THE
MILBANK QUARTERLY
A MULTIDISCIPLINARY JOURNAL OF POPULATION HEALTH AND HEALTH POLICY

Perspective

Improving Equitability and Inclusion for Testing and Detection of Lead Poisoning in US Children

CHRISTINA SOBIN,*
MARISELA GUTIéRREZ-VEGA,†
GISEL FLORES-MONTOYA,‡ MICHELLE DEL RIO,§
JUAN M. ALVAREZ,‖ ALEXANDER OBENG,#
JALEEN AVILA,** and GANGA HETTIARACHCHI††

**Public Health Sciences, University of Texas; †Psicología, Universidad Autónoma Ciudad Juárez; ‡Psychology, Carleton College; §Environmental and Occupational Health, School of Public Health, Indiana University; ‖School of Public Health, University of Texas Health Science Center at Houston; #School of Public Health, Texas A&M University; **Public Health Sciences, University of Texas; ††Soil and Environmental Chemistry, Kansas State University*

Policy Points:

- Child lead poisoning is associated with socioeconomic inequity and perpetuates health inequality.
- Methods for testing and detection of child lead poisoning are ill suited to the current demographics and characteristics of the problem.
- A three-pronged revision of current testing approaches is suggested.
- Employing the suggested revisions can immediately increase our national capacity for equitable, inclusive testing and detection.

Abstract: Child lead poisoning, the longest-standing child public health epidemic in US history, is associated with socioeconomic inequity and perpetuates health inequality. Removing lead from children's environments ("primary

The Milbank Quarterly, Vol. 0, No. 0, 2023 (pp. 1-26)

prevention") is and must remain the definitive solution for ending child lead poisoning. Until that goal can be realized, protecting children's health necessarily depends on the adequacy of our methods for testing and detection. Current methods for testing and detection, however, are no longer suited to the demographics and magnitude of the problem. We discuss the potential deployment and feasibility of a three-pronged revision of current practices including: 1) acceptance of capillary samples for final determination of lead poisoning, with electronic documentation of "clean" collection methods submitted by workers who complete simple Centers for Disease Control and Prevention–endorsed online training and certification for capillary sample collection; 2) new guidance specifying the analysis of capillary samples by inductively coupled plasma mass spectrometry or graphite furnace atomic absorption spectrometry with documented limit of detection ≤ 0.2 μg/dL; and 3) adaptive "census tract–specific" universal testing and monitoring guidance for children from birth to 10 years of age. These testing modifications can bring child blood lead level (BLL) testing into homes and communities, immediately increasing our national capacity for inclusive and equitable detection and monitoring of dangerous lower-range BLLs in US children.

Keywords: child lead poisoning, social justice, social-structural inequity, child health disparity.

Introduction

Lead poisoning in US children, the longest-standing child public health epidemic in US history,[1] is driven by social, economic, and racial disparities. Although all children are vulnerable, lead poisoning is found overwhelmingly among minority children living in lower income neighborhoods with old, unrenovated housing, and/or situated near major lead hazard sources that contaminate the local air, water, and soil.[1-5] Caused largely by social structural inequities, the irreversible effects of childhood lead poisoning perpetuate life-long health disparities, representing yet another manifestation of systemic racism.[6]

The definitive solution for protecting children from lead exposure is removing lead from children's environments. This has not been achieved, however, for many complex reasons,[1] and lead remains ubiquitous in our modern environments,[7-10] a testimony to the impact of unregulated industry on the health of our nation.[11] A recent study determined that approximately half of the current US population has been exposed to dangerous levels of lead in early childhood.[12] Another study using

National Health and Nutrition Examination Survey (NHANES) data and census tract–reported factors associated with increased risk of child lead exposure estimated that at least 1.2 million US children were annually exposed to environmental lead, yielding blood lead levels (BLLs) >10 μg/dL (the "action threshold" until January 2012).[13] In another set of reports by Reuters using aggregated national data, in over 3,000 US cities, the rates of lead poisoning among tested children (in 2018, BLL ≥ 5.0 μg/dL) were found to be double those reported during the height of the Flint, Michigan, child lead exposure crisis; in an estimated 1,100 US cities, the rates were at least three times higher.[14,15] A reanalysis of data is needed to determine the numbers of tested children with BLLs greater than or equal to the current benchmark of 3.5 μg/dL (determined in October 2021), but it is undoubtedly higher. These rates are unacceptable by any standard.

Lead is a remarkably potent neurotoxin, and there is now broad acceptance that no level of lead exposure is "safe" for children. An abundance of evidence has shown that chronic exposure to environmental lead yielding child BLLs as low as 2.0 μg/dL[16] (US National Toxicology Program,[17] 2012[18]) disrupts cognitive and motor functions during childhood and adolescence,[19-29] damages the brain and peripheral organs,[25,30-35] and increases the risk of later cardiovascular disease, obesity, and mortality.[2,3] The annual economic burden of child lead exposure has been estimated to be \$5.9 million in long-term medical care costs and an estimated \$50.9 billion in lost economic productivity.[39]

Finally, solving the problem of lead poisoning in US children will require solutions for how we approach primary prevention and secondary prevention. With regard to primary prevention, new, feasible, and broadly effective approaches are needed for identifying and removing lead hazard sources from children's environments *before* exposure occurs. With regard to secondary prevention, major gaps in how we currently test for and detect child lead poisoning must be addressed. In a study that modeled the numbers of lead-exposed children likely "missed" for testing each year (based on NHANES child BLL data from 1999 to 2010), it was estimated that, each year, at least 500,000, and possibly more than 2 million, highest-risk children are never even tested.[13,15] Another recent study that analyzed geocoded birth certificate data and BLL results from 2011 to 2018 in North Carolina showed that 30% of highest-risk children were never tested.[40] National estimates are similar, with 35% of a Medicaid cohort never receiving a first test, and

50% of children never receiving critical follow-up monitoring.[41] It is critical to note that inclusive, equitable, and accurate child BLL testing simultaneously provides valid and reliable surveillance data for a given point in time, which is essential for demonstrating funding needs for primary prevention goals.

This paper addresses the major gaps in secondary prevention, that is testing for and detection of child lead poisoning. We consider alternative testing strategies that can increase our national capacity for inclusive and equitable child BLL testing and that yield accurate, precise results for dangerous lower-range child BLLs. We briefly summarize historical details that shaped the current testing practices, discuss how current approaches may inadvertently miss testing hundreds of thousands of high-risk children each year, and suggest how revised practices can be deployed to substantially improve our case detection success among high-risk children with dangerous lower-range BLLs. We also consider the material, analytic, and time costs of the alternative strategies suggested.

Some Historical Details Relevant to Current Clinical Practices for Child BLL Testing

Our interpretation of child BLLs has changed dramatically over the past 70 years. In the 1960s, clinical action was recommended for child BLLs >60 μg/dL. Advances in assay technology during the 1950s and 1960s allowed for increasingly precise estimates of heavy metals in aqueous media,[42] and the harm to children of exposure to lead yielding BLLs well below 60 μg/dL became apparent. Landmark studies of workers and children living near the Asarco Smelter in El Paso, Texas, were the first to quantify "silent effects" on cognitive and motor function in children with BLLs <30μg/dL,[43-46] and in 1979, the "clinical benchmark" for lead poisoning was lowered to 25 μg/dL. Studies accumulated showing damaging effects associated with lower and lower levels of lead exposure, prompting gradual benchmark changes, first to 10 μg/dL (1991), then to 5 μg/dL (2012), and, most recently, to 3.5 μg/dL (2021).[47]

Since at least 1990, the Centers for Disease Control and Prevention (CDC) has repeatedly warned that no level of lead exposure should be considered "safe" for children because no lower value could be identified at which toxic effects did not occur. Meanwhile, statistically based and biologically arbitrary "reference values" or "action thresholds" were defined to guide child intervention. Over the years, loss of the distinction between "toxicity" and "reference value" complicated understanding of the problem. For decades in many states, levels below a given benchmark were neither reported nor monitored, leaving us without meaningful estimates of the numbers of children affected.

The current approaches for testing and identifying children with lead poisoning were largely defined in 1988, when the benchmark for intervention was 25 μg/dL. Enactment of the Lead Contamination Control Act formally authorized local and state agencies to create state-based, CDC-funded child lead poisoning prevention programs (CLPPPs).[48] Today, in many states, CLPPP funding, although grossly underresourced, continues to be the backbone of child lead poisoning prevention efforts. Every state provides information online that includes different combinations of topics for parents on, for example, common child lead hazard sources, the risks to children's health of lead exposure, expected timepoints and frequencies for child lead testing, how children are identified for testing, and tiered interventions for managing different levels of lead exposure.

Summary of Current Child BLL Testing Practices

"Universal testing," first instituted in 1991,[49] aimed to reach the largest numbers of children possible. Youngest children were targeted for testing because studies of higher-range BLLs[4] suggested that blood lead concentrations increased between approximately 6 and 12 months of age, then decreased after 3 years of age, which is attributable to a combination of hand-to-mouth behavior and crawling, which exposed children to lead-contaminated household dust and soil residue. Studies have yet to be conducted that examine whether these trends are similar for exposures yielding dangerous lower-range BLLs. Today, states diverge broadly in guidelines and expectations for child lead testing. No state requires, monitors, and enforces child BLL testing on a child-by-child basis. As of 2017, ten states recommended "universal" testing at 12 and 24 months of age; in two of these states, guidance included children up to 6 years of age, depending on their lead testing history.[50] In the remaining 40 states, a “targeted testing” approach is used that relies on parent completion of “personal risk questionnaires.” These query factors known to be associated with child lead poisoning, for example, peeling paint in the home and age of residence. The answers are used to determine whether a child should be referred for testing. If the parents’ answers to one or more items is “yes,” and, in some states, “don’t know,” a blood test using a venous sample (drawn from the child’s arm vein) is required to determine lead poisoning.

In some cases, a child may be first “screened” using a point-of-care device to analyze a finger-stick blood sample (recalls of these devices will be discussed below). If the initial screen is considered negative for lead exposure, further testing is not expected but may be conducted. If the point-of-care device screen is considered positive for lead exposure, a BLL test of a venous sample is required for “final determination” of lead exposure. In past years, the inappropriate use of the point-care-devices to analyze venous samples resulted in an unknown number of false-positive results, prompting a US Food and Drug Administraton (FDA) safety warning[51] against this practice. Venous samples are expected to be sent to a laboratory for analysis by one of several possible assay methods (e.g., graphite furnace atomic absorption spectroscopy [GFAAS], atomic absorption spectroscopy, inductively coupled plasma optical emission spectrometry, inductively coupled plasma mass spectrometry [ICPMS]); however, guidance regarding an acceptable limit of detection (LOD) for the child blood lead assay is not provided. Laboratory results are typically returned within 2-4 weeks.

As of 2020, in 43 states,[52] medical health care workers and medical facilities are required to report all child BLL results to state agencies for surveillance purposes. In the remaining states, reporting is based on a state-defined benchmark. States differ broadly regarding which child BLLs trigger which level of intervention. A BLL of 3.5 μg/dL triggers home testing in three states; in all other states, parent education is provided for lower-range exposures and home testing begins only on detection of a BLL of $\geq$10 μg/dL; a few states include the provision of early intervention services for lead-exposed children. In some states, two venous sample tests within 3 months yielding a child BLL of $\geq$15 μg/dL, or one venous sample test yielding a child BLL $\geq$20 μg/dL, are required before any action is taken to identify and remove possible home lead hazard sources.

Reorienting Clinical Practice to Promote Equitability, Inclusion, and Accuracy in Child BLL Testing

Three aspects of current testing practices may be directly contributing to gaps in child BLL testing and can be amended as discussed below and summarized in Table 1.

1. Accept results based on capillary sample blood draws for determination of lead poisoning with "clean" collection methods electronically documented by certified BLL sample collectors

All states currently require a venous sample blood test as a "final determination" of child lead poisoning, and this may be the single biggest barrier to equitable, inclusive child testing. The challenge of getting children at risk for lead exposure into child clinics and doctors' offices—of explicit concern to the CDC in 1997[53]—remains a concern today. Venous sample blood collection is uncomfortable and frightening to children and to some parents, decreasing the likelihood of compliance. The discomfort during the procedure is dependent on the skill of the phlebotomist. In many underserved areas, particularly rural areas, pediatric phlebotomists can be scarce or nonexistent. Because phlebotomists are required for venous sample blood draws, they are costly.

More broadly speaking, research has shown that compliance with pediatric preventive services are significantly associated with socioeconomic factors.[54] Regardless of insurance issues, parents may be working one or more jobs, making visits to doctor's offices not only expensive but also difficult if not impossible to navigate. Immigration concerns of parents and/or relatives can discourage parents from seeking services or guidance from anyone they may perceive as an authority figure. In many states, depending on whether parents are insured or their type of insurance, parents may be expected to pay some or all of the cost for a venous sample BLL test (e.g., \$70-\$120 per child). Medical expenses have become the number one reason for new family bankruptcies in the United States,[55] and for good reason; insured and uninsured parents alike fear adding medical expenses to their monthly budgets.

The original 1991 CDC guidance for child BLL testing described venous sample blood tests as the "preferred" method for confirming child lead exposure.[53] Note, however, that this guidance also stated that

Table 1. Summary of Current Practices, How Current Practices "Miss" Lead-Exposed Children, and Feasible Alternatives to Current Practices

Current Practice	How Current Practice "Misses" Children	Alternative Approach
1. Venous sample blood test requiring medical office/clinic visit	High-risk children are less likely to get to doctors' offices or clinics; venous draws reduce compliance; venous samples are high cost and require certified pediatric phlebotomists.	Accept capillary finger-stick blood samples collected using document "clean" methods by specially trained and certified sample collectors as "determination" of lead poisoning; this allows testing to be conducted in trusted neighborhood settings where children/parents/families already gather (e.g., Head Start programs, WIC offices, schools, synagogues, churches, mosques, daycare centers, and YMCAs).
2. No specific assay method recommended for testing blood samples for lead; no LOD recommended	Assay methods other than ICPMS and GFAAS may not have the precision and/or accuracy needed to detect and monitor dangerous lowest-range BLLs; point-of-care devices lacking accuracy and precision for dangerous, lower-range BLLs continue to be used in an unknown number of offices and clinics.	Specify ICPMS or GFAAS with an LOD $\leq$0.2 μg/dL for all child BLL tests.

Continued

Table 1. (Continued)

Current Practice	How Current Practice "Misses" Children	Alternative Approach
3. Referral system used in 72% of states with referral based on parent's responses to a questionnaire; testing focused on infants and toddlers; one or two negative BLL tests before 2-3 or 5-6 years of age (depending on the state) are taken as evidence that the child is "not exposed"	Parents must get to a location where the risk questionnaire is administered; self-report measures have high potential for bias and include items that can be perceived as stigmatizing; children above 6 years old are also vulnerable to neurotoxic effects of lead exposure; lead absorption in children is biologically complex; BLLs are meaningful but imperfect estimates of circulating blood lead and should be expected to fluctuate; one or two BLL tests in early childhood cannot be assumed to rule out exposure.	Set adaptive census tract–specific universal testing guidelines using baseline results to determine the frequency of testing needed for children 0-10 years old; depending on risk determined by two consecutive baseline tests, testing could range from monitoring once every 3 years to minimum biannual testing.

capillary blood samples were a "reasonable option" if specific methods to maximally reduce the chances of lead contamination from the surface of the skin were strictly followed.[53] Over time, the venous sample blood test became *the* standard for definitive confirmation because of concerns that capillary samples (e.g., finger stick) were too vulnerable to contamination and could result in false-positive results.

Without question, if capillary samples were approved for the determination of lead poisoning, strict and enforceable guidelines for hand-cleaning would have to be instituted, particularly given the current child BLL reference value of 3.5μg/dL. This is not an insurmountable obstacle. To ensure "clean" capillary samples, a standard hand-cleaning protocol could be defined that uses proven methods for removing lead from the surface of the skin. Hand-cleaning methods have proven efficacy and are simple and inexpensive to carry out. Rigorous studies conducted by researchers at the CDC/National Institute for Occupational Safety and Health showed the high efficacy of using two consecutive isostearamidopropyl morpholine lactate/citric acid wipes (marketed as LeadOff™, Hygenall Corporation, Huntsville, AL) with a clear water rinse for cleaning lead from the surface of the skin (lead retrieval from skin surface > 99%).[56]

Confirming adherence to a defined cleaning protocol is, of course, critical. There are many low-cost, technology-assisted approaches that could be used. For example, CDC-sponsored certification for the collection of capillary samples could be required for all sample collection workers. The training could include documentation of minimum education expectations and completion of a short test. The protocol for the capillary draw itself could require two certified capillary collection workers, one who executes the stepwise hand-cleaning procedure and one who observes and documents the procedure on an electronic form. For each child, the observer would record the completion of each required step in "real time" using an official data- and time-stamped CDC-issued form with simple checkboxes. Both workers could be required to sign each form. Providing the form virtually through a secure CDC-sponsored website for use on a laptop or tablet with immediate upload of information and signatures could be the basis for a national registry database of child blood sample collection. Information from the date- and time-stamped electronic collection forms can be readily used to evaluate positive child BLL test results.

Capillary sample collection is considered by the FDA to be a "noninvasive" procedure with practically no medical risk other than momentary discomfort on the fingertip for some children. Finger-stick blood samples are now widely used among adults for in-home testing of, for example, blood sugar and coagulation, and many children recognize and easily cooperate with the procedure. Child-specific lancets ensure the greatest comfort, and many children may not even feel the stick; small vibrating gadgets are effective in distracting worried children while blocking perception of the finger stick (Pain Care Labs Buzzy™, Atlanta, GA).

Using certified capillary sample collectors trained in deployment and electronic reporting of hand-cleaning methods that ensure uncontaminated capillary samples would be far more cost-effective, feasible, and child and parent friendly than requiring venous sample blood collection by pediatric phlebotomists. It would also allow child BLL testing to be conducted in community locations that children and families know, trust, and frequent. In our research studies, for example, we formed strong and lasting alliances for child BLL testing with local public elementary schools.[27,57] Ideal locations would likely vary depending on the characteristics and needs of the community. These might include public schools, Head Start centers, Special Supplemental Nutrition Program for Women, Infants, and Children locations, YMCAs/YWCAs, libraries, churches, synagogues, mosques, and/or repurposed COVID-19 vaccination sites. Capillary samples could also be collected by mother/infant/child intervention specialists who have already established relationships during the provision of other support services. Given the magnitude and demographics of the current child lead exposure problem in the United States, future success in reducing child lead poisoning in the United States will likely require the use of capillary samples for determination of exposure.

2. Provide guidance for the analysis of capillary samples by ICPMS or GFAAS with documented LOD <0.2 μg/dL.

For a given range of lead concentrations, the assay method used for estimating BLL from whole blood samples determines the precision and accuracy of the result and, thus, its practical value for surveillance and monitoring. The median child BLL in the United States has decreased overall in recent decades, and the current problem of child lead poisoning largely concerns values in the dangerous lower range (e.g., <10 μg/dL) and requiring assay methods with high precision and accuracy. Because no level of lead exposure is "safe" for children, precise and accurate detection of BLLs below the current statistically determined reference value (3.5 μg/dL) is critical for long-term surveillance and prediction and for ensuring meaningful integration of national-level data.

When the 1991 CDC guidance was issued, relatively few feasible assay methods were available. That situation has changed substantially in the intervening 31 years. Most assay technologies have improved,[58,59] but the LOD can vary depending on calibration parameters.[58,60] With recent technological advances, both ICPMS and GFAAS provide the lowest elemental detection limits for lead.[61,62] Since its introduction in 1980,[63] ICPMS has become a "gold-standard" method for precisely and accurately estimating lowest level elements in aqueous solutions such as human blood,[58,60,64,65] and its LOD can be assumed to be low (e.g., well below 0.2 μg/dL). For GFAAS, an LOD $\leq$0.2 is readily achievable with using a Zeeman effect background correction (spectral splitting by magnetic field).[61,66] Importantly, both methods require no more than 50 μL of "clean" whole blood capillary samples, which can be collected via child-sized finger-stick lancets, or arm-stick (Tasso, Inc., Seattle, WA) methods.

There are many options for ensuring the feasibility of this new guidance. For example, a nationwide network of CDC-approved ICPMS- or GFAAS-equipped laboratories, with annually documented LODs of $\leq$0.2 μg/dL, could be established. Federal contracts could lower the cost per sample, and state "buy-in" costs for testing could be automatically deducted from state-level CLPPP grants. (Not all states currently have CLPPP funding, and this benefit could meaningfully incentivize states who do so.) Samples would be accepted for analysis only if the requisite electronic child sample documentation form (including verification of hand-cleaning protocol) had been uploaded by the trained and certified sample collectors. Anonymized child BLL results, including the child's sex, age, race/ethnicity, and census tract, could be uploaded into a national registry database, providing new capacity to geographically map "hot spots" in real time and monitor child BLLs across time. These results would be used to determine "census tract–specific" expectations for child BLL testing described below.

It is important to briefly address the limitations of point-of-care devices for child BLL screening. The numbers of doctors' offices that continue to use point-of-care devices is not known. The BLL estimates from these devices have been repeatedly shown to be of questionable reliability at levels below 10 μg/dL.[18,67-69] Given the current reference value of 3.5 μg/dL, these devices are no longer appropriate for managing the current problem[68,70] and could seriously undermine meaningful child surveillance and monitoring.

3. Provide adaptive "census tract–specific" universal testing guidance for children 0-10 years.

Once a hallmark of CLPPPs nationwide,[49] universal child testing for lead exposure is no longer recommended in 72% (36/50) of US states.[52] Instead, the CDC recommends that state and local health authorities develop their own targeted screening and intervention guidelines based on local risk factors and available resources. Most states use a targeted referral system for determining which children should be tested. Referrals for child lead testing can come from a variety of sources; in most cases, the process relies on parent responses to a "personal risk" questionnaire, administered in a clinic, doctor's office, well-child health care visit, or other public health service center. Thus, at the core of screening, compliance is the willingness and intention of medical providers. Barriers to screening can include the number of well-child screenings required in one office visit and/or lack of knowledge among providers regarding both the current state recommendations and the dangers associated with chronic lead exposure. When providers pursue child BLL screening via the targeted referral approach, the form used queries of child and home characteristics that are known risk factors for child lead exposure, and the items vary somewhat by state. A "yes," and, in some states, "don't know," response will trigger a referral for child BLL testing. The forms are designed to be "parent friendly" and "parent appropriate" and are available in different languages specific to the community. Nonetheless, the unreliability of self-report has been extensively studied and described.[71] With regard to parents of children at risk of lead exposure, any of the following could impact whether a parent would be willing and/or able to provide accurate information regarding child lead exposure risk factors: whether the form was completed without assistance, leaving the interpretation of questions and/or answers up to the parent; whether a trained and sensitive worker is available to check and confirm answers as needed; how many other forms were completed at the same time; whether the parent was comfortable requesting a form written in their language of choice; and whether "I don't know" responses were provided as an option.

It is also important to consider the potential implications of the questions for parents, particularly those querying conditions of the living environment. Parents who face economic challenges can have many practical reasons for feeling that they need to carefully manage how medical authority figures perceive the home they provide for their children. Any of the following factors can also directly impact how parents respond to home environment questions: whether the parent was previously undomiciled, whether the parent has faced child custody issues, whether the parent receives or has had challenges obtaining public assistance, whether the parent lives in public housing, and the extent to which the parent perceives any of the questions on the form as a reflection of the quality of home environment they provide for their children.

The extent to which the current "personal risk" referral system misses detection of lead-exposed children would be difficult to estimate, but a return to some form of "universal" child BLL testing guidance for all states would simply remove issues related to the current referral approach.

Because the current problem appears to cluster largely in underserved neighborhoods, strategic use of baseline testing to define adaptive "census tract–specific" guidelines for child BLL testing could ensure that resources are targeted to the highest-needs areas. For example, two rounds of comprehensive child BLL testing conducted over one 6-month interval could quickly reveal which census tracts require ongoing surveillance by twice-per-year BLL monitoring and which appear to be relatively low risk, with follow-up testing every 3 years, for example.

Another issue that is not managed by current clinical practices concerns ages of risk. In many states, BLL testing is recommended only for infants and toddlers up to 3 years of age. Even in states that have maintained "virtual universal testing," testing recommendations stop at 5 or 6 years of age. Although smaller children have higher risk of exposure through hand-to-mouth behavior and more readily absorb lead because of their small body size, this does not mean that older children are not also at risk. Studies from at least the past 15 years, including children older than 6 years of age, have quantified their vulnerability to the neurotoxic effects of lead exposure and that the severity of these are mediated by common genetic variants.[27,72-76] These findings are corroborated by research investigating neurodevelopment during the "forgotten years" (e.g., 6-12 years of age) when the brain continues to undergo critical periods of growth and change.[77-79] Because recommendations for BLL testing and reporting have been limited to the youngest children, data are largely not available regarding how many school-age children might also be chronically exposed to lead. Including school-age children in adaptive "census tract–specific" universal testing is critical for understanding the scope of the current problem and for increasing knowledge on the effects of lead exposure in these middle school years. To ensure feasibility, for the initial deployment, ages included could be from birth to 10 years of age and expanded to preadolescence for communities over time with demonstrated higher risk.

The recommended frequency of BLL testing is also important to consider. In most states, if children are tested, they are tested once or twice before 2 or 3 years of age. Some states repeat testing once or twice before the age of 5 or 6 years depending on whether earlier tests were provided. A few states recommend annual testing for highest-risk children up to 5 or 6 years of age. In the vast majority of states, one or two negative blood lead level tests conducted during infancy or toddlerhood are used to rule out child lead exposure.

A blood test provides the best available approximation of circulating lead, but it is an imperfect surrogate marker. The physiology of lead absorption in infants, toddlers, and children, and thus the amount of lead available for detection in a blood sample at a given point in time, is influenced by complex interacting physiological and environmental factors that fluctuate.[80]

The amount of circulating lead available to be detected is necessarily dependent on the timing of absorption from children's lungs[81-84] and/or gut[85-88] and the ratio of lead deposited in organs,[89,90] both of which involve the interaction of dynamic mechanisms influenced by individual developmental differences, developmental stage, and genetics.[91-93] These processes are in turn influenced by varying environmental factors, including, for example, the route of exposure (inhalation vs. ingestion),[94,95] type of lead hazard source and frequency of exposure,[83] and socioeconomic factors that result in, for example, empty stomachs,[96,97] low calcium stores,[98] and other nutritional deficits that can increase lead absorption and decrease the body's capacity to excrete toxins, depending on the age of the child.[25,99,100] The amount of lead available for detection in blood is also dependent on its half-life—estimated to be 28-35 days for single exposures, but it is a far more difficult calculation for children chronically exposed to lead.[101-103] In recent longitudinal studies of 193 children 6 months to 16 years of age residing in neighborhoods previously designated "high risk" for lead exposure, BLLs within individuals varied significantly over a 24-month period, and with repeated testing, age was not a significant predictor of BLL.[104]

Unless a lead hazard source is available to a child to ingest or inhale in some highly consistent way, all other things being equal, child BLLs would be expected to fluctuate over time rather than stay the same, particularly those from exposures to multiple lower-level sources. Fluctuating child BLLs, however, cannot be assumed to represent fluctuating risk to the developing brain and other organs. The instability of BLLs can guide recommendations for child testing at least twice per year for children living in census tracts determined to be at "high risk" of lead exposure.

Feasibility: Material, Analytic, and Time Costs for Capillary Sample Collection with ICPMS

The following estimated costs are based on our experiences over the past 15 years using the above-described methods in six elementary schools and two local churches for the collection of over 1,000 child finger-stick capillary blood samples, collected using documented "clean" methods and analyzed by ICPMS. We began our studies in 2007 by using LeadCare devices until we realized their limitations for reliably detecting dangerous lower-range child BLLs,[68] at which point we used only ICPMS analysis of finger-stick capillary samples collected following a strict and documented hand-cleaning protocol using a collector/observer protocol similar to that described above.

The total material costs of BLL assays, including materials, supplies, and ICPMS analyses, were between $36 and $42 per child sample. Based on conversations with other laboratories, the estimated cost of GFAAS would be comparable or less. With a team of as few as two specially trained workers—one worker to complete document hand-cleaning, complete protocol tracking forms, and organize paperwork, and one worker to collect samples—in public elementary school settings, we were able to complete capillary sample collection for 50-60 children during a regular school day (10 children at a time called en masse from each of six 40-min Physical Education periods), yielding 250-300 samples in one 5-day week. For an elementary school of approximately 500 children, all children in the school could be tested in one 2-week period. Special testing times (usually early morning) were designated for sample collection from infants and toddlers. Repeat testing was conducted at 4- to 6-month intervals. For a given elementary school, for example, biannual testing could be scheduled for one 2-week period in the fall and spring terms. Importantly, a "universal" biannual testing approach ensures that, for children with identified lead poisoning, follow-up monitoring following intervention becomes routine. When BLLs are monitored over time, geographically mapping[105] (e.g., via ArcGIS) is valuable for determining exposure "hot spots" and also for identifying areas in which no children have BLLs >1 μg/dL, for example. With biannual testing, patterns of exposure can be examined within 2 months of sample collection, and decisions can be made regarding how the testing strategy should be modified to best manage different BLL result outcomes.

Conclusion

Although removing lead from our children's environments must remain our central goal (primary prevention), lead continues to be ubiquitous in the United States. Once exposed, there are no interventions that can reverse the potentially devastating effects of lead exposure, particularly those associated with dangerous lower-range BLLs.[106] As this child public health epidemic continues, we are dependent on accurate and precise detection of lead poisoning to limit its short- and longer-term effects. Current clinical approaches for identifying children with lead poisoning are ill-suited to the magnitude and demographics of the problem and, each year, inadvertently "miss" testing for hundreds of thousands of children. Attention and resources must focus on substantially improving our national capacity to provide inclusive, equitable, and precise BLL testing for all children, particularly those at highest risk of exposure to lead, yielding dangerous lower-range BLLs. Revising federal guidance to accept capillary blood samples collected with verified "clean" sampling methods analyzed by ICPMS or GFAAS with a minimum LOD of

<0.2 μg/dL for determination of child lead exposure with the frequency of repeated monitoring for children 0-10 years of age determined according to adaptive "census tract–specific" schedules would remove current systemic barriers to testing for highest-risk children and dramatically increase our national capacity for inclusive and equitable detection and monitoring of lead poisoning in US children.

References

1. Breysse PN. Lead elimination for the 21st century. *J Public Health Manag Pract*. 2019;25(Suppl 1):S3-S4.
2. Egan KB, Cornwell CR, Courtney JG, Ettinger AS. Blood lead levels in U.S. children ages 1–11 years, 1976–2016. *Environ Health Perspect*. 2021;129(3):37003.
3. Brown RW, Longoria T. Multiple risk factors for lead poisoning in Hispanic sub-populations: a review. *J Immigr Minor Health*. 2010;12(5):715-725.
4. Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. *J Pediatr*. 2002;140(1):40-47.
5. Yeter D, Banks EC, Aschner M. Disparity in risk factor severity for early childhood blood lead among predominantly African-American Black children: the 1999 to 2010 US NHANES. *Int J Environ Res Public Health*. 2020;17(5):1552.
6. Sampson RJ, Winter A. The racial ecology of lead poisoning: toxic inequality in Chicago neighborhoods, 1995–2013. *DuBois Review: Social Science Research on Race*. 2018;13(2).
7. Centers for Disease Control and Prevention. *Preventing Lead Poisoining in Young Children*. US Department of Health and Human Services, Public Health Service; 1991.
8. Garside M. *Mine Production of Lead in the Leading Countries Worldwide, 2010–2020*. Statista, Inc. 2020.
9. Gottesfeld P. Time to ban lead in industrial paints and coatings. *Front Public Health*. 2015;3:144.
10. Klochko K. *Mineral Industry Surveys: Lead*. US Geological Survey; 2021.
11. Markowitz G, Rosner D. *Lead Wars: The Politics of Science and Fate of America's Children*. University of California Press; 2014.
12. McFarland MJ, Hauer ME, Reuben A. Half of US population exposed to adverse lead levels in early childhood. *Proc Natl Acad Sci U S A*. 2022;119(11): e2118631119.

13. Roberts EM, Madrigal D, Valle J, King G, Kite L. Assessing child lead poisoning case ascertainment in the US, 1999–2010. *Pediatrics*. 2017;139(5):e20164266.
14. Pell MB, Schneyer J. The thousands of U.S. locales where lead poisoning is worse than in Flint. *Reuters Investigates*. December 19, 2016. Accessed September 28, 2022. https://www.reuters.com/investigates/special-report/usa-lead-testing/
15. Schneyer J, Pell MB. Millions of American children missing early lead tests, Reuters finds. *Reuters Investigates*. June 9, 2016. Accessed September 28, 2022. https://www.reuters.com/investigates/special-report/lead-poisoning-testing-gaps/
16. Lanphear BP, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect*. 2005;113(7):894-899.
17. Health effects of low-level lead. US National Toxicology Program. February 25, 2020. Accessed September 28, 2022. https://ntp.niehs.nih.gov/whatwestudy/assessments/noncancer/completed/lead/index.html
18. Lanphear BP. Low-level toxicity of chemicals: no acceptable levels? *PLoS Biol*. 2017;15(12):e2003066.
19. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics*. 1992;90(6):855-861.
20. Byers RK. Lead poisoning; review of the literature and report on 45 cases. *Pediatrics*. 1959;23(3):585-603.
21. Byers RK, Lord EE. Late effects of lead poisoning on mental development. *Am J Dis Child*. 1943;66:471-494.
22. Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL. Early exposure to lead and juvenile delinquency. *Neurotoxicol Teratol*. 2001;23(6):511-518.
23. Gibson JL. A plea for painted railings and painted rooms as the source of lead poisoning amongst Queensland children. *Public Health Rep*. 2005;120(3):301-304.
24. Needleman HL, McFarland C, Ness RB, Fienberg SE, Tobin MJ. Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicol Teratol*. 2002;24(6):711-717.
25. Rădulescu A, Lundgren S. A pharmacokinetic model of lead absorption and calcium competitive dynamics. *Sci Rep*. 2019;9(1):14225.
26. Sanders T, Liu Y, Buchner V, Tchounwou PB. Neurotoxic effects and biomarkers of lead exposure: a review. *Rev Environ Health*. 2009;24(1):15-45.

27. Sobin C, Flores-Montoya MG, Gutierrez M, Parisi N, Schaub T. δ-Aminolevulinic acid dehydratase single nucleotide polymorphism 2 (ALAD2) and peptide transporter 2*2 haplotype (hPEPT2*2) differently influence neurobehavior in low-level lead exposed children. *Neurotoxicol Teratol*. 2015;47:137-145.
28. Flores-Montoya MG, Alvarez JM,, Sobin C. Olfactory recognition memory is disrupted in young mice with chronic low-level lead exposure. *Toxicol Lett*. 2015;236(1):69–74.
29. Flores-Montoya MG, Sobin C. Early chronic lead exposure reduces exploratory activity in young C57BL/6J mice. *J Appl Toxicol*. 2015;35(7):759-765.
30. Basgen JM, Sobin C. Early chronic low-level lead exposure produces glomerular hypertrophy in young C57BL/6J mice. *Toxicol Lett*. 2014;225(1):48-56.
31. Dominguez S, Flores-Montoya MG, Sobin C. Early chronic exposure to low-level lead alters total hippocampal microglia in pre-adolescent mice. *Toxicol Lett*. 2019;302:75-82.
32. Flores-Montoya MG, Bill CA, Vines CM, Sobin C. Early chronic low-level lead exposure reduced C-C chemokine receptor 7 in hippocampal microglia. *Toxicol Lett*. 2019;314:106-116.
33. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. *Brain* 2003;126(Pt 1):5-19.
34. Sobin C, Montoya MG, Parisi N, Schaub T, Cervantes M, Armijos RX. Microglial disruption in young mice with early chronic lead exposure. *Toxicol Lett*. 2013;220(1):44-52.
35. Dribben WH, Creeley CE, Farber N. Low-level lead exposure triggers neuronal apoptosis in the developing mouse brain. *Neurotoxicol Teratol*. 2011;33(4):473-480.
36. Lanphear BP, Rauch S, Auinger P, Allen RW, Hornung RW. Low-level lead exposure and mortality in US adults: a population-based cohort study. *Lancet Public Health*. 2018;3(4):e177-e184.
37. Leasure JL, Giddabasappa A, Chaney S, et al. Low-level human equivalent gestational lead exposure produces sex-specific motor and coordination abnormalities and late-onset obesity in year-old mice. *Environ Health Perspect*. 2008;116(3):355-361.
38. Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ. Lead exposure and cardiovascular disease—a systematic review. *Environ Health Perspect*. 2007;115(3):472-482.
39. Trasande L, Liu Y. Reducing the staggering costs of environmental disease in children, estimated at $76.6 billion in 2008. *Health Aff (Millwood)*. 2011;30(5):863–870.
40. Kamai EM, Daniels JL, Delamater PL, Lanphear BP, Gibson JM, Richardson DB. Patterns of children's blood lead screening and

blood lead levels in North Carolina, 2011-2018-who is tested, who is missed? *Environ Health Perspect*. 2022;130(6):67002.

41. Knighton AJ, Payne NR, Speedie S. Lead testing in a pediatric population: underscreening and problematic repeated tests. *J Public Health Manag Pract*. 2016;22(4):331-337.
42. National Research Council (US) Committee on Measuring Lead in Critical Populations. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*. National Academies Press (US); 1993.
43. Landrigan PJ, Baker EL Jr., Feldman RG, et al. Increased lead absorption with anemia and slowed nerve conduction in children near a lead smelter. *J Pediatr*. 1976;89(6): 904-910.
44. Landrigan PJ, Gehlbach SH, Rosenblum BF, et al. Epidemic lead absorption near an ore smelter. The role of particulate lead. *N Eng J Med*. 1975;292(3):123-129.
45. Landrigan PJ, Whitworth RH, Baloh RW, Staehling NW, Barthel WF, Rosenblum BF. Neuropsychological dysfunction in children with chronic low-level lead absorption. *Lancet*. 1975;1(7909):708-712.
46. Morse DL, Landrigan PJ, Rosenblum BF, Hubert JS, Housworth J. El Paso revisited: epidemiologic follow-up of an environmental lead problem. *JAMA*. 1979;242(8):739-741.
47. *Timeline of childhoood lead poisoning prevention highlights*. October 25, 2022. Accessed September 28, 2022. https://www.cdc.gov/nceh/lead/about/timeline.html.
48. Lead Contamination Control Act of 1988. HR 4939. 100th Congress, Public Law 100-572 (1988).
49. Centers for Disease Control and Prevention. *Preventing Lead Poisoning in Young Children: A Statement by the U.S. Department of Health and Human Services (No. 537)*. US Department of Health and Human Services, Public Health Service; 1985.
50. Dickman J. *Children at Risk: Gaps in State Lead Screening Policies*. Safer Chemicals, Healthy Families; 2017.
51. Blood lead safety alert. Centers for Disease Control and Prevention. July 30, 2019. Accessed September 28, 2022. https://www.cdc.gov/nceh/lead/news/blood-lead-safety-alert.htm
52. Michel JJ, Erinoff E, Tsou AY. More guidelines than states: variations in U.S. lead screening and management guidance and impacts on shareable CDS development. *BMC Public Health*. 2020;20(1):127.
53. Centers for Disease Control and Prevention. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health*

Officials. US Department of Health and Human Services, Public Health Service; 1997.

54. Jones MN, Brown CM, Widener MJ, Sucharew HJ, Beck AF. Area-level socioeconomic factors are associated with noncompletion of pediatric preventive services. *J Prim Care Community Health*. 2016;7:143-148.
55. Himmelstein DU, Lawless RM, Thorne D, Foohey P, Woolhandler S. Medical bankruptcy: still common despite the Affordable Care Act. *Am J Public Health*. 2019;109(3):431-433.
56. Esswein EJ, Ashley K. Handwipe method for removing lead from skin. *J ASTM Int*. 2011;8(5):1-10.
57. Alvarez J, Del Rio M, Mayorga T, Dominguez S, Flores-Montoya MG. A comparison of child blood lead levels in urban and rural children ages 5–12 years living in the border region of El Paso, Texas. *Arch Environ Contam Toxicol*. 2018;75(4):503-511.
58. Caldwell KL, Cheng P-Y, Jarrett JM, et al. Measurement challenges at low blood lead levels. *Pediatrics*. 2017;140(2):e20170272.
59. Deibler K, Basu P. Continuing issues with lead: recent advances in detection. *Eur J Inorg Chem*. 2013;2013(7):1086-1096.
60. World Health Organization. *Brief Guide to Analytic Methods for Analyzing Lead in Blood*. 2nd ed. World Health Organization; 2011.
61. Pacer EJ, Palmer CD, Parsons PJ. Determination of lead in blood by graphite furnace atomic absorption spectrometry with Zeeman background correction: improving a well-established method to support a lower blood lead reference value for children. *Spectrochim Acta B: At Spectrosc*. 2022;190:106324.
62. Trzcinka-Ochocka M, Brodzka R, Janasik B. Useful and fast method for blood lead and cadmium determination using ICP-MS and GF-AAS; validation parameters. *J Clin Lab Anal*. 2016;30(2):130-139.
63. Houk RS, Fassel VA, Flesch GD, Svec HJ, Gray AL, Taylor CE. Inductively coupled argon plasma as an ion source for mass spectrometric determination of trace elements. *Anal Chem*. 1980;52(14):2283-2289.
64. Laur N, Kinscherf R, Pomytkin K, Kaiser L, Knes O, Deigner H-P. ICP-MS trace element analysis in serum and whole blood. *PLoS One*. 2020;15:e0233357.
65. Wilschefski SC, Baxter MR. Inductively coupled plasma mass spectrometry: introduction to analytical aspects. *Clin Biochem Rev*. 2019;40(3):115-133.

66. Slavin W. Accuracy in furnace atomic absorption spectroscopy. *J Res Nat Bur Stand*. 1988;93(3):445-446.
67. Gilbert SG, Weiss B. A rationale for lowering the blood lead action level from 10 to 2 microg/dL. *Neurotoxicology* 2006;27(5):693-701.
68. Sobin C, Parisi N, Schaub T, de la Riva E. A Bland-Altman comparison of the Lead Care® System and inductively coupled plasma mass spectrometry for detecting low-level lead in child whole blood samples. *J Med Toxicol*. 2011;7(1):24-32.
69. Magellan Diagnostics recalls LeadCare II, LeadCare Plus, and LeadCare Ultra blood lead tests due to risk of falsely low results. US Food and Drug Administration. March 31, 2022. Accessed September 28, 2022. https://www.fda.gov/medical-devices/medical-device-recalls/magellan-diagnostics-recalls-leadcareleadcarepluanddcareultrbloodd
70. Mason J, Ortiz D, Pappas S, Quigley S, Yendell S, Ettinger AS. Response to the US FDA LeadCare Testing Systems recall and CDC Health Alert. *J Public Health Manag Pract*. 2019;25(Suppl 1):S91-S97.
71. Tanur J. *Questions About Questions: Inquiries into the Cognitive Bases of Surveys*. Russell Sage Foundation; 1992.
72. Sobin C, Gutierrez M, Alterio H. Polymorphisms of delta-aminolevulinic acid dehydratase (ALAD) and peptide transporter 2 (PEPT2) genes in children with low-level lead exposure. *Neurotoxicology*. 2009;30(6):881-887.
73. Takeuchi H, Taki Y, Nouchi R, et al. Lead exposure is associated with functional and microstructural changes in the healthy human brain. *Commun Biol*. 2021;4(1):912.
74. Reuben A, Elliott ML, Abraham WC, et al. Association of childhood lead exposure with MRI measurements of structural brain integrity in Midlife. *JAMA*. 2020;324(19):1970-1979.
75. Reuben A, Schaefer JD, Moffitt TE, et al. Association of childhood lead exposure with adult personality traits and lifelong mental health. *JAMA Psychiatry*. 2019;76(4): 418-425.
76. Beckley AL, Caspi A, Broadbent J, et al. Association of childhood blood lead levels with criminal offending. *JAMA Pediatr*. 2018;172(2):166-173.
77. Franke K, Luders E, May A, Wilke M, Gaser C. Brain maturation: predicting individual BrainAGE in children and adolescents using structural MRI. *Neuroimage*. 2012;63(3):1305-1312.
78. Mah VK, Ford-Jones EL. Spotlight on middle childhood: rejuvenating the 'forgotten years.' *Paediatr Child Health*. 2012;17(2):81-83.

79. Somsen RJM, van't Klooster BJ, van der Molen MW, van Leeuwen HM, Licht R. Growth spurts in brain maturation during middle childhood as indexed by EEG power spectra. *Biol Psychol*. 1997;44(3):187-209.
80. Del Rio M, Sobin C, Hettiarachchi GM. Biological factors that impact variability of lead absorption and BLL estimation in children: implications for child blood lead level testing practices. *J Environ Health*. 2022;85(5):18-26.
81. Geiser M, Kreyling WG. Deposition and biokinetics of inhaled nanoparticles. *Part Fibre Toxicol*. 2010;7:2.
82. James AC, Stahlhofen W, Rudolf G, et al. Annexe D. Deposition of inhaled particles. *Ann ICRP*. 1994;24(1-3):231-299.
83. Kastury F, Smith E, Lombi E, et al. Dynamics of lead bioavailability and speciation in indoor dust and x-ray spectroscopic investigation of the link between ingestion and inhalation pathways. *Environ Sci Technol*. 2019;53(19):11486-11495.
84. Nemmar A, Hoet PH, Vanquickenborne B, et al. Passage of inhaled particles into the blood circulation in humans. *Circulation*. 2002;105(4):411-414.
85. Deshommes E, Prévost M. Pb particles from tap water: bioaccessibility and contribution to child exposure. *Environ Sci Technol*. 2012;46(11):6269-6277.
86. Deshommes E, Tardif R, Edwards M, Sauvé S, Prévost M. Experimental determination of the oral bioavailability and bioaccessibility of lead particles. *Chem Cent J*. 2012;6(1):138.
87. Jaishankar M, Tseten T, Anbalagan N, Mathew BB, Beeregowda KN. Toxicity, mechanism and health effects of some heavy metals. *Interdiscip Toxicol*. 2014;7(2):60-72.
88. Mushak P. Gastro-intestinal absorption of lead in children and adults: overview of biological and biophysico-chemical aspects. *Chem Speciation Bioavailability*. 1991;3(3-4):87-104.
89. Lentini P, Zanoli L, Granata A, Signorelli SS, Castellino P, Dell'Aquila R. Kidney and heavy metals - the role of environmental exposure (review). *Mol Med Rep*. 2017;15(5):3413-3419.
90. Barry PS. Concentrations of lead in the tissues of children. *Br J Ind Med*. 1981;38(1):61-71.
91. Hopkins MR, Ettinger AS, Hernández-Avila M, et al. Variants in iron metabolism genes predict higher blood lead levels in young children. *Environ Health Perspect*. 2008;116(9):1261-1266.
92. Replogle RA, Li Q, Wang L, Zhang M, Fleet JC. Gene-by-diet interactions influence calcium absorption and bone density in mice. *J Bone Miner Res*. 2014;29(3):657-665.

93. Bonny O, Bochud M. Genetics of calcium homeostasis in humans: continuum between monogenic diseases and continuous phenotypes. *Nephrol Dial Transplant*. 2014;29(Suppl 4):iv55-iv62.
94. Asgharian B, Ménache MG, Miller FJ. Modeling age-related particle deposition in humans. *J Aerosol Med*. 2004;17(3):213-224.
95. Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, Fomon SJ. Absorption and retention of lead by infants. *Pediatr Res*. 1978;12(1):29-34.
96. James HM, Hilburn ME, Blair JA. Effects of meals and meal times on uptake of lead from the gastrointestinal tract in humans. *Hum Toxicol*. 1985;4(4):401-407.
97. Liu J, Xu X, Wu K, et al. Association between lead exposure from electronic waste recycling and child temperament alterations. *Neurotoxicology*. 2011;32(4):458-464.
98. Elias SM, Hashim Z, Marjan ZM, Abdullah AS, Hashim JH. Relationship between blood lead concentration and nutritional status among Malay primary school children in Kuala Lumpur, Malaysia. *Asia Pac J Public Health*. 2007;19(3):29-37.
99. Blake KC, Mann M. Effect of calcium and phosphorus on the gastrointestinal absorption of 203Pb in man. *Environ Res*. 1983;30(1):188-194.
100. Walter SD, Yankel AJ, von Lindern IHv. Age-specific risk factors for lead absorption in children. *Arch Environ Health*. 1980;35(1):53-58.
101. Dignam TA, Lojo J, Meyer PA, Norman E, Sayre A. Reduction of elevated blood lead levels in children in North Carolina and Vermont, 1996–1999. *Environ Health Perspect*. 2008;116(7):981-985.
102. Leggett RW. An age-specific kinetic model of lead metabolism in humans. *Environ Health Perspect*. 1993;101(7):598-616.
103. Todd AC, Wetmur JG, Moline JM, Godbold JH, Levin SM, Landrigan PJ. Unraveling the chronic toxicity of lead: an essential priority for environmental health. *Environ Health Perspect*. 1996;104(Suppl 1):141-146.
104. Del Rio M. *Sta bility of Blood Lead Levels in Children with Chronic Low-Level Lead Absorption*. Dissertation. The University of Texas at El Paso; 2021.
105. Child Lead Poisoning Prevention Program Geographic Information System Workgroup. *Using GIS to Assess and Direct Childhood Lead Poisoning Prevention: Guidance for State and Local Childhood Lead Poisoning Prevention Programs*. Washington, D.C Centers for Disease Control and Prevention; 2004.

106. Dietrich KN, Ware JH, Salganik M, et al. Effect of chelation therapy on the neuropsychological and behavioral development of lead-exposed children after school entry. *Pediatrics*. 2004;114(1):19-26.

Funding/Support:

Conflicts of Interest: The authors declare they have nothing to disclose.

Address correspondence to: Christina Sobin, The University of Texas at El Paso, 500 West University Ave, College of Health Sciences and School of Nursing Building, Rm 401, El Paso, TX 79968 (email: casobin@utep.edu).