Moderately Elevated Blood Lead Levels: Effects on Neuropsychologic Functioning in Children

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ABSTRACT. Investigations of moderately elevated lead levels and children's cognitive functioning have yielded conflicting results, although studies showing no effects used measures of limited sensitivity and breadth. In this study, a comprehensive neuropsychologic battery was used to determine whether deficits would be revealed. An experimental group of 15 children with a past history of moderately elevated lead levels, but subsequently without increased lead levels for at least a year, were comapred with a control group matched by residential area, socioeconomic status, parental IQ, age, and gender. The experimental groups' performance was lower on the battery overall and on measures of motor skill, memory, language, advanced spatial functions, and concentration. The results suggest that exposure to moderately elevated lead levels exerts significant and nontransient effects on cognitive functions. *Pediatrics* 1987;80:623–629; *lead, neuropsychology, toxic agent.*

William Blake once said, "One does not know what is enough until one knows what is too much." Although not referring to lead levels, Blake's statement describes a crucial problem. Undue lead absorption is matched by few other pediatric health problems and may place large numbers of children at risk, but we do not know much lead absorption is too much.[1,2]

When lead produces clinical signs of toxicity, medical examination can detect and delineate adverse effects. It is unknown, however, whether lead causes permanent damage when toxic signs are absent, and overt signs of toxicity are rare with body burdens of less than 60 μg/100 mL.[1] Metabolic abnormalities have been found at much lower levels, but they are of uncertain clinical significance and do not necessarily aid in patient management.[3,4]

Physical injury or toxic agents can impact negatively on behavior or cognitive functioning, even in the absence of detectable physical anomalies.[5] Thus, with agents such as lead, direct assessment of behavior or cognitive functioning may help to determine impact. There is evidence that lead levels greater than 60 μg/100 mL are associated with a reduction in overall intelligence and an increase in behavioral or attentional difficulties.[6] However, results have varied in studies evaluating lower elevations. Significant but small magnitude differences in cognitive functioning have been found by some researchers at or less than the 30- to 60-μg band, whereas other researchers have obtained no differences or differences that disappear when sociocultural factors are controlled.[7–22]

Methodologic problems limit the interpretive value of many studies on the cognitive effects of lead levels at or less than the 30- to 60-μg range and may explain the contrasting outcomes. These problems include failure to obtain control groups or to match experimental and control groups on critical variables, such as socioeconomic status.[6,12,20,23,24] Cognitive measures have varied widely in different studies and have often been restricted to global measures of intelligence or to instruments of limited sensitivity or breadth.

Neuropsychologic assessment methods are more sensitive to cognitive deficit than standard intelligence tests alone or limited cognitive batteries.[25] Studies on increased lead levels in which less sensitive techniques were used and in which nonsignificant findings were obtained may thus represent false-negative errors. Although some investigators have used sections of neuropsychologic test batteries, to our knowledge, none have administered a

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comprehensive set of neuropsychologic tests, such as the Halstead-Reitan battery.[26]

The current study was conducted to determine whether and what cognitive deficits would be found when a comprehensive neuropsychologic battery was administered to children with moderately increased blood lead levels. The primary hypothesis addressed was: children with a past history of moderately elevated blood lead levels will show significant cognitive deficits when their performance on a comprehensive neuropsychologic battery is compared with matched control children. A secondary exploratory hypothesis was: children with moderately elevated blood lead levels will show a consistent pattern of cognitive deficits.

MATERIALS AND METHODS

Subjects

A total of 15 experimental children and 15 matched control children were recruited. Participation was voluntary, and informed consent was obtained from both the parents and the children. Experimental subjects came from the Pediatric Lead Clinic of the Rhode Island Hospital, which is affiliated with the state's department of health and which services the entire state. Clinic records for the preceding 5 years were reviewed to obtain potential subjects meeting four inclusionary criteria: (1) 5 to 12 years of age, (2) blood lead levels between 30 and 60 μg/100 mL obtained previously on at least two consecutive assessments separated by at least 3 months, (3) blood lead levels greater than 60 μg/100 mL never obtained, and (4) levels less than 30 μg/100 mL maintained for at least the most recent year as demonstrated by a minimum of two laboratory assessments. This last criterion was used to help separate transient from more stable effects. Only 28 children met all of the criteria, a relatively small percentage of the lead clinic patients. Many failed to meet criteria 3 or 4 above.

Exclusionary criteria included: (1) gross mental retardation; (2) maintenance on a psychopharmacologic agent; (3) major sensory or motor handicaps; (4) prenatal, perinatal, or postnatal stresses associated with CNS dysfunction; and (5) medical conditions effecting the CNS (eg, epilepsy). Information regarding exclusionary criteria was obtained through review of the medical records and, as necessary, interview with parents. Four potential subjects with medical conditions possibly associated with CNS dysfunction were excluded.

We were able to contact the families of 18 of the remaining 24 potential subjects. The first 15 that came in for scheduled appointments were included in the study. Within the group, highest recorded lead levels ranged from 36 to 59 μg/100 mL (mean 52.2 μg/100 mL). The average of the three highest lead levels ranged from 35 to 55 μg/100 mL (mean 44.6 μg/100 mL). Age at which lead was first detected and treated ranged from 14 to 72 months (mean 38.9 months). The length of time between the date subjects' lead levels decreased to less than 30 μg/100 mL and subsequently remained so on all further blood tests and the date they participated in the study ranged from 12 to 83 months (mean 43.3 months). At the time of participation in the study, lead levels ranged from 11 to 29 μg/100 mL (mean 19.1 μg/100 mL).

We planned to recruit siblings of experimental subjects as control subjects, but many of the previously tested siblings also had elevated lead levels. Thus, obtaining current blood levels on previously untested siblings (which only measures recent lead uptake) seemed an insufficient safeguard for eliminating those with previously elevated lead levels. Only two siblings who were suitable for the study had been tested on more than one past occasion and had blood levels of less than 30 μg/100 mL consistently.

Additional control children were recruited from a primary care clinic in the Rhode Island Hospital. Through a computer search we identified a pool of subjects that matched for age, gender, and residential area. Medical records were reviewed for the exclusionary criteria. With their primary physician's permission, letters were sent to the parents of potential subjects and follow-up calls were made requesting participation but explaining the study's voluntary nature. It was necessary to send 26 letters to obtain the remaining control subjects.

Demographic characteristics of the groups appear in Table 1. Socioeconomic status was determined using the procedure of Hollingshead and Redlich.[27]

TABLE 1. Demographic Characteristics

	Group	
	Experimental	Control
Age (mo)		
Mean	93.5	93.7
Range	69–128	70–128
SD	17.9	17.0
Gender (No. of children)		
Male	6	7
Female	9	8
Socioeconomic status*		
Mean	4.2	4.2
Range	3–5	2–5
SD	0.83	0.98
Parental IQ (%)		
Mean	64	60
Range	9–95	12–95
SD	28.4	23.4

* Hollingshead and Redlich's procedure.[27]

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Scores ranged from 1 (highest status) to 5 (lowest). Most children came from disadvantaged families. To provide an estimate of parental intelligence, mothers of subjects completed the Raven's Progressive Matrices,[28] a nonverbal test of intellectual functioning that reduces the role of sociocultural factors. Scores are listed in percentiles. The scores of the experimental group (64%) and the control group (60%) were within the average range. Overall, the two groups matched closely. As planned, we did not match children on intelligence. Lead absorption may create cognitive deficits that lower IQ, and thus, IQ differences may result from the effects of lead and not artifact.

Measures

The comprehensive neuropsychologic battery is adopted from the work of Luria.[29] The battery consists primarily of standardized psychometric tests, although a number of clinical tests are used. Extensive normative data are available on all of the tests and items. Item difficulty and scoring are adjusted for age.

Six general areas of functioning are tested: psychomotor, memory, visual-motor and spatial, language and associated functions (eg, reading), attention and concentration, and reasoning. Testing in each of these general areas is comprehensive, as detailed in Table 2. The battery consists of a series of divisions (eg, Motor: speed, strength, dexterity), and two of the divisions are further divided into subdivisions (eg, Spatial: rudimentary, advanced). One or more tests may be used to assess functioning within divisions or subdivisions.

Certain of the divisions and subdivisions may not be self-explanatory. "First-trial learning" refers to the amount of information retained following the initial presentation of material, and "incremental learning" refers to increases in retained information over repeated presentations of materials. "Visual-verbal associative learning" describes the ability to learn pairings between visual symbols and words or sounds. Rudimentary spatial functions include such basic operations as identifying objects by sight and distinguishing left from right; advanced functions involve more complex operations, such as spatial analysis and synthesis.

The Child Behavior Checklist was obtained to provide a check on the external validity of the neuropsychologic assessment results. The Child Behavior Checklist is of demonstrated reliability and validity in the assessment of behavior and adjustment.[30-32] Forms vary depending on age and gender, and children are rated along a series of dimensions derived from multivariate analyses. Dimensions include depression, social withdrawal, hyperactivity, aggressiveness, and social activities and competencies.

Procedure

When subjects arrived, a blood sample was obtained before testing was initiated. An interview was conducted separately with the parent(s) by the senior author to obtain any needed background information and to explain procedures for completing the Child Behavior Checklist and the Raven's Progressive Matrices test. The neuropsychologic tests were administered in a set order and scored in a standard fashion by a trained technician unaware of the subjects' group status.

All subjects completed all measures without undue difficulty. Testing required 3½ hours to five hours, with rest periods provided as needed. Results were discussed with parents by the senior author. For any child evidencing problems, we offered to consult with the school to aid in educational planning.

TABLE 2. Areas, Divisions, and Subdivisions of Functioning Assessed

Area	Divisions	Subdivisions
Motor	Speed, strength, dexterity	
Memory	Verbal	Immediate, remote, rote, conceptually based, storage, retrieval, first-trial learning, incremental learning
	Visual	
	Visual-verbal associative learning	
Visual-motor/spatial	Visual-motor	
	Spatial	Rudimentary, advanced
Language	Fluency, naming, repetition, vocabulary, oral comprehension, reading, writing	
Attention/concentration	Attention, concentration	
Reasoning abilities	Abstract reasoning, concept formation, inductive/deductive	

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RESULTS

Because of concerns about certain assumptions of parametric methods and because tests within the battery use different levels of measurement, nonparametric statistics were used. To compare overall performance, mean scores were calculated for the control and experimental groups for the 42 tests administered. The comparative frequency with which the control group achieved higher mean test scores (35) v lower means scores (7) was analyzed using the Sign Test, with a resulting z score of 4.17 ($P < .01$). This result supports our first hypothesis, which stated that children with a history of moderately increased lead levels will perform at a lower level, overall, than matched control subjects.

The comparative performance of experimental and control subjects in major areas and their divisions within the battery are represented graphically in the Figure. The mean scores on the test or tests making up divisions and areas were converted to percentile scores for this purpose. The light and darkened bars represent performance levels for the experimental and control groups, respectively. The horizontal line cutting across divisions and areas demarks average performance and provides a standard for evaluating the performance of each group. Within each area represented in the Figure, performance in divisions appears first, followed by performance in that area overall.

The significant difference in overall performance was due to the consistency with which the control group outperformed the experimental group and not because of large but select differences (Figure). The largest contrast between groups (Motor: speed) represented about 1 SD, and most differences were much smaller, representing about 0.5 SD or less. The scores of both groups were less than average in most areas, further demonstrating the importance of control groups in studies on elevated lead levels.

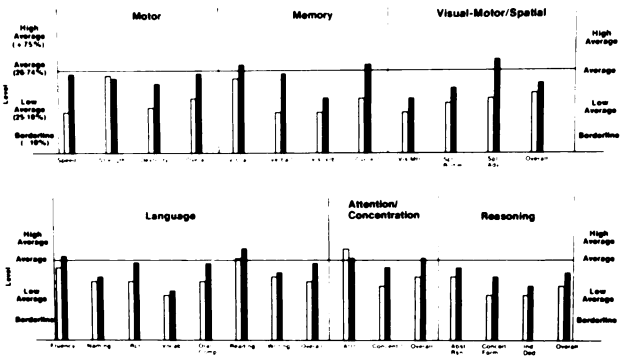

Figure. Performance levels on sections and subsections of battery. Abbreviations: Vis.-Vrb., visual-verbal associative learning; Vis.-Mtr., visual-motor; Spt.-Rudim., spatial-rudimentary; Spt.-Adv., spatial-advanced; Rpt., repetition; Vocab., vocabulary; Oral. Comp., oral comprehension; Attn., attention; Concent., concentration; Abst. Rsn., abstract reasoning; Concept Form., concept formation; Ind.-Ded., inductive and deductive reasoning.
* $P < .05$ (one-tailed).

The results displayed in the Figure were subject to separate statistical analysis. Control and experimental children were paired by age, and their relative performance on each test was then evaluated. For the six areas and for most subdivisions, the Sign Test was used to assess the relative frequency of higher and lower scores across the pairs of experimental and control subjects. The Wilcoxin signed-ranks test, a more powerful procedure, could be applied in the few cases in which only one test was used to assess a division of functioning (eg, inductive and deductive reasoning).

Comparison of the groups for overall performance in the motor area yielded a z score of 2.39 ($P < .01$, one-tailed), thus indicating significantly lower performance by the experimental group. Within divisions, the experimental group obtained significantly lower scores on a measure of speed (N = 15, $T = 10$, $P < .05$, one-tailed) and on a measure of dexterity (N = 15, $T = 21$, $P < .05$, one-tailed) but not on a measure of strength.

A significant difference was found for overall performance in the memory area ($z = 4.86$, $P < .01$, one-tailed). However, within divisions, significant differences were found only in verbal memory and not in visual or visual-verbal memory. Numerous subdivisions of verbal memory were evaluated (Table 2), and significant differences were found in all but one of these subdivisions (rote recall). These results indicate broad-based problems in verbal memory, including not only decreased capacity to store and retrieve information following its initial presentation but also reduced gain from repeated presentations of material and more rapid loss (forgetting) of information over time.

Overall performance levels were not significantly different in the visual-motor/spatial area ($z = 1.06$, $P < .05$, one-tailed). Analysis of divisions yielded nonsignificant differences for visual-motor and rudimentary spatial functions but a significant difference for advanced spatial functions ($z = 2.23$, $P < .05$, one-tailed).

A significant difference was found for language functions overall ($z = 2.49$, $P < .01$, one-tailed). Differences across divisions were relatively small and nonsignificant, although the control group outperformed the experimental group in every case.

There was no significant difference in overall performance level in the area of attention/concentration ($z = .92$, $P > .05$) or for the division of attention. Differences in concentration, however,

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were significant ($z = 2.23$, $P < .05$, one-tailed). In the area of reasoning, the control group outperformed the experimental group consistently, but differences across the divisions, and overall ($z = .92$, $P > .05$), were not significant.

Various analyses were conducted to test hypothesis 2, which stated that a consistent pattern of deficits would be found among experimental subjects. These analyses included within-group comparisons of performance across areas, correlational analysis of mean performance pattern for the group and individual performance patterns, and clinical analysis of profiles. None of the analyses yielded strong or consistent support for a pattern of performance deficit that characterized all or most of the subjects. Trends did appear, which included significantly better performance on attentional measures v measures of motor functions, memory functions, advanced spatial functions, and concentration and significantly better performance on measures of rudimentary v advanced spatial functions.

The experimental and control groups differed only slightly and inconsistently on the factors and scales of the Child Behavior Checklist. Thus, it was not surprising that scores on the composite battery did not show a significant relationship with the Child Behavior Checklist factor scores or with scores on the individual subtests for aggressive and hyperactive behavior. Given these results, further analysis was not performed.

DISCUSSION

The experimental group's lower overall performance supports the hypothesis that children with a past history of moderately increased blood lead levels will show cognitive deficits when compared with matched control children. The subjects had never attained markedly elevated blood levels, were generally treated early and aggressively, and had maintained levels of less than 30 μg/100 mL for at least 1 year prior to testing. It is conceivable that even relatively short-term elevations in blood lead levels exert a nontransient, negative impact on cognitive functions. Epidemiologic studies suggest that, in the United States alone, perhaps more than 1 million children are at higher risk than our subjects.[2]

Our results are obviously consistent with studies showing decreased cognitive functioning in association with moderate or lower lead exposure and conflict with studies showing no decrement. Uncovering systematic patterns that might underlie differing outcomes across these studies is an immense challenge, especially given broad differences in subjects, methods, timing of assessment, etc. Perhaps a unique feature of the present study, however, is the breadth of the measures used. These measures detected significant differences among relatively small subject groups or with an experimental design considerably less powerful than that achieved with larger groups. Recall further that it was the general consistency of lower scores, rather than isolated but large magnitude differences, that accounted for the statistically significant contrast in overall performance. Thus, it remains possible that at least some studies reporting nonsignificant findings represent false-negative errors stemming from measures of insufficient breadth and sensitivity. In planning future work, investigators might consider reducing subject number to permit more complete neuropsychologic assessment. Even well-designed studies with large subject groups may fail to detect true differences if measures lack sensitivity; negative findings obtained under such conditions will remain ambiguous.

It might seem inappropriate to indicate that our experimental subjects showed "deficits." If percentile scores are translated into standard scores and one assumes a normal distribution, the differences between the experimental and control subjects averaged less than 0.5 SD. Relatively small differences have also been reported in other studies, and some investigators have concluded that these differences are not of much importance, particularly when compared with the more powerful associations found for social factors.

We would urge great caution before disregarding even seemingly small differences in functioning. As Rutter[6] has argued, if the IQ scores of all children decreased by less than 0.5 SD, twice as many children would be within the retarded range. Furthermore, because our current measurement techniques are far from perfectly sensitive, and because variable responses among children can mask even large differences among select subgroups, obtained differences could substantially underestimate true effects. Innumerable times, agents once thought innocuous were later shown to be harmful as measurement techniques improved. In addition, what is "minor" now may not remain so later. A recent report suggests that earlier lead exposure is related to cerebellar calcification among elderly individuals.[33]

Finally, demonstrations that the experimental or statistical control of potentially confounding variables, such as socioeconomic status, reduces or eliminates obtained statistical relationships between lead exposure and cognitive functioning are suggestive but not definitive. There is an obvious

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need to exercise control in the design and execution of studies, as witness the generally below average level of performance shown by both of our subject groups. However, it is never certain that the correct variables have been controlled or whether one has inadvertently mitigated true differences. For example, suppose that lower parental IQ not only reflects but also partially accounts for social disadvantage, ie, that individuals of below average intelligence less frequently attain higher strata.[34] Suppose further that lead exposure decreases IQ. Given these two possibilities and the high correlation obtained between maternal and perinatal lead levels,[19] then "matching" subjects on parental IQ and socioeconomic status may actually eliminate differences that are truly related to lead exposure. The study of lead will not yield to any simple experimental approaches; the excellent methodologic critiques provided by several authors merit our careful attention in the design and interpretation of studies.[6,20,23,24]

The secondary aim of our study, to identify a specific pattern of cognitive deficit, was not achieved. The experimental group evidenced substantial heterogeneity, and neither large nor select differences were found across or within groups. However, the small subject groups decreased the probability that anything less than dramatic and highly consistent differences would achieve statistical significance. A more powerful design might confirm trends in our data, including relatively greater difficulties in fine-motor skills, verbal memory and learning, advanced spatial functions, and concentration.

Whether a specific or consistent pattern of deficit exists is partially independent of questions about the specific effects of lead and whether the severity and expression of such effects vary in relation to other parameters. Shaheen,[34] for example, has postulated one such specific effect: that lead exposure impedes myelination. If Shaheen is correct, then type and severity of cognitive deficit should vary in relation to phase and stage of the myelination process and, thus, relative to age of exposure.

The investigation of specific effects and their relationship to other parameters is critically important. It remains essential to establish a threshold for safe exposure. Should the effects of lead vary in relation to other parameters, such as age of exposure, length of exposure, recurrence of elevated levels, nutritional status, or premorbid intelligence, to name a few possibilities, then threshold cannot be determined by lead levels alone. In turn, determining risk and priorities for prevention and intervention will also need to take these parameters into account.

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