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ABSTRACT

This report presents an overview of research on childhood lead exposure and poisoning, and the related social issues. The report first summarizes the history of lead poisoning and its prevalence in the United States, and discusses the basis for recent changes in guidelines for lead exposure by the Centers for Disease Control (CDC). The report then reviews the animal and human research literature on lead exposure and poisoning, focusing on the neurobehavioral effects of lead poisoning, methodological issues, and directions for future research. It then discusses the public policy implications of lead exposure and the financial costs of abatements, finding that the removal of lead from children's environments has proven to be more expensive, complicated, and time-consuming than once thought. The report concludes that while the effects of high levels of lead exposure have been documented for nearly a century, the specific effects of lower levels of lead (10 to 15 micrograms per deciliter of blood) are less clear, although the data seem to support that even low-level lead exposure causes cognitive and behavioral deficits. (Contains 68 references.) (MDM)

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Developmental Effects of Lead Exposure in Children

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The past few decades have witnessed a dramatic interest in the effects of childhood lead poisoning. Although the toxic effects of lead have been acknowledged for generations and the production of lead dates to ancient civilizations, only recently has it been known that even low levels of lead exposure may have serious effects on children's development. In 1991, driven by accumulating research findings, the Centers for Disease Control (CDC) published new guidelines regarding the treatment of children exposed to lead. In these guidelines the blood lead level of intervention was lowered from 25 to 10 micrograms per deciliter (µg/dL).

Low levels of lead exposure have been associated with a broad array of childhood problems ranging from cognitive and behavioral problems in preschool through high school (see Environmental Protection Agency [EPA], 1986, and Needleman, 1992, for a summary of findings) to hearing problems (Fox, 1992). New insights regarding the effects of lead have come from many sources. In particular, evidence from animal research has been greatly influential, and many recent, well-designed and carefully conducted cross-sectional and longitudinal studies carried out in several countries have added to our understanding of the effects of lead on development.

Sadly, in the end it appears that there may be no safe level of exposure to lead and that some neurotoxic effects may be irreversible (American Academy of Pediatrics, 1993).[1] However, lead poisoning continues to be entirely preventable: remove the source of lead from the child's environment, and remove the problem; never expose the child to lead, never have a problem. Unfortunately, removing the sources of lead from children's environments has proved to be a much more difficult and costly procedure than once thought, although there may be some success stories, as in the case of restricting leaded gasoline.

This report presents an overview of childhood lead exposure and poisoning, and the related social issues. The first section briefly summarizes the history of lead poisoning and its prevalence in the U.S., and discusses the basis for recent changes in the CDC guidelines. The next section reviews the animal and human literature on lead poisoning. The purpose of this review is to highlight current findings and to illustrate some of the mixed results and methodological issues relevant to studies of children, not to provide exhaustive coverage of the extensive literature on lead effects. A third section proposes strategies for future research. Finally, policy is-

sues and legislative action related to managing lead hazards in the home and community are discussed, and conclusions are drawn.

Background

Ancient History

Lead was one of the original metals used by man. It was utilized by ancient civilizations as a source of silver and in the production of items as diverse as dishes, pigments, sling shots, weights, and coins. In the Roman Empire, lead was an integral part of the aqueduct system which supplied the drinking water for the city. Some have speculated that the fall of Rome resulted from the widespread use of lead (Gilfillan, 1965). Others point out that it is difficult to ascertain how much lead was actually in Roman drinking water because the waters came from springs in and around as Rome as well as from the aqueducts. It has been suggested that more problematic was the use of sapa, a sweetening agent made from grapes cooked in lead pots (Nriagu, 1993).

Nicander, a Greek poet-physician in the second century B.C., theorized that exposure to white lead and the sugar of lead resulted in what are now recognized as the classic signs of lead poisoning—palsy, pallor, colic, and constipation. During the Middle Ages the effects of lead poisoning apparently received little attention; not until the Industrial Revolution was an interest in lead revived (Cory-Slechta, 1984).

Modern History

Australia was one of the first countries to recognize and treat lead poisoning. In the 1890s a physician named Turner identified children with symptoms ranging from headache to vomiting and foot- and wrist-drop which he attributed to a leaded water tank. Gibson, another Australian physician, published a paper in 1904 titled *A Plea for Painted Railing and Painted Walls as the Source of Poisoning Among Queensland Children* (for further historical details, see Lin-Fu 1992). He argued that the source of lead poisoning for the majority of Australian children was the leaded paint that covered railings and porches. Moreover, he determined that exposure was greatest during the warmer months when children were playing outside and had opportunities to have their hands coated with lead dust and engage in hand-to-mouth activities.

Physicians in the U.S. were initially skeptical of the Australian findings, but by the 1920s were forced to confront the issue because reports of lead poisoning had become more prevalent. One significant outbreak of lead poisoning occurred in the Baltimore area, during the Depression, and was found to be due to the burning of battery casings for domestic fuel (Williams, Schultze, Rothchild et al., 1933, cited in Lin-Fu, 1992). This was only one source of lead exposure; others appeared in the years following the Depression. In particular, leaded paint and leaded gasoline have been, and continue to be, the predominant sources of lead poisoning in the U.S.

Concern about lead poisoning and exposure increased slowly in the 1940s and 1950s. This was in part because most lead poisoning was thought to be primarily a problem of slum-dwelling children and the result of pica, whereby children consume nonfood items, in this case peeling and chipping lead paint (Lin-Fu, 1992). It wasn't until the 1960s that lead poisoning was recognized as an important pediatric problem (Griggs, Sunshine, & Newill, 1964).

In 1967 Jane Lin-Fu, writing for the Department of Health, Education, and Welfare (now the Department of Health and Human Services [DHHS]), published a paper entitled *Lead Poisoning in Children* that spelled out for the first time that lead poisoning was a serious threat to children's health causing neurological problems, mental retardation, and even death. Following this publication, efforts to educate the general population regarding the threat of lead poisoning increased dramatically.

In 1971 federally assisted screening of chil-

dren for lead poisoning began. From these screenings it was determined that 20% to 45% of children tested had blood lead levels over 40 µg/dL. It was also discovered that lead-poisoned children did not just reside in inner-city slums, nor did they all suffer from pica. Hence, the importance of lead dust in the environment coupled with typical infant hand-to-mouth activity was confirmed (Lin-Fu, 1992).

Since the mid-1960s the incidence of lead poisoning has decreased significantly due to mass education and the reduction of lead in both gasoline and food processing (lead solder used in canning; Lin-Fu, 1987). Despite these efforts, however, childhood lead poisoning remains one of the most prevalent preventable childhood health problems in the U.S. today.

There have been great improvements since the early 1970s in the technology of measuring lead in microsamples of blood and in other sources. Advances in the past decade, including the introduction of improved instrumentation for the atomic absorption spectrometry and anodic stripping voltammetry, have made it possible to obtain highly accurate and precise measures of lead in microsamples of blood. Prior to 1970 venous samples of 5–20 µL of blood, usually obtained from children by external jugular or femoral vein puncture, were required for the older colorimetric procedures. At that point, a single technician could process only 8 to 10 samples per day. Currently, a technician can test up to 80 samples per day. Without these technologies the research of the past 20 years would not have been possible (Julian Chisolm, personal communication, July, 1994).

Prevalence

In October 1993, during National Child Safety Month, DHHS cited lead poisoning as the most hazardous health threat to children under the age of 6. In 1992 the EPA had estimated that approximately 3 million children in the United States had blood lead levels greater than 10 µg/dl , levels high enough to possibly affect intelligence and development. Recent evidence suggests that lead levels above 10 µg/dL may occur more frequently in urban than suburban areas (Harvey, 1994; Nordin, Rolnick, & Griffin, 1994).

Despite efforts to reduce the level of lead in the environment through prohibition of lead in residential paint (a ruling made in 1977 by the U.S. Consumer Product Safety Commission) and reductions of lead in gasoline, there is still a considerable environmental threat. The EPA (1992) has estimated that dust and soil in many parts of the country continue to be contaminated by leaded gasolines and paints and that 74% of all housing units built before 1980 contain lead-based paint. Faced with this information on prevalence and other accumulating evidence on the effects of lead poisoning, the CDC changed its lead policies in 1991 (CDC, 1991).

CDC Policy Changes

In its 1991 revised policies, the CDC made several recommendations, including that the blood lead level of "concern" be lowered to 10 µg/dL and that virtually all children be screened for lead exposure and poisoning. It is notable that the American Academy of Pediatrics supported the CDC's policies, stating that "pediatric care providers should increase their efforts to screen children for lead exposure. Blood lead screening should be a part of routine health supervision for children" (1993, p. 181). These recommendations have proven to be somewhat controversial (Harvey, 1994).

Requiring screening. On the question of screening, some physicians have argued that selective rather than universal screening would be less costly. By carefully questioning patients, using a lead poisoning questionnaire developed by the CDC (1991) or similar questions, the physician can determine the extent of risk and the need for further blood testing.

Selective screening has been shown to be a cost-effective procedure in several studies (see

Harvey, 1994). However, a recent study found that only 12% of the 556 physicians surveyed were currently testing for the blood lead levels of their patients and that their knowledge of the effects of lead exposure and poisoning was limited (Bar-on & Boyle, 1994). Thus, how feasible it might be to institute selective screening on a broad scale is unclear.

Lowering the blood lead level of concern. The CDC's decision to lower the blood lead level of concern was based on several important research findings (CDC, personal correspondence, 1993). For example, two meta-analyses of relevant studies indicated that lead exposure, even at low levels, does affect the intelligence of children (Bellinger & Needleman, 1992; Needleman & Gastonis, 1990). Moreover, findings of long-term effects of lead poisoning in a group of 132 predominantly Caucasian children also influenced the CDC decision (Needleman, Schell, Bellinger, Leviton, & Allred, 1990). In this study adolescents who had been exposed to lead in early childhood were more likely to drop out of school, be absent more, have reading disabilities, have lower class standings, and have lower vocabulary and verbal reasoning scores, poorer hand-eye coordination, longer reaction times, and slower finger tapping.[2]

In 1986 the EPA estimated that with each raised level of blood lead there is an associated decline in IQ points: 1 to 2 points at levels of 15 to 30 µg/dL; 4 points at levels of 30 to 50 µg/dL; and 5 points at levels > 50 µg/dL. This loss of IQ points was seen as problematic by the CDC because of the potential for increased numbers of children in the severe deficit category and decreased numbers in the above-average category.

It should be noted that some researchers (see, for example, Ernhart, Landa, & Wolf, 1986) have claimed that the effects of lead on IQ are minimal. Ernhart has argued that when the confounding variable of parental IQ was controlled for, the apparent effects of lead on intelligence were reduced (Ernhart, Landa, & Schell, 1981). Ernhart's work has been criticized by researchers in the field, however, for not controlling for another confounding variable, namely that some of her research subjects had alcoholic mothers; the effects of prenatal exposure to alcohol on cognitive functioning are well documented.

Other research supporting the CDC changes included findings on the effects of lead on hearing (Fox, 1992) and on physical stature and several biochemical indices (CDC, 1991). Finally, animal research also contributed to the CDC decision. For example, the CDC (personal correspondence, 1993) was concerned about findings from primate studies showing that low levels of lead interfered with cognitive and behavioral functioning.

Review of Research

Neurobehavioral Effects of Lead Poisoning

Animal Studies

Animal studies provide important evidence on lead exposure effects. The fact that the genetic make-up of research animals as well as their environments, unlike those of typical lead-poisoned children, can be more carefully controlled makes it easier to pinpoint whether deficits are the result of lead rather than some other source.

Mice and rats. Many studies of rats and mice have documented the effects of lead poisoning in several areas of development. In one study, for example, mice that were chronically exposed to lead were slower to open their eyes (thought to indicate delayed CNS development) and had reduced body weights throughout their lives (Donald, Cutler, & Moore, 1987). Interestingly, children who are prenatally exposed to lead have been found to have low birthweights or to be small for gestational age (Schwartz, 1992).

The male lead-exposed rats in this same study exhibited significantly shorter latencies to aggression than their nonexposed counterparts, and the lead-exposed rats overall demonstrated

an increase in exploratory behavior. Aggressive behavior in lead-exposed mice has been documented by other researchers as well (Engellenner, Burright, & Donovick, 1985; Holloway & Thor, 1986). Many human studies have found that lead increases activity level and aggressive behavior (e.g., Marlowe, Stellern, & Errera, 1982).

The effects of lead exposure on the cognitive functioning of rats and mice have also been well documented. These deficits have been found to be particularly apparent in learning paradigms. Deficits range from problems with the acquisition of brightness, spatial, auditory, and tactual stimulus control to differential patterns of responding to contingencies of reinforcement, which have been found to be sensitive indicators of neurological toxicity (for a critical review, see Cory-Slechta, 1984).

Monkeys. Findings reported in the primate literature have also helped clarify the effects of lead on behavior and development. One review states that "behavioral impairments have been observed in every group of primates tested with no evidence for a threshold " (Rice, 1992, p. 150). Deficits, with seemingly irreversible effects, have been found in monkeys with blood lead levels as low as 11 µg/dL. Included are attentional difficulties, learning problems, and decreased memory capacities.

Other researchers have documented behavioral and learning problems in primates similar to those observed in lead-exposed children. Lead exposure in monkeys has been found to result in decreased muscle tone, increased agitation, and reduced visual attentiveness (Levin, Schneider, Ferguson, Schantz, & Bowman, 1988).

Human Studies

Decrements in IQ. The effects of lead exposure and poisoning on intellectual functioning have received a great deal of attention, and, as already mentioned, evidence is mixed. Many studies have shown decreases in overall IQ scores as well as specific deficits in verbal and performance areas to be associated with lower ranges of lead exposure, but such deficits have been absent in some other studies.

In one study, for instance, of a clinical group consisting of children with lead levels > 15 µg/dL, and controls with levels < 15 µg/dL, children were administered the WISC-R and WRAT (Yule, Lansdown, Millar, & Urbanowicz, 1981). The clinical group showed deficits in the verbal section of the WISC-R but not on the performance scale. In addition, the clinical group had lower reading and spelling achievement scores.

A more recent study also found an inverse association between lead exposure and verbal IQ interacting with the child's age at exposure (Bellinger, Stiles, & Needleman, 1992). Elevated lead level at 24 months, only, was associated with lower IQ scores at age 10 years. In this study 249 children were tested for lead exposure and placed in one of three groups (low = < 3 µg/dL, below the 10th percentile; medium = 6.5 µg/dL, approximately the 50th percentile; and high = > 10 µg/dL, above the 90th percentile). Blood lead levels were measured at 6, 12, 18, 24, and 57 months. Cognitive assessments were conducted using the Bayley Scales of Infant Development at age 24 months and the WISC-R and K-TEA at age 10. Controlling for possible confounds, i.e., family social class, maternal education and IQ, life events, and blood lead history, analyses showed that children's blood lead level, at age 24 months, was significantly associated with children's verbal IQ at age 10 years. Performance IQ was unaffected at all assessment periods. Regarding the K-TEA scores, only blood lead level at 24 months of age were significantly associated with decreased scores, predominantly in the area of spelling and mathematics. The composite score at age 10 decreased 8.9 points for each increment of 10 µg/dL measured at 24 months.

It should be noted that Bellinger et al. (1992) did not carry out any blood lead measurements between 24 and 57 months of age. However, blood lead is known to rise steadily from 16 to 24 months and peak at approximately 24

months and then very slowly decline (Julian Chisolm, personal communication, July, 1994). Thus the 24-month blood lead levels were most probably representative of the peak lead level of the subjects.

In yet another study (Bellinger, Leviton, Waternaux, Needleman, & Rabinowitz, 1987) umbilical cord blood lead measures from 249 fetuses were used to assign subjects to three prenatal exposure groups: the low group < 3 µg/dL, the medium group 6–7 µg/dL, and the high group > 10 µg/dL. Postnatal lead exposure was determined by further blood lead measures. The child's postnatal development was first assessed at age 6 months and then semiannually, utilizing the Mental Development Index (MDI) of the Bayley Scales of Infant Development. Only infants in the high prenatal exposure group scored lower on the MDI during the first 2 years of life.

International studies. Studies conducted outside the U.S. have further substantiated the ill effects of lead on children's intellectual development, although not without some mixed results as to the specific pattern of deficits created by lead poisoning. In a study examining 494 infants born in South Australia (Baghurst et al.,1992), blood lead samples were drawn from umbilical cord at birth, and then lead level was further assessed at ages 6 and 15 months, at 2 years, and annually thereafter. Blood lead levels ranged from a mean lifetime average concentration of 7–11 µg/dL (low) to 18–28 µg/dL (high). The children were tested for intellectual abilities using the McCarthy at ages 2 and 4 years. At age 7, they were tested with the WISC-R. The IQ scores of lead-exposed children whose lead levels had risen from 10 to 30 µg/dL (across their lifetimes) were between 4.4 to 5.3 points lower than those of nonexposed children; and those who performed poorly at age 4 on the McCarthy did not improve their performance at age 7, suggesting that IQ decrements due to early lead exposure may persist.

A study of a group of British children ages 2 to 5 (Harvey, Hamlin, Kumar, & Delves, 1984) did not find a relationship between blood lead level and measures of intelligence. In this study the assessment included four cognitive tasks from the British Ability Scales, psychomotor tasks from the Stanford-Binet, and behavioral ratings based on observations made in testing. Blood lead levels ranged from 6 to 30 µg/dL. Maternal IQ was assessed with the Ravens Progressive Matrices and Mill Hill Vocabulary Scales. Health and marital information was also obtained from the parents. Although blood lead level and intelligence measures were found to be unrelated, it should be noted that nearly half the children (53 of 133) had incomplete data, making the findings inconclusive.

In Germany, an assessment of 1,879 school-aged children between 6 and 11 years of age with blood lead levels ranging from 5 to 60 µg/dL revealed blood lead level associated with some skills and not others (Winneke, Brockhaus, Ewers, Kramer, & Neuf, 1990). A neurobehavioral battery included assessments of IQ (WISC), visual motor integration skills (Bender-Gestalt and the Trail Making Test–TMT), reaction performance abilities (Delayed Reaction Time–DRT and Vienna Reaction Device–VRD), and general behavior (as rated by parents and teachers). No differences were found in general intellectual performance or in less standardized measures of neurobehavioral function such as behavioral ratings. Higher lead levels were related, however, to disrupted performance on the visual-motor integration and reaction performance tasks. These results replicated findings from an earlier study showing that lead-exposed children exhibited deficits in perceptual motor tasks (Winneke & Kramer, 1984).

Interestingly, another recent study found blood lead levels (in this case neonatal levels) to be associated with deficits in motor skills at age 6. Specifically, lead level related to visual-motor control, fine-motor skills, and upper-limb speed. The researchers point out that "motor developmental outcomes may be more sensitive indicators of lead's adverse effects on the central nervous system, as they are probably less con-

founded by social factors" (Dietrich, Berger, & Succop, 1993, p. 301).

In an investigation of the relationship between low lead exposure and children's learning disabilities and need for special education (Lyngbye, Hansen, Trillingsgaard, Beese, & Grandjean, 1990), 177 children, aged 6 to 7, were selected from a variety of public and private schools in Aarhus, Denmark. Dentine lead levels were used to divide the children into a high-lead group (> 18.7 µg/dL) and a comparison group (< 5 µg/dL). At the end of the second grade, children's intellectual performance was assessed using the WISC-R. Children in the high-lead group scored significantly lower on the verbal skills domain than did the comparison group. No performance effects were noted.

Overall these studies conducted in both the U. S. and abroad support the idea that intellectual performance varies with lead exposure. However, what specific areas of functioning are affected remains unclear. Is it verbal, performance, or both areas of functioning that are affected? The mixed results may be explained in part by a study conducted by Shaheen (1984) comparing 18 children aged 4 to 6 with histories of lead exposure to a nonexposed comparison group on a six-factor cognitive neuromotor battery that measured language/linguistic skills, visual-motor integration, rapid learning and attention, motor skills, spatial reasoning, and visual analysis. The clinical group was divided into subgroups representing early exposure to lead (before 24 months), middle exposure (24 to 36 months), and late exposure (after 36 months). The early exposure group's deficits were in the linguistic/language area, whereas the mid-exposure group exhibited poor performance on spatial tasks. The clinical late-exposure group showed no performance decrement relative to the comparison group and the other exposure groups on either spatial or linguistic tasks. These results suggest that early exposure to lead is associated with deficits in the verbal domain; while late exposure is associated with deficits in the performance domain. These findings may help explain the inconsistency of some other research.

Discrepant findings may also relate to socioeconomic factors. For example, the subject population of the Bellinger et al. study (1992), in which effects on functioning in the verbal realm were found, consisted of middle- and upper-middle class subjects. Dietrich et al.'s study (1993) was of low-income black children and found differences in the performance areas. As Dietrich et al. point out, these differences may well reflect that functioning in the performance domain is less susceptible to cultural effects than functioning in the verbal area.

Behavioral Effects

Hyperactivity. Perhaps the most compelling findings of lead research are repeated reports of behavioral problems linked to lead exposure and poisoning. Investigators have found that lead appears to be related to children's irritability, distractibility, lethargy, emotional underreactivity, disciplinary problems, hyperactivity, classroom overactivity, impulsivity, and decreased attentional skills.

For example, a recent study conducted in Scotland of 501 children documented a relationship between lead exposure and children's behavior (Thomson et al., 1989). Behavioral assessments of the children were provided by both teachers and parents, using the Rutter behavioral scales. Multiple regression analyses showed a significant relationship between measures of lead and teachers' ratings on the total Rutter behavior index, the aggressive/anti-social index, and the hyperactivity score. These results suggest an association between low-level lead exposure (mean blood lead level = 10.4 µg/dL) and antisocial behavior and hyperactivity in children.

In another study of school-aged children (Yule, Urbanowicz, Lansdown, & Millar, 1984), it was reported that teachers' ratings of the children's hyperactivity, impulsivity, time spent daydreaming, ease to frustration, and inattention all increased with higher lead levels. The 166 children, aged 6 to 12 years, were measured for

blood lead level. Lead levels were grouped into four categories ranging from low (7–11 µg/dL) to high (17–32 µg/dL). Teachers completed a behavior rating battery consisting of an 11-item questionnaire (Needleman et al., 1979); the 26-item Teacher Rating Scale B(2), which is widely used in Britain to screen general behavior and emotional difficulties; and the 40-item Conners measure, which is used in studies of hyperactivity. The questionnaire results showed an association between lead exposure and higher ratings of hyperactivity, overactivity, and ease to frustration. The Rutter ratings showed children in the upper 50% of lead levels exhibiting more squirmy behaviors, fighting, inertness, disobedience, and overactivity. On every subscale of the Conners scale, children with higher blood lead values were rated as more deviant, with scores statistically significant for hyperactivity, conduct problems, and inattention.

A further study found a significant relationship between blood lead level in children and an increase in frequency of hyperactive behavior ratings (Silva, Hughes, Williams, & Faed, 1988). This study, conducted in New Zealand, assessed a sample of 579 11-year-old children with mean blood lead level of 11 µg/dL, with range 4 to 50 µg/dL. The assessment was composed of the WISC, the Burt Word Reading Test, the Rutter Parent and Teacher Behavior Questionnaire, and teacher and parent reports. Correlations were found between children's lead level and ratings of hyperactive behavior and inattention.

Attention span. A 3-year follow-up study (Bellinger, Needleman, Hargrave, & Nichols, 1981) investigated the relationship between lead level and children's off-task behavior in the classroom. Using a random subsampling of children from an earlier study (Needleman, Davidson, Sewell, & Shapiro, 1974), researchers observed the classroom behavior of children divided into dentine lead-level groups, with low-lead < 10 µg/dL, mid-lead < 13 µg/dL, and high-lead > 20 µg/dL. The children were observed at quiet academic study for four 4-minute periods. At 7-second intervals, the children were scored by the examiners as either on- or off-task. Dentine lead level was found to be significantly related to distracted off-task behavior, i.e., looking at peers, looking at the observer, and looking away from work.

Another study followed 1,923 children from an advantaged white, middle- and upper-middle class population from birth to age 6 (Leviton et al., 1993). Parents completed a questionnaire and provided a sample of the child's shed teeth. The children's teachers completed a questionnaire assessing seven aspects of classroom behavior and academic performance across seven clusters: behavior, hyperactivity, reading, arithmetic, following directions, daydreaming, and completing tasks. The mean umbilical cord blood lead level for this population was 6.8 µg/dL, and the mean dentine lead level was 3.3 µg/dL. Girls with an elevated dentine lead level exhibited a higher level of dysfunction in interactions with peers and completing tasks. Males with elevated lead levels suffered from an inability to attend to and follow directions.

Abuse. Lead-exposed children with behavioral problems may be more difficult to teach and also more difficult to parent. In fact, a recent study found that children suspected of being physically abused had significantly higher blood lead levels (27 times higher) than a comparison group matched for race, sex, income, and housing status (Bithoney, Vandeven, & Ryan, 1993).

Methodological Issues

Role of Environmental Factors in the Development of Lead-Exposed Children

While lead poisoning does appear to affect children's behavior, as well as their learning, the contribution of negative environmental factors cannot be discounted. Several studies have pointed to differential effects of lead exposure related to socioeconomic status. For example, in a study in Germany (Winneke & Kramer, 1984)

only lower-class children who had been exposed to lead showed difficulties with reaction time and visual-motor integration tasks. In their study of British children Harvey et al. (1984) found IQ to be inversely related to blood lead level only in a group of children whose parents were manual workers, no relationship was found for the comparison group whose parents were nonmanual workers. Yet another study (Dietrich, Krafft, Bornschein, Hammond, & Hoffman, 1987) found differences in performance on the Bayley to be related to SES. Thus, there appears to be an interaction between lead exposure and SES, with children of lower SES status being more vulnerable to effects.

Other factors associated with low SES may put these children at greater risk for deficits due to lead exposure. Families in poverty may be less able to maintain stable homes for their children and less able to provide opportunities for their intellectual development. Also, low-income parents are likely to have greater difficulty in providing for their children's medical and nutritional needs.

Poor nutrition is a serious issue, particularly in the case of lead exposure and poisoning. Foods high in fat are problematic because they tend to increase lead absorption in humans, whereas foods high in calcium and iron help the body absorb less lead (Pennsylvania Department of Health, 1991). To reduce the risk of lead poisoning, children should consume fresh fruits and vegetables, dairy products, and lean meats, all of which are difficult to provide on a tight budget.

Methods of Determining Lead Levels

There are several methods for monitoring the amount of lead in the body: these include measuring the lead levels in blood, teeth, urine, hair, and bone. Each offers advantages and disadvantages that must be carefully weighed in evaluating research findings.

Blood. Probably the most commonly used method of monitoring the presence of lead in the body involves testing for lead in the blood. To obtain samples of blood lead, two methods are used: the capillary sample and the venous puncture. The capillary sample is obtained from a heel stick, typically used with children under age 1, and a finger stick for older children. The advantage of capillary samples is that this method is a quick, expedient, and simple procedure for trained medical personnel. Capillary samples are easily contaminated, however, because lead can lodge in the grooves of the fingerprint and elevate the amount of lead detected in the sample (CDC, 1991). To minimize these drawbacks, the CDC has prescribed guidelines for obtaining capillary specimens. And in response to the problems with capillary sampling, the CDC recommends that the "venous blood is the preferred specimen for analysis and should be used for lead measurement whenever practical" (CDC, 1991, p. 45). Current procedure commonly involves testing initially using a finger stick, and then performing confirmatory venous puncture if an elevated lead level is found.

Blood lead level is often a short-term reflection of recent changes in exposure to lead (Mushak, 1992). If a child consumes a large quantity of lead, for example, his or her level rises quickly over a period of several hours, and a very high blood lead measure can be obtained if testing is done during this time.

While low-level lead exposure in adults is thought to be reflected in a stable blood lead level, this is not the case for developing children (Rabinowitz, Leviton, & Needleman, 1984). Particularly in infants and young children up to age 2, blood lead levels may be extremely labile due to developmental changes as well as shifts in environmental lead. Blood lead levels in children may change by as much as 5 µg/dL in a week (Ernhart, 1992). For this reason serial, rather than single, blood measurements are the most informative.

These instabilities in children's blood lead level create an obvious research problem, calling for study designs that control for dramatic shifts in blood lead levels in young children. One common method is to obtain several samples and

average them into a "mean lifetime lead exposure."

Teeth. Some researchers have chosen to avoid the problems of blood lead altogether in favor of measuring lead in teeth, generally in shed deciduous, or "baby," teeth. The amount of lead in these teeth reflects exposure across the entire time the tooth is in the mouth. In that researchers can simply request that parents save the teeth their children lose, this method has proven to be extremely efficient. Its main drawback is that the amount of lead in teeth varies depending on position (e.g., eye tooth versus molar) and the amount of decay (Mushak, 1992). Bellinger et al. (1992) also note that dentine levels may not prove to be good markers of lead when the research questions pertain to very low levels of lead.

Urine and hair. Research designs have utilized measures of lead in urine as well as in hair. Both have proved unreliable and disappointing; assessments vary greatly within individual and between individuals (Mushak, 1992).

Bone. A fourth method, which is strictly a research procedure at this point, involves examining lead in the bone. Because lead is ultimately deposited in bone, this method may provide a more accurate picture than the others. Interestingly, the XRF (a special type of low-energy X-ray) measurement of lead in the tibular bone of children has been found to provide an accurate measure of lead (Rosen et al., cited in Mushak, 1992), but the routine use of this method is unfortunately too costly at this point.

In sum, there appear to be pluses and minuses to each type of sampling. At this point, it appears that serial blood lead measurements are probably one of the best options. Research that uses some of the more controversial methods should be interpreted with caution.

Directions for Future Research

While the effects of lead exposure and poisoning have been extensively studied, questions remain. One has to do with the effects of low-level lead exposure (i.e., 10 to 15 µg/dL) on development. While some of the studies discussed showed deficits at lower levels, additional data are sorely needed. More studies are needed to specify the impact of low-level exposure on cognitive functioning and neurodevelopment. Studies must also isolate the effects of lead from the effects of other factors in the environment, such as poverty, lack of environmental stimulation in the home, and family history of learning problems.

Another important issue is raised by Shaheen's study (1984). As discussed previously, this study found differential effects of lead related to the age at which the child was exposed. While these findings are very exciting, it should be noted that this study utilized a very small sample, and lacked a non–lead-poisoned group for comparison. A large scale replication of the Shaheen design could prove very informative.

One way to address some of these questions may be through animal studies, which utilize methodologies that are freer of "cultural bias" than studies of humans. For example, lead exposure in monkeys has been found to result in reduced visual attentiveness (Levin et al., 1988). Habituation paradigms like those used in this study have been used in studies of children, with interesting results. For example, Ross, Tesman, Auld, and Nass (1992) found that premature infants with mild intraventricular hemorrhage differed from those without hemorrhage on a visual attention task. Assessing the performance of lead-exposed children, using a similar habituation paradigm, could be informative.

While the effects of lead exposure on attention behavior are well documented at this point, children may be experiencing other types of behavior problems yet to be identified. In our laboratory we are currently exploring this issue through a study of 45 children (ages 7 to 10) who

have been treated and or followed by the Polyclinic Medical Center's Childhood Lead Poisoning Prevention Program. Preliminary analyses of data obtained through the Achenbach Child Behavior Checklist, completed by teachers, indicate that the problems exhibited by lead-exposed children are wide ranging (Tesman, Morrow, & Varma, 1994). For example, male children were found to exhibit attentional problems and delinquent behaviors, which is consistent with other research (Wicks-Nelson & Israel, 1991), but surprisingly they were also above the mean in the withdrawn, somatic complaint, and anxious/depressed categories. A significant portion of the boys were also unexpectedly classified as internalizing (i.e., fearful, inhibited, and overcontrolled).

Another surprise came from the female subjects. Although girls are typically less prone to behavior problems, teacher ratings of the female subjects in our study showed a wide range of difficulties, including attention and aggressive behavior problems and behaviors categorized as withdrawn. The girls were often found to fall in the externalizing category (i.e., aggressive, antisocial, and undercontrolled). These preliminary data suggest that lead-exposed children, of both genders, are at risk for a range of behavioral problems—problems that may have lifelong implications.

Policy

Whatever deficits are documented, or yet to be documented, the troubling fact remains that lead exposure continues to be an entirely preventable problem. Children who never come in contact with lead are free from exposure; if exposure is minimized so will their risk be. Unfortunately, the removal of lead from children's environments has proven to be a more expensive, complicated, and time-consuming process than once thought.

Abatement

Congress and government agencies are not unaware of the need to address the lead hazard and the issue of its removal. In 1988 the Agency for Toxic Substance and Diseases Registry estimated that 42 to 47 million houses in the U.S. contained lead paint (cited in Lin-Fu, 1992). According to the 1991 CDC guidelines, "eradicating childhood lead poisoning requires a long-term active program of primary lead-poisoning prevention, including abatement of paint hazards in homes, day-care centers, and other places where young children play and live" (1991, p. 65).

In 1990 the U.S. Department of Housing and Urban Development (HUD) published a comprehensive guide to abatement titled *Lead-based Paint Interim Guidelines for Hazard Identification in Public and Indian Housing*. These guidelines outline proper abatement procedures, complete with time frames for investigations and interventions; emergency and long-term measures for abatement; descriptions of the three categories of abatement and the procedures to follow with each; and specific standards for the amount of allowable lead following abatement procedures.

Three different abatement procedures can be followed in removing lead paint from buildings: replacement, encapsulation, or paint removal. Replacement involves totally removing a lead paint-covered part of a building, such as a window, and replacing it with a new part. While this method is very efficient, it can be costly. It is also impractical for certain parts of buildings such as the walls.

Encapsulation is the process whereby lead painted surfaces are covered with a material that prevents any access to lead paint or lead dust. Unfortunately, methods of encapsulating have been difficult to locate and develop. Ordinary paint does not lend itself to encapsulation, nor does any other material that peels or flakes over time—as does paint.

Paint removal is perhaps the most dangerous process, because these procedures create large amounts of lead residue, usually in the form of lead dust. HUD has suggested that machine sanding, chemical stripping, and removal of paint with a heating device are hazardous to both workers and building occupants. Because of the risks, lead abatement done improperly can create even more trouble than leaving the lead paint intact. Obviously the need for skilled workers in the case of removal is crucial.

Abatement must continue to be a central goal. Even though it is costly, fully abated housing will allow *generations of children* to grow up protected from lead poisoning. Abatement is not a simple task, however. Each procedure has its disadvantages, and ultimate effectiveness is in question. Even after abatement is completed, further testing is needed to determine if lead remains—once again requiring skilled workers, expensive equipment, and laboratory facilities. Finally, recent monitoring of some instances of abatement has revealed that even when abatement is carried out following current guidelines, the goal of lowered blood lead levels sometimes remains elusive (e.g., Lin-Fu, 1992; Weitzman et al., 1993).

Parent Education

One promising approach to reducing children's blood lead levels without relying on abatement was reported in a study of 490 children, aged 6 to 71 months, who lived in the vicinity of a defunct lead smelter in homes built before the 1920s (Kimbrough, LeVois, & Webb, 1994). A program of parent education was found to be effective in lowering blood lead levels.

The parents in this study underwent extensive training in how to cope with the environmental presence of lead. They were instructed to wash their children's hands before meals and before bed, to keep their fingernails clipped short, and to provide a well-balanced diet. Parents were shown housekeeping techniques, including how to carefully remove chipping and peeling paint and how to put up barriers to keep children away from heavily leaded areas. Parents were also instructed to seek expert guidance before conducting any renovations.

The result of this intensive program was dramatic. The children's mean blood lead level decreased over a 4-month period from 15 μg/dL to 7.8 μg/dL; and a follow-up 1 year later found that blood lead levels continued to be low (mean = 9 μg/dL). Interestingly, the Illinois Department of Public Health has had similar success with a counseling intervention (Kimbrough et al., 1994).

While these results are promising, it should be noted that the subjects in this study were a motivated group of parents with children with elevated blood levels. The families had limited access to health care and lead abatement resources and were "at a loss as to what to do." Also, the counseling provided was intensive, consisting of several 30- to 45-minute sessions conducted with the whole family (Kimbrough et al., 1994). Further research with matched controls who do not receive counseling is needed.

Current Legislative Action

It is obvious that the most important step to preventing lead poisoning is abatement. On October 28, 1992, Congress passed the Residential Lead-based Hazard Reduction Act of 1992 (P.L. 102–550). Probably the most comprehensive legislation on lead to be passed in the last two decades, this Act stands to have far-reaching implications. Among the many provisions, the EPA is required to set up regulations and to establish centers to train professionals in the handling and removal of lead-based paint. The sale of homes is also regulated, such that sellers have to notify prospective home buyers regarding the presence of lead paint, and prospective buyers will have the option of conducting their own inspections.

HUD abatement demonstration grants have been awarded to several cities, with initial funding of $150 million provided by the House Ap-

propriations Committee. The committee has also appropriated $37 million for the CDC's lead poisoning prevention grants.

The need for monies for abatement is pressing. In March 1993 the Health Subcommittee of the House Ways and Means Committee approved a health care reform bill (H.R. 2479) that includes elements of the Lead Abatement Trust Fund legislation. It remains to be seen if this bill will pass through both houses of Congress. The proposed funding source for this bill is a tax on the lead industry. The lead industry is arguing that taxing lead will cost jobs.

On May 25, 1994, the Senate passed the Lead Exposure Reduction Act of 1994 (S. 729) sponsored by senators Reid and Lieberman. The bill includes provisions for (1) establishing limits for particular products that contain unsafe amounts of lead; (2) setting up programs to reduce and restrict lead in several items, e.g., plumbing fixtures, ink, curtain rods, etc.; (3) requiring the EPA to maintain an inventory of all uses of lead sold and distributed commercially, and to devise labeling procedures for new and already marketed products; (4) providing mandatory recycling of batteries and a method to link retailers and smelters; (5) setting up a federal program to fund inspections for lead paint at schools and day-care centers built prior to 1980; and finally (6) establishing a National Center for the Prevention of Lead Poisoning (*Congressional Record*, 1994a, 1994b). It should be noted that a companion bill is yet to be introduced in the House.

Conclusions

The effects of lead poisoning and exposure on the development of children have been documented for almost 100 years and certainly suspected centuries before that. Lead poisoning, especially at higher levels, causes serious, perhaps irreversible deficits in biological functioning, cognition, and behavior, as supported by numerous studies.

The specific effects of lower levels of lead on children's development are somewhat less clear-cut. However, the data also seem to support that even low-level lead exposure causes cognitive and behavioral deficits. Apparently no level of lead in the body may be viewed as safe. But the need for further research is pressing. In particular, we must promote the use of good research design; animal models may continue to be helpful here.

Finally, the most urgent question is how to deal with the millions of homes and buildings in the U.S. that are contaminated by lead. In that current abatement procedures are both time-consuming and costly, and of questionable effectiveness, there is not going to be an easy solution to this problem. Other promising new avenues need further exploration.

It should be noted that it is possible that some effects of lead may be reversible. A recent study (Ruff, Bijur, Markowitz, Ma, & Rosen, 1994) demonstrated that lowering the lead level of lead-poisoned children resulted in a modest improvment in cognitive functioning. One must question, however, how much of the IQ gains in this study were due to practice effects.

The results of this study and other research conducted in the Needleman lab have been repeatedly questioned by Ernhart and Scarr. For a summary of this controversy, see Palca, 1991. In March 1994 the Office of Research Integrity, serving DHHS, cleared Needleman of any misconduct and ruled that although the research contained numerous errors and misstatements, these "did not necessarily alter the conclusion [of the Needleman 1979 article]" (Hilts, 1994, p. A22).

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