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Developmental lead exposure and adult criminal behavior: A 30-year prospective birth cohort study

John Paul Wright^{a,*}, Bruce P. Lanphear^b, Kim N. Dietrich^c, Michelle Bolger^d, Lisa Tully^e, Kim M. Cecil^{c,e}, Catherine Sacarellos^a

^aSchool of Criminal Justice, University of Cincinnati, OH 45221, United States of America

^bFaculty of Health Sciences, Simon Fraser University and Child and Family Research Institute, British Columbia Children's Hospital, Vancouver, British Columbia, Canada

^cDepartment of Environmental Health, Division of Epidemiology, University of Cincinnati College of Medicine, Cincinnati, OH 45267, United States of America

^dDepartment of Social Science, Criminal Justice Program, DeSales University, Center Valley, PA 18034, United States of America

^eDepartment of Radiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH 45229, United States of America

Abstract

Using a variety of research designs and measures of lead absorption, numerous studies link childhood lead exposure to a range of cognitive and behavioral deficits, including low IQ, impulsivity, juvenile delinquency, and criminal behavior in adolescence and early adulthood. In this study, we tested the association between multiple measures of blood lead concentration assessed in childhood with criminal behavior in adulthood and across the life-course. Prospective data from the Cincinnati Lead Study (CLS) included blood lead measures quarterly across the first 78 months of life and the number of times a person was arrested across the life-course (from age 18 to 33 years) and in later adulthood (age 27 to 33 years). Childhood blood lead concentration prospectively predicted variation in adult arrests and arrests over the life-course, indicating lead absorption is implicated in the etiology of crime—especially in geographic areas where environmental sources of lead are more prevalent and concentrated. Efforts to decrease lead exposure in both developed and developing countries should be part of a comprehensive strategy to reduce social dislocation and crime.

Keywords

Lead absorption; Crime; Criminal behavior; Prospective study

* Corresponding author at: School of Criminal Justice, University of Cincinnati, Cincinnati, OH 45221, United States of America. john.wright@uc.edu (J.P. Wright).

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1. Introduction

Lead (Pb), a recognized developmental neurotoxicant, is associated with a diverse range of inter-related, pervasive, and cascading negative effects that compromise healthy physiological and nervous system functioning (Basha et al., 2005; Grandjean and Landrigan, 2006; World Health Organization, 2010). Indeed, over 5 decades of animal (Aykin-Burns et al., 2003; Delville, 1999) and human research (Bellinger et al., 1992; Chiodo et al., 2004) has documented the variety of biological systems pathologically influenced by lead absorption. Lead absorption has been found to disrupt synaptogenesis, neurotransmission and, at higher levels, cause encephalopathy (Bunn et al., 2001; Emory et al., 1999).

Adverse biological consequences of early lead exposure prompted investigations into the effects of lead on other developmental outcomes. Chief amongst these outcomes were IQ scores (Landrigan et al., 1975; Needleman et al., 1979). These studies, which have used a variety of measures of body lead burden, including blood lead levels, dentine levels, and K-line X-ray fluorescence spectroscopy, have converged to show that early life lead ingestion is associated with significantly decreased scores on standardized measures of intelligence (Canfield et al., 2003; Lanphear et al., 2005; U. S. Department of Health and Human Services, 2012; Wasserman et al., 2003). Indeed, three meta-analytic studies and a pooled analysis of the lead-IQ relationship found that childhood blood lead concentration was associated with a reduction in IQ scores of between 2 and 7 points; the deleterious effects of lead on IQ have also been documented at low levels of lead in blood, below 10 µg/dL (Lanphear et al., 2005; Nation and Gleaves, 2001; Needleman and Gatsonis, 1990; Schwartz, 1994).

Research into the effects of lead on human development has included a broader array of behavioral outcomes. These studies, which include exposure levels measured at the national (Nevin, 2000, 2007), county (Haynes et al., 2011; Stretesky and Lynch, 2001), and individual level, have linked lead absorption to ADHD (Braun et al., 2006), childhood conduct problems (Braun et al., 2008), and poor school performance (Magzamen et al., 2013). Of particular relevance to this study are the empirically established connections between early childhood body lead burden and aggression (Olympio et al., 2010), juvenile delinquency (Dietrich et al., 2001; Needleman et al., 2002; Needleman et al., 1996), and crime in adolescence and early adulthood (Wright et al., 2008). These studies are particularly important because chronic antisocial behavior often develops early in the life-course (DeLisi, 2006), is situationally and temporally stable (Lynam et al., 2009), and is associated with a life-course marred by imprisonment, criminal victimization, and premature mortality (Maughan et al., 2014). Several scholars note that serious and frequent adult criminal behavior virtually requires childhood antisocial conduct (Robins, 1978). Identifying early-life factors associated with persistent offending thus takes on a special urgency.

One possible factor related to problem behavior across the life-course is lead. Studies at the aggregate level report positive associations between air lead concentrations and crime rates. Nevin (2000, 2007), for example, reported striking temporal associations between crime rates and environmental lead levels. Analyzing air lead concentrations found in 3111 counties in the United States, Stretesky and Lynch (2001) reported air lead concentrations

significantly predicted homicide rates. In a study of 88 counties in Ohio, Haynes et al. (2011) found that CDC reported air lead levels significantly predicted county-level adjudication rates for adolescent offenders. Finally, Boutwell et al. (2017) analyzed the association between blood lead levels and violent crime across the 106 Census tracts that compose St. Louis, MO. Boutwell et al., geocoded the addresses of 59,645 children under the age of 6 who were tested between 1996 and 2007, aggregating the average blood lead level to their geocoded census tract. All violent crimes reported to the St. Louis Metropolitan Police during this period were also geocoded and aggregated to their respective census tracts. Their results revealed significant associations between aggregated blood lead levels and firearms crimes, assault, robbery, and homicide.

At the individual level, numerous studies link early life lead ingestion to a diverse array of behavioral problems, including delinquent and criminal behavior (Froehlich et al., 2009; Marcus et al., 2010). A meta-analysis of 19 studies of 8561 individuals between the ages of 4 and 18 reported a modest positive effect of lead on child and adolescent problem behavior ($r = 0.19$, $d = 0.39$; Marcus et al., 2010). Effect sizes varied by lead measure, with hair analyses producing the largest correlation, followed by blood lead, dentine levels, and bone K-line -X-ray fluorescence spectroscopy. Effect sizes were not significantly modified by confounders, such as SES or home environment (Marcus et al., 2010).

Analyzing bone lead levels of 194 adjudicated youth and 146 non-adjudicated youth, Needleman et al. (2002) and his colleagues found that elevated bone lead levels were 4-times greater in adjudicated youth. Using data from the Cincinnati Lead Study, Dietrich et al. (2001) and his colleagues found that childhood blood lead concentrations significantly predicted self-reported delinquency. In a follow-up study of the same prospective sample, they found that prenatal, 78-month, and average childhood blood lead measures significantly predicted the frequency of arrest from age 18 to 24 (Wright et al., 2008). Significant positive associations between dental enamel lead concentrations and self-reported problem behaviors were recently reported in a sample of 173 Brazilian youth, although dental lead levels were not associated with a composite measure of self-reported delinquency (Olympio et al., 2010). Finally, Sampson and Winter (2018) followed a subgroup of children ($n = 212$) from the longitudinal, multicohort Project on Human Development in Chicago (PHDCN). Average blood lead levels, measured before age 6, significantly predicted scores on the CBC one-year later and again at age 17. No association was found, however, between early blood lead levels and future arrests.

The current study examines the potential effects of early lead absorption on later criminal offending by extending a previously published examination of the Cincinnati Lead Study (CLS) cohort by Wright et al. (2008) with arrest counts from the latest data collection period—a period that spans 10 years since the last collection wave. As such, we examine the link between early life lead absorption, assessed through multiple blood lead measurements taken over the first 78 months of life, and adult criminal behavior measured from 2003 to 2013. Since the CLS contains detailed arrest counts spanning the adult life-course, we are also able to test the association between early lead absorption and a cumulative measure of lifetime arrests (from 1998 to 2013). Moreover, we test whether arrests that occurred when respondents were between the ages of 18 and 24 account for the association between early

lead absorption and arrests in adulthood (from 2003 to 2013). Collectively, our analyses span the impact of childhood lead absorption on arrests that occur 25 to 30 years in the future.

2. Materials and methods

2.1. Subjects

The Cincinnati Lead Study (CLS) is a prospective longitudinal birth cohort of women and their offspring recruited from neighborhoods within Cincinnati, Ohio from 1979 to 1984 (Wright et al., 2008). Pregnant women in the first or second trimester receiving services in prenatal clinics within the catchment area, which were characterized by high levels of poverty and a high prevalence of lead poisoning, were approached for participation within the study. Women who had diabetes, presented with psychiatric problems, were addicted to drugs or alcohol, or refused participation were excluded. Offspring weighing less than 1500 g, with less than a 35-week gestational age, with a five-minute APGAR score less than 6, or with serious medical issues, including defined genetic syndromes, were also excluded (Dietrich et al., 1987).

Initially, 376 newborns were recruited with 305 infants undergoing developmental and medical examinations at three and six months. Offspring were assessed quarterly until age 60 months and semiannually from 66 months to 78 months. Children were assessed again at 10 years of age, 16–17 years of age, between the ages of 19 to 24 years and, more recently, when subjects ranged in age from 27 to 33 years. A total of 254 participants constitute the current cohort with sample sizes ranging from 190 to 220 depending on the blood lead measure. Missing cases were primarily the result of death ($n = 4$) or state custody ($n = 24$). Compared to subjects not under custody, those in state custody had significantly higher average lifetime blood lead concentrations ($t = 2.12$, $p = .022$), average late childhood blood lead concentrations ($t = 2.88$, $p = .004$), and late childhood blood lead concentrations ($t = 3.10$, $p = .003$). No mean differences were found using a measure of prenatal blood lead concentrations ($t = 1.10$, $p = .143$).

Examinations of this cohort have not detected significant differences in blood lead levels or developmental indices due to missing data, nor did we detect nonrandom attrition due to death or incarceration (Dietrich et al., 2001; Hornung et al., 2009; Wright et al., 2008). We obtained informed consent from mothers at each stage of assessment and from their children at age 18 years and beyond. CLS study protocols have been approved and are under continuous annual review by the Institutional Review Boards at the University of Cincinnati, College of Medicine and Cincinnati Children's Hospital Medical Center.

2.2. Blood lead measures

We assessed blood lead concentrations prenatally, and during the first or early second trimester. Thereafter, venous blood lead concentrations were measured up to 23 times across childhood—quarterly from three months to 60 months and semiannually from 66 to 78 months. Blood lead data are available for 89%–92% of the cohort from 3 months to 5 years of age. Whole blood was analyzed by anodic stripping voltammetry at the Hematology and Environmental Chemistry Laboratory of the Kettering Laboratory, Department of

Environmental Health. The performance of this former national reference laboratory was uniformly excellent throughout the course of these studies (Roda et al., 1988). We used four blood lead indices: prenatal maternal blood lead concentrations ($\mu\text{g/dL}$), available for 87% of the sample (219/250); average childhood blood lead concentrations ($\mu\text{g/dL}$); average late childhood blood lead concentrations assessed across 60 to 78 months ($\mu\text{g/dL}$); and late childhood blood lead concentrations measured at 78 months. For 23 cases that were missing data on the 78-month blood lead test, we used the measure at 72 months (Wright et al., 2008).

2.3. Criminal behavior measures

Our dependent variable is a count measure of the number of criminal arrests. We obtained adult arrest histories from public records in Hamilton County, OH when subjects were between the ages of 18 and 24 years, and again when subjects were between the ages of 27 and 33 years. Public data on arrest histories are updated frequently and provided a relatively comprehensive picture of the nature of the offenses and any resulting dispositions. Arrest represents a conservative measure of criminal involvement. First, several studies find that measures of arrest are downwardly biased indicators of actual criminal behavior (Hindelang et al., 1981). In a longitudinal study of 411 boys from London, England, Farrington et al. (2014) found that self-reported crimes of convicted offenders were 30 times greater compared to non-convicted individuals. In an analysis of 506 boys from Pittsburgh, offenders reported an average of 80 self-reported crimes for every 1 arrest (Farrington et al., 2007). Second, restricting criminal records searches to a single county, as we have done, potentially underestimates a respondent's true criminal involvement. Some offenders may have committed crimes in other jurisdictions that resulted in arrest—arrests that would not be counted in our analyses. Third, studies indicate that the majority of reported and detected crimes do not result in arrest and that arrest decisions are based predominately on the seriousness of the offense, on the offender's prior record, on the victim's request, and on any mandatory arrest policies that may exist (Smith and Visser, 1981). Finally, measures of arrest are the least influenced by criminal justice system processing—that is, they represent the entry point into the criminal justice system and thus are not as subject to the various legal and extra-legal biases that keep some cases in the system but that exclude other cases.

Two reviewers independently coded subjects' arrest histories and the nature of the arrest. These reviewers categorized arrests into violent offenses (murder, rape, robbery, assault, domestic violence, and weapons possession), property offenses (burglary, larceny, theft by deception), drug offenses (distribution, manufacturing, possession), fraud, obstruction of justice, and motor vehicle offenses (serious and non-serious). Differences between reviewers in the coding of arrests, which occurred in less than 1% of cases, were resolved by a third reviewer. Individuals who committed or who were charged with multiple crimes from a single criminal event were coded as a single arrest incident.

We analyzed a total of five arrest measures. The first four were from the 2003–2013 follow-up period and include the total number of arrests for each subject, exclusive of minor vehicle arrests, the total number of arrests for violent crimes, the total number of arrests for drug crimes, and the total number of arrests for property crimes. The summated arrest counts

for violent crimes, property crimes, and drug crimes account for the majority of all arrests from the 2003–2013 measurement period. The CLS, however, contains the complete arrest histories of subjects from the age of 18 years onward. Capitalizing on the longitudinal nature of the data, we also created a measure of the total number of arrests from ages 18 to 33 years or from 1998 to 2013. This measure reflects the accumulated number of arrests over the respondent's lifetime.

Assessments of criminal conduct at one point in time are modestly to strongly correlated with the same assessments of criminal behavior in the future. This is true also in the CLS, where the measure of prior arrests (ages 18 to 24 years) correlates with contemporaneous measures of arrest ($r = 0.51$). In later analyses, we control for arrests between ages 18 and 24 to predict future adult arrests. This strategy allows for a conservative assessment of the effects of the lead measures on adult arrests.

2.4. Potential confounds

The CLS contains a large number of variables, including potential confounds. We selected confounds that prior studies have linked to variation in neuropsychological functioning since lead is believed to jeopardize neuropsychological health. Our analyses thus control for birth weight (grams), maternal age at delivery, APGAR scores taken at 1 min, self-reported maternal drug use during pregnancy that includes reports of alcohol, marijuana and tobacco use, maternal IQ measured by the WAIS-R, and the Home Observation for Measurement of the Environment (HOME) Inventory scores across the first 3 years. Results of these analyses concurred with prior analyses of these data, revealing that confounding in the CLS is limited (Dietrich et al., 2001; Hornung et al., 2009; Wright et al., 2008). Even so, we included these independent variables in each analysis and report the adjusted incident rate ratios. Table 1 in Appendix A presents the bivariate correlation matrix of key measures, while Table 2 in Appendix A presents negative binomial results for the total number of arrests from 2003 to 2013 and for lifetime arrests, inclusive of all statistical controls for each blood lead measure.

2.5. Statistical analyses

Arrest counts are positively skewed and Poisson regression results indicated over-dispersion. To account for over-dispersion, all models were estimated using negative binomial regression with Huber-White corrected standard errors. Multiplicative interactions between lead and sex, and lead and race were estimated across all equations. No significant interactions were detected. We report incident rate ratios with corresponding 95% confidence intervals and use two-tailed statistical tests. For sake of scaling, we converted all blood lead measures to reflect 5 µg/dL intervals. All analyses were conducted in Stata 14 (StataCorp, 2013).

3. Results

3.1. Descriptive analysis

Table 1 presents descriptive statistics on sample mothers and their offspring as well as descriptive information on the total and sex-specific distribution of arrests. As also discussed in Wright et al. (2008), the CLS is 90% African-American, is split evenly between males

and females, and 84% of the households were headed by an unmarried mother. Families in the CLS were economically disadvantaged, with 73% scoring in the lowest two levels of the Hollingshead Index and slightly over 50% of mother's having graduated from high school at the time of delivery. Maternal IQ scores, measured by use of the WAIS-R, were relatively low, with full scale scores averaging 75. Overall, the sample is relatively homogenous racially and economically owing, in part, to the geographic location of the catchment areas.

Excluding minor traffic offenses, 254 CLS participants generated a total of 1429 total independent lifetime arrests and 1129 independent arrests from October 2003 through June 2013. The average CLS subject experienced 5.6 lifetime arrests and 4.2 arrests from 2003 through 2013 (ages 27 to 33 years). Males ($n = 121$) averaged 8.15 arrests over their adult lifetime, generating 986 independent arrests, while females ($n = 133$) averaged 3.3 arrests and generated 443 total arrests. In the current wave, 14 individuals (12 males) were found incarcerated in local and state institutions. Overall, 22% of respondents had never been arrested, including 18.2% of males, and 25.6% of females. From 2003 to 2013 22% of males had not been arrested compared to 30.6% of females. These proportions parallel those found in other urban samples (Brame et al., 2014).

Fig. 1 graphically depicts the blood lead levels for CLS participants averaged across the first 72 months of life. The majority of the sample had average blood lead levels in excess of 10 $\mu\text{g}/\text{dL}$ across childhood ($\bar{x} = 14.4 \mu\text{g}/\text{dL}$, $s = 6.6 \mu\text{g}/\text{dL}$). Only three subjects in the CLS cohort received a diagnostic chelation; none received therapeutic chelation. This was in concert with the national standard of care for lead exposed infants and children during the early years of this long-term prospective study.

3.2. Multivariate analysis

Because our analyses involve multiple dependent variables and multiple blood lead measures, we present reduced form results in Tables 2 and 3. A correlation matrix and the full results for adult arrests and lifetime arrests are presented in Appendix A. Examining first the results in Table 2, we found that total adult arrests from 2003 to 2013 were significantly predicted by prenatal blood lead levels ($RR = 1.15$, 95% $CI = 1.03\text{--}1.27$), by average late childhood blood lead levels ($RR = 1.07$, 95% $CI = 1.01\text{--}1.14$), and by 6-year blood lead levels ($RR = 1.07$, 95% $CI = 1.00\text{--}1.14$). Violent arrests and arrests for property crimes were not significantly predicted by the blood lead measures, however, prenatal, late childhood, and 6-year blood lead measures predicted drug arrests. Incident rate ratios ranged from 1.07 for average childhood blood lead levels to 1.20 for prenatal maternal blood lead levels.

The last column in Table 2 presents the results for the cumulative measure of arrests over a subject's lifetime, from age 18 to 33. Three of the blood lead measures prospectively predicted variation in lifetime arrests: Prenatal blood lead ($RR = 1.17$, 95% $CI = 1.06\text{--}1.28$), average late childhood blood lead ($RR = 1.07$, 95% $CI = 1.01\text{--}1.14$), and 6-year blood lead levels ($RR = 1.08$, 95% $CI = 1.01\text{--}1.16$). Overall, the results presented in Table 2 reveal that measures of childhood body lead burden are prospectively related to arrests in adulthood, arrests over the lifetime, and especially arrests for drug law violations. The association between early lead absorption and arrests for violent crime and property crime was consistently null.

Table 3 presents the negative binomial results for the blood lead measures on adult arrests (2003 – 2013) including the control variables but with the addition of the total number of arrests of respondents from 1998 to 2003. Recall that criminal behavior, as measured by arrest, can be relatively stable over time with arrests occurring earlier in life predicting future arrests. In many instances, controls for prior criminal behavior attenuate or account completely for many reported associations. By including a measure of arrests that occurred earlier in life, we provide a conservative test of the linkages between the measures of early blood lead levels and adult criminal conduct. For sake of contrast, we also present the estimates from Table 2 for adult arrests, labeled baseline estimates.

In 2 of the 4 equations, the blood lead assessments significantly predicted adult arrests, including average childhood blood lead levels (RR = 1.05, 95% CI = 1.01–1.09) and average late childhood blood lead levels (RR = 1.06, 95% CI = 1.01–1.12). Conversely, the association between prenatal and 6-year blood lead assessment and adult arrests was null. Overall, a 5 µg/dL increase in childhood blood lead levels corresponded to a 5 to 6% increase in the rate of change in arrests over time.

4. Discussion

The CLS is the longest running prospective study on the effects of early life lead exposure. Data from this sample have provided statistical evidence that body lead burden in early in life is statistically predictive of juvenile delinquency and arrests in young adults. The current study extends these results by now showing that early childhood blood lead levels predict variation in arrests well into adulthood and across the life-course. A variety of blood lead measures assessed prenatally and through the first 78 months of life, predicted variation in adult arrests when subjects were between 27 and 33 years of age, including a measure of total arrests, arrests for violent crime, and arrests for drug crimes. Arrests for property crimes were unrelated to the blood lead indices. Measures of childhood blood lead levels, however, also predicted arrests across respondent's life-course, from age 18 to 33. Moreover, the effect of early blood lead levels on adult crime withstood a control for arrests accumulated earlier in life, lending confidence that our blood lead measures are not spuriously associated to adult arrests. At least in the CLS data, the signature left by early life lead ingestion remained detectable into the 3rd decade of life.

Lead in early childhood imparts greater risk for atypical developmental than does lead absorption occurring in adulthood (Dietert and Piepenbrink, 2006; Hornung et al., 2009; Rogan et al., 2001). Reasons for increased early susceptibility are varied. Maternal bone lead stores, for example, become bioavailable during pregnancy, with lead crossing through the blood-brain barrier and preferentially binding to calcium (Ca^{2+}) and zinc (Zn^{2+}) binding sites (Hornung et al., 2009; Markowitz, 2000). Children living in homes or neighborhoods with elevated dust, paint, or air lead levels absorb a greater fraction of available lead than do adults living in the same household. In a direct test of the age of greatest susceptibility, Hornung et al. (2009) found that the ratio of 6-year blood lead levels to 2-year blood lead levels had the strongest adverse impact on Wechsler IQ scores assessed at 6 years of age and on arrests occurring in early adulthood. We evaluated the ratio of 6-year blood lead

levels to 2-year levels across all outcomes. The results were consistently null suggesting that susceptibility may be more closely related to arrest onset than long-term arrest patterns.

The precise mechanism linking early lead to criminal behavior remains unknown, however, we offer two possibilities. First, as a calcium mimicking ion, lead ingestion has been linked to activation of protein kinase C, damage to N-Methyl-D-Aspartate receptors, interference with DNA repair through its effects on apurinic/apyrimidinic endonuclease 1, and increased demyelination and axonal degeneration within mammalian white matter (Brubaker et al., 2009; Hwang et al., 2002; Marchetti, 2003; McNeill et al., 2004; Windebank, 1986). Imaging studies of the CLS cohort found childhood blood lead levels predictive of altered myelination and reduced axonal integrity (Brubaker et al., 2009), reduced gray matter volume, and region-specific reductions in the anterior cingulate cortex (ACC) and the prefrontal cortex, especially for males (Brubaker et al., 2010; Cecil et al., 2008). Other studies have linked hypoactivity in the prefrontal cortex and dysregulation in the ACC with impulsive violent behavior (Raine, 2002). Deficient executive functions, particularly low impulse control, have consistently been linked to criminal behavior across a broad range of studies (Moffitt et al., 2011). Early lead exposure, ingestion, and subsequent absorption likely compromises regions of the brain associated with behavioral regulation.

Second, although less explored, animal and human studies suggest that early lead absorption may reduce dopaminergic responsivity, potentially leading to deficiencies in attentional control and to drug use and abuse (Jones and Miller, 2008). Animals exposed to lead prior to or shortly after birth have been found to self-administer opiates at higher relative dosages and frequencies than those not exposed (Rocha et al., 2004). In a study of female intravenous drug users, Fishbein et al. (2008) found tibial bone lead concentrations 1.8 times higher than age-adjusted non-using women, and significant statistical interactions between tibial lead levels and measures of cognitive flexibility and risky decision-making. In the most recent imaging study with 155 CLS participants, Beckwith et al., 2018 reported that approximately 52% of the cohort tested positive for illicit drug use, primarily marijuana. (Beckwith et al., 2018) Our findings, too, suggest a link between childhood blood lead levels and future drug-related arrests. While we were unable to differentiate arrests for drug distribution from arrests for drug use, our findings add to a small but growing body of evidence implicating lead absorption in the etiology of drug related conduct.

The majority of observational studies on the link between childhood lead absorption and later problem behaviors have been limited to outcomes measured in adolescence or early adulthood. Recently, however, Beckley et al. (2018) reported results from their analysis of data from Dunedin, New Zealand on a cohort ($n = 553$) of individuals with blood lead levels measured at age 11 and cumulative criminal conviction data out to age thirty-eight. Overall, Beckley et al. (2018) found weak and inconsistent evidence linking childhood blood lead levels to future criminal convictions. Our results, however, contrast with theirs by showing a link between multiple indices of early life blood lead absorption and arrests from age 18 to 33. The Dunedin data contained a single blood lead measure, assessed relatively late in childhood, and relied on a measure of criminal conviction as an outcome variable. The use of criminal conviction, as opposed to a measure of arrest, combined with a single assessment

of blood lead concentration, may have attenuated their results. Moreover, we note that neither the CLS nor the Dunedin data are confounded by SES.

Average blood lead levels have dropped precipitously in Western countries over the last several decades. At the same time, lead exposure has emerged as a major health concern in several developing countries (Burki, 2012; Dooyema et al., 2012). While we are just now beginning to understand the long-term legacy of childhood lead ingestion in Western society, childhood lead absorption at levels that far exceed those historically detected in industrialized countries have been reported in developing countries—countries without adequate healthcare and without adequate environmental protections. Morbidity and mortality rates associated with childhood lead absorption in these regions will likely be one part of a broader constellation of negative effects associated with lead—a constellation of effects that include antisocial behavior expressed over the life-course.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix A

Table 1A

Pearson product moment correlation coefficients of key variables.

	1	2	3	4	5	6	7	8	9	10	11	12	13
Age	1.0												
Sex	−0.01	1.0											
Race	−0.19	0.01	1.0										
WISC-R	−0.00	−0.16	−0.10	1.0									
Prenatal BL	0.22	0.06	0.20	−0.02	1.0								
Average Childhood BL	0.28	0.01	−0.09	−0.16	0.30	1.0							
Late Childhood BL	0.37	0.01	−0.08	−0.22	0.33	0.91	1.0						
Six-Year BL	0.24	0.05	−0.01	−0.25	0.30	0.81	0.89	1.0					
Violent Arrests	−0.04	0.25	−0.04	−0.13	0.13	0.09	0.09	0.11	1.0				
Drug Arrests	−0.02	0.38	0.11	−0.11	0.11	0.12	0.11	0.14	0.36	1.0			
Property Arrests	0.03	0.23	−0.09	−0.12	0.01	0.14	0.14	0.12	0.46	0.21	1.0		
Total Arrests 2003–2013	−0.03	0.33	0.08	−0.19	0.16	0.15	0.17	0.19	0.74	0.74	0.55	1.0	
Lifetime Arrests	0.10	0.34	0.03	−0.18	0.21	0.16	0.20	0.24	0.72	0.72	0.45	0.92	1.0

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Fig. 1.
The Distribution of Average Lifetime Blood Lead Levels with Associated Standard Errors.

Table 1

Descriptive statistics for maternal and offspring CLS participants.

Variables	Mean	Std. deviation	Range	Total
Age of respondent (2013)	30	1.52	27–33	
Race (0 = Black, 1 = White)	0.90	0.30	0–1	
Sex (1 = Female)	0.50	0.50	0–1	
Maternal IQ	75	9.26	55–110	
Birth weight in grams	3144	454	1814–4340	
APGAR 1 Minute	7.91	1.30	1–9	
HOME Index (36 Month)	32	6.56	13–48	
Maternal drug use	0.95	1.00	0–5	
Full scale IQ	87	11.30	62–118	
Arrests 2003–2013	4.35	5.94	0–34	1129
Males	5.58	7.27	0–34	754
Females	2.80	3.33	0–19	375
Lifetime arrests	5.63	8.27	0–48	1429
Males	8.15	10.65	0–48	986
Females	3.33	4.10	0–20	443

Table 2

Negative binomial regression results for the association between childhood blood lead measures, adult arrests 2003–2013, and lifetime arrests.

Blood lead variable	Adult arrests	Violent arrests	Drug arrests	Property arrests	Lifetime arrests
IRR (95% C.I.)					
Prenatal blood lead	1.15 (1.03–1.27) ^a	1.17 (0.98–1.40)	1.21 (1.02–1.43) ^a	0.96 (0.80–1.14)	1.16 (1.06–1.28) ^a
Average childhood blood lead	1.03 (0.99–1.08)	1.01 (0.94–1.09)	1.06 (0.98–1.15)	1.03 (0.96–1.11)	1.02 (0.97–1.07)
Average late childhood blood lead	1.07 (1.01–1.13) ^a	1.04 (0.96–1.14)	1.13 (1.03–1.24) ^a	1.02 (0.93–1.12)	1.06 (1.00–1.13) ^a
6-year blood lead	1.07 (1.00–1.14)	1.08 (0.96–1.21)	1.17 (1.02–1.33) ^a	1.00 (0.89–1.12)	1.08 (1.01–1.16) ^a

^a Coefficients significant at $P < .05$, two-tailed tests, Huber-White standard errors with age, race, sex, maternal IQ, birth weight (in grams), HOME 36 Month, Maternal age at delivery, Apgar 1-min, Maternal drug use during pregnancy, and WISC-R controlled in each equation.

Table 3

Negative Binomial Regression Results of the Association Between Childhood Blood Lead Measures and Adult Arrests 2003–2013 with Controls for Prior Arrests from 1998 to 2003.

Blood lead variable	Adult arrests	Adult arrests	Adult arrests	Adult arrests
IRR (95% C.I.)				
Prenatal blood lead	1.09 (0.99–1.20)			
Baseline estimate	1.15 (1.03–1.27) ^a			
Average childhood Blood lead		1.05 (1.00–1.09) ^a		
Baseline estimate		1.03 (0.99–1.08)		
Average late childhood blood lead			1.06 (1.01–1.12) ^a	
Baseline estimate			1.07 (1.01–1.13) ^a	
6-Year blood lead				1.04 (0.98–1.11)
Baseline estimate				1.07 (1.00–1.14)

^aCoefficients significant at $P < .05$, two-tailed tests, Huber-White standard errors with age, race, sex, maternal IQ, birth weight (in grams), HOME 36 month, Maternal age at delivery, APGAR 1-min, Maternal drug use, WISC-R, and arrests from 1998 to 2003 controlled in each equation.