

Bone lead levels in adjudicated delinquents A case control study

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Abstract

Background: Lead exposure shares many risk factors with delinquent behavior, and bone lead levels are related to self-reports of delinquent acts. No data exist as to whether lead exposure is higher in arrested delinquents. The goal of this study is to evaluate the association between lead exposure, as reflected in bone lead levels, and adjudicated delinquency. **Methods:** This is a case-control study of 194 youths aged 12–18, arrested and adjudicated as delinquent by the Juvenile Court of Allegheny County, PA and 146 nondelinquent controls from high schools in the city of Pittsburgh. Bone lead was measured by K-line X-ray fluorescence (XRF) spectroscopy of tibia. Logistic regression was used to model the association between delinquent status and bone lead concentration. Covariates entered into the model were race, parent education and occupation, presence of two parental figures in the home, number of children in the home and neighborhood crime rate. Separate regression analyses were also conducted after stratification on race. **Results:** Cases had significantly higher mean concentrations of lead in their bones than controls (11.0 ± 32.7 vs. 1.5 ± 32.1 ppm). This was true for both Whites and African Americans. The unadjusted odds ratio for a lead level ≥ 25 vs. < 25 ppm was 1.9 (95% CL: 1.1–3.2). After adjustment for covariates and interactions and removal of noninfluential covariates, adjudicated delinquents were four times more likely to have bone lead concentrations > 25 ppm than controls (OR = 4.0, 95% CL: 1.4–11.1). **Conclusion:** Elevated body lead burdens, measured by bone lead concentrations, are associated with elevated risk for adjudicated delinquency.

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1. Introduction

Most studies of childhood lead exposure have focused on cognitive function, using IQ tests as the outcome of interest. There are a number of reasons to believe that antisocial behavior may be a more sensitive and consequential outcome. This is not a new notion. Pediatricians who treat lead poisoning have frequently been told by parents that, after recovery, their offspring became oppositional, aggressive or

violent. In 1943, Randolph Byers was stimulated to design the first follow-up study of lead-poisoned children. The precipitant was his discovery that children referred to him for evaluation of violent behavior were former patients who had been treated for lead poisoning. Of the 20 subjects he studied, 19 had severe behavior problems or were learning disordered [5].

Published controlled studies of the lead-delinquency hypothesis are limited to three. Denno [9] found that the strongest predictor of arrest in Philadelphia youths enrolled in the Collaborative Perinatal Project was a history of lead poisoning. In 1996, we studied a cohort of 301 boys in the Pittsburgh School System. Bone lead levels at 12 years of age were significantly related to parents' and teachers' Child Behavior Checklist ratings of aggression, attention and delin-

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quency. The subjects' self-reports of delinquent acts were also positively associated with bone lead concentrations [19]. Dietrich et al. [10] have recently reported that prenatal exposure to lead was associated with increased parent reports of antisocial behavior and postnatal exposure with increased reports of delinquent acts by the subjects themselves.

To more directly examine the relationship between lead exposure and criminality, we conducted a case-control study of bone lead levels in 194 male youths arrested and adjudicated as delinquent by the court.

2. Methods

2.1. Subjects

Cases were youths who resided in Allegheny County, PA, who were arrested and adjudicated by the Juvenile Court as delinquent. Controls were nondelinquent youths attending high schools in Pittsburgh. Two programs of the Allegheny County Juvenile Court serve serious delinquents: the Community Intensive Supervision Program (CISP) and the Allegheny Academy. Both programs function either as alternatives to incarceration of serious offenders or provide aftercare following incarceration. Three CISP centers located in Allegheny County participated in our study. Thirty-nine percent of the CISP enrollees were serious offenders discharged from incarceration to the program. Other enrollees were classified by the court program as drug dealers, assaulters, probation violators, firearm possessors, auto thieves or were guilty of robbery or other crimes.

Those offenders from neighborhoods in Allegheny County that did not have CISP homes were sent to the Allegheny Academy, a facility for delinquents in a neighboring township. The distribution of offenses in the Academy and CISP was similar. At the end of their public school session, students were picked up at home and taken by bus to the Academy or to the CISP center. At about 9 p.m., students were returned to their homes and, at 11 p.m., a telephone bed check made. Almost all enrollees in the CISP program were African American; in the Academy, the proportion of White youths was approximately 35%.

During the period of our study, 547 males were enrolled in the two programs. From the CISP program, we recruited 100 delinquent males and from the Academy 95 males. Of those we did not recruit, 166 were released or transferred from the programs before we could make contact; 72 candidates declined to participate and we were unable to reach the parents of 108. Two were excluded for medical conditions and four had incomplete data collection.

We initially designed our study to match controls with cases within each high school classroom. When we began the study, school administration policy had changed and we were barred from direct contact with potential controls. High school principals became the contact with potential subjects. Those high school principals who chose to coop-

erate sent letters describing the study to their students' families. Each letter contained a reply addressed to our laboratory. We contacted parents who expressed interest and made appointments for them and their offspring. We recruited from 6 of 11 Pittsburgh high schools. Of the five high schools that were not included, one refused, three were magnet schools offering special educational programs and one served a distinctly higher socioeconomic population. Controls were screened by telephone to eliminate those with arrest histories, seizures or taking neuroactive medications. From 283 respondents, 200 male controls were recruited; 19 refused, 25 were unable to be reached and 39 were excluded by the telephone screen.

Subjects and controls were given the Self-Report of Delinquency (SRD) [14], a 36-item inventory of antisocial acts committed over the previous 6 months, scaled from 0 to 4 depending on frequency of acts committed. Because many delinquents are not arrested and therefore are not known to the juvenile justice system, we attempted to minimize unidentified delinquents in our control group by excluding from analysis controls with either a Juvenile Court record or an SRD score ≥ 20 , the 90th percentile. Of the 200 controls we recruited, 50 were excluded, 34 because of court records, 1 because of a diagnosis of autism, 13 because of high SRD scores and two because of a history of lead poisoning. To evaluate potential bias produced by excluding the latter two groups, we reintroduced them into the model and compared unadjusted odds ratios with and without them. Because a number of cases attended different high schools than controls, we evaluated the potential bias by conducting an additional logistic regression including only cases and controls who attended the same high schools. We then compared odds ratios for reduced models from the full sample with that from subjects attending the same schools.

Data for cases and controls are given in Tables 1 and 2. One case and four controls were not included in the stratified analysis because their race was indeterminate. White high school students responded to our control recruitment letter at a higher rate than African Americans, and our case-to-control ratio was different across racial groups (Whites: 36 cases/95 controls, African Americans: 158 cases/51 controls).

Subjects were studied between April 1996 and August 1998. Informed consent was obtained at the time of study, at which time they were paid US\$30.00 and their parents US\$20.00. This study was reviewed and approved by the University of Pittsburgh Institutional Review Board.

2.2. Bone lead measurements

Tibial bone lead concentrations were estimated with *in vivo* X-ray fluorescence (XRF) employing 88.03 keV photons from a ^{109}Cd source to induce characteristic lead K X-rays, measured with a backscatter counting geometry [31]. Bone lead concentrations were estimated from the lead K $\beta_{1,3}$ X-rays (84.94 and 84.45 keV). A 30-min tibia exposure

Table 1
Descriptive variables of cases and controls stratified by race

	<i>n</i>	African American cases ^a	<i>n</i>	African American controls ^a	<i>P</i> value *	<i>n</i>	White cases ^a	<i>n</i>	White controls ^a	<i>P</i> value *
Age (mean ± S.D.)	158	15.8 ± 1.4	51	15.5 ± 1.1	.0662	36	15.7 ± 1.3	95	15.8 ± 1.1	.6829
Grade (mean ± S.D.)	158	9.5 ± 1.5	51	9.8 ± 0.9	.0446	36	9.3 ± 1.2	95	10.3 ± 1.1	<.0001
Parent education (mean ± S.D.)	109	12.7 ± 2.0	51	13.0 ± 1.8	.3621	34	12.6 ± 2.2	94	12.9 ± 1.6	.4609
Parent occupation code (mean ± S.D.)	112	2.4 ± 2.6	51	2.5 ± 2.5	.7387	36	2.4 ± 2.95	94	3.4 ± 2.8	.1038
Two parental figures in home	112	–	51	–	–	36	–	94	–	–
Yes (%)	38	33.9%	28	54.9%	.0114	17	47.2%	75	79.8%	.0003
Two biological parents (% yes)	15	13.4%	11	21.6%	.1862	7	19.4%	61	64.9%	<.0001
Spouse education (mean ± S.D.)	36	12.1 ± 2.2	28	12.7 ± 2.2	.3144	17	12.4 ± 1.5	75	12.7 ± 2.1	.5704
Spouse occupation (mean ± S.D.)	38	2.3 ± 2.3	27	3.4 ± 3.1	.0869	17	2.8 ± 2.4	75	3.9 ± 2.3	.0716
No. of children living in home (mean ± S.D.)	112	2.0 ± 1.3	51	2.4 ± 1.4	.0650	36	2.1 ± 1.3	95	1.9 ± 1.3	.4294
Neighborhood crime rate (mean ± S.D.)	155	83.8 ± 41.1	50	124.5 ± 312.8	.3634	36	42.2 ± 35.3	94	53.1 ± 26.8	.1005
Self-reported delinquency score (mean ± S.D.)	158	23.9 ± 16.5	51	6.5 ± 5.0	<.0001	36	31.5 ± 21.2	95	4.5 ± 4.7	<.0001

^a One case and four controls not included because not identified as Black/biracial or White.

* *P* values based on *t* test for means or χ^2 for frequencies.

resulted in an effective dose of 17/19 nSv, corresponding to 0.001% of the average natural environmental radiation dose (3 mSv). The XRF protocol was approved by the University of Pittsburgh Radiation Safety Committee.

Lead K $\beta_{1,3}$ X-rays and coherent scatter peak areas were obtained with a nonlinear minimization program. Spectral data were modeled as Gaussian peaks superimposed on monotonic background functions. Instrument quality control was achieved by daily monitoring lead K X-ray peak widths and locations using a lead plug check standard.

A set of bone lead phantoms (lead-doped plaster of Paris) furnished by the National Institute of Standards and Technology (NIST) was used to recalibrate our instrument and validate our spectrum analyses. A plot of lead concentration estimates obtained with our analytical protocol against NIST-reported lead values shows close agreement (Fig. 1).

Conservative criteria were used to set our minimum detectable concentration (MDC) values [1]. Instrumental MDC was estimated as 4.65 θ , the standard deviation of replicate low-lead “blank” NIST phantom measurements. For 16 replicates, an MDC of 14.7 μ g Pb/g plaster, corresponding to an MDC of 21.5 μ g Pb/g bone mineral, was obtained. When the lead K X-ray signal is small, the masking effect of the Poisson-distributed background fre-

quently produces negative bone lead concentrations. These were expected and observed. To deal with this in our analyses, we dichotomized our lead estimates at 25 ppm, the 80th percentile of the distribution, and just above the alternate MDC estimates described above. Dichotomizing at 20 and 30 ppm resulted in similar splits.

2.3. Data analysis

After data checking and verification, univariate distributions of variables were calculated (Table 1). To deal with the

Table 2
Bone lead concentrations for cases and controls stratified by race

	Cases		Controls		<i>P</i> value
	<i>n</i>	Mean ± S.D.	<i>n</i>	Mean ± S.D.	
All subjects	195	11.0 ± 32.7	150	1.5 ± 32.1	.007
African American ^a	158	9.0 ± 33.6	51	– 1.4 ± 31.9	.05
White ^a	36	20.0 ± 27.5	95	3.5 ± 32.6	.008

^a One case and 4 controls not included because not identified as black/biracial or white.

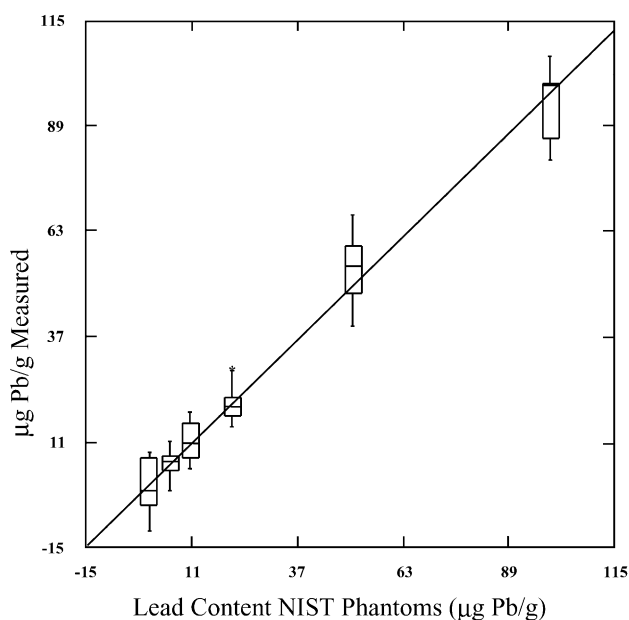


Fig. 1. XRF spectroscopy performance using NIST lead phantoms. For each of six lead-doped plaster of Paris phantoms, actual micrograms of lead per gram of plaster are presented on the abscissa. Box plots showing medians, quartiles and range of replicate ($N \geq 16$) XRF measurements are displayed on the ordinate. The diagonal line shows the Pearson correlation ($r = .98$).

race-related differential recruitment of cases and controls, bone lead levels were compared after stratification by race (Table 2).

Cognizant that our controls were a self-selected group within the underlying population from which our cases came, we adjusted our analysis for both individual and community potential confounders. The association of bone lead, dichotomized at 25 ppm, with delinquency was modeled by logistic regression. Covariates were selected on the basis of *a priori* information on risk factors; all but one were dichotomized. Included were race, parental education (number of years), parental occupation (Hollingshead occupational scale) [12], presence of two parental figures in the home, number of children in the home and neighborhood crime rate. Because, in many cases, the distribution of covariates was sparse or uneven, we elected to categorize most covariates using *a priori* rules creating categories that seemed logical. Two interaction terms were created on the basis of exploratory analyses employing variates in pairs. They were: lead \times race and lead \times single parent.

To evaluate the influence of neighborhood factors, from the 1992 Pittsburgh Police Statistical Report and the Pennsylvania Uniform Crime Report, we obtained the number of serious crimes committed in each Pittsburgh neighborhood. From census data, we calculated crimes per 1000 residents/year for each neighborhood. This neighborhood crime rate was stratified at the median (63 cases/1000) and assigned to each subject, according to neighborhood residence at time of testing.

The initial logistic model evaluated all subjects. An unadjusted odds ratio was computed and all covariates then entered into the model. Two interaction terms were then created and the model reduced, eliminating noninfluential covariates. To deal with the imbalance in proportion of controls and cases between Whites and African Americans, and to examine the effects of lead and other covariates while holding race constant, we stratified the subjects by race and analyzed two separate logistic models within racial strata. Table 3 presents the odds ratios for

lead, covariates and interaction terms for all models. Table 4 presents the cross-tabulation of subjects by three covariates: race, presence of two parental figures and parental occupation.

3. Results

Table 2 shows the mean \pm S.D. bone lead concentrations for all cases and controls and stratified by race. Delinquents had significantly higher bone lead levels than controls (11.0 ± 32.7 vs. 1.5 ± 32.1 ppm; $P=.007$). Limiting subjects to those attending the same high schools and comparing bone lead levels, we found almost identical bone lead levels (11.1 ± 27.6 vs. 1.5 ± 32.1 ppm). White delinquents had higher bone lead levels than African American delinquents (20 ± 27.5 vs. 9.0 ± 33.6 ppm). Both White and African American cases had achieved lower grade levels than controls at the time studied.

For all subjects, the unadjusted odds ratio was 1.9 (95% CL: 1.1–3.2) (Table 3). Adding nondelinquent controls with high SRD scores and the three lead-poisoned subjects to the model had minimal effect on the odds ratio (OR = 1.96, 95% CL: 1.1–3.4).

After covariate adjustment, the odds ratio for bone lead was 3.7 (95% CL: 1.3–10.5). The odds ratio (all subjects) in the reduced model was 4.0 (95% CL: 1.4–11.1). The odds ratio evaluating only subjects attending the same high schools was 3.2 (95% CL: 0.4–25.3).

Race and absence of two parental figures were influential covariates. After stratification, White subjects had an unadjusted odds ratio of 3.4 (95% CL: 1.4–8.1). Covariate adjustment increased the odds ratio to 3.8 (95% CL: 1.1–13.3). The odds ratio for the reduced model was 3.6 (95% CL: 1.1–12.3). African American subjects showed a similar pattern. The unadjusted odds ratio in this group of 1.5 increased to 2.2 in the full model and 2.6 in the reduced model. While African American race and single parenthood were themselves risk factors for delinquency,

Table 3
Odds ratios: full and reduced logistic models for all subjects, Whites and African Americans

Variable	All subjects		Whites		African Americans	
	Unadjusted OR = 1.9 (1.1–3.2)		Unadjusted OR = 3.4 (1.4–8.1)		Unadjusted OR = 1.5 (0.7–3.6)	
	Full model	Reduced model	Full model	Reduced model	Full model	Reduced model
Bone lead	3.7 (1.3–10.5)	4.0 (1.4–11.1)	3.8 (1.1–13.3)	3.6 (1.1–12.3)	2.2 (0.5–10.0)	2.6 (0.6–11.0)
Race	7.6 (3.6–15.8)	5.6 (3.0–10.4)	–	–	–	–
Lead \times race	0.6 (0.2–2.4)	0.6 (0.2–2.0)	–	–	–	–
Single parent	3.2 (1.7–6.1)	3.4 (1.9–6.3)	5.1 (1.6–15.6)	4.5 (1.5–13.5)	3.0 (1.3–6.8)	3.0 (1.4–6.5)
Lead \times single parent	0.5 (0.1–1.7)	0.5 (0.1–1.7)	0.2 (0.0–1.8)	0.4 (0.1–2.6)	0.5 (0.1–3.3)	0.5 (0.1–2.9)
Parent education	1.0 (0.8–1.1)	–	1.1 (0.8–1.4)	–	0.9 (0.7–1.1)	–
Parent occupation	1.3 (0.7–2.4)	–	2.7 (1.0–7.2)	2.4 (1.0–5.9)	0.9 (0.4–1.9)	–
No. of children	0.7 (0.4–1.4)	–	2.3 (0.8–6.1)	1.9 (0.7–5.0)	0.4 (0.2–0.9)	0.5 (0.2–1.0)
Neighborhood crime rate	0.5 (0.3–1.0)	–	0.3 (0.1–1.0)	–	0.6 (0.3–1.4)	–

Table 4
The influence of social risk factors on the number of low lead cases and odds ratios

Strata	Bone lead (ppm)	Cases	Controls	Odds Ratios
<i>Race</i>				
African American subjects	≥25	35	8	1.5 (0.7–3.6)
	<25	123	43	
White subjects	≥25	14	15	3.4 (1.4–8.1)
	<25	22	80	
<i>Two parental figures</i>				
No	≥25	25	10	1.3 (0.6–3.0)
	<25	68	35	
Yes	≥25	15	13	2.6 (1.1–5.9)
	<25	41	91	
<i>Parent occupation</i>				
Manual/mental/unemployed	≥25	25	11	1.7 (0.8–3.7)
	<25	67	49	
Skilled/clerical/professional	≥25	15	12	2.3 (1.0–5.4)
	<25	42	77	

White subjects and children with two parents had higher odds ratios for lead.

4. Discussion

These findings of higher concentrations of lead in delinquents and of an association of bone lead levels with delinquency after covariate adjustment support those we reported in 1996 on the relationship between bone lead and antisocial behavior [19], and are consistent with both Denno's [9] and Dietrich et al.'s findings [10]. The effect is substantial. With all subjects in the model, bone lead level was the second strongest risk factor, exceeded only by race. In the stratified models, with race eliminated, bone lead was exceeded as a risk only by single parent status.

The case for lead as a causal factor in antisocial behavior is also given credence by two recent ecological studies. Stretesky and Lynch reported positive correlations between homicide rates (National Center for Health Statistics) and air lead levels for 3111 US counties. After adjustment for 15 confounding variables, a four-fold increase in homicides in the highest lead counties compared to the lowest lead counties was found [28]. Nevin [21] reported a statistically significant association between gasoline lead sales and violent crime after adjustment for unemployment and percent of population in the high crime age group.

Selection of controls is always a critical issue in case-control studies and presents a possible challenge to validity in this study. Although we attempted to recruit controls similar in background to our delinquent sample, volunteer bias was encountered. African Americans from our potential control pool responded at a much lower rate than Whites to our recruitment letters. Within racial strata, the differences between cases and controls were few: A higher proportion

of controls were White; controls also tended to be in a higher school grade ($P=.04$, African Americans; $P<.0001$, Whites), more often have two parental figures in the home and have parents with higher Hollingshead employment status. Adjustment for these factors in the logistic model increased the effect size for lead. Control of race by stratification demonstrated a lead effect within both Black and White strata, and disclosed an increased effect size for lead in White subjects. Limiting our sample to only those subjects attending the same high schools ($n=43$ cases, 145 controls), the odds ratio was reduced by 25% from 4 to 3.1 and the confidence limits widened, crossing 1. Although we cannot exclude the possible role of unmeasured confounders, the relative stability of the effect size after adjustment for important confounders is reassuring.

Most studies of the causes of criminal behavior have focused on social risk factors; less attention has been given to the influence of brain dysfunction [24,25]. Of seven reviews on the influence of brain lesions or other central nervous system (CNS) disorders on violent offending published between 1974 and 1989, however, six concluded that there was a positive association [17]. Some neurotoxins, notably alcohol, amphetamines and other drugs of abuse, are acknowledged as facilitators of criminal behavior. Other neurotoxins, including lead, have largely been ignored.

Abnormal CNS function is frequently found in the most serious delinquents. Those delinquents who display anti-social behavior early in life persist in their behavior, while those who start later tend to give it up in their late teens. Those offenders in the group of life-persistent delinquents have a higher rate of impaired neuropsychological function when compared to those who begin later [18]. This early onset/life persistent group, approximately 6% of the population, is responsible for 50% of the crime.

PET scans of brain function have demonstrated decreased glucose metabolism in the prefrontal lobe of murderers compared to controls [23]. Impulsivity, a critical precursor of antisocial behavior, is mediated in the prefrontal lobes of the cerebral cortex and damage in this area is associated with behavioral disturbances. Lesions in the prefrontal cortex are also associated with many characteristic behaviors of ADHD [6]. ADHD, if accompanied by conduct disorder or defiance, is a strong risk factor for antisocial behavior and later arrests [2,22,26]. In numerous studies from the United States and around the world, lead-exposed children have been reported to display impulsivity and attentional problems [4,11,26,27,30,33,34]. This observation confirms that found by Byers in his follow-up study of 20 lead-poisoned children published in 1943:

Behavior difficulties were common throughout the series. Much of this behavior could be classified as "forced reaction to stimuli in the environment" described by Strauss and Werner as evidence of cortical damage. It was usually described as unreliable impulsive behavior, cruel impulsive behavior, short attention span and the like. [5]

The specific biological mechanisms underlying lead's effect on aggression and impulsivity are not known. Lead acts at a large number of CNS sites, some of which are involved in impulse control. Lead interferes with synaptogenesis [3], diminishes the inhibition of brain phosphokinase C [15], decreases norepinephrine-induced inhibition [29] and lowers brain levels of serotonin or 5-HIAA [13,32]. Lead exposure is associated with increased levels of D-aminolevulinic acid, which may antagonize GABA inhibition [16]. Lead also enhances both D₁ and D₂ dopamine sensitivity, and alters NMDA receptor sensitivity [7,8].

In addition to its direct action on the brain and impulse control, lead exposure can increase risk for delinquency through a separate, indirect route: impaired cognitive function and classroom performance. Early lead exposure has been shown to be associated with a seven-fold increase in the rate of high school failure and a six-fold increase in reading disabilities [20]. Students who do poorly in school, read poorly and fail out are more likely to become law-breakers. In the sample reported here, cases had lower grade achievement than controls. This could be due to grade retention or to time spent in incarceration.

Adjustment for covariates in the logistic models in this study increased the odds ratio for bone lead. While African American race, absence of two parental figures and low status parental occupation are independent risk factors for criminality, the effect size for lead was larger in White subjects, in families with two parental figures and with parents in higher status employment. This apparent paradox occurs because nonlead social factors that raise the risk of delinquency increase the number of subjects in the low lead/delinquent case group. This becomes apparent in examination of Table 4. In each higher risk stratum, the proportion of delinquents in the low lead cell is higher and the resultant lead odds ratio lower.

Raine reported a similar finding in his PET scan study of murderers. Those subjects whose rearing histories were rated more favorably had greater impairment in prefrontal glucose metabolism than those with severe social deficits. He suggested that among violent offenders without deprived home backgrounds, the "social push" to violence is minimized and "consequently brain abnormalities provide a relatively stronger predisposition to violence" [23].

A limitation of this study is the smaller number of African American controls, resulting in wider confidence limits in this stratum. Given the sizable difference in bone lead levels, it seems unlikely that this limited control sample biased the study towards a false positive conclusion.

These data are the first reported bearing on the association between lead at asymptomatic doses and adjudicated delinquency. If other studies find a similar association between lead and delinquency, a sizable segment of this important societal problem of delinquency and violence would become accessible to primary prevention. Future epidemiological studies of the causes of criminality should include lead and other neurotoxic agents as risk factors.

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