not shown in the table.

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 mis-reporting. 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.

[46] These 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.

In Table 7, being arrested or charged with a crime show elasticities of 0.38 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. In order 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.[47] For violent crime, the results are insignificant. For property crime, the elasticity is significant and substantial in both samples. Alternate specifications using a spline of blood lead yield similar results, with the primary difference being an effect for violent crime that is almost significant (elasticity 0.45 with a p-value of 1.6). Given the questions raised by these sub-category results, further work will test the robustness of these crime results in greater detail.

VII. 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 0.2 for child behavior problems, 1.0 for teenage pregnancy, 0.4 for teenage aggressive behavior, and 0.5 for teenage criminal behavior. These estimates are robust 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 as appropriate. Together, they paint a picture that exposure to lead in early childhood triggers an unfolding

[47] Note that these two categories do not include all types of crime delineated in the surveys, as listed above. The main types not included are drug crimes, traffic offenses, and other offenses to public order.

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 relevant to our understanding of environmental history or social behavior. It would be a moot point. 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, the societal-level effects on social behavior that are implied by the above results may not be hypothetical, but rather 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 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." I believe that the current results may present a slightly different 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, rather, 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, and current work in progress uses 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. The current paper suggests that these effects could be substantial.

VIII. Conclusion

This project aims to bring together and strengthen three largely separate 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, lead to lower IQ and higher prevalence of learning disabilities, and lead to more impulsive and aggressive behavior. Numerous studies investigate a range of exposure levels to

many different toxicants, but they 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. 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 societal benefits. This paper, and the larger research agenda of which it is a part, aims to join and strengthen these three threads.

Simply put, the results in this paper suggest that lead may be a crucial missing link in social scientists' explanations of social behavior. More broadly, the missing link may be the environmental toxicants that impair behavior and the regulations altering exposure to those toxicants. 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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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%	

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	1.5	0.3	1.3	1.5	1.7
Grams of lead per gallon of gasoline, 1980 to 1984	0.6	0.2	0.5	0.6	0.7
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

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

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

	Linear	*Log*	*Spline*
Blood Lead	0.230 **		
	(0.029)		
Log Blood Lead		1.776 **	
		(0.205)	
Blood Lead 0 to 5 mcg/dL			0.362 **
			(0.140)
Blood Lead 5 to 10 mcg/dL			0.325 **
			(0.055)
Blood Lead 10+ mcg/dL			0.025
			(0.059)
Age	0.012	0.018	0.021
	(0.051)	(0.051)	(0.051)
Female	-1.191 **	-1.189 **	-1.203 **
	(0.142)	(0.142)	(0.142)
Race - Black	-1.349 **	-1.131 **	-0.987 **
	(0.260)	(0.236)	(0.275)
Race - Hispanic	-0.233	-0.253	-0.250
	(0.272)	(0.272)	(0.272)
Race - Other	-0.109	-0.106	-0.099
	(0.201)	(0.201)	(0.201)
Mother's education - HS grad	-1.921 **	-1.919 **	-1.915 **
	(0.228)	(0.227)	(0.227)
Mother - teen mother	-0.268 *	-0.311 **	-0.310 **
	(0.159)	(0.159)	(0.160)
Income ($k)	-0.030 **	-0.029 **	-0.029 **
	(0.003)	(0.003)	(0.003)
Elasticity BPI wrt lead, at lead = 4	0.088 **	0.162 **	0.223 **
	(0.010)	(0.018)	(0.043)
Elasticity BPI wrt lead, at lead = 11	0.210 **	0.139 **	0.009 **
	(0.021)	(0.013)	(0.005)

Table 5a. Elasticities of Child Behavior Problems indices with respect to Blood Lead.

	Age 4-12	*Age 6-7*	*Age 8-9*	*Age 11-12*
Total	0.174 **	0.066	0.258 **	0.205 **
	(0.030)	(0.055)	(0.062)	(0.071)
Oppositional	0.188 **	0.103	0.249 **	0.192 **
	(0.035)	(0.065)	(0.068)	(0.080)
Antisocial	0.160 **	0.069	0.237 **	0.166 **
	(0.036)	(0.073)	(0.073)	(0.083)
Hyperactive	0.161 **	0.028	0.144 **	0.179 **
	(0.036)	(0.061)	(0.070)	(0.093)
Headstrong	0.226 **	0.197 **	0.303 **	0.285 **
	(0.045)	(0.088)	(0.092)	(0.103)
Behavior/Emotional Problems	0.247 **			
	(0.118)			

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

	Age 4-12	
	Sometimes	**Often**
Impulsive	0.040	0.145
	(0.036)	(0.125)
Bullies	0.111	0.257
	(0.074)	(0.168)
Strong Temper	0.148 **	0.416 **
	(0.046)	(0.118)

	Age 6-7		*Age 8-9*	
	Sometimes	**Often**	**Sometimes**	**Often**
Impulsive	-0.089	-0.391 *	0.032	0.114
	(0.046)	(0.216)	(0.066)	(0.227)
Bullies	0.021	0.045	0.243 *	0.552 *
	(0.153)	(0.329)	(0.152)	(0.325)
Strong Temper	0.131	0.369	0.214 *	0.626 *
	(0.089)	(0.230)	(0.099)	(0.250)

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		1.932 **
		(0.844)
Pregnant by age 17	1.041 **	0.913 **
	(0.558)	(0.343)
Pregnant by age 19	1.142 **	0.896 **
	(0.439)	(0.209)
Got partner pregnant by age 19		0.572
		(0.728)
Substance Use		
Alcohol by age 13	0.556 **	0.234 *
	(0.189)	(0.140)
Cigarettes by age 13	0.257	0.121
	(0.195)	(0.133)
Marijuana by age 17	0.568 **	0.044
	(0.159)	(0.079)

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	0.517 ** (0.171)	
Hit someone by age 17	0.542 ** (0.134)	
Hurt someone badly by age 17	0.346 ** (0.140)	
Attacked someone by age 13		0.499 ** (0.237)
Attacked someone by age 17		0.358 ** (0.127)
Crime (by age 17)		
Arrested		0.375 ** (0.173)
Charged		0.513 ** (0.244)
Convicted	0.601 ** (0.285)	0.643 ** (0.324)
Convicted of a violent crime	0.729 (0.778)	0.207 (0.807)
Convicted of a property crime	2.155 ** (0.552)	0.693 ** (0.523)

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		