23. Screening for Elevated Lead Levels in Childhood and Pregnancy

RECOMMENDATION

Screening for elevated lead levels by measuring blood lead at least once at age 12 months is recommended for all children at increased risk of lead exposure. All children with identifiable risk factors should be screened, as should all children living in communities in which the prevalence of blood lead levels requiring individual intervention, including residential lead hazard control or chelation therapy, is high or is undefined (see ***Clinical Intervention***). Evidence is currently insufficient to recommend an exact community prevalence below which targeted screening can be substituted for universal screening. Clinicians can seek guidance from their local or state health department. There is insufficient evidence to recommend for or against routine screening for lead exposure in asymptomatic pregnant women, but recommendations against such screening may be made on other grounds. There is also insufficient evidence to recommend for or against counseling families about the primary prevention of lead exposure, but recommendations may be made on other grounds. Recommendations regarding the primary prevention of lead poisoning by population-wide environmental interventions are beyond the scope of this chapter.

Burden of Suffering

Prevalence. The prevalence of elevated blood lead levels in the U.S. population has declined 78% in the past decade,[1] due primarily to marked declines in lead in gasoline, soldered cans, and air.[1–6] In a 1988–1991 national survey of children aged 1–5 years, 9% and 0.5% had blood lead levels ≥10 µg/dL and ≥25 µg/dL, respectively, down from 88% and 9% a decade before.[7] (The units µg/dL will be used throughout this chapter; to convert to µmol/L, divide by 20.72.) Prevalence varies widely among different communities and populations, however, with studies reporting 2–41% of children having blood lead levels ≥10 µg/dL, 0–3% ≥25 µg/dL, and 0–0.5% ≥40 µg/dL.[8–19] Current national data for pregnant women

have not been published, but only 0.5% of U.S. women aged 12–49 years of age have blood lead levels ≥10 µg/dL.[7] Two large surveys of low-income pregnant women found 0%[20] and 6%[21] with blood lead levels >15 µg/dL.

Risk Factors for Elevated Lead Levels. The highest mean blood lead levels in the U.S. occur in children aged 1–2 years (mean 4.1 µg/dL) and in adults ≥50 years of age (4.0 µg/dL), with the lowest in adolescents (1.6 µg/dL).[7] Among adults, geometric mean levels are significantly higher in males than in females. Correlates of higher blood lead levels at all ages include minority race/ethnicity, central city residence, low income, low educational attainment, and residence in the Northeast region of the U.S.[7,22] These factors are associated with increased exposure to important lead sources, including dilapidated, pre-1950 housing with lead-based paint, lead-soldered pipes and household lead dust; and lead in dust and soil from heavy traffic and industry.[22–27] Other potential sources of household lead exposure include clothing or waste material brought home by workers in lead-based industries or hobbies, lead-based paint and dust contamination in pre-1950 housing that is undergoing remodeling or renovation, dietary intake from lead-soldered cans and lead-based pottery, and traditional ethnic remedies.[23,24,28]

Neurotoxic Effects of Lead Exposure in Children. Very high levels of inorganic lead exposure can produce serious neurologic complications, which may result in death or long-term sequelae.[23] A growing number of studies have reported associations between neurotoxic effects and blood lead levels once thought to be harmless. Adequately designed and conducted prospective cohort studies from a broad range of child populations have reported that a rise in blood lead from 10 to 20 µg/dL is associated with a likely decrement of about 2 points (reported range –6 to +1) in intelligence test scores (IQ).[29–35] In these studies, the mean blood lead levels at age 1–2 years (7.7–35.4 µg/dL) were higher than the current U.S. mean for this age group (4 µg/dL), but most levels were below 35 µg/dL. A meta-analysis[36] that included the five oldest of these cohort studies concluded that a doubling of blood lead levels from 10 to 20 µg/dL measured at age 2 years was associated with a statistically significant mean reduction of 1–2 IQ points; evidence was inconclusive regarding an association of IQ with mean postnatal blood lead levels. Although most cross-sectional studies evaluating the association of tooth and blood lead with IQ suffer from methodologic problems such as selection bias and limited adjustment for covariates, they have been generally consistent in reporting small negative effects of elevated lead levels on IQ.[e.g., in 36,37] A meta-analysis that included studies of whole tooth lead published since 1979 reported a statistically significant 1 point reduction in IQ associated with a doubling of tooth lead from 5 to 10 µg/g.[36] Evidence is not sufficient to quantify the exact nature

of the relationship between IQ and higher blood lead levels (i.e., 40–100 µg/dL). Cross-sectional studies[38–42] have consistently reported small, inverse associations between blood or tooth lead and reaction (attentional) performance, but studies evaluating the effect of mildly elevated lead levels on other measures of neurodevelopmental function (e.g., behavior, learning disorders, auditory function) have produced inconclusive results. These have been less thoroughly evaluated than IQ, however.

In most studies, the size of the estimates of lead effects on IQ are reduced when adjusted for potentially confounding variables,[36] suggesting that some of the observed association may be due to imperfectly measured or unmeasured covariates. Studies in rodents and primates, however, which can avoid most of the methodologic weaknesses of observational studies in humans, report cognitive, attentional, and behavioral deficits, as well as auditory and visual dysfunction, with mildly elevated blood lead levels,[43–45] supporting a causal relationship between low-level lead exposure and neurotoxic effects in children. Studies demonstrating laboratory abnormalities (e.g., impaired vitamin D metabolism) in persons with blood lead levels as low as 10–15 µg/dL[23,46–48] also support a causal relationship.

Adverse Effects of Lead Exposure on Pregnancy Outcomes. The effects of very high blood lead levels during pregnancy on reproductive outcomes such as abortion and stillbirth have been recognized for many years.[23] Observational studies in pregnant women with blood lead levels <30 µg/dL have reported associations between elevated levels and birth weight, length of gestation (including preterm delivery), and neonatal head circumference.[49–56] The associations have been small, variable in direction of effect, and not statistically significant in most studies. These studies failed to detect important effects on other reproductive outcomes. Inconsistent results may be due in part to imprecise measures of fetal lead exposure.[55–59] All but one[34] of six previously cited cohort studies,[29–34] as well as the meta-analysis described above,[36] reported no association between antenatal or perinatal maternal blood lead levels and full-scale IQ measured at preschool or school age. Although very high lead levels in pregnancy are clearly hazardous, the adverse effects on the fetus of antepartum lead levels in the range typically found in the U.S. are not established.

Other Adverse Effects of Lead Exposure. Lead exposure affects many organ systems, including cardiovascular, renal, and hepatic, but most clinically apparent (i.e., symptomatic) effects occur with blood lead levels ≥50 µg/dL.[23,60–63] Small increases in systolic blood pressure have been associated with mildly elevated blood lead levels (i.e., 1–3 mm Hg for a rise in blood lead from 10 to 20 µg/dL) in most large, population-based, cross-sectional studies evaluating nonpregnant adults and pregnant women.[64–70] In children, evidence of blood pressure effects is more limited: one cross-

sectional study found no association between elevated blood lead levels (range 7–70 μg/dL) and elevated blood pressure.[71] Adverse effects on height from lead levels well below 40 μg/dL have been suggested by analyses of national cross-sectional data,[72,73] but cohort studies with more extensive covariate adjustment report either transient or no effect of elevated lead levels (peak sample means 11–17 μg/dL) on growth.[35,74,75]

Accuracy of Screening Tests

Screening tests considered for detecting lead exposure include blood lead and free erythrocyte (or zinc) protoporphyrin levels. Blood lead concentration is the more sensitive of the two for detecting modest lead exposure, but its accuracy, precision and reliability can be affected by environmental lead contamination during blood collection, day-to-day biologic variability, and laboratory analytic variation. Lead contamination of collecting equipment, and skin contamination during capillary sampling, may each positively bias blood lead levels by up to 1.0 μg/dL, on average, although individual effects of skin contamination may be much greater.[76–80] Studies defining abnormal results as blood lead levels above 10 or 20 μg/dL have reported false-positive rates of 3–9% for capillary sampling compared to simultaneously collected venous blood lead.[77–78] Day-to-day biologic variability and trends over time contribute to higher false-positive rates for initial capillary samples when compared to results from venous testing done at a later date.[77,81] False-negative rates with capillary sampling appear to be lower, reported in one study as 1–8% compared to venous blood.[78] In published surveys,[76,82] 80–90% of clinical laboratories participating in proficiency testing programs met performance criteria for blood lead (within ±4 μg/dL of target values, for values <40 μg/dL[82]); unpublished national data show >95% of participating laboratories meeting these criteria and >80% achieving accuracy to within ±2 μg/dL of target values (unpublished data, Centers for Disease Control and Prevention, November 1993). Nonparticipating laboratories are likely to be less proficient. Reported blood lead values may differ by as much as 5 μg/dL from true values due to these sources of variability and bias, which may affect the predictive value of a positive test. Results from capillary samples may vary even more, although recent studies suggest the positive bias can be reduced with increased attention to reducing skin lead contamination.[77,78]

The erythrocyte protoporphyrin (EP) test, an indirect measure of lead exposure based on lead's effects on the hematopoietic system, is unaffected by contamination with environmental lead and is easily performed on capillary blood specimens, making it more acceptable for use with young patients. Erythrocyte (or zinc) protoporphyrin is insensitive, however, to modest elevations in blood lead levels.[21,83–89] The test also lacks

specificity,[21,83,84,86,87,90] thus limiting its predictive value. In one study, EP measurements were taken on 47,230 suburban and rural children; although 4.7% of the children had an elevated erythrocyte protoporphyrin level, only 0.6% had elevated blood lead levels.[91]

In communities where there is a low prevalence of lead levels requiring individual intervention with chelation or residential lead hazard control, blood lead screening will have a low yield, and many unaffected children will be tested at potentially high cost and inconvenience. A questionnaire that can predict those at high risk for elevated lead levels would allow targeted screening in low prevalence areas, increasing the yield of blood testing by increasing the pretest probability of elevated lead levels in those who are tested. Cross-sectional studies[13–15,92–93a] in urban and suburban, mostly midwestern, populations have shown that one or more positive responses to five questions (about exposures to deteriorated paint from older or renovated housing, to other lead-poisoned children, or to lead-related hobbies or industry)[128] detects 64–87% of children with blood lead levels ≥10 μg/dL. Three studies reported higher sensitivities (81–100%) for blood lead levels ≥15–20 μg/dL.[15,92,93a] None of these studies evaluated the ability of questionnaires to detect levels above 20 μg/dL, in part because so few patients had levels so high. Specificity among the studies ranged from 32% to 75%. In the samples with a lower prevalence (2–7%) of levels ≥10 μg/dL, the proportion of those with a negative questionnaire who had elevated blood lead levels was predictably low (0.2–3.5%), but increased to 19% when the population prevalence of elevated lead levels was higher (17–28%).

Effectiveness of Early Detection

Detection of lead exposure before the development of potentially irreversible complications permits the clinician to recommend environmental interventions to limit further exposure and, when necessary, to begin medical treatment with chelating agents. Early detection may also result in interventions that prevent exposure of other children to lead (the child with elevated blood lead level acting as a sentinel for a hazardous environment). There is relatively little convincing evidence that these interventions improve health, however. One issue is that most available studies in asymptomatic children evaluate the effects of various interventions on blood lead levels rather than on clinical outcomes. Second, blood lead levels typically decline with the passage of time. On average, blood lead levels in childhood decrease with age after peaking at about 2 years of age, even without intervention.[7] Longitudinal studies of asymptomatic children with elevated lead levels have shown reductions in blood lead levels after short- and long-term follow-up in the absence of any intervention,[94,95] a result at-

tributable at least in part to regression to the mean, random variation, and laboratory error. To evaluate adequately the effects of interventions on blood lead levels, studies must take into account these changes over time, preferably by the use of controls who do not receive the intervention.

Effect of Screening on Clinical Outcomes. Evidence is not available to demonstrate that universal screening for blood lead results in better clinical outcomes than either screening targeted to high-risk persons or individualized testing in response to clinical suspicion. Several older studies reported that, compared to historical results from individualized testing, intensive screening programs targeted to children in high-risk neighborhoods reduced case fatality rates, mortality rates, and proportions of children detected with very high blood lead levels or who developed symptomatic lead poisoning.[96–98] In the absence of concurrent controls, it is not clear whether the reported reductions in mortality and case fatality rates were due to screening, or to improvements in medical care over time. Reductions in mean lead levels may also have been due to secular trends, changes in screening tests, and to screening greater numbers of children, including many at low risk for severe lead poisoning. Thus, the available evidence regarding the efficacy of screening programs is weak.

Effect of Interventions to Lower Blood Lead Levels on Clinical Outcomes. In contrast to substantial evidence that chelating agents benefit children with symptomatic lead poisoning, few studies have compared potential clinical benefits of chelation therapy with its adverse effects in asymptomatic children. Ethical considerations preclude such trials for children with blood lead levels above 45 µg/dL. A large randomized controlled trial assessing the effect of chelation therapy on IQ in young children with venous blood lead concentrations of 20–45 µg/dL is currently under way (G. Rhoads, personal communication, Environmental and Occupational Health Sciences Institute, Piscataway, NJ, January 1994). An observational study[99,100] compared children with blood lead levels between 13 and 46 µg/dL (median 30 µg/dL), who did and did not receive EDTA chelation therapy depending on the results of a lead mobilization test. There was no effect of chelation on IQ at either 7 weeks or 6 months follow-up after controlling for age and initial IQ. Changes in concentrations of blood lead, bone lead, and EP also did not differ significantly between chelated and unchelated children. The greatest reductions in blood lead were associated with the highest initial lead levels, independent of chelation. The method of treatment assignment (i.e., based on a positive mobilization test) was most likely to have biased the study toward finding an effect of chelation, yet no effect was observed. There is thus little evidence presently available to confirm a clinical benefit from chelation therapy for children with lead levels <45 µg/dL. A comprehensive literature review found no studies evaluating

clinical effects of residential lead hazard control. Their effects on blood lead levels are reviewed below.

Effects of Chelation Therapy on Blood Lead Levels. In uncontrolled experiments and case series in asymptomatic children with initial blood lead levels ranging from 40 to 471 µg/dL, chelating agents reduced blood lead levels substantially, to levels <40–70 µg/dL (varying with initial levels); these reductions were maintained for weeks to years after therapy was discontinued.[101–105] Most of these children were also returned to homes that had undergone lead hazard reduction, and the effect of this additional intervention was not specifically evaluated. Chelating agents have caused short-term reductions in blood lead levels in children whose pretreatment values ranged from 20 to 49 µg/dL in nonrandomized comparative trials, cohort studies, and uncontrolled experiments; these reductions have not been sustained over longer periods in the absence of repeated or continuing chelation therapy or environmental interventions.[104,106–109] Most of these studies did not report whether chelation therapy was combined with environmental interventions. With such weak evidence, including the previously cited cohort study reporting no effects of chelation on IQ,[99] it is difficult to make a convincing argument that chelation therapy to lower moderately elevated blood lead levels has a long-term benefit.

Effect of Residential Lead Hazard Control on Blood Lead Levels. For most asymptomatic children with elevated lead levels, the primary goal of intervention is to reduce exposure to lead-contaminated paint, dust, and soil in the child's home environment, since these sources account for most excessive lead exposure. Residential lead-based paint hazard control methods have become increasingly effective for reducing exposure to lead paint and lead-contaminated dust.[25,110,111] These new techniques are now replacing the older strategies, which often created lead dust during the intervention process, but there are currently few published studies of their effect on blood lead levels.

Because most published studies used older, less effective techniques, the effects of residential interventions on blood lead reported in the literature, and outlined below, probably indicate the minimum possible benefit of residential lead-paint and lead-contaminated dust hazard control. In an early cohort study[112] of 184 children with initial blood lead levels ≥50 µg/dL, children discharged after chelation therapy to lead-free (i.e., new or completely gutted and renovated) housing had significantly lower mean blood lead levels when compared to children exposed to "legally abated" or to inadequately abated housing (28.8 µg/dL vs. 38.5 and 57 µg/dL, respectively). Children in lead-free housing also had fewer recurrences of levels ≥50 µg/dL at 12 and 24–30 months follow-up. A nonrandomized trial[113] of households with children having initial blood lead levels >29 µg/dL

compared more intensive experimental lead-reduction procedures with the lead-reduction procedures commonly in use in the study community. Neither intervention had any effect on mean dust or blood lead levels as tested 6 months after abatement, but an untreated control group was not included. Published[114] and unpublished[115] retrospective cohort studies suggest that residential lead paint hazard control is associated with modest declines (4–10 μg/dL) in mean blood lead levels in children with initial blood lead levels ≥ 25 μg/dL, although in one study[114] those with initial blood lead levels <35 μg/dL benefitted little from intervention. Case series and uncontrolled experiments, both weak study designs, have also evaluated lead-paint hazard control efforts in children with initial blood lead levels of 25–55 μg/dL;[100,116–119] several were published only as abstracts or summaries.[115,120] These studies reported statistically significant declines in mean blood lead levels, ranging from 2.5 to 10.2 μg/dL, 6–12 months after residential lead-based paint hazard control. All of the studies cited suffer from important design flaws, such as substantial drop-out rates and inadequate control for confounding variables such as season and age. Despite their flaws, the consistency of the results from these studies suggests a small, beneficial effect of lead-based paint hazard control on blood lead levels. As noted, there are as yet no published studies evaluating the effects on blood lead levels of newer residential lead hazard control techniques.

There are important problems with using one-time residential lead-paint hazard control as the sole method to reduce lead exposure in children.[121] Poor, inner-city families tend to move frequently, so that treating the current residence may have limited long-term benefit to the child, although benefit may accrue to other children moving in to that residence (see below). Residential lead-paint hazard control is costly and labor-intensive, resulting in low rates of intervention, especially in poor communities.[24,122] Lead dust is ubiquitous and highly mobile, so that recontamination by nearby lead sources, including soil lead, may occur after lead-paint hazard control efforts take place in a dwelling.[110,123,124] These problems indicate a need for additional individual interventions, as well as more comprehensive community-based interventions, to reduce household lead exposure.

The small effect noted in studies evaluating lead-paint hazard control methods may be attributable in part to recontamination of the dwelling by nearby lead sources and from subsequent deterioration of painted surfaces.[110,123,124] Several studies have evaluated measures designed to reduce ongoing lead-dust contamination from lead-contaminated paint and soil. In a nonrandomized controlled trial among children with blood lead levels of 30–49 μg/dL, having a research team wet-mop all lead-contaminated interior surfaces twice a month with a high-phosphate detergent cleanser resulted in significantly greater adjusted declines in mean blood lead lev-

els of children in intervention households compared to children in control households (6.9 vs. 0.7 μg/dL) at 1-year follow-up.[125] There have been no controlled studies to evaluate whether counseling families to perform similar cleaning would be equally effective in reducing blood lead levels. In one uncontrolled experiment, the families of 78 children with blood lead levels of 10–35 μg/dL, who were living in the vicinity of a defunct lead smelter, received intensive (30–45 minutes) in-home education and literature on prevention of lead exposure.[126] The mean blood lead levels in the 51 (65%) children who had follow-up blood lead levels at 4 months declined from 15.0 to 7.8 μg/dL (and maximum levels from 35.0 to 12.7 μg/dL). Without concurrent controls, it is not possible to determine how much regression to the mean and seasonal and age variations contributed to these reductions in blood lead levels. There is also evidence that clinician counseling at the worksite to reduce lead dust ingestion by workers (e.g., through personal hygiene practices) can significantly reduce mean blood lead levels at 1-year follow-up,[127] but this study also lacked controls and may not be generalizable to the residential setting.

A third focus of residential lead hazard control is exposure to soil lead. In a randomized controlled trial[123] of young children with initial blood lead levels of 7–24 μg/dL, extensive soil abatement, one-time dust abatement, and removal of loose interior paint resulted in a statistically significant reduction in mean blood lead levels of 1.2–1.3 μg/dL compared to loose paint removal alone. This clinically insignificant decline was associated with a substantial reduction in soil lead from a median 2,000 to 105 ppm. Preliminary results of the U.S. Environmental Protection Agency's Three City Urban Soil Lead Abatement Demonstration Project similarly suggest that substantial declines in soil lead cause only modest reductions in mildly elevated blood lead concentrations.[124] The small effect was due at least in part to rapid recontamination with dust lead in households undergoing soil abatement. Among children living near a closed lead smelter, only 3% of the variance in blood lead levels was attributable to soil lead.[127a]

An important potential benefit of residential lead hazard control is its effect on the lead levels or clinical outcomes of other children who live in the same household as a child identified with elevated lead levels, or who subsequently move into the remediated residence. The literature review revealed no published evidence evaluating the effect of residential lead hazard control measures on such children. Based on the biokinetics of lead,[23] it is reasonable to believe that environmental interventions conducted before children are exposed are likely to prevent increases in blood lead levels more effectively than the same interventions in children who have already been exposed.

Effect of Nutritional Interventions on Blood Lead Levels. In most settings, neither residential lead-based paint or dust hazard control nor chelation therapy is routinely offered to children with blood lead levels <20 μg/dL, but some experts have recommended offering these children dietary counseling to reduce their blood lead levels.[128] Diets deficient in calories, calcium, and zinc have been associated with increased gastrointestinal absorption of lead,[129,130] but there is only limited evidence that counseling to correct such nutritional inadequacies will reduce blood lead levels or prevent further increases. Results of experimental studies of the effects of iron deficiency on lead absorption and retention in adult humans have been equivocal.[129,131] In a cohort study of children with initial blood lead levels of 13–46 μg/dL,[99] all children who were iron deficient or depleted were prescribed iron supplementation. Although most children were still iron deficient at the end of the study, there were improvements in ferritin level that were not associated with either declines in blood lead or improvements in cognitive function. Cross-sectional and cohort studies have failed to establish a clear association between mean blood lead levels and measures of iron status in women at midpregnancy or delivery, in newborns (cord blood), or in children.[57,99,131–134]

Adverse Effects of Screening and Intervention. The most common adverse effects of screening for elevated lead levels are false-positive fingerstick results, and the anxiety, inconvenience, work or school absenteeism, and financial costs associated with return visits and repeat tests. An EDTA lead mobilization test, used for some children with blood lead levels of 30–44 μg/dL,[135] requires intramuscular or intravenous infusion, a stay at the clinical center for at least 8 hours, and for young children, application of urine collection bags.[136] Residential lead-based paint and dust hazard control, when improperly done,[25] may produce acute increases in blood lead levels in resident children and abatement workers, occasionally necessitating hospitalization and chelation therapy.[113,116,137–139] Currently recommended techniques for lead hazard reduction are likely to reduce these adverse effects.[25] Chelating agents for asymptomatic lead poisoning have also been associated with important adverse effects. EDTA and dimercaprol (BAL) have transient renal, hepatic, and other toxicity, require intravenous or intramuscular injection, and generally require hospitalization for administration.[128,140,141] Common adverse effects of d-penicillamine are penicillin-like sensitivity reactions and transient nephrotoxicity; there are rare life-threatening reactions.[96,105,107,128] Succimer (meso-2,3-dimercaptosuccinic acid, or DMSA) causes mild gastrointestinal and systemic symptoms, rashes, and transient elevations in liver function tests, in up to 10% of cases.[104,106,108,142]

Recommendations of Other Groups

Several states mandate either universal screening for lead exposure or selective screening of populations at high risk for lead exposure.[143] Periodic screening of children with blood lead measurement is also required for Medicaid's Early and Periodic Screening, Diagnostic, and Treatment Program.[144] The American Academy of Pediatrics[145] and the Bright Futures guidelines[146] recommend: *(a)* screen all children for lead exposure at about 12 months of age, and possibly again at about 24 months of age; *(b)* take a history of lead exposure (using questionnaires provided with the guidelines) between the ages of 6 months and 6 years to identify high-risk children who should be screened earlier or more frequently; and *(c)* provide education to parents on safe environmental, occupational, nutritional and hygiene practices to protect their children from lead exposure. Follow-up screening intervals should be based on risk assessment and previous blood lead levels. The Centers for Disease Control and Prevention (CDC) recommends screening all children at 12 months of age using a blood lead test, except in communities where no childhood lead poisoning problem exists; high-risk children require earlier and more frequent screening.[128] The American Academy of Family Physicians (AAFP)[147] and the Canadian Task Force on the Periodic Health Examination[148] recommend screening all children who are at high risk of lead exposure (e.g., due to exposure to heavy traffic and industry, or to dilapidated older housing). The recommendations of the AAFP are currently under review. The American Medical Association recommends regularly screening all children under the age of 6 years for lead exposure through history-taking and, when appropriate, blood lead testing.[149] They recommend that the decision to employ universal or targeted screening be made based on prevalence studies of blood lead levels in the local pediatric population.

No major organizations currently recommend screening pregnant women for elevated lead levels.

Discussion

There is fair evidence that screening for elevated lead levels in asymptomatic children at increased risk for lead exposure will improve clinical outcomes. Because there have been no controlled trials directly evaluating screening for elevated lead levels, this conclusion is based on a chain of evidence constructed from studies of weaker design. First, in young asymptomatic children, blood lead levels as low as 10 μg/dL are associated with measurable neurodevelopmental dysfunction. Second, although the national prevalence of elevated lead levels has declined substantially in the past decade, a high prevalence persists in some communities, particularly

poor urban communities in the northeastern U.S. Third, measurement of venous blood lead concentration is a convenient, reliable, precise and reasonably valid screening test for assessing lead exposure. Fourth, current interventions, including residential lead hazard control and chelation therapy, can reduce blood lead levels in children identified with levels ≥25 μg/dL, although the quality of evidence supporting their effectiveness is weak and a beneficial effect on IQ or other clinical outcomes has not yet been demonstrated. There is also weak evidence that screening high-risk children for elevated lead levels results in improved clinical outcome compared to historical controls identified by case-finding. Based on this evidence of the current burden of suffering and the effectiveness of early detection, the Task Force recommends screening children at increased risk for lead exposure.

While no studies have evaluated a specific age at which to screen, the natural history of blood lead levels in children, which increase most rapidly between 6 and 12 months and peak at age 18–24 months, suggests that screening at about 12 months of age is likely to be most effective for the early detection of elevated lead levels.

For those children who are screened and found to have initial blood lead levels <25 μg/dL, there is as yet little evidence regarding the effectiveness of early detection and intervention, or of repeated screening to detect further increases in blood lead. Longitudinal and cross-sectional studies suggest that in children ≥2 years, most such levels will decline naturally with time, but elevated levels may persist in children who are chronically exposed.[101]

There is no direct evidence comparing the outcomes of universal screening with the outcomes from targeted screening for elevated lead levels. Recent studies indicate that the prevalence of elevated blood levels in the U.S. has declined dramatically in the past decade, but that local prevalence is highly variable, with more than 10-fold differences between communities. In a community with a low prevalence of elevated blood lead levels, universal screening may result in disproportionate risks and costs relative to benefits. The prevalence level at which targeted screening can replace universal screening is a public health policy decision requiring consideration of factors in addition to the scientific evidence for effectiveness of early detection, such as available resources, competing public health needs, and costs and availability of alternative approaches to reducing lead exposure. Good quality analyses are needed to determine the population prevalence below which universal lead screening is not cost-effective. Clinicians can consult with their local or state health department regarding appropriate screening policy for the local child population.

In communities where data suggest that universal screening is not indicated, there may nevertheless be some children who are at increased risk

of blood lead levels in the range for which individual intervention by chelation therapy or residential lead hazard control has been demonstrated to be effective. These children may have had exposure to lead sources such as lead-based hobbies or industries, traditional ethnic remedies, or lead-based pottery. Selective blood lead screening of such high-risk children is appropriate even in low prevalence communities. There is fair evidence that a validated questionnaire of known and acceptable sensitivity and specificity can identify those at high risk. In several studies, the CDC[128] and similar questionnaires correctly identified 64% to 87% of urban and suburban children who had blood lead levels ≥10 μg/dL. These questionnaires have not been adequately evaluated as a screening tool to detect higher blood lead levels (e.g., ≥20–25 μg/dL), or to detect exposure in other populations (e.g., migrant workers, rural communities). Locale-specific questionnaires that inquire about likely local sources of lead exposure may lead to improved prediction.

As is the case in children, there are no controlled trials evaluating screening for elevated lead levels in pregnant women, nor are there sufficient data to construct an adequate chain of evidence demonstrating benefit. The prevalence of levels >15 μg/dL appears to be quite low in pregnant women. There is fair evidence that mildly elevated lead levels during pregnancy are associated with small increases in antepartum blood pressure, but limited evidence that these levels have important adverse effects on reproductive or other outcomes, including intelligence of offspring. An extensive literature search failed to identify studies evaluating screening or intervention for lead exposure in pregnant women. There are potentially important adverse effects of chelation therapy on the fetus, and of residential lead hazard control on both the pregnant woman and fetus if they are not performed according to established standards. Removal to a lead-free environment would theoretically be effective in reducing lead exposure but has not been specifically evaluated in pregnancy. There is thus insufficient evidence to recommend for or against screening pregnant women for the detection of elevated lead levels.

Population-based interventions for the primary prevention of lead exposure are likely to be more effective, and may be more cost-effective, than office-based screening, treatment and counseling. Community, regional, and national environmental lead hazard reduction efforts, such as reducing lead in industrial emissions, gasoline, and cans, have proven highly effective in reducing population blood lead levels.[1–6,150,151] Remaining important sources of lead (e.g., lead paint and pipes in older homes, lead-contaminated soil) are, however, more difficult to address on a population-wide basis. Studies of community-based efforts to reduce lead exposure from these and other sources in order to prevent the occurrence of elevated lead levels are ongoing.[25,110,152] Evaluation of the effectiveness

of community-based interventions, and recommendations regarding their use, are beyond the scope of this document.

CLINICAL INTERVENTION

Screening for elevated lead levels by measuring blood lead at least once at age 12 months is recommended for all children at increased risk of lead exposure ("B" recommendation). All children with identifiable risk factors should be screened, as should children living in communities in which the prevalence of blood lead levels requiring individual intervention, including chelation therapy or residential lead hazard control, is high or is undefined. If capillary blood is used, elevated lead levels should be confirmed by measurement of venous blood lead. The optimal frequency of screening for lead exposure in children, or for repeated testing of children previously found to have elevated blood lead levels, is unknown and is left to clinical discretion; consideration should be given to the degree of elevation, the interventions provided, and the natural history of lead exposure, including the typical peak in lead levels at 18–24 months of age.

In communities where the prevalence of blood lead levels requiring individual intervention is low, a strategy of targeted screening, possibly using locale-specific questionnaires of known and acceptable sensitivity and specificity, can be used to identify high-risk children who should have blood lead testing. Examples of individual risk factors include: *(a)* living in or frequently visiting an older home (built before 1950) with dilapidated paint or with recent or ongoing renovation or remodeling, *(b)* having close contact with a person who has an elevated lead level, *(c)* living near lead industry or heavy traffic, *(d)* living with someone whose job or hobby involves lead exposure, *(e)* using lead-based pottery, or *(f)* taking traditional ethnic remedies that contain lead.[128] There is currently insufficient evidence to recommend an exact population prevalence below which targeted screening can be substituted for universal screening. The results of cost-benefit analyses, available resources and public health priorities are among the determinants of the prevalence below which targeted screening is recommended for a community. Clinicians can seek guidance from their local or state health department.

There is insufficient evidence to recommend for or against routine screening for lead exposure in asymptomatic pregnant women ("C" recommendation). Recommendations against such screening may be made on the grounds of limited and conflicting evidence regarding the current burden of suffering, high costs, and the potential for adverse effects from intervention.

There is insufficient evidence to recommend for or against trying to prevent lead exposure by counseling families to control lead dust by re-

peated household cleaning, or to optimize caloric, iron, and calcium intake specifically to reduce lead absorption ("C" recommendation). For high-risk individuals or those living in high-prevalence communities, such recommendations may be made on other grounds, including minimal risk of adverse effects from the cleaning or the dietary advice, and the additional, unrelated benefits from optimizing nutrition (see Chapter 22, Screening for Iron Deficiency Anemia, and Chapter 56, Counseling to Promote a Healthy Diet).

Recommendations regarding community- or population-based interventions for the primary prevention of lead poisoning, assessment of community lead contamination, or the setting of community priorities for lead hazard reduction, are beyond the scope of this document.

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