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Lead Screening During Pregnancy and Lactation

Abstract: Prenatal lead exposure has known adverse effects on maternal health and infant outcomes across a wide range of maternal blood lead levels. Adverse effects of lead exposure are being identified at lower levels of exposure than previously recognized in both children and adults. In 2010, the Centers for Disease Control and Prevention issued the first guidelines regarding the screening and management of pregnant and lactating women who have been exposed to lead.

Prenatal lead exposure has known adverse effects on maternal health and infant outcomes across a wide range of maternal blood lead levels (1). Adverse effects of lead exposure are being identified at lower levels of exposure than previously recognized in both children and adults (2–7). In 2010, the Centers for Disease Control and Prevention issued the first guidelines regarding the screening and management of pregnant and lactating women who have been exposed to lead (8).

Background

Environmental policies and public health education programs have led to significant reductions in cases of lead exposure in the United States (9). Despite these improvements, approximately 1% of women of childbearing age (15–49 years) have blood lead levels greater than or equal to 5 micrograms/dL (8).

Although no threshold has been found to trigger the adverse health effects of lead (8), in nonpregnant adults blood lead levels less than 5 micrograms/dL are considered normal, blood lead levels between 5 micrograms/dL and 10 micrograms/dL require follow-up, and blood lead levels greater than 10 micrograms/dL are managed with environmental assessment and abatement of exposures. Chelation therapy is considered at blood lead levels greater than 40 micrograms/dL for symptomatic individuals, and levels greater than 70 micrograms/dL are considered a medical emergency. In children, treatment is recommended at blood lead levels of 45 micrograms/dL or greater.

The main target for lead toxicity is the nervous system, both in adults and children (10). High levels of

exposure can result in delirium, seizures, stupor, coma, or even death. Other overt signs and symptoms of lead toxicity may include hypertension, peripheral neuropathy, ataxia, tremor, headache, loss of appetite, weight loss, fatigue, muscle and joint aches, changes in behavior and concentration, gout, nephropathy, lead colic, and anemia. Health effects of chronic low-level exposure in adults include cognitive decline, hypertension and other cardiovascular effects, decrements in renal function, and adverse reproductive outcome. The developing nervous systems in children make them more susceptible to the neurologic effects of lead toxicity.

Adverse Health Effects of Prenatal Exposure

Lead readily crosses the placenta by passive diffusion and has been detected in the fetal brain as early as the end of the first trimester (8). Elevated lead levels in pregnancy have been associated with several adverse outcomes, including gestational hypertension, spontaneous abortion, low birth weight, and impaired neurodevelopment (11–14).

Lead exposure has been associated with an increased risk of gestational hypertension, but the magnitude of the effect, the exposure level at which risk begins to increase, and whether risk is most associated with acute or cumulative exposure remain uncertain. Also, it is unclear whether lead-induced increases in blood pressure during pregnancy lead to severe hypertension or preeclampsia (11, 15–18).

Evidence shows that maternal exposure to high levels of lead increases the risk of spontaneous abortion (19).

However, data for an association between low or moderate lead levels and spontaneous abortion are inconsistent. The strongest available evidence comes from a prospective study of 668 pregnant women in Mexico City that demonstrated a statistically significant dose–response relationship between low-to-moderate maternal blood lead levels and the risk of spontaneous abortion (12). Yet, another longitudinal study of 351 women in Japan showed no difference in blood lead levels between spontaneous abortion cases (n=15) and ongoing pregnancies (20). Larger prospective studies are needed to further clarify the effects of low and moderate levels of lead on spontaneous abortion risk.

More recent and well-designed studies suggest that maternal lead exposure during pregnancy is inversely related to fetal growth, as reflected by duration of pregnancy and infant size. One study that used a registry-based approach found that offspring of mothers occupationally exposed to lead had an increased risk of low birth weight (relative risk [RR], 1.34; confidence interval [CI], 1.12–1.6) compared with infants of women not exposed to lead (13). A case–control study in Mexico City found umbilical cord blood lead levels to be higher in preterm infants (mean value, 9.8 micrograms/dL) compared with term infants (mean value, 8.4 micrograms/dL) (21). A birth cohort study, also conducted in Mexico City, found maternal bone lead burden to be inversely related to offspring weight (22), length, and head circumference at birth (23).

A large number of studies provide evidence that prenatal lead exposure impairs children’s neurodevelopment. Some prospective studies have included children with low levels of prenatal lead exposure and continue to detect inverse associations with neurodevelopment, although these data are less consistent than those related to the high levels of lead exposure. In one study, each 1 microgram/dL increase in umbilical cord blood lead was found to be associated with a reduction of 0.6 points in the mental development index scores of the Bayley Scales of Infant Development at age 3 months, with similar results at age 6 months (14, 24). However, another prospective cohort study found that the relationship between prenatal blood lead levels and early childhood IQ is not linear, with the strongest postnatal effects noted at low levels of prenatal exposure (25). The available data are inadequate to establish the presence or absence of an association between maternal lead exposure and major congenital anomalies in the fetus.

Lead Exposure During Breastfeeding

Although the benefits of breastfeeding generally outweigh the risks of environmental exposure, the effects of breastfeeding on infant lead levels have been studied. Lead has been detected in the breast milk of women in population-based studies; however, the availability of high-quality data to assess the risk for toxicity to the breastfeeding infant is limited (8). Although infant blood lead levels

have been correlated with the duration of breastfeeding (26), the ratio of breast milk lead levels to blood lead levels has been found to be less than 3% (27). According to the American Academy of Pediatrics, because of the contribution of lead levels found in infant formula and other infant foods, breastfed infants of mothers with normal blood lead levels are actually exposed to slightly less lead than if they were not breastfed (28).

Screening and Management

Pregnancy

The Centers for Disease Control and Prevention (CDC) and the American College of Obstetricians and Gynecologists do not recommend blood lead testing of all pregnant women in the United States. Obstetric health care providers should consider the possibility of lead exposure in individual pregnant women by evaluating risk factors for exposure as part of a comprehensive health risk assessment and perform blood lead testing if a single risk factor is identified. Assessment of lead exposure should take place at the earliest contact with the pregnant patient.

Important risk factors for lead exposure in pregnant women are listed in [Box 1](#). Lead-based paint is less likely to be an important exposure source for pregnant women than it is for children, except during renovation or remodeling in older homes. Women should take precautions when repainting surfaces with deteriorated paint or performing any remodeling or renovation work that disturbs painted surfaces, such as scraping off paint or tearing out walls (8).

For pregnant women with blood lead levels of 5 micrograms/dL or higher, sources of lead exposure should be identified and women should receive counseling regarding avoidance of further exposure. Confirmatory and follow-up blood lead testing should be performed in accordance with the CDC’s recommended schedules ([Table 1](#)) and maternal or umbilical cord blood lead levels should be measured at delivery (8). Women with confirmed blood lead levels of 45 micrograms/dL or more should be treated in consultation with clinicians experienced in the management of lead toxicity and high-risk pregnancy. Once the source of lead exposure is identified and eliminated, the initial decrease in blood lead level occurs relatively rapidly because of lead’s short (35-day) initial half-life in blood (29). This initial rapid decrease is followed by a slow, continuous decrease over several months to several years because of mobilization of lead from stores in the bone (8). Recommendations for the frequency of blood lead follow-up tests are included in [Table 1](#).

Adequate dietary intake of calcium, iron, zinc, vitamin C, vitamin D, and vitamin E is known to decrease lead absorption (30, 31). Iron-deficiency anemia is associated with elevated blood lead levels and may increase lead absorption. During pregnancy and lactation, lead from prior exposures can be mobilized from bones because

Box 1. Risk Factors for Lead Exposure in Pregnant and Lactating Women ↵

- Recent emigration from or residency in areas where ambient lead contamination is high—women from countries where leaded gasoline is still being used (or was recently phased out) or where industrial emissions are not well controlled.
- Living near a point source of lead—examples include lead mines, smelters, or battery recycling plants (even if the establishment is closed).
- Working with lead or living with someone who does—women who work in or who have family members who work in an industry that uses lead (eg, lead production, battery manufacturing, paint manufacturing, ship building, ammunition production, or plastic manufacturing).
- Using lead-glazed ceramic pottery—women who cook, store, or serve food in lead-glazed ceramic pottery made in a traditional process and usually imported by individuals outside the normal commercial channels.
- Eating nonfood substances (pica)—women who eat or mouth nonfood items that may be contaminated with lead, such as soil or lead-glazed ceramic pottery.
- Using alternative or complementary substances, herbs, or therapies—women who use imported home remedies or certain therapeutic herbs traditionally used by East Indian, Indian, Middle Eastern, West Asian, and Hispanic cultures that may be contaminated with lead.
- Using imported cosmetics or certain food products—women who use imported cosmetics, such as kohl or surma or certain imported foods or spices that may be contaminated with lead.
- Engaging in certain high-risk hobbies or recreational activities—women who engage in high-risk activities (eg, stained glass production or pottery making with certain leaded glazes and paints) or have family members who do.
- Renovating or remodeling older homes without lead hazard controls in place—women who have been disturbing lead paint, creating lead dust, or both or have been spending time in such a home environment.
- Consumption of lead-contaminated drinking water—women whose homes have leaded pipes or source lines with lead.
- Having a history of previous lead exposure or evidence of elevated body burden of lead—women who may have high body burdens of lead from past exposure, particularly those who have deficiencies in certain key nutrients (calcium or iron).
- Living with someone identified with an elevated lead level—women who may have exposure in common with a child, close friend, or other relative living in the same environment.

Modified from Centers for Disease Control and Prevention. Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta (GA): CDC; 2010. Available at: <http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf>. Retrieved March 7, 2012.

Table 1. Frequency of Maternal Blood Lead Follow-up Testing During Pregnancy ↵

Venous Blood Lead Level*	
(micrograms/dL)	Perform Follow-up Test(s) [†]
Less than 5	• None (no follow-up testing is indicated)
5–14	• Within 1 month • Obtain a maternal blood lead level [‡] or cord blood lead level at delivery
15–24	• Within 1 month and then every 2–3 months • Obtain a maternal blood lead level [‡] or cord blood lead level at delivery • More frequent testing may be indicated based on risk factors
25–44	• Within 1–4 weeks and then every month • Obtain a maternal blood lead level [‡] or cord blood lead level at delivery
45 or more	• Within 24 hours and then at frequent intervals depending on clinical interventions and trend in blood lead levels • Consultation with a clinician experienced in the management of pregnant women with blood lead levels in this range is strongly advised • Obtain a maternal blood lead level or cord blood lead level at delivery

*Venous blood sample is recommended for maternal blood lead testing.

[†]The higher the blood lead level on the screening test, the more urgent the need for confirmatory testing.

[‡]If possible, obtain a maternal blood lead level before delivery because blood lead levels tend to increase over the course of pregnancy.

Modified from Centers for Disease Control and Prevention. Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta (GA): CDC; 2010. Available at: <http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf>. Retrieved March 7, 2012.

of the increased bone turnover. Pregnant and lactating women with a current or past blood lead level of 5 micrograms/dL or higher should receive specific nutritional recommendations regarding calcium and iron supplementation. A balanced diet that contains 2,000 mg of calcium and 60–120 mg of iron daily is recommended (8). This can be achieved through either food intake or supplementation. Supplements should be divided into doses of 500 mg of calcium and 60 mg of iron to improve absorption.

Lactation

Women with risk factors for elevated lead levels (Box 1) who have not been screened during pregnancy should be screened postpartum if they plan to breastfeed. Initiation of breastfeeding should be encouraged postpartum in a woman with a blood lead level less than 40 micrograms/dL.

A woman with a confirmed blood lead level of 40 micrograms/dL or higher should not initiate breastfeeding and should be advised to pump and discard her breast milk until her blood lead level has decreased to less than 40 micrograms/dL. Blood lead measurements should be repeated every 1–2 weeks after the source of exposure has been identified and removed. At maternal blood lead levels of 5–39 micrograms/dL, breastfeeding should be initiated and accompanied by sequential testing of infant blood lead levels to monitor trends. If the infant’s blood lead level is greater than 5 micrograms/dL, breastfeeding should be discontinued until the maternal blood lead level decreases. If no external source is identified, and the maternal blood lead level is greater than 20 micrograms/dL and the infant blood lead level is 5 micrograms/dL or more, breast milk should be suspected as the source and temporary interruption of breastfeeding until the maternal blood lead level decreases should be considered.

In addition to removing the source of lead exposure for the mother and infant, several nutritional interventions have been studied. Calcium supplementation (1,200 mg daily) has been associated with a 5–10% decrease in breast milk lead levels among women over the course of lactation (31–34), suggesting that calcium supplementation also may be an intervention strategy to reduce lead in breast milk from both current and previously accumulated sources. Among women in the postpartum period, increased intakes of vitamin C also have been associated with decreased levels of lead in breast milk.

Conclusions and Recommendations

- Routine blood lead testing of all pregnant women is not recommended.
- Risk assessment of lead exposure should take place at the earliest contact with pregnant or lactating women, and blood lead testing should be performed if a single risk factor is identified (Box 1).
- Elevated lead levels in pregnancy have been associated with gestational hypertension, spontaneous abortion, low birth weight, and impaired neurodevelopment.
- Prenatal lead exposure has known adverse effects on maternal health and infant outcomes across a wide range of maternal blood lead levels.
- Pregnant women with blood lead levels of 5 micrograms/dL or higher should be treated as follows:
 - Sources of lead exposure should be identified.
 - Women should receive counseling regarding avoidance of further exposure and receive specific nutritional recommendations regarding calcium and iron supplementation because these strategies can decrease their lead levels.
 - Confirmatory and follow-up blood lead testing should be performed in accordance with the CDC’s recommended schedules (Table 1).

- Women with confirmed blood lead levels of 45 micrograms/dL or more should be treated in consultation with clinicians experienced in the management of lead toxicity and high-risk pregnancy.
- Initiation of breastfeeding should be encouraged postpartum in a woman with a blood lead level less than 40 micrograms/dL.
- A breastfeeding woman with a confirmed blood lead level of 40 micrograms/dL or higher should be advised to pump and discard her breast milk until her blood lead level has decreased to less than 40 micrograms/dL.
- If no external source is identified, and the maternal blood lead level is greater than 20 micrograms/dL and the infant blood lead level is 5 micrograms/dL or more, breast milk should be suspected as the source and temporary interruption of breastfeeding until the maternal blood lead level decreases should be considered.

References

1. Bellinger DC. Teratogen update: lead and pregnancy. *Birth Defects Res A Clin Mol Teratol* 2005;73:409–20. [PubMed] [Full Text] ↩
2. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *N Engl J Med* 2003;348:1517–26. [PubMed] [Full Text] ↩
3. Jusko TA, Henderson CR, Lanphear BP, Cory-Slechta DA, Parsons PJ, Canfield RL. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. *Environ Health Perspect* 2008;116:243–8. [PubMed] [Full Text] ↩
4. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. Low-level environmental lead exposure and children’s intellectual function: an international pooled analysis. *Environ Health Perspect* 2005;113:894–9. [PubMed] [Full Text] ↩
5. Menke A, Muntner P, Batuman V, Silbergeld EK, Guallar E. Blood lead below 0.48 micromol/L (10 microg/dL) and mortality among US adults. *Circulation* 2006;114:1388–94. [PubMed] [Full Text] ↩
6. Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ. Lead exposure and cardiovascular disease--a systematic review. *Environ Health Perspect* 2007;115:472–82. [PubMed] [Full Text] ↩
7. Tellez-Rojo MM, Bellinger DC, Arroyo-Quiroz C, Lamadrid-Figueroa H, Mercado-Garcia A, Schnaas-Arrieta L, et al. Longitudinal associations between blood lead concentrations lower than 10 microg/dL and neurobehavioral development in environmentally exposed children in Mexico City. *Pediatrics* 2006;118:e323–30. [PubMed] [Full Text] ↩
8. Centers for Disease Control and Prevention. Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta (GA): CDC; 2010. Available at: <http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf>. Retrieved March 7, 2012. ↩

9. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, et al. The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *JAMA* 1994;272:284–91. [PubMed] ↩
10. Agency for Toxic Substances and Disease Registry. Toxicological profile for lead. Atlanta (GA): ATSDR; 2007. Available at: <http://www.atsdr.cdc.gov/ToxProfiles/tp13.pdf>. Retrieved March 7, 2012. ↩
11. Rabinowitz M, Bellinger D, Leviton A, Needleman H, Schoenbaum S. Pregnancy hypertension, blood pressure during labor, and blood lead levels. *Hypertension* 1987; 10:447–51. [PubMed] [Full Text] ↩
12. Borja-Aburto VH, Hertz-Picciotto I, Rojas Lopez M, Farias P, Rios C, Blanco J. Blood lead levels measured prospectively and risk of spontaneous abortion. *Am J Epidemiol* 1999;150:590–7. [PubMed] [Full Text] ↩
13. Irgens A, Kruger K, Skorve AH, Irgens LM. Reproductive outcome in offspring of parents occupationally exposed to lead in Norway. *Am J Ind Med* 1998;34:431–7. [PubMed] ↩
14. Dietrich KN, Krafft KM, Bornschein RL, Hammond PB, Berger O, Succop PA, et al. Low-level fetal lead exposure effect on neurobehavioral development in early infancy. *Pediatrics* 1987;80:721–30. [PubMed] ↩
15. Rothenberg SJ, Manalo M, Jiang J, Cuellar R, Reyes S, Sanchez M, et al. Blood lead level and blood pressure during pregnancy in South Central Los Angeles. *Arch Environ Health* 1999;54:382–9. [PubMed] ↩
16. Magri J, Sammut M, Savona-Ventura C. Lead and other metals in gestational hypertension. *Int J Gynaecol Obstet* 2003;83:29–36. [PubMed] [Full Text] ↩
17. Vigh M, Yokoyama K, Mazaheri M, Beheshti S, Ghazizadeh S, Sakai T, et al. Relationship between increased blood lead and pregnancy hypertension in women without occupational lead exposure in Tehran, Iran. *Arch Environ Health* 2004;59:70–5. [PubMed] ↩
18. Yazbeck C, Thiebaugeorges O, Moreau T, Goua V, Debotte G, Sahuquillo J, et al. Maternal blood lead levels and the risk of pregnancy-induced hypertension: the EDEN cohort study. *Environ Health Perspect* 2009;117:1526–30. [PubMed] [Full Text] ↩
19. Hertz-Picciotto I. The evidence that lead increases the risk for spontaneous abortion. *Am J Ind Med* 2000;38:300–9. [PubMed] ↩
20. Vigh M, Yokoyama K, Kitamura F, Afshinrokh M, Beygi A, Niroomanesh S. Early pregnancy blood lead and spontaneous abortion. *Women Health* 2010;50:756–66. [PubMed] [Full Text] ↩
21. Torres-Sanchez LE, Berkowitz G, Lopez-Carrillo L, Torres-Arreola L, Rios C, Lopez-Cervantes M. Intrauterine lead exposure and preterm birth. *Environ Res* 1999;81:297–301. [PubMed] ↩
22. Gonzalez-Cossio T, Peterson KE, Sanin LH, Fishbein E, Palazuelos E, Aro A, et al. Decrease in birth weight in relation to maternal bone-lead burden. *Pediatrics* 1997;100:856–62. [PubMed] ↩
23. Hernandez-Avila M, Peterson KE, Gonzalez-Cossio T, Sanin LH, Aro A, Schnaas L, et al. Effect of maternal bone lead on length and head circumference of newborns and 1-month-old infants. *Arch Environ Health* 2002;57:482–8. [PubMed] ↩
24. Dietrich KN, Krafft KM, Shukla R, Bornschein RL, Succop PA. The neurobehavioral effects of early lead exposure. *Monogr Am Assoc Ment Defic* 1987;8:71–95. [PubMed] ↩
25. Wasserman GA, Liu X, Popovac D, Factor-Litvak P, Kline J, Waternaux C, et al. The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. *Neurotoxicol Teratol* 2000;22:811–8. [PubMed] ↩
26. Lozoff B, Jimenez E, Wolf AW, Angelilli ML, Zatakia J, Jacobson SW, et al. Higher infant blood lead levels with longer duration of breastfeeding. *J Pediatr* 2009;155:663–7. [PubMed] [Full Text] ↩
27. Gulson BL, Yui LA, Howarth D. Delayed visual maturation and lead pollution. *Sci Total Environ* 1998;224:215–9. [PubMed] ↩
28. Lead exposure in children: prevention, detection, and management. American Academy of Pediatrics Committee on Environmental Health. *Pediatrics* 2005;116:1036–46. [PubMed] [Full Text] ↩
29. Rabinowitz MB, Wetherill GW, Kopple JD. Kinetic analysis of lead metabolism in healthy humans. *J Clin Invest* 1976; 58:260–70. [PubMed] [Full Text] ↩
30. Mahaffey KR. Environmental lead toxicity: nutrition as a component of intervention. *Environ Health Perspect* 1990; 89:75–8. [PubMed] [Full Text] ↩
31. Gulson BL, Mizon KJ, Palmer JM, Korsch MJ, Taylor AJ, Mahaffey KR. Blood lead changes during pregnancy and postpartum with calcium supplementation. *Environ Health Perspect* 2004;112:1499–507. [PubMed] [Full Text] ↩
32. Ettinger AS, Tellez-Rojo MM, Amarasiriwardena C, Peterson KE, Schwartz J, Aro A, et al. Influence of maternal bone lead burden and calcium intake on levels of lead in breast milk over the course of lactation. *Am J Epidemiol* 2006;163:48–56. [PubMed] ↩
33. Ettinger AS, Hu H, Hernandez-Avila M. Dietary calcium supplementation to lower blood lead levels in pregnancy and lactation. *J Nutr Biochem* 2007;18:172–8. [PubMed] [Full Text] ↩
34. Ettinger AS, Lamadrid-Figueroa H, Tellez-Rojo MM, Mercado-Garcia A, Peterson KE, Schwartz J, et al. Effect of calcium supplementation on blood lead levels in pregnancy: a randomized placebo-controlled trial. *Environ Health Perspect* 2009;117:26–31. [PubMed] [Full Text] ↩

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