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MANAGEMENT

MEMORANDUM

OLEM Directive 9285.6-54

SUBJECT: Recommendations for Assessing Short-Term Exposure Scenarios Involving Lead at Superfund Sites

FROM: Dana Stalcup, Director 
Assessment and Remediation Division
Office of Superfund Remediation and Technology Innovation

TO: Superfund National Program Managers, Regions 1 - 10

The purpose of this memorandum is to transmit the Technical Review Workgroup for Metals and Asbestos (TRW) Technical document entitled "Recommendations for Assessing Short-Term Exposure Scenarios Involving Lead at Superfund Sites." This document describes an option for assessing short-term exposures to lead-contaminated media.

Neither the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEBUK) nor the Adult Lead Model (ALM) was designed to assess short-term (exposure for less than 90 days), periodic (exposure less frequently than 1 exposure per 1 week), or acute (less than 14 days) exposures. A minimum of 90 days is considered to be the minimum exposure to produce a quasi-steady-state blood lead concentration.

For situations where short-term exposures are expected, the model exposure scenario may need to be adjusted to meet the minimum exposure frequency and duration, or alternative modeling approaches may be explored. When alternative approaches are employed, users are encouraged to characterize the results in terms of resulting peak blood lead concentration from the exposure and risk of exceeding a range of blood lead concentrations (*e.g.*, 5, 10, 15, and 20 ug/dl) during peak blood lead concentration and the duration of the elevated blood lead concentration.

This report contains case studies as examples.

This report and other efforts related to addressing lead in soil can be found on the Internet at <https://www.epa.gov/superfund/lead-superfund-sites-technical-assistance>. Please contact Michele Burgess at Burgess.Michele@epa.gov or (703) 603-9003 if you have questions or concerns.

Attachment

1. "Recommendations for Assessing Short-Term Exposure Scenarios Involving Lead at Superfund Sites."

cc: Mathy Stanislaus, OLEM
Nitin Natarajan, OLEM
Barry Breen, OLEM
Reggie Cheatham, OLEM/OEM
Barnes Johnson, OLEM/ORCR
David Lloyd, OLEM/OBLR
Charlotte Bertrand, OLEM/FFRRO
Carolyn Hoskinson, OLEM/OUST
Cyndy Mackey, OECA/OSRE
Richard Albores, OECA/FFEO
John Michaud, OGC/SEWRLO
OSRTI Managers
Regional Superfund Branch Chiefs, Regions 1 – 10
Kristin Giacalone, Superfund Lead Region Coordinator, Region 2
NARPM Co-Chairs
TRW Committee Members

Recommendations for Assessing Short-Term Exposure Scenarios Involving Lead at Superfund Sites

Background

Case reports of acute lead poisoning from accidental or intentional ingestion of lead-containing products have been associated with health effects in adults and children (*e.g.*, Abraham et al., 2002; Barber and Jacyna, 2011; Krishnan et al., 2012; Ogawa et al., 2008; Toto et al., 2012). The Agency for Toxic Substances and Disease Registry (ATSDR) (2007) reported an acute (≤ 14 days) oral lowest-observed-adverse-effect level (LOAEL) of approximately 0.02 mg/kg-day for decreases in aminolevulinate dehydratase activity based on two studies using human volunteers (Cools et al., 1976; Stuik, 1974).¹ Thus, acute exposure to lead can result in adverse health effects in children and adults, and thus the ability to accurately predict short-term blood lead concentration (PbB) may be important for certain hazardous waste sites.

Neither the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model, U.S. EPA 2010) nor the Adult Lead Methodology (ALM, U.S. EPA, 2003b) was designed to assess short-term (exposure for less than 90 days), periodic (exposure less frequently than 1 exposure per 1 week), or acute (≤ 14 days) exposures. Instead, the IEUBK model and the ALM simulate PbB associated with continuous exposure of sufficient duration to result in a quasi-steady state (U.S. EPA, 1994, 1996). Based on estimates of the first-order elimination half-time for lead in blood of approximately 30 days for adults and children (U.S. EPA, 2003b), a constant lead intake rate over a duration of 90 days would be expected to achieve a PbB that is sufficiently close to the quasi-steady state. Infrequent and non-continuous exposures (*i.e.*, less than 1 day per week over a minimum duration of 90 days) would be expected to produce oscillations in PbB associated with the absorption and subsequent clearance of PbB between each exposure event. Thus, the IEUBK model and the ALM can only provide an approximation of a quasi-steady-state PbB concentration for periodic exposures. These approximations may also be underestimated if short-term exposures are time averaged over the entire year (Lorenzana et al., 2005; U.S. EPA, 2003a).²

¹ In addition, U.S. EPA's Integrated Science Assessment for Lead (2013) reported that studies in rats suggest that there is the potential for an increase in blood pressure following short-term lead treatment (daily injections for one week or two weeks of drinking water exposure). It is possible that the increases in blood pressure following short- and long-term lead exposures are occurring through separate mechanisms; however, studies using both short- and longer-term lead exposure have correlated increased blood pressure with an activation of the renin-angiotensin system (*i.e.*, increase in angiotensin converting enzyme activity). (Fiorim et al., 2011; Sharifi et al., 2004; Simões et al., 2011; as cited by U.S. EPA, 2013).

² For non-residential settings, the TRW Lead Committee recommends a default adult lead exposure frequency (EF) value of 219 days/year for the ALM. This estimate corresponds to the average time spent at work by both full-time

Considerations for Modeling the Exposure Scenario

For exposure scenarios at Superfund sites that do not meet the recommended minimum exposure frequency and duration (i.e., less than 1 day per week and of duration shorter than 90 consecutive days.) for either the IEUBK model or ALM (U.S. EPA, 2003a), users have the option of modifying the exposure scenario to meet the minimum or using an alternative model to predict PbB associated with the short-term of intermittent exposure. Several models of lead pharmacokinetics are available that could be used to assess shorter exposure durations (Bert et al., 1989; Leggett, 1993; O'Flaherty, 1993; Rabinowitz et al., 1976). While none of these have been broadly accepted by EPA for use in supporting regulatory decisions at Superfund sites, they have the functionality to simulate PbB concentrations associated with acute, short-term, or highly intermittent exposure scenarios. One model in particular, the International Commission on Radiological Protection (ICRP, 1994) model (which is based on the Leggett model), has been used to explore relationships between intermittent exposures and blood lead concentrations (Abrahams et al., 2006; Khoury and Diamond, 2003; Lorenzana et al., 2005).³ This model has been particularly useful for these types of assessments because it was designed to simulate daily or weekly intakes of Pb and associated biokinetics. EPA lead risk assessment models (IEUBK and ALM) have shown that parameter values in the ICRP model can be set to predict similar profiles of blood lead concentrations to the IEUBK model for similar exposure scenarios (Khoury and Diamond, 2003). Thus, with some caution the ICRP model may be used to explore alternative exposure scenarios where exposure duration is less than 90 days. While these alternative approaches may be used, none of these are readily available to risk assessors for Superfund risk assessments and are only considered acceptable for research applications. The EPA is developing an All-Ages Lead Model (AALM, U.S. EPA, 2005) that will allow users to implement the Leggett and O'Flaherty models explore short-term exposure scenarios.

and part-time workers engaged in contact intensive activities (based on 1991 data from the Bureau of Labor Statistics). The time-weighted approach assumes that the adverse health effects of lead are related to long-term average PbB concentrations. While this has been established for chronic effects of lead, the health effects (acute or chronic) of elevated PbB levels that occur after acute exposures resulting in short-term PbB concentrations less than 20 µg/dL are not well understood. As a result, the temporary increase in PbB lead concentration that occurs following intermittent exposure may be underestimated when using a time-weighted average approach (EPA, 2003a).

³ In general it is recommended that users select a model that allows a time step of 1/10th (or smaller) than the exposure period of interest.

Considerations for EPA's Risk Reduction Goal for Soil Lead Exposures

The EPA's health protection goal (U.S. EPA, 1994, 1998) is intended for long-term exposures to lead, EPA has not established risk-based targets for short-term exposures (*i.e.*, the long-term health protection goal of 5% not to exceed 10 µg/dL established by the Superfund program [U.S. EPA, 1994, 1998]). Thus this health goal may not necessarily be appropriate for acute or highly intermittent exposures.⁴ When establishing cleanup goals for hazardous waste sites where intermittent exposures occur, risk managers should consider the potential for adverse health effects that might result from short-term (weeks or months) increases in PbB, since increased levels of PbB over short-temporal periods can have adverse effects on children (U.S. EPA, 2006). While the scientific evidence has not established a minimum duration of lead exposure that is without risk of adverse health effects, research has demonstrated that acute increases in PbB can occur in association with short-term increases in lead intake, and that concurrent PbB is more strongly associated with IQ losses than other temporal measurements (*e.g.*, the average PbB over the child's lifetime up to the time of the IQ test, or the highest PbB measured over that time period) (U.S. EPA, 2006). These studies suggest that increased levels of lead intake over short-temporal periods (*e.g.*, a few months) may pose risk of neurocognitive effects to children (U.S. EPA, 2006). In addition, the evidence demonstrates increased vulnerability to adverse effects of lead during some relatively short developmental periods (*e.g.*, prenatal), which further suggests the importance of exposure periods as short as a few months (U.S. EPA, 2006). Hence, although short-term lead exposures cannot be adequately modeled with existing EPA risk assessment tools (*i.e.*, IEUBK model, ALM) or evaluated against a risk reduction goal, consideration should be given to preventing short-term exposures to high concentrations of lead in environmental media.

Limitations for Exposure Scenarios Involving Pica

The IEUBK model was not designed to assess the health impact of pica events (or geophagy which is the intentional consumption of soil). Pica behavior violates assumptions of modeling using the IEUBK model. The IEUBK model is based on the assumption that soil-dust ingestion is due to inadvertent ingestion of particles that adhere to hands, etc. If soil is intentionally ingested (geophagy), then a larger quantity of soil is consumed and the particle size may also be larger. In addition, pica

⁴ The IEUBK model should be run using an appropriate level of concern for your site. Since 1994, OSWER programs have utilized a risk reduction goal of limiting exposure to soil lead levels such that children would have no more than 5% risk of exceeding a PbB of 10 µg/dL. Until EPA revises this policy, that risk reduction goal is appropriate to support Superfund site cleanup decisions. The Centers for Disease Control and Prevention (CDC, 2012) adopted the 97.5th percentile PbB from National Health and Nutrition Examination Survey (NHANES) (currently 5 µg/dL) as a reference value to target intervention for individual children and communities with PbB at and above that concentration. That PbB reference value may be used as a health goal for Superfund sites where it is considered appropriate and there are sufficient resources available.

(geophagy) behavior is typically highly intermittent and does not meet the minimum exposure frequency and duration necessary to use the IEUBK model.

While other models may accept the highly intermittent nature of pica behavior (*i.e.*, as discussed earlier, Leggett and O'Flaherty can simulate highly intermittent exposure scenarios), these models have not been adequately evaluated for simulating the biokinetics that would follow ingestion of very high amounts of lead (that may be ingested through pica). At these very high intakes of Pb and soil, the absorption fraction assumptions in the IEUBK model and the ALM would likely have to be adjusted (since they are based on much lower intakes) because the assumptions in these models is that the absorption fraction is independent of both Pb and soil intake.

Also, because pica behavior is different from the typical exposure pathway for soil ingestion (incidental ingestion is soil-dust adhered to hands), the sieving recommendations and bioavailability assumptions typically used for soil may not apply.

Limitations for Assessing Intermittent Airborne Lead Exposures

If the objective is also to simulate absorption of inhaled lead, the IEUBK model is not the best tool. This conclusion is especially true when the air exposures are highly variable. Inhaled particles deposit in the different regions of the respiratory tract as a function of particle size (U.S. EPA, 2013). Lead associated with smaller particles, which are predominantly deposited in the pulmonary region, may (depending on solubility) be absorbed into the general circulation or be transported via phagocytic cells to the gastrointestinal tract (U.S. EPA, 2013). Lead associated with larger particles that are predominantly deposited in the upper and large airways (*e.g.*, nasal pharyngeal and tracheobronchial regions of respiratory tract), may be transported by mucocilliary transport into the esophagus and swallowed, thus also making its way to the gastrointestinal tract (U.S. EPA, 2013). The IEUBK model parameter for absorption of inhaled Pb is intended to represent the combined outcome of above processes. The default value, 32%, reflects a specific particle size distribution that was considered to represent exposures to airborne Pb exposure from an active smelter (U.S. EPA, 1989) See discussion in Section 4.1.10 of the 1994 Guidance Manual (U.S. EPA, 1994).

Particle size can influence deposition in the lung and subsequent absorption (pulmonary or gastric) (U.S. EPA, 2013). Also, the form of lead can influence absorption (U.S. EPA, 2013). In general, the IEUBK model and ALM were not designed to address this level of complexity in airborne lead exposures. For example, small particles with greater surface area may have greater absorption in the lung, and large particles may deposit in the upper and large airways of the respiratory tract and be swallowed. If detailed information is available on particle size fractions and lead concentration in the particle size

fractions, alternative models such as the ICRP model, which implements the Human Respiratory Tract model (ICRP, 1994), and Leggett's biokinetic model (Leggett, 1993), could be used to explore how that information impacts lead intake and uptake. These tools offer more options for modeling air exposures to humans and may be more appropriate for some sites.

Khoury and Diamond (2003) describe one approach to estimating soil lead levels from air lead levels, using the blood lead concentration that would trigger medical monitoring as a benchmark for exposure levels in air to be avoided for acute exposures. See also the National Ambient Air Quality Standards (NAAQS) risk assessment (U.S. EPA, 2007) which discusses the NAAQS 3-month rolling average as an air lead concentration to be avoided for airborne lead exposure.

Recommendations

Based on the observation of the acute rise in PbB following an exposure and because the pharmacokinetics of lead indicate that lead is readily transferred from the blood to the nervous system, it is recommended that acute, short-term exposures be assessed and addressed where possible.

Because of the limitations in the Superfund risk assessment tools, the TRW Lead Committee recommends that users not apply the IEUBK model or the ALM to assess exposure frequency less than 1 day per week and of duration shorter than 90 consecutive days. Ninety days is considered to be the minimum exposure to produce a quasi-steady-state PbB concentration (U.S. EPA, 2003b). The reliability of these models for predicting PbB concentrations for exposure durations less than 90 consecutive days has not been assessed (U.S. EPA, 2003a). For such situations, the exposure scenario may need to be adjusted to meet the minimum exposure frequency and duration (as discussed in U.S. EPA, 2003a), or alternative modeling approaches may be explored. When alternative approaches are employed, users are encouraged to characterize the results in terms of peak PbB that results from the exposure as well as risk of exceeding a range of PbB (such as 5, 10, 15, and 20 $\mu\text{g/dL}$) during the peak PbB and the duration of the elevated PbB. The magnitude and duration of the peak PbB resulting from the acute exposure may be considered in terms of a response action. Several case studies are provided below as examples.

Case Study No. 1: Goose Meal

Exposure Scenario/Objective

The objective was to determine the impact on PbB from consuming a goose where the meat was contaminated with lead. For the simulation, two meals were assumed given a two pound annual limit for the goose meat distribution program (hunters providing game to food banks). Because the exposure does not meet the exposure frequency (EF) and exposure duration (ED) minimum for the IEUBK model, the ICRP model was used.

Approach

The mean lead level in goose breast meat was 0.37 ppm (wet weight), and the 95th percentile is 1.5 ppm. Exposure assumptions were two meals a year (reasonable given the limited availability of this food) at 90 grams (wet weight) per meal (total meat consumption rate for a 3–6-year old child from Exposure Factors Handbook [U.S. EPA, 2011], assuming that all meat consumed on those days would be from the goose). This yields approximately 40 µg Pb/meal or 150 µg Pb/meal. Variables from the IEUBK model were used as inputs for the ICRP model.

Bioavailability of lead in the goose was assumed to be 30% at age 2-7 years, the ICRP model default.

Results

Figures 1a–d show the predicted PbB for 2- to 7-year old children for consumption of goose meat at 40 or 150 µg Pb/day for two consecutive days with different baseline PbB concentrations (1 or 2.5 µg/dL).

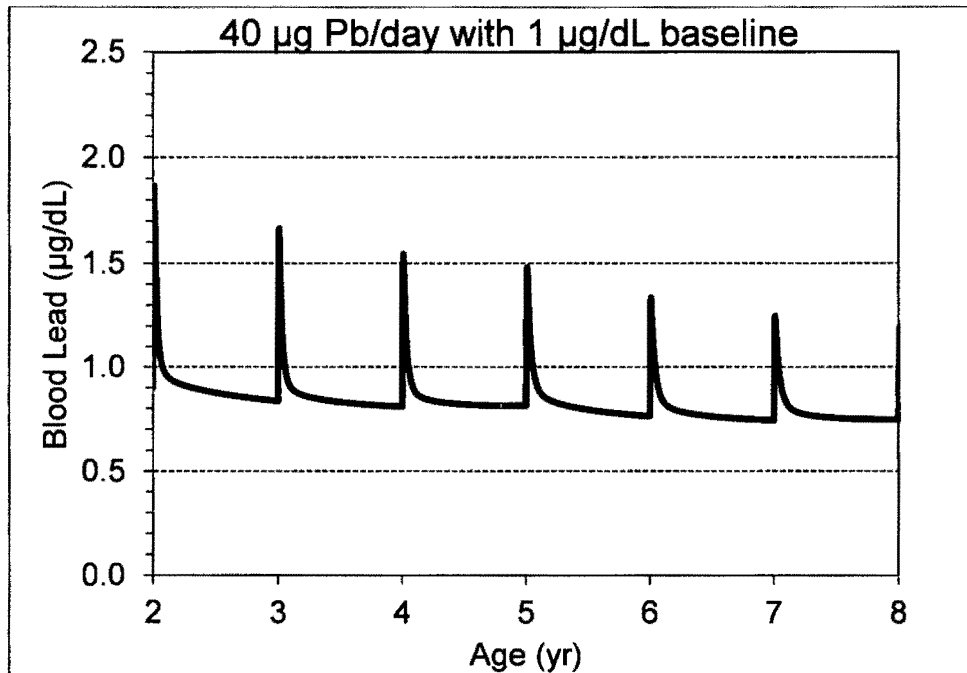


FIGURE 1a. Plot shows the predicted relationship between blood lead concentration (PbB) for 2- to 7-year old children for consumption of goose meat at 40 µg Pb/day for two consecutive days with baseline PbB concentration 1 µg/dL.

IEUBK Model Variables

Pb Soil Res: 200 µg/g

Pb Air Res: 0.1 µg/m³

MSD: 0.7

Dust/air: 100

Soil/dust: 0.45

Pb Dust Res: 150 µg/g

IR SD Res: 0.1 g/day

IN Pb SD Res: 17.25 µg/day

RAC: 0.485

IN non-Soil BKG: 3.2 µg/day

IN Pb SD Res: 0 µg/day

IN Pb Res: 3.2 µg/day

Start Site: 1095 day

ED site: 14 days

End Site: 1109 day

ED site 24: hr

Pb Air site: 0.15 µg/m³

V: 0.445 m³/hr

IN Pb Air Site: 1.6 µg/day

Pb Pb Soil Site: 623 µg/g

IR Pb Soil Site: 0.1 g/day

IN Pb Soil Site: 62.3 µg/day

IN Pb Soil Site: µg/day

Pb Soil Site: 0 µg/g

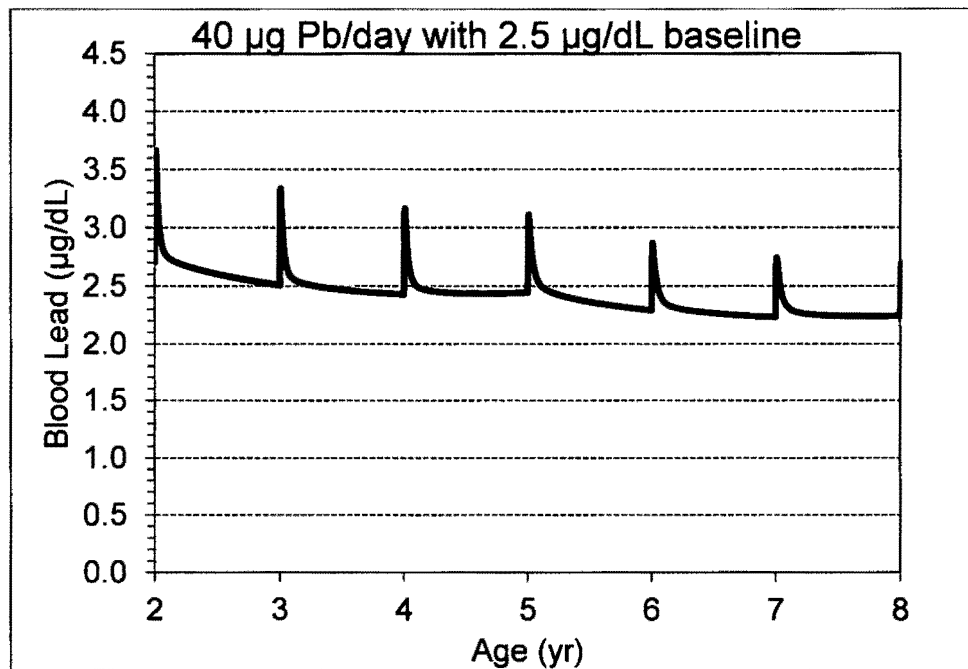


FIGURE 1b. Plot shows the predicted relationship between blood lead concentration (PbB) for 2- to 7-year old children for consumption of goose meat at 40 µg Pb/day with soil ingestion for two consecutive days with baseline PbB concentration 2.5 µg/dL.

IEUBK Model Variables

Pb Soil Res: 200 µg/g
Pb Air Res: 0.1 µg/m³
MSD: 0.7
Dust/air: 100
Soil/dust: 0.45
Pb Dust Res: 150 µg/g
IR SD Res: 0.1 g/day
IN Pb SD Res: 17.25 µg/day
RAC: 0.485
IN non-Soil BKG: 3.2 µg/day
IN Pb SD Res: 13.6 µg/day
IN Pb Res: 16.8 µg/day
Start Site: 1095 day
ED site days: 14
End Site: 1109 day
ED site hr: 24
Pb Air site: 0.15 µg/m³
V: 0.45 m³/hr
IN Pb Air Site: 1.6 µg/day
Pb Pb Soil Site: 623 µg/g
IR Pb Soil Site: 0.1 g/day
IN Pb Soil Site: 62.3 µg/day
IN Pb Soil Site: µg/day
Pb Soil Site: 0 µg/g

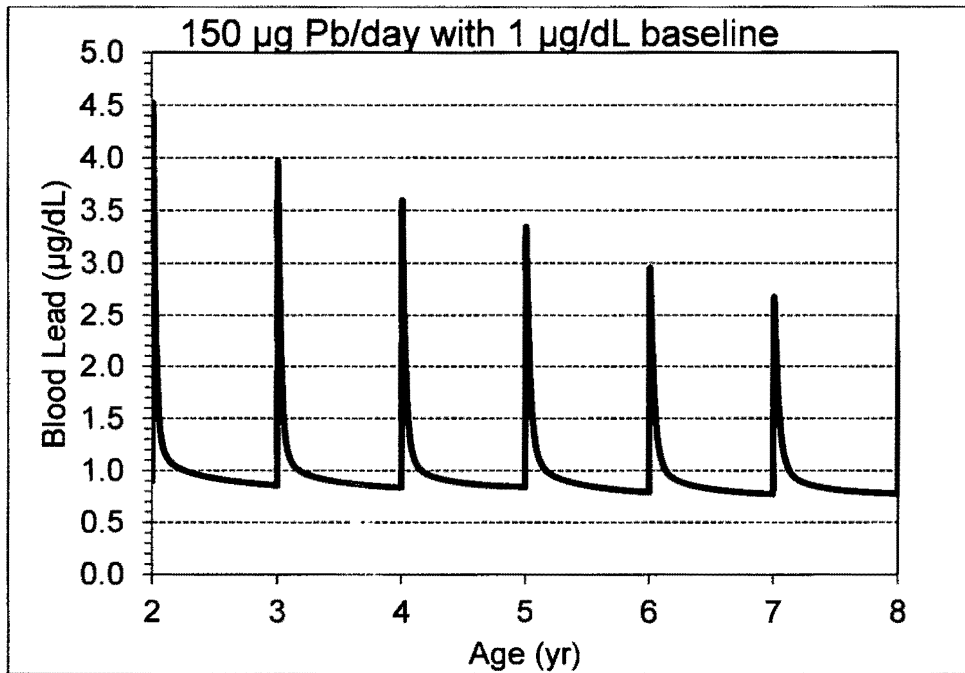


FIGURE 1c. Plot shows the predicted relationship between blood lead concentration (PbB) for 2- to 7-year old children for consumption of goose meat at 150 µg Pb/day for two consecutive days with baseline PbB concentration 1 µg/dL.

IEUBK Model Variables

Pb Soil Res: 200 µg/g

Pb Air Res: 0.1 µg/m³

MSD: 0.7

Dust/air: 100

Soil/dust: 0.45

Pb Dust Res: 150 µg/g

IR SD Res: 0.1 g/day

IN Pb SD Res: 17.25 µg/day

RAC: 0.485

IN non-Soil BKG: 3.2 µg/day

IN Pb SD Res: 0 µg/day

IN Pb Res: 3.2 µg/day

Start Site: 1095 day

ED site: 14 days

End Site: 1109 day

ED site: 24 hr

Pb Air site: 0.15 µg/m³

V: 0.445 m³/hr

IN Pb Air Site: 1.6 µg/day

Pb Pb Soil Site: 623 µg/g

IR Pb Soil Site: 0.1 g/day

IN Pb Soil Site: 62.3 µg/day

IN Pb Soil Site: µg/day

Pb Soil Site: 0 µg/g

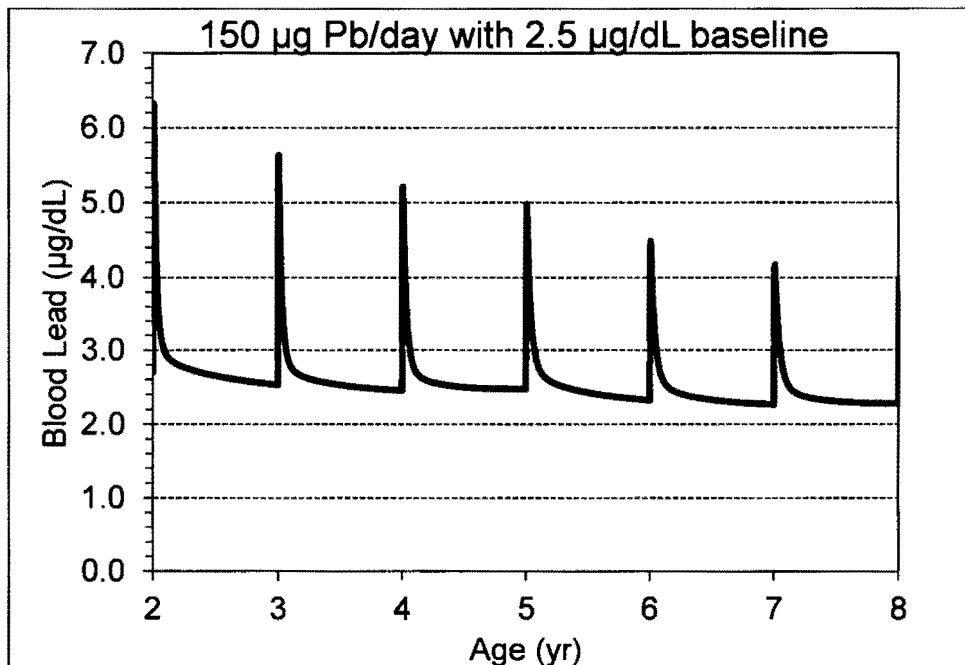


FIGURE 1d. Plot shows the predicted relationship between blood lead concentration (PbB) for 2- to 7-year old children for consumption of goose meat at 150 µg Pb/day with soil ingestion for two consecutive days with different baseline PbB concentration 2.5 µg/dL.

IEUBK Model Variables

Pb Soil Res: 200 µg/g
Pb Air Res: 0.1 µg/m³
MSD: 0.7
Dust/air: 100
Soil/dust: 0.45
Pb Dust Res: 150 µg/g
IR SD Res: 0.1 g/day
IN Pb SD Res: 17.25 µg/day
RAC: 0.485
IN non-Soil BKG: 3.2 µg/day
IN Pb SD Res: 13.6 µg/day
IN Pb Res: 16.8 µg/day
Start Site: 1095 day
ED site: 14 days
End Site: 1109 day
ED site: 24 hr
Pb Air site: 0.15 µg/m³
V: 0.445 m³/hr
IN Pb Air Site: 1.6 µg/day
Pb Pb Soil Site: 623 µg/g
IR Pb Soil Site: 0.1 g/day
IN Pb Soil Site: 62.3 µg/day
IN Pb Soil Site: µg/day
Pb Soil Site: 0 µg/g

Case Study No. 2: Short-term Childhood Exposure to Lead⁵

Exposure Scenario/Objective

The objective of this example was to derive acute soil criteria for lead for residential exposure scenarios based on a target blood lead concentration of 20 µg/dL (*i.e.*, not more than 5% of the exposed target population having a total blood lead concentration exceeding 20 µg/dL). The target receptor for the residential soil criterion was a young child (6-months to 7-years old). The target duration of exposure was 30 days or less (simulating exposure to a breached cap over contaminated soil).

The 20 µg/dL target PbB was based on an interpretation of the CDC recommendation that PbB levels in the range of 20–44 µg/dL would result in a home visit by a public health agency within 24 hours of the referral from a physician. This interpretation contrasts the response recommended with the report of PbB in the range of 15–19 µg/dL where a home visit is initiated only with persistent elevation as defined by two consecutive blood lead measurements taken more than 3 months apart. Thus, 20 µg/dL could be considered as a short-term elevation in PbB that would trigger an action (a consequence to be avoided).

Approach

Under this exposure scenario, steady-state conditions are not achieved. The exposure scenario does not meet the minimum for exposure frequency (EF) and exposure duration (ED) minimum for the IEUBK model (U.S. EPA, 2009). Therefore, the IEUBK model is not appropriate for use under the acute exposure scenario. EPA is currently developing the AALM. The AALM provides a more appropriate basis for estimating the relationship between soil lead and PbB under the acute exposure scenario. When this simulation was run, an earlier version that was similar to the ICRP model was used. This model is also referred to as the Leggett (1993) model. This model is a dynamic physiologically-based pharmacokinetic (PBPK) model that calculates lead concentration in various tissues from a variety of media as a one-day step function. It is, therefore, able to predict the PbB resulting from short-term exposures. Although EPA has not finalized several exposure related aspects of the overall model package, the PBPK portion of the model is well established (U.S. EPA, 2005).

The ICRP model was used to simulate the acute scenario, specifically: 400 mg soil/day; 1-, 10-, and 30-day exposure duration; 2-year old child; absorption fraction from soil = 0.5 (default assumption); and background PbB = 1.5 µg/dL. Although the model can accommodate lead exposures from multiple

⁵ Adapted from “Report of the NJ Department of Environmental Protection Science Advisory Board Standing Committee on Public Health in Response to the Charge Questions on Development of Acute Soil Criteria” (NJDEP, 2011). The NJDEP SAB report was accepted by the full SAB and has been posted on the NJDEP web site (<http://www.state.nj.us/dep/sab/final-acute-soil-standards-report.pdf>). The recommendations reflect the SAB comments and not necessarily the policy of the NJDEP.

environmental media and routes of exposure, the model calculations in this case were restricted to ingestion of soil. For these conditions, the model predicts a linear relationship between soil lead concentration and PbB (see Figures 2a–c). The results are provided for both the peak and mean PbB. For the same duration of exposure and the same target blood concentration, the mean PbB is achieved with a higher soil concentration than the peak PbB. This difference is because each single daily intake of Pb results in a daily oscillation in blood Pb (see Figure 1a) as absorbed Pb is eliminated between doses. The mean reflects the PbB profile over the averaging time, which was in each case, exposure period. The soil concentration corresponding to the mean and peak converge as the exposure duration (and averaging time) decrease (see Figure 2c). Thus, at the time that the peak PbB is achieved, the mean concentration reflects the lower concentrations at each time point leading up to the peak concentration.

Results

Figures 2a–c show the predicted PbB for a 2-year old over a range of lead daily intakes for exposures of 30, 10, and 1 days.

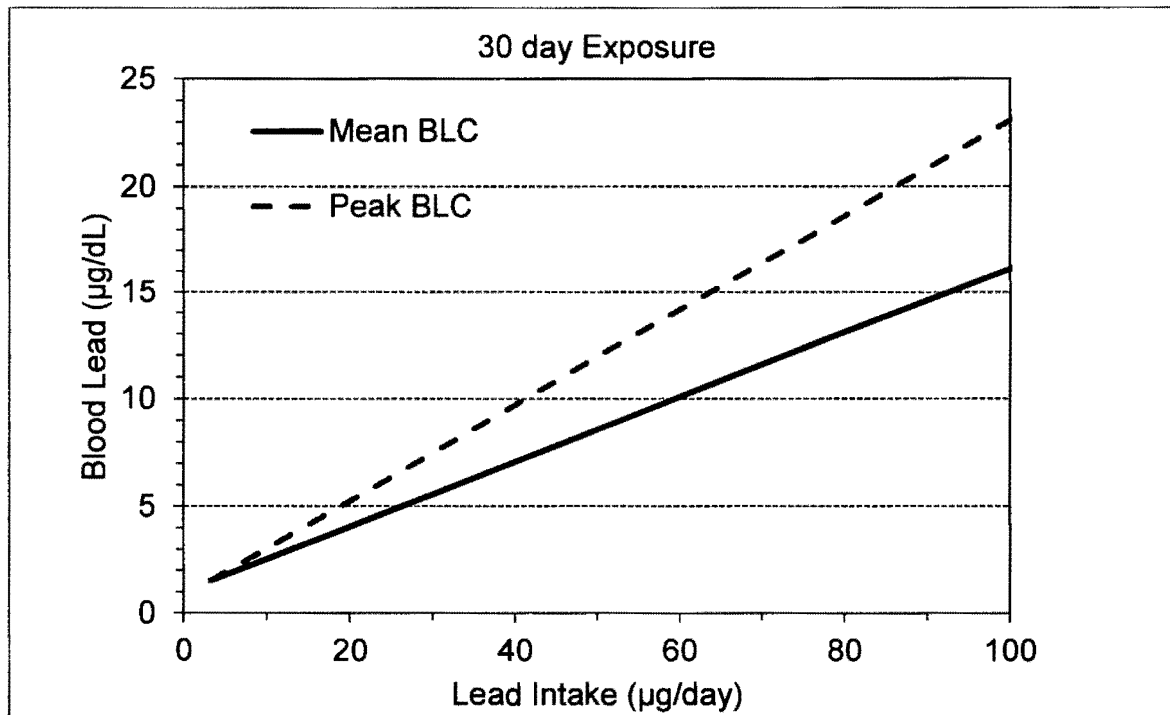


Figure 2a. Plot shows predicted relationship between lead intake (μg lead ingested/day) and blood lead concentration (BLC) for a 30-day exposure beginning at age 2 years. Mean BLC: mean age 730–760 days. Peak BLC: peak age 730–760 days.

ICRP Model Variablese

Air exposure: zero

Ingest baseline: $3.2 \mu\text{g/day}$ (PbB at age 730 days = $1.5 \mu\text{g/dL}$)

Short-term exposure: $0 - 100 (+3.2) \mu\text{g/day}$

Short-term exposure duration: 30 days (age 730–760 days)

Absorption fraction: 0.5

Soil ingestion rate: 400 mg/day

Soil lead concentration: total ingestion (baseline + short-term exposure)/soil ingestion rate

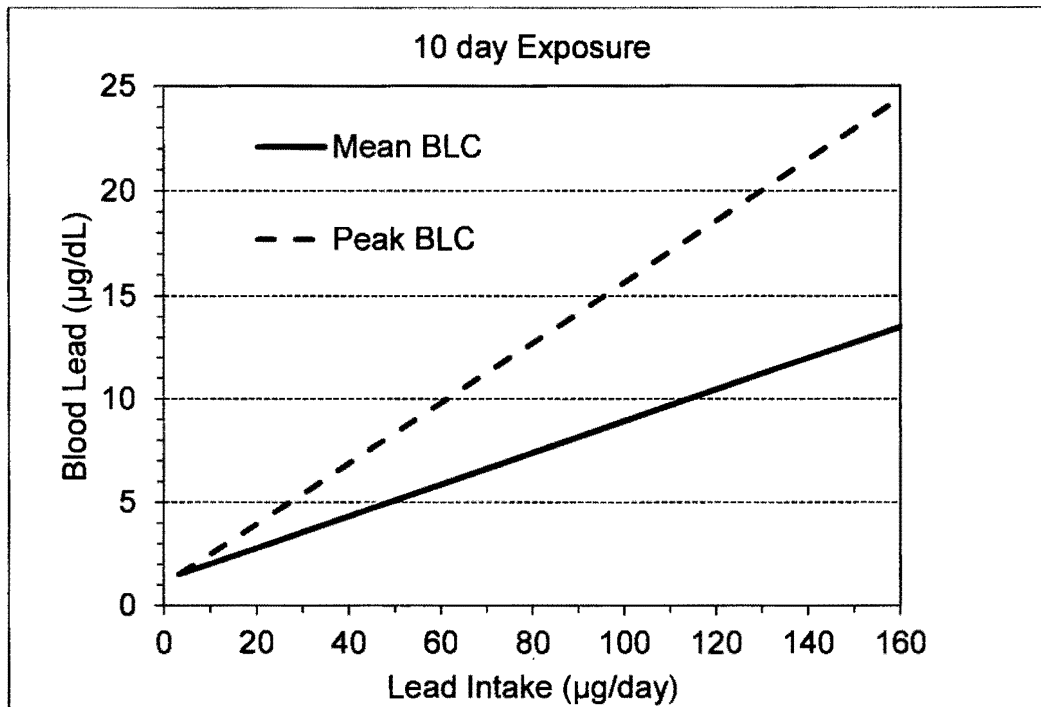


Figure 2b. Plot shows predicted relationship between lead intake (μg lead ingested/day) and blood lead concentration (BLC) for a 10-day exposure beginning at age 2 years. Mean BLC: mean age 730–740 days. Peak BLC: peak age 730–740 days.

ICRP Model Variables

Air exposure: zero

Ingest baseline: $3.2 \mu\text{g/day}$ (PbB at age 730 days = $1.5 \mu\text{g/dL}$)

Short-term exposure: $0 - 100 (+3.2) \mu\text{g/day}$

Short-term exposure duration: 10 days (age 730–740 days)

Absorption fraction: 0.5

Soil ingestion rate: 400 mg/day

Soil lead concentration: total ingestion (baseline + short-term exposure)/soil ingestion rate

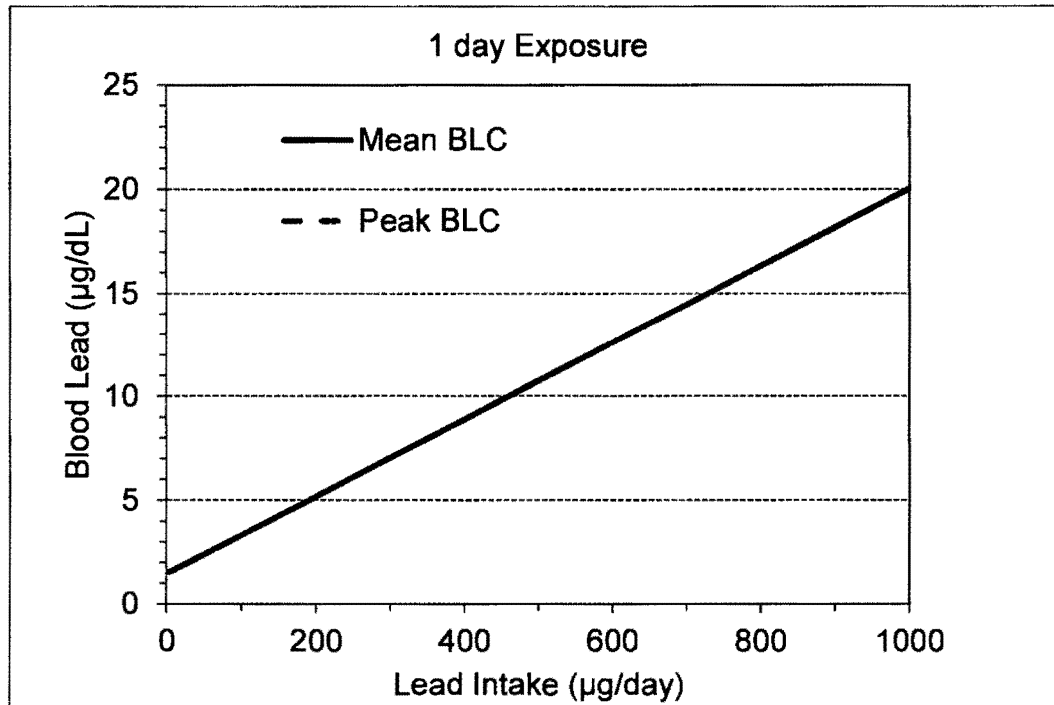


Figure 2c. Plot shows predicted relationship between lead intake (μg lead ingested/day) and blood lead concentration (BLC) for a 1-day exposure beginning at age 730 days. Mean BLC: mean for age 2 years, 730–731 days. Peak BLC: peak age 730–731 days.

ICRP Model Variables

Air exposure: zero

Ingest baseline: $3.2 \mu\text{g/day}$ (blood lead concentration at age 730 days = $1.5 \mu\text{g/dL}$)

Short-term exposure: 0 – 1,000 ($+3.2$) $\mu\text{g/day}$

Short-term exposure duration: 30 days (age 730–731 days)

Absorption fraction: 0.5

Soil ingestion rate: 400 mg/day

Soil lead concentration: total ingestion (baseline + short-term exposure)/soil ingestion rate

References

- Abraham, R. Loomba, R., and Pandian, J.D. 2002. δ -ALA levels in serum and urine – A diagnostic tool for possible lead poisoning. *Indian J. Clin. Biochem.* 17(2): 64–67.
- Abrahams, P.W., Follansbee, M.H., Hunt, A., Smith, B., and Wragg, J. 2006. Iron nutrition and possible lead toxicity: An appraisal of geophagy undertaken by pregnant women of UK Asian communities. *Appl. Geochem.* 21(1): 98–108.
- ATSDR (Agency for Toxic Substances and Disease Registry). 2007. Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry: Atlanta, GA. August. Available online at: <http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>.
- Barber, T. and Jacyna, M. 2011. Acute lead intoxication from medications purchased online presenting with recurrent abdominal pain and encephalopathy. *J. R. Soc. Med.* 104(3):120–123.
- Beck, B.D., Mattuck, R.L., Bowers, T.S., Cohen, J.T., and O'Flaherty, E. 2001. The development of a stochastic physiologically-based pharmacokinetic model for lead. *Sci. Total Environ.* 274(1-3): 15–19.
- Bert, J.L., Van Dusen, L.J. and Grace, J.R. 1989. A generalized model for the prediction of lead body burdens. *Environ. Res.* 48(1): 117–127.
- CDC (Centers for Disease Control and Prevention). 2012. CDC Recommendations on Children's Blood Lead Levels (BLLs). Centers for Disease Control and Prevention: Atlanta, GA. Available online at: <http://www.cdc.gov/nceh/lead/data/definitions.htm>.
- Cools, A., Salle, H.J.A., Verberk, M.M., and Zielhuis, R.L. 1976. Biochemical response of male volunteers ingesting inorganic lead for 49 days. *Int. Arch. Occup. Environ. Health* 38(2): 129–139.
- Fiorim, J., Ribeiro Júnior, R.F., Silveira, E.A., Padilha, A.S., Vescovi, M.V., de Jesus, H.C., Stefanon, I., Salices, M., and Vassallo, D.V. 2011. Low-level lead exposure increases systolic arterial pressure and endothelium-derived vasodilator factors in rat aortas. *PLoS One* 6(2): e17117.
- ICRP (International Commission on Radiological Protection). 1994. Human Respiratory Tract Model for Radiological Protection. A Report of a Task Group of the International Commission on Radiological Protection. No. 66. Elsevier Science Health Science Division: Tarrytown, NY. 482 pp.
- Khoury, G. and Diamond, G. 2003. Risks to children from exposure to lead in air during remedial or removal activities at Superfund sites: A case study of the RSR lead smelter Superfund site. *J. Expo. Anal. Environ. Epidemiol.* 13(1): 51–65.
- Krishnan, A.V., Park, S.B., Huynh, W., Lin, C.S., Henderson, R.D., and Kiernan, M.C. 2012. Impaired energy-dependent processes underlie acute lead neuropathy. *Muscle Nerve* 46(6): 957–961.
- Leggett, R.W. 1993. An age-specific kinetic model of lead metabolism in humans. *Environ. Health Perspect.* 101(7): 598–616.
- Lorenzana, R.M., Troast, R., Klotzbach, J.M., Follansbee, M.H., and Diamond, G.L. 2005. Issues related to time averaging of exposure in modeling risks associated with intermittent exposures to lead. *Risk Anal.* 25(1): 169–178.

- NJDEP (New Jersey Department of Environmental Protection). 2012. Response to the Charge Questions on Development of Health-based Acute Criteria. Summary Report of the NJDEP Science Advisory Board. Available online at: <http://www.state.nj.us/dep/sab/final-acute-soil-standards-report.pdf>.
- O'Flaherty, E.J. 1993. Physiologically based models for bone-seeking elements. IV. Kinetics of lead disposition in humans. *Toxicol. Appl. Pharmacol.* 118(1): 16–29.
- Ogawa, M., Nakajima, Y., Kubota, R., and Endo, Y. 2008. Two cases of acute lead poisoning due to occupational exposure to lead. *Clin. Toxicol. (Phila)* 46(4): 332–335.
- Rabinowitz, M.B., Wetherill, G.W., and Koppel, J.D. 1976. Kinetic analysis of lead metabolism in health humans. *J. Clin. Invest.* 58(2): 260–270.
- Sharifi, A.M., Darabi, R., Akbarloo, N., Larijani, B., and Khoshbaten, A. 2004. Investigation of circulatory and tissue ACE activity during development of lead-induced hypertension. *Toxicol. Lett.* 153(2): 233–238.
- Simões, M.R., Ribeiro Júnior, R.F., Vescovi, M.V., de Jesus, H.C., Padilha, A.S., Stefanon, I., Vassallo, D.V., Salaices, M., and Fiorese, M. 2011. Acute lead exposure increases arterial pressure: Role of the Renin-Angiotensin system. *PLoS ONE* 6(4): e18730.
- Stuick, E.J. 1974. Biological response of male and female volunteers to inorganic lead. *Int. Arch. Arbeitsmed.* 33(2): 83–97.
- Toto, M., De Giacomo, A., Petruzzelli, M.G., Dicuonzo, F., Santoro, N., Del Vecchio, G.C., Craig, F., and Margari, L. 2012. Guillain-Barré-like syndrome in a child with lead poisoning. *Neuropediatrics* 43(4): 217–220.
- U.S. EPA (U.S. Environmental Protection Agency). 1989. (OAQPS Staff Paper). Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation; Report No. EPA-450/2-89/011; U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (U.S. Environmental Protection Agency). 1994. OSWER Directive: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. Office of Solid Waste and Emergency Response: Washington, DC. OSWER Directive #9355.4-12. August. Available online at: <http://epa.gov/superfund/lead/products/oswerdir.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 1996. Soil Screening Guidance: User's Guide. Office of Solid Waste and Emergency Response: Washington, DC. Publication No. 9355.4-23. July. Available online at: <http://www.epa.gov/superfund/resources/soil/ssg496.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 1998. OSWER Directive: Clarification to the 1994 Revised Interim Soil Lead (Pb) Guidance for CERCLA Sites and RCRA Corrective Action Facilities. Office of Solid Waste and Emergency Response: Washington, DC. EPA/540/F-98/030, PB98-963244, OSWER Directive #9200.4-27P. August. Available online at: <http://epa.gov/superfund/lead/products/oswer98.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 2003a. Assessing Intermittent or Variable Exposures at Lead Sites. Office of Solid Waste and Emergency Response: Washington, DC. EPA-540-R-03-008, OSWER Directive #9285.7-76. Available online at: <http://epa.gov/superfund/lead/products/twa-final-nov2003.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 2003b. Recommendations of the Technical Review Workgroup for Lead for an Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil. Technical Review Workgroup for Lead: Washington, DC. EPA-540-R-03-001.

- January. Available online at:
<http://www.epa.gov/superfund/health/contaminants/lead/products/adultpb.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 2005. All-Ages Lead Model (AALM) Version 1.05 (External Draft Report). Office of Research and Development: Research Triangle Park, NC. October. Available online at: <http://cfpub.epa.gov/ncea/cfm/recorddisplay.cfm?deid=139314>.
- U.S. EPA (U.S. Environmental Protection Agency). 2006. Air Quality Criteria Document for Lead. National Center for Environmental Assessment, Office of Research and Development: Research Triangle Park, NC. EPA/600/R-05/144aF. October. Available online at: <http://cfpub.epa.gov/ncea/cfm/recorddisplay.cfm?deid=158823>.
- U.S. EPA (U.S. Environmental Protection Agency). 2007. Lead: Human Exposure and Health Risk Assessments for Selected Case Studies. Office of Air Quality Planning and Standards: Research Triangle Park, NC. EPA-452/R-07-014a. October. Available online at: http://www.epa.gov/ttn/naaqs/standards/pb/data/20071101_pb_ra_body.pdf.
- U.S. EPA (U.S. Environmental Protection Agency). 2010. Integrated Exposure Uptake Biokinetic Model for Lead in Children, Windows® version (IEUBKwin v1.1 build 11). Office of Superfund Remediation and Technology Innovation: Washington, DC. February. Available online at: <http://www.epa.gov/superfund/lead/products.htm#change>.
- U.S. EPA (U.S. Environmental Protection Agency). 2011. Exposure Factors Handbook. Office of Research and Development: Washington, DC. EPA/600/R-090/052F. September. Available online at: <http://www.epa.gov/ncea/efh/pdfs/efh-complete.pdf>.
- U.S. EPA (U.S. Environmental Protection Agency). 2013. Integrated Science Assessment for Lead. Office of Research and Development. National Center for Environmental Assessment: Research Triangle Park, NC. EPA/600/R-10/075F. June. Available online at: <http://cfpub.epa.gov/ncea/isa/recorddisplay.cfm?deid=255721>.