

Early exposure to lead and juvenile delinquency

Kim N. Dietrich^{a,b,c,*}, M. Douglas Ris^{b,c}, Paul A. Succop^a,
Omer G. Berger^{b,c}, Robert L. Bornschein^a

^aDepartment of Environmental Health, Division of Epidemiology and Biostatistics,
University of Cincinnati College of Medicine, Cincinnati, OH 45267-0056, USA

^bDepartment of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, OH, USA

^cChildren's Hospital Medical Center of Cincinnati, Cincinnati, OH, USA

Received 15 March 2001; received in revised form 14 August 2001; accepted 18 September 2001

Abstract

Cross-sectional studies have reported an association between lead (Pb) levels in bone and delinquent behavior in later childhood and adolescence. This is the first prospective longitudinal study of Pb and child development to address this question with comprehensive assessments of toxicant exposure and other developmental cofactors. A prospective longitudinal birth cohort of 195 urban, inner-city adolescents recruited between 1979 and 1985 was examined. Relationships between prenatal and postnatal exposure to Pb (serial blood Pb determinations) and antisocial and delinquent behaviors (self- and parental reports) were examined. Prenatal exposure to Pb was significantly associated with a covariate-adjusted increase in the frequency of parent-reported delinquent and antisocial behaviors, while prenatal and postnatal exposure to Pb was significantly associated with a covariate-adjusted increase in frequency of self-reported delinquent and antisocial behaviors, including marijuana use. Use of marijuana itself by Cincinnati Lead Study (CLS) teens was strongly associated with all measures of delinquent and antisocial behavior. This prospective longitudinal study confirmed earlier clinical observations and recent retrospective studies that have linked Pb exposure with antisocial behavior in children and adolescents. Both prenatal and postnatal exposure to Pb were associated with reported antisocial acts and may play a measurable role in the epigenesis of behavioral problems independent of the other social and biomedical cofactors assessed in this study. © 2001 Elsevier Science Inc. All rights reserved.

Keywords: Lead; Delinquency; Development; Toxicology; Adolescence; Prospective studies

1. Introduction

More than 50 years ago, clinicians reported a high prevalence of severe behavior problems in a series of schoolchildren who survived acute Pb encephalopathy [5]. A recent prospective case study of a young female with symptomatic Pb poisoning noted dramatic regressions in social behavioral functioning following high-level intoxication [9]. In a survey of Philadelphia youths enrolled in the Collaborative Perinatal Project, it was found that the strongest predictor of criminality in males was a history of Pb poisoning [7]. In an investigation of approximately 300 juveniles enrolled in the Pittsburgh Youth Study, teachers

and parents reported a significant Pb-related association with the Delinquency and Aggressive clusters of the Achenbach Child Behavior Checklist (A-CBC). Higher bone Pb concentration was associated with a significantly increased risk of exceeding the clinical score on the Attention, Aggression and Delinquency subscales of the A-CBC following adjustment for potential confounding cofactors [20]. In a case-control study of 216 adjudicated delinquent youths, cases were found to have significantly higher bone Pb concentrations than sociodemographically matched controls from high schools in the city of Pittsburgh [21].

Lead is associated with a large number of alterations in central nervous system physiology in *in vitro* and *in vivo* animal models. Several of these pathological processes could potentially be involved in the epigenesis of antisocial behavior. Early exposure to Pb may interfere with synaptogenesis, disrupt apoptosis, lower levels of serotonin and increase levels of D-aminolevulinic acid, which may antagonize GABA inhibition. Lead also enhances both D₁ and

* Corresponding author. Department of Environmental Health, Division of Epidemiology and Biostatistics, University of Cincinnati College of Medicine, Cincinnati, OH 45267-0056, USA. Tel.: +1-513-558-0531 (office), +1-513-621-2654 (clinic); fax: +1-513-621-2812.

E-mail address: kim.dietrich@uc.edu (K.N. Dietrich).

D₂ dopamine sensitivity and alters *N*-methyl-D-aspartate receptor sensitivity. Effects of Pb on mitochondrial energy metabolism may also be important in the pathogenesis of neurological effects [29].

While the retrospective/observational work previously reported is suggestive [5,7,9,20,21], these findings need to be replicated in a prospective study design, which enables conclusions to be drawn about the relative incidence of events as they unfold in time. The purpose of this investigation was to appraise the relationship between early exposure to Pb and neuropsychological and later social functioning in middle adolescence. It was hypothesized that early exposure to Pb, as assessed by serial blood Pb (PbB) determinations from the prenatal period to approximately 6.5 years of age, would be associated with a higher frequency of delinquent acts as measured by standardized questionnaires administered to both the adolescent and primary caregiver.

2. Methods

The subjects of this investigation were recruited from the Cincinnati Lead Study (CLS), a birth cohort of approximately 300 subjects that have been followed since prenatal recruitment began in 1979 and concluded in early 1985 [10,12,13]. Women were recruited from obstetrical clinics located in the catchment area. Women known to be addicted to drugs, alcoholic, diabetic or those with proven neurologic disorders, psychoses or mental retardation were excluded. Infants less than 35 weeks gestation or less than 1500 g birth weight were excluded from postnatal recruitment. Infants with defined genetic syndromes or other serious medical conditions at birth were also ineligible for follow-up. The microanalytical laboratory at the University of Cincinnati Department of Environmental Health participates in several quality control programs for the measurement of Pb in whole blood. The performance of this laboratory has been uniformly superior throughout the course of this study and others conducted by Cincinnati investigators [24].

The study succeeded in recalling 195 CLS subjects for follow-up examinations between 1997 and 1999. Reasons for attrition since the last published follow-up assessment at 6.5 years [10] ($n=253$) included refusals ($n=4$), chronically missed appointments ($n=6$), inability to determine the current location of subjects' families ($n=38$), long-term incarceration ($n=4$), homicide ($n=2$), severe developmental disability ($n=2$) and the lack of a psychometrician on the day of appointment ($n=2$). Subjects in this analysis did not differ significantly from those lost to follow-up in terms of measures of Pb exposure, perinatal health, early school-age intelligence or socioeconomic status (SES).

Subjects were between approximately 15 and 17 years of age ($M=15.6$, $S.D.=0.8$ years). Ninety-two percent of the sample were African-American and 53% were male. Seventy-four percent of subjects' families were in the lowest two of the five levels on Hollinghead's Four-Factor Index of

Social Position [15]. Average parental IQ was low in this cohort ($M=75.3$, $S.D.=9.2$), but has been highly predictive of child IQ throughout the study [10]. The average level of education of the primary caregiver was 11.4 years ($S.D.=1.5$). Three out of four CLS households were headed by single parents.

Because of missing data for prenatal PbB and some essential covariates, the numbers of subjects available for the multivariable analyses were less than the total sample of 195. Thus, for prenatal PbB, $N=157$, whereas for analyses of postnatal PbB indices, $N=186$.

Prenatal (maternal) PbB levels were low ($M=8.9$, $S.D.=3.9$). By contemporary standards, some CLS subjects were exposed postnatally to high levels of environmental Pb during the first 6 years of life. Fig. 1 presents mean PbB concentrations over time for subjects divided into four groups on the basis of average childhood exposures. Blood was sampled from the subjects shortly after birth and on a quarterly basis thereafter until CLS children reached 5 years of age. Blood Pb levels were also assessed at 66, 72 and 78 months. During the first 5 years of life, 35% of the cohort had at least one PbB concentration in excess of 25 $\mu\text{g}/\text{dL}$, 79% in excess of 15 $\mu\text{g}/\text{dL}$ and over 99% exceeded 10 $\mu\text{g}/\text{dL}$, the current action level cited by the United States Centers for Disease Control [28]. When interviewed as adolescents, the CLS cohort had an average PbB concentration of only 2.8 ($S.D.=1.3$) $\mu\text{g}/\text{dL}$.

These analyses focused a priori on first trimester maternal prenatal PbB as an index of intrauterine exposure and two indices of postnatal Pb exposure (average childhood PbB or the mean of 22 samples collected on a quarterly and [after 5 years] on a biannual basis from birth to 6 years of age, and a late measure of body burden, PbB at approximately 6.5 years). Late measures of body burden have been particularly predictive of neuropsychological outcomes in several prospective studies of Pb and child development, including the CLS [7] and another conducted in Port Pirie, Australia [1]. Although speculative, higher PbB concentrations in late childhood may reflect individual differences in lead metabolism that may have a genetic or nutritional basis (i.e., retention/excretion). The possibility exists that this might make some children more vulnerable to Pb's late developmental neurotoxicity. For the purpose of data analysis, these PbB variables were treated as both continuous ($\mu\text{g}/\text{dL}$) and categorical (lowest to high) to help clarify dose-effect relationships.

All testing and interviews took place at a pediatric clinic located in the heart of the catchment area. Neuropsychological tests and interviews were conducted by a developmental psychologist (KND) or by trained assistants who were under daily supervision by the principal investigator. This investigation employed self- and parental report methods of documenting antisocial or delinquent behavior because they are more valid than official records in documenting socially deviant behavior. With the advent of reliable measures of self- and parental reports of delinquency, researchers have

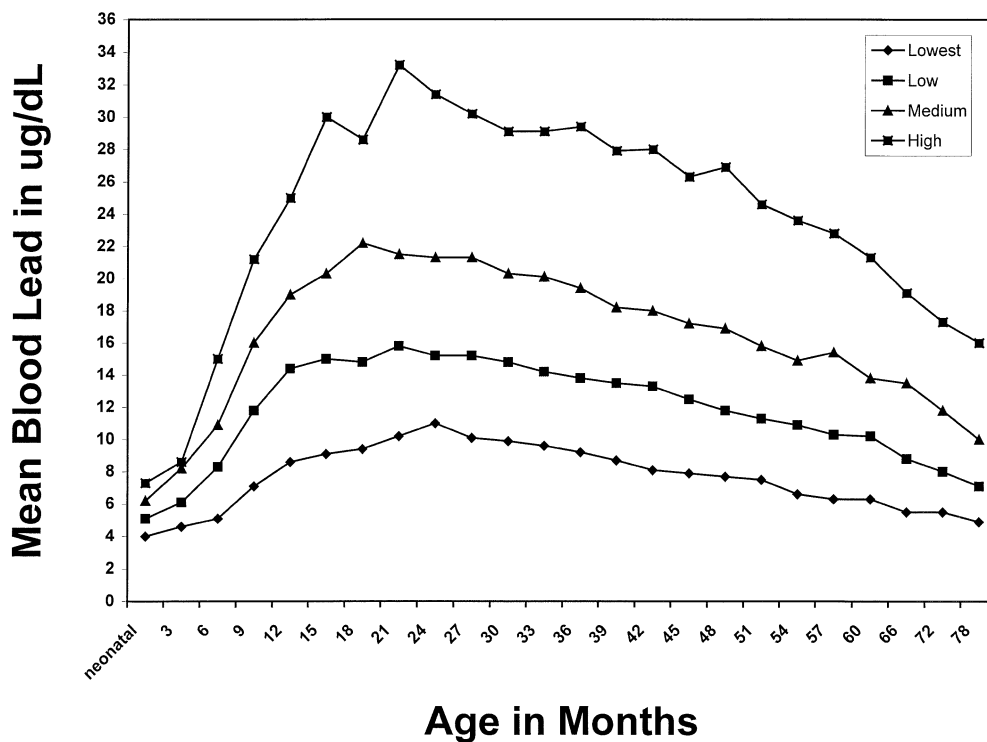


Fig. 1. Longitudinal PbB profiles for four groups of Cincinnati Lead Study subjects based upon average childhood PbB concentrations—the mean of 22 quarterly postnatal PbB determinations (lowest <10 µg/dL, $N=63$; low 10–15 µg/dL, $N=63$; medium 16–20 µg/dL, $N=43$; high >20 µg/dL, $N=26$).

learned that arrest statistics reflect only a small portion of antisocial acts actually committed by adolescents [27].

Each subject was administered the Self-Report of Delinquent Behavior (SRDB) [14]. This questionnaire conceptualizes delinquent conduct as behavior in violation of legal statutes, which involve some risk of arrest. Items on the questionnaire include offenses against property, persons and other illegal activities such as driving without a license or disorderly conduct. Self-report measures of delinquent and antisocial behavior are widely utilized in national surveys as reliance on official (e.g., police, court) sources of information introduces layers of potential bias between actual behavior and the data. Furthermore, the reliability and validity of recently developed self-report instruments such as the one utilized in the present investigation have been well-established [27].

Parents or legal guardians were administered the Parental Report of Predelinquent and Delinquent Behavior (PRDB) [11]. This survey questions the parent about problems the child may be having in school (e.g., truancy, fighting, suspensions), home (e.g., fighting, talking back, stealing from family members, staying out too late, running away) and community (e.g., police contact, court appearance, adjudicated delinquent, institutional placement). The staff's long-term relationship with CLS families and assurances of confidentiality enabled the study to collect these data on sensitive matters with considerable confidence in their validity.

These instruments yield a very large number of variables. However, for the purposes of this analysis, a limited number of outcomes were selected a priori to investigate. For self-reports, the total number of delinquent acts committed during the last 12 months was calculated. For the primary analysis, the total SRDB score was examined. For parental/guardian reports, total problems scores were calculated for delinquent/antisocial offenses occurring during the preceding 12 months in school, home and community. A total PRDB problems score was calculated by summing the number of problems reported to have occurred in school, the home and the community. Consumption of alcohol and drugs as well as use of tobacco by CLS subjects was also assessed with a self-report measure. [30] Previous work with this instrument has demonstrated its sensitivity to alcohol and drug related problems in young adults [22].

Because exposure to Pb is often correlated with other factors early in development that may contribute to cognitive and behavioral problems, a number of developmental cofactors were measured and examined for their confounding potential [12]. These included indices of obstetrical and perinatal complications, parental substance abuse, birth weight and gestational age, early measures of the quality of caregiving in the home environment, SES, parental IQ, subjects' gender and age. Variables entertained as potential covariates or confounders are listed in Table 1. Due to the small number of non-African-American subjects, we did not examine ethnicity as a covariate.

Table 1

Candidate covariates and confounders

Perinatal and child health factors

Maternal age at birth of subject
Birth weight
Birth length
Neonatal head circumference
Gestational age by physical exam [2]
Gestational age by dates
Apgar at 1 and 5 min
Obstetrical Complications Scale score [16]
Postnatal Complications Scale score [16]
Cigarette consumption during pregnancy (1/2 packs/day)
Alcohol consumption during pregnancy (yes/no)
Marijuana consumption during pregnancy (yes/no)
Narcotics use during pregnancy (yes/no)
Number of previous abortions
Number of previous stillbirths
Gravidity
Parity
Otitis media (number of discrete infections to 5 years of age)
Child's iron status at 1–6 years (hemoglobin, hematocrit and total iron binding capacity)
Consumption of alcohol and tobacco during adolescence [30]

Sociohereditary factors

Subject's gender
Subject's age at assessment
SES [15]
Mean HOME score (average of total score at 6, 12, 24 and 36–48 months) [6]
Maternal intelligence [26]
Highest grade attained by primary caregiver
Family on public assistance
Number of adults in home
Number of children in home
Subject attended a preschool program

The data analytic strategy employed by the CLS has been described previously in considerable detail [12,13]. Covariates were pretested for their confounding potential by examining their bivariate relationship with both PbB and the SRDB and PRDB total scores. Following both backward elimination and forward inclusion step-wise multiple regression analyses, those covariates which were independently related to either the SRDB and/or PRDB at $P \leq .10$ were included in all subsequent multiple regression analyses. For substantive reasons, certain variables were included in each regression analysis regardless of the statistical significance of their relationship to the SRDB or PRDB. These included mean HOME score, parental IQ and current SES. Each measure of antisocial/delinquent behavior was examined independently for their relationship to selected measures of Pb dose (prenatal PbB, average childhood PbB and 78-month PbB).

Consumption of alcohol and drugs as well as use of tobacco by CLS subjects was also assessed with a self-report measure.[30] Previous work with this instrument has demonstrated its sensitivity to alcohol and drug related problems in young adults [22]. Data analyses utilized multiple linear regression and analysis of covariance techniques that exam-

ined the relationships between blood indices of prenatal/postnatal Pb exposure and delinquent and antisocial behavior at approximately 16 years of age. All data analyses employed the Statistical Analysis System (SAS). Statistical significance for PbB variables was defined as a two-tailed P value $\leq .05$. Only covariate-adjusted PbB parameter estimates and group means are presented in this report.

3. Results

3.1. Delinquent and antisocial behavior

Self-reported acts of delinquent behavior were common. For example, CLS subjects reported an average of 3.5 (S.D. = 3.6, range = 0–22) behavioral events in the preceding 12 months. Self-reported acts of delinquent behavior were evenly distributed across the categories of assaults on persons, property and other antisocial acts. Parents/guardians reported that 33.8% of subjects had at least one court appearance for offense(s) in the last year and 19.5% were adjudicated delinquent. Contrary to the literature [25], measures of delinquent behavior selected for these analyses did not significantly differ by gender. Females and males were equally likely to self-report antisocial/delinquent behavioral events (including violence against others), have police contact, appear in court, be adjudicated delinquent and sentenced to institutional placement. Parental reports of behavioral problems at home, school or in the community were also unrelated to gender. Furthermore, no statistically significant PbB by gender interactions were observed. The Pearson correlation between self- and parent-reported total problems scores was .52 ($P \leq .0001$).

3.2. Self-report of delinquent behavior

As expected, measures of marijuana and alcohol use by the adolescent over their lifetime and/or during the preceding 12 months were correlated with self-reported delinquent behavior. For example, the correlation between lifetime frequency of marijuana consumption rated on a scale of 0 (no occasions) to 6 (40+ occasions) and total SRDB score was $r = .51$, $P \leq .0001$. This correlation was not an artifact of being arrested for the offense as only one subject reported being apprehended by legal authorities for use of the drug. However, 28% of CLS subjects admitted using marijuana at least once during their lifetime. Marijuana use was also moderately correlated with average childhood Pb exposure ($r = .27$, $P \leq .01$). Because the use of controlled substances itself constitutes illegal conduct, substance abuse was not entertained as a potential covariate or confounder in these analyses.

Developmental cofactors in the final multivariable models for SRDB included birth weight, parental IQ, average total HOME scores and SES. Following covariate-adjustment, all PbB variables were significantly associated with

Table 2

Covariate adjusted parameter estimates for the association between PbB concentrations ($\mu\text{g}/\text{dL}$) and total SRDB scores

PbB variable ($\mu\text{g}/\text{dL}$)	β^a	S.E.	Partial R^2	P
Prenatal	.192	.076	.049	.002
78 months	.193	.061	.055	.002
Average childhood	.101	.047	.031	.036

^a Parameter estimates for PbB are adjusted for birth weight, HOME scores, SES and parental IQ. Covariables independently associated with SRDB total at $P < .05$ included parental IQ and birth weight.

an increase in self-reported delinquent behavior. Table 2 presents the covariate-adjusted parameter estimates for prenatal and postnatal PbB variables.

To clarify the dose–effect relationships, PbB indices were transformed to categorical variables and least-square means were calculated from an analysis of covariance procedure. Fig. 2 presents the dose-effect relationship between PbB categories and adjusted total SRDB scores.

The magnitude of the dose–effect relationship across PbB categories is of practical interest. For example, on average subjects in the highest prenatal PbB category engaged in 2.3 more delinquent acts over the preceding 12 months than subjects in the lowest category; subjects in the medium and highest average childhood PbB category engaged in approximately 1.5 more delinquent acts than subjects in the lower categories; and subjects in the highest 78-month PbB category engaged in 4.5 more delinquent acts than subjects in the lowest category.

Table 3

Covariate adjusted parameter estimates for the association between PbB indices and total PRDB scores

PbB variable ($\mu\text{g}/\text{dL}$)	β^a	S.E.	Partial R^2	P
Prenatal	.194	.089	.045	.032
78 months	.131	.072	.022	.070
Average childhood	.090	.056	.012	.109

^a Parameter estimates for PbB are adjusted for birth weight, HOME scores, SES and parental IQ. Covariables independently associated with PRDB total scores at $P < .05$ included parental IQ and birth weight.

3.3. Parental report of predelinquent and delinquent behavior

As with the SRDB, marijuana consumption by the subject was associated with total behavioral problems reported by the parent ($r = .36$, $P \leq .001$), but was not entertained as a confounder in these analyses. Table 3 presents the covariate-adjusted parameter estimates for PbB indices and the PRDB total problems score. Only prenatal maternal PbB was significantly associated with adjusted total PRDB scores.

4. Discussion

This is the first prospective longitudinal study of child development to examine the relationship between toxicant exposure and measures of juvenile delinquency and anti-

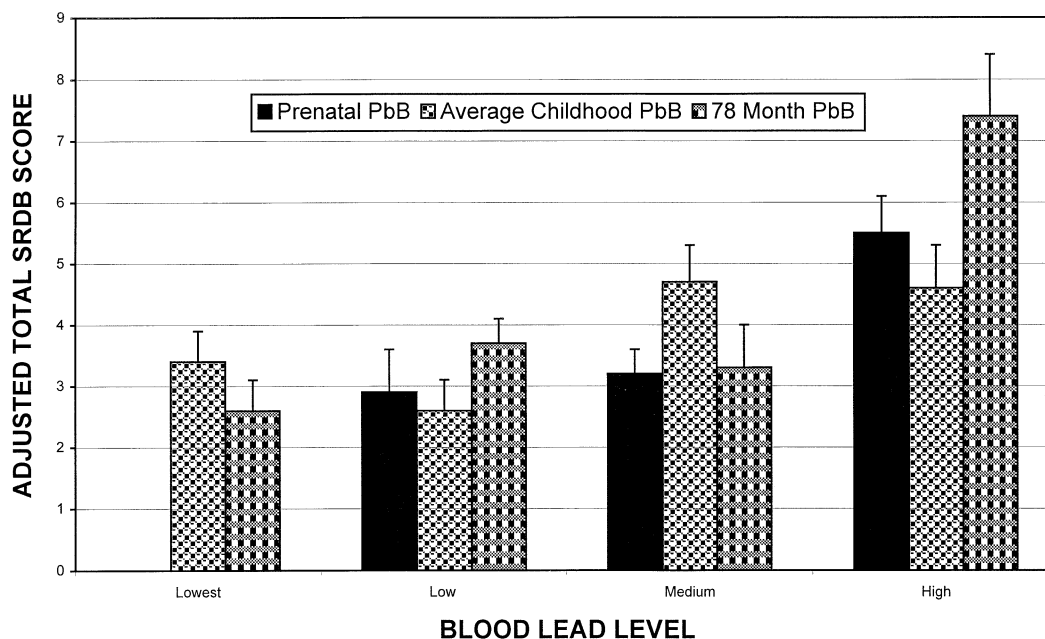


Fig. 2. Adjusted dose–effect relationships between prenatal and postnatal PbB categorical indices and adjusted total Self-Report of Delinquent Behavior scores. Prenatal PbB: low $\leq 5 \mu\text{g}/\text{dL}$, medium 6–10 $\mu\text{g}/\text{dL}$, high $>10 \mu\text{g}/\text{dL}$; average childhood PbB: lowest $\leq 5 \mu\text{g}/\text{dL}$, low 10–15 $\mu\text{g}/\text{dL}$, medium 16–20 $\mu\text{g}/\text{dL}$, high $>20 \mu\text{g}/\text{dL}$; PbB78: lowest $\leq 5 \mu\text{g}/\text{dL}$, low 6–10 $\mu\text{g}/\text{dL}$, medium 10–15 $\mu\text{g}/\text{dL}$, high $>15 \mu\text{g}/\text{dL}$. One-way bars represent standard errors. Model R^2 ranged from .11 to .16. SRDB means adjusted for birth weight, HOME scores, SES and parental IQ. As in the previous analyses, covariables independently associated with SRDB total scores at $P < .05$ were parental IQ and birth weight. When treated categorically, PbB variables were statistically significant (two-tailed test) for prenatal ($P \leq .02$) and 78-month ($P \leq .0007$) PbB, but not for average childhood PbB ($P \leq .09$).

social behavior. To the authors' knowledge, this is also the first study to report a significant relationship between low level prenatal Pb exposure and behavioral problems in adolescents. The association between early exposure to Pb and juvenile delinquency noted in previous cross-sectional and case-control studies are generally supported by these results. However, the magnitude of these associations was modest as reflected in partial R^2 values in the linear regression analyses that never exceeded 6%. Nevertheless, the strength of these associations is not atypical for the Pb literature in general [29].

Overall, we were not able to account for a great deal of the variance in antisocial behavior as indexed by self- or parental report. Residual and unmeasured confounding are always of concern in observational studies where all possible covariates cannot be assessed and those available are not measured with equal precision. However, we examined a wide range of developmental cofactors using reliable and valid instruments. The low model R^2 may also be due to the fact that the sample was sociodemographically homogeneous as well as the lack of measures that may have proven to be independently predictive such as, for example, maternal depression. The lack of gender differences (typically a strong predictor of antisocial and delinquent behavior), though intriguing, also resulted in models with low predictive power. The inclusion of neuropsychological variables examined in this sample such as measures of executive functioning, attention, and IQ may have amplified the predictive vigor of the models. However, controlling for neurocognitive variables known or suspected to be affected by developmental Pb exposure [8] would obviously be inappropriate. The possibility that early exposure to Pb may lead to a higher risk of antisocial behavior in later life through its effects on neuropsychological functions is interesting and will be the subject of future analyses of these data. Variables independently associated with measures of antisocial behavior included maternal intelligence and lower birth weight. The association with lower parental IQ was not unexpected and a few studies suggest that delinquency is related to medical complications at birth.[4] Nevertheless, while one could argue that the effect size for the toxicant of interest is small, Pb may be one of many important variables in the epigenesis of antisocial behavior by producing a bias in the developmental trajectories of exposed children.

Lead was more consistently associated with self as opposed to parental reports. This may be due, in part, to the fact that the SRDB is a much more comprehensive measure of antisocial and delinquent behaviors that has well documented validity and reliability in studies of this kind [14,27]. The use of drugs, particularly marijuana, was strongly associated with self- and parental reports of antisocial and delinquent behaviors in the home, school and community. However, these variables were not regarded as candidate confounders owing to the fact that their use constitutes an illegal activity and some animal model studies suggest that developmental Pb exposure may increase

anxiety and appetite for mood altering substances [19]. Indeed, postnatal PbB indices were modestly but significantly associated with both marijuana and alcohol consumption by CLS teens.

There is always a possibility of response bias in studies where the disease and its anticipated effects are known to the subject. Thus, one explanation for these results might be that CLS subjects, being aware of their previously "high" exposures to lead during the first 4–5 years of life, might be more likely to recall their antisocial acts and perhaps overstate the severity of their transgressions.

Such an accounting of these findings seems unlikely. First, when the study was conducted the link between juvenile delinquency and exposure to lead was not widely discussed in media our subjects were likely to be exposed to such as magazines, newspapers, radio and television. Second, the level of lead exposure considered developmentally neurotoxic in the early 1980s when these subjects were preschoolers was considerably higher than it is today. In our base sample of approximately 250 subjects, only 8.5% underwent a diagnostic chelation and just 5% were chelated therapeutically. The reader should bear in mind that the standard of care for lead-poisoned children is different today where effective outpatient therapies are available with orally administered chelating agents such as dimercaptosuccinic acid. Furthermore, the action level for environmental and medical intervention was substantially revised long after CLS subjects reached their peak PbB concentrations [28]. Third, the dose–response relationship for fetal exposure as indexed by maternal prenatal PbB cannot be explained by response bias as neither women nor their children were aware of these values which were all below the level of obstetrical concern for pregnant women at the time recruitment was underway (1979–1984). Fourth, as is typical in this kind of research a dose–response methodology was utilized. Spurious findings related to response bias would require that the subjects have a fairly sophisticated understanding of their exposure. In other words, it would require an implicit or explicit appreciation for their *degree* of exposure, as related to other subjects, and a corresponding *degree* of bias. This seems highly unlikely. Finally, it is also important to note that the assessment of juvenile delinquency and antisocial behavior in the CLS occurred in a single, day long session that included a comprehensive assessment of neuropsychological status, personality and other outcomes. It would not be apparent to the subject that we were particularly focused on self- or parent-reported antisocial behavior as a core outcome. In any event, it seems unlikely that by late adolescence, CLS subjects were conscious of their PbB levels when they were infants and preschoolers. Nevertheless, however, improbable the possibility of response bias in this and any other study of its kind cannot be completely ruled out.

Although the removal of Pb from paint and most motor fuels has resulted in dramatic reductions in children's PbB levels [23], many children in the United States still have

unacceptably high concentrations of Pb in their bodies. Furthermore, cases of symptomatic poisoning, although rare, continue to occur [9]. Only a small fraction of studies have specifically examined the association between early exposure to Pb and delinquency. This is the first prospective longitudinal study with comprehensive documentation of exposure to Pb and other key developmental cofactors to do so. The neurobiological and neuropsychological pathways that may mediate the demonstrated relationship between early Pb exposure and delinquency have yet to be worked-out. Although the determinants of delinquency and antisocial behavior are complex, an environmental neurotoxicant that is potentially avoidable with adequate primary prevention may play a role in its etiology among urban, inner-city children.

The weight of the evidence supports a relationship between developmental Pb exposure and modest deficits in IQ and academic attainment [3]. In this investigation, early exposure to Pb was associated with an increased frequency of self- and parent-reported delinquent acts—a relationship that could not be explained on the basis of various medical, social and family risk factors. It appears that the neurodevelopmental effects of this avoidable environmental disease of childhood may not be limited to declines in IQ or academic abilities.

A prominent theorist in the area of delinquency research has pointed out that temporary and situational antisocial behavior is quite common in adolescent populations [17]. However, persistent, stable antisocial behavior is found among a relatively small number of teens who seem destined to pursue a criminal career [18]. Further research on this cohort may help to determine if the delinquent, antisocial behavioral course on which some have begun is adolescence-limited or life-course-persistent. We are continuing to follow this cohort into their early twenties with various assessments of antisocial behavior to determine if early exposure to Pb is associated with adult criminality.

Acknowledgments

This research was supported by a grant from the National Institute of Environmental Health Sciences, National Institutes of Health (RO1ES08158). This study has been reviewed and approved by the Institutional Review Board of the University of Cincinnati College of Medicine.

References

- [1] P.A. Baghurst, A.J. McMichael, N.R. Wigg, G.V. Vimpani, E.V. Robertson, R.J. Roberts, S.L. Tong, Environmental exposure to lead and children's intelligence at the age of seven years, *N. Engl. J. Med.* 327 (1992) 1279–1284.
- [2] J.L. Ballard, K.K. Novak, M.A. Driver, A simplified score for assessment of fetal maturation in newly born infants, *J. Pediatr.* 95 (1979) 769–774.
- [3] D. Bellinger, K.N. Dietrich, Low-level lead exposure and cognitive function in children, *Pediatr. Ann.* 23 (1994) 600–605.
- [4] P.A. Brennan, S.A. Mednick, Medical histories of antisocial individuals, in: D.M. Stoff, J. Breiling, J.D. Maser (Eds.), *Handbook of Antisocial Behavior*, Wiley, New York, 1997, pp. 269–279.
- [5] R.K. Byers, E.E. Lord, Late effects of lead poisoning on mental development, *Am. J. Dis. Child.* 66 (1943) 471–494.
- [6] B. Caldwell, R. Bradley, *Home Observation for Measurement of the Environment*, University of Arkansas, Little Rock, AR, 1979 (unpublished manual).
- [7] D.W. Denno, *Biology and Violence*, Cambridge Univ. Press, New York, NY, 1990.
- [8] K.N. Dietrich, Environmental neurotoxicants and psychological development, in: K.O. Yeates, M.D. Ris, H.G. Taylor (Eds.), *Pediatric Neuropsychology: Research, Theory, and Practice*, Guilford Press, New York, NY, 1999, pp. 206–234.
- [9] K.N. Dietrich, O.G. Berger, A. Bhattacharya, Symptomatic lead poisoning in infancy: A prospective case analysis, *J. Pediatr.* 137 (2000) 568–571.
- [10] K.N. Dietrich, O.G. Berger, P.A. Succop, P.B. Hammond, R.L. Bornschein, The developmental consequences of prenatal and postnatal lead exposure: Intellectual attainment in the Cincinnati lead study cohort following school entry, *Neurotoxicol. Teratol.* 15 (1993) 37–44.
- [11] K.N. Dietrich, T.D. Hill-Hornsby, *Parental Report of Pre-Delinquent and Delinquent Behaviors*, University of Cincinnati College of Medicine, Cincinnati, OH, 1995 (unpublished manual).
- [12] K.N. Dietrich, K.M. Kraft, R.L. Bornschein, P.B.O.G. Berger, P.A. Succop, M. Bier, Low-level fetal lead exposure effect on neurobehavioral development in early infancy, *Pediatrics* 80 (1987) 721–730.
- [13] K.N. Dietrich, P.A. Succop, O.G. Berger, P.B. Hammond, R.L. Bornschein, Lead exposure and the cognitive development of urban preschool children: The Cincinnati lead study cohort at age 4 years, *Neurotoxicol. Teratol.* 13 (1991) 203–211.
- [14] D.S. Elliott, D. Huizinga, S.S. Ageton, *Explaining Delinquency and Drug Use*, Sage, Beverly Hill, CA, 1995.
- [15] A.B. Hollingshead, *Four-Factor Index of Social Position*, Yale University, New Haven, CT, 1985 (unpublished manual).
- [16] B. Littman, A.H. Parmelee, Medical correlates of infant development, *Pediatrics* 61 (1978) 470–474.
- [17] T.E. Moffitt, Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy, *Psychol. Rev.* 100 (1993) 674–701.
- [18] T.E. Moffitt, Adolescence-limited and life-course persistent offending: A complementary pair of developmental theories, in: T.P. Thornberry (Ed.), *Developmental Theories of Crime and Delinquency*, Transaction Publishers, London, 1997, pp. 11–54.
- [19] J.R. Nation, D.M. Baker, B. Taylor, D.E. Clark, Dietary lead increases ethanol consumption in the rat, *Behav. Neurosci.* 100 (1986) 525–530.
- [20] H.L. NeedLeman, C. Mcfarland, R. Ness, M. Tobin, J. Greenhouse, Bone lead levels in adjudicated delinquents: A case-control study, *Pediatr. Res.* 47 (2000) 155A.
- [21] H.L. NeedLeman, J.A. Riess, M.J. Tobin, G.E. Biesecker, J.B. Greenhouse, Bone lead levels and delinquent behavior, *J. Am. Med. Assoc.* 275 (1996) 363–369.
- [22] R.B. Noll, R.A. Zucker, H.E. Fitzgerald, W.J. Curtis, Cognitive and motoric functioning of sons of alcoholic fathers: The early childhood years, *Dev. Psychol.* 28 (1992) 665–676.
- [23] J.L. Pirkle, R.B. Kaufmann, D.J. Brody, T. Hickman, E.W. Gunter, Exposure of the US population to lead, *Environ. Health Perspect.* 106 (1998) 745–750.
- [24] S.M. Roda, R.D. Greenland, R.L. Bornschein, P.B. Hammond, Modification of an anodic stripping voltammetry procedure for improved accuracy of blood lead analysis, *Clin. Chem.* 34 (1988) 563–567.
- [25] M. Rutter, Antisocial behavior: Developmental psychopathology perspectives, in: D.M. Stoff, J. Breiling, J.D. Maser (Eds.), *Handbook of Antisocial Behavior*, Wiley, New York, NY, 1997, pp. 115–124.
- [26] A.B. Silverstein, Two-and four-subtest short forms of the Wechsler

- adult intelligence scale-revised, *J. Consult. Clin. Psychol.* 50 (1982) 415–418.
- [27] T.P. Thornberry, M.D. Krohn, The self-report method of measuring delinquency and crime, in: D. Duffee (Ed.), *Measurement and Analysis of Crime and Justice: Criminal Justice 2000*, vol. 4, US Department of Justice, Office of Justice Programs, Washington, DC, 2000, pp. 33–83.
- [28] United States Centers for Disease Control, Preventing Lead Poisoning in Young Children—A Statement by the United States Centers for Disease Control—October 1991, United States Department of Health and Human Services, Public Health Service, Centers for Disease Control, Atlanta, GA, 1991.
- [29] World Health Organization, Environmental health criteria 165: Inorganic lead, W.H.O. International Programme on Chemical Safety, Geneva, Switzerland, 1995.
- [30] R.A. Zucker, H.E. Fitzgerald, R.B. Noll, Drinking and Drug History Questionnaire, Michigan State University, Department of Psychology, Lansing, MI, 1990 (unpublished manual).