

**Recommendations of the
Technical Review Workgroup for Lead for an
Approach to Assessing Risks Associated with Adult
Exposures to Lead in Soil**



Preface

This report was updated in 2003 to add two appendices and remove "interim" from the title. The change in the title reflects publication of the TRW's Adult Lead Model Review Report. **With the exception of the following, the guidance is unchanged from the December 1996 publication.** The report now includes an appendix showing the format for the spreadsheet form of the model (Appendix B) and an explanation of how the guidance is to be applied (Appendix C). The NHANES Report (March 2002) should be used in conjunction with this report. Based on the TRW's analysis of the data collected in the completed NHANES III survey (Phases 1 and 2), updated ranges for the baseline adult PbB and GSDi adult parameters should be in the EPA ALM spreadsheet. Although the use of these updated ranges in the EPA ALM spreadsheet would not appreciably change PRGs calculated with the methodology, it is recommended that data from both phases of NHANES III be used in all PbB analyses; this is consistent with CDC recommendations. **The results of the NHANES III Report are not included in this document except by reference.**

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1. INTRODUCTION

This report describes a methodology for assessing risks associated with non-residential adult exposures to lead in soil. The methodology focuses on estimating fetal blood lead concentration in women exposed to lead contaminated soils. This approach also provides tools that can be used for evaluating risks of elevated blood lead concentrations among exposed adults. The methodology is the product of extensive evaluations by the Technical Review Workgroup for Lead (TRW) which began considering methodologies to evaluate nonresidential adult exposure in 1994 (Balbus-Kornfeld, 1994; U.S. EPA, 1994a). In 1995, the TRW reviewed a methodology developed by EPA Region 8 for deriving risk-based remediation goals (RBRGs) for nonresidential soil at the California Gulch NPL site (U.S. EPA, 1995). A TRW committee on adult lead risk assessment was formed in January, 1996 to further develop the ideas and information gathered as part of these previous efforts into a generic methodology that could be adapted for use in site-specific assessments.

This report provides technical recommendations of the TRW for the assessment of adult lead risks using this methodology. An overriding objective in the development of this methodology was the immediate need for a scientifically defensible approach for assessing adult lead risks associated with nonresidential exposure scenarios. The TRW recognizes that other adult lead models may provide useful information. In particular, models providing more detailed representations of lead kinetics may be useful in supporting more detailed predictions about the time course of blood lead concentrations among individuals who receive brief acute exposures to lead or whose exposures otherwise change markedly with time. The methodology presented here uses a simplified representation of lead biokinetics to predict quasi-steady state blood lead concentrations among adults who have relatively steady patterns of site exposures (as described in this report). The TRW believes that this approach will prove useful for assessing most sites where places of employment are (or will be) situated on lead contaminated soils. This information is expected to promote consistency in assessments of adult lead risks. The methodology described in this report is an approach that is recommended for use pending further development and evaluation of integrated exposure biokinetic models for adults. The TRW is undertaking review of other models and will provide reviews on other approaches as appropriate. The Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children (U.S. EPA, 1994b,c) is the recommended approach for assessing residential lead risks.

The recommended approach for assessing nonresidential adult risks utilizes a methodology to relate soil lead intake to blood lead concentrations in women of child-bearing age. It is conceptually similar to a slope factor approach for deriving RBRGs that had been proposed by Bowers et al. (1994) and which was adapted for use at the California Gulch NPL site in Region 8 (U.S. EPA, 1995). This report describes the basic algorithms that are used in the methodology and provides a set of default parameter values that can be used in cases where high quality data are not available to support site-specific estimates. The rationale for each parameter default value is provided in the Appendix.

2. OVERVIEW OF THE APPROACH

The methodology described in this report relates soil lead concentrations to blood lead concentrations in the exposed population according to the algorithms described below. Note that the algorithms may consist of variables that include superscripts and/or subscripts. The convention adopted in this report is to use superscripts as exponents (i.e., a mathematical operation), whereas subscripts represent key words that provide additional information to distinguish between similar variables. The basis for the calculation of the blood lead concentration in women of child-bearing age is the algorithm given by Equation 1:

$$PbB_{adult,central} = PbB_{adult,0} + \frac{PbS \cdot BKSF \cdot IR_s \cdot AF_s \cdot EF_s}{AT} \quad (\text{Equation 1})$$

where:

$PbB_{adult,central}$ = Central estimate of blood lead concentrations ($\mu\text{g/dL}$) in adults (i.e., women of child-bearing age) that have site exposures to soil lead at concentration, PbS .

$PbB_{adult,0}$ = Typical blood lead concentration ($\mu\text{g/dL}$) in adults (i.e., women of child-bearing age) in the absence of exposures to the site that is being assessed.

PbS = Soil lead concentration ($\mu\text{g/g}$) (appropriate average concentration for individual).

$BKSF$ = Biokinetic slope factor relating (quasi-steady state) increase in typical adult blood lead concentration to average daily lead uptake ($\mu\text{g/dL}$ blood lead increase per $\mu\text{g/day}$ lead uptake).

IR_s = Intake rate of soil, including both outdoor soil and indoor soil-derived dust (g/day).

AF_s = Absolute gastrointestinal absorption fraction for ingested lead in soil and lead in dust derived from soil (dimensionless).

EF_s = Exposure frequency for contact with assessed soils and/or dust derived in part from these soils (days of exposure during the averaging period); may be taken as days per year for continuing, long term exposure.

AT = Averaging time; the total period during which soil contact may occur; 365 days/year for continuing long term exposures.

The basis for the RBRG calculation is the relationship between the soil lead concentration and the blood lead concentration in the developing fetus of adult women that have site exposures. As a health-based goal, EPA has sought to limit the risk to young children of having elevated blood lead concentrations. Current Office of Solid Waste and Emergency Response (OSWER) guidance calls

for the establishment of cleanup goals to limit childhood risk of exceeding 10 µg/dL to 5% (U.S. EPA, 1994a). Equation 2 describes the estimated relationship between the blood lead concentration in adult women and the corresponding 95th percentile fetal blood lead concentration ($PbB_{fetal, 0.95}$), assuming that $PbB_{adult, central}$ reflects the geometric mean of a lognormal distribution of blood lead concentrations in women of child-bearing age. If a similar 95th percentile goal is applied to the protection of fetuses carried by women who experience nonresidential exposures, Equation 2 can be rearranged to reflect a risk-based goal for the central estimate of blood lead concentrations in adult women using Equation 3:

$$PbB_{fetal, 0.95} = PbB_{adult, central} \cdot GSD_{i, adult}^{1.645} \cdot R_{fetal/maternal} \quad (\text{Equation 2})$$

$$PbB_{adult, central, goal} = \frac{PbB_{fetal, 0.95, goal}}{GSD_{i, adult}^{1.645} \cdot R_{fetal/maternal}} \quad (\text{Equation 3})$$

where:

$PbB_{adult, central, goal}$ = Goal for central estimate of blood lead concentration (µg/dL) in adults (i.e., women of child-bearing age) that have site exposures. The goal is intended to ensure that $PbB_{fetal, 0.95, goal}$ does not exceed 10 µg/dL.

$PbB_{fetal, 0.95, goal}$ = Goal for the 95th percentile blood lead concentration (µg/dL) among fetuses born to women having exposures to the specified site soil concentration. This is interpreted to mean that there is a 95% likelihood that a fetus, in a woman who experiences such exposures, would have a blood lead concentration no greater than $PbB_{fetal, 0.95, goal}$ (i.e., the likelihood of a blood lead concentration greater than 10 µg/dL would be less than 5%, for the approach described in this report).

$GSD_{i, adult}$ = Estimated value of the individual geometric standard deviation (dimensionless); the GSD among adults (i.e., women of child-bearing age) that have exposures to similar on-site lead concentrations, but that have non-uniform response (intake, biokinetics) to site lead and non-uniform off-site lead exposures. The exponent, 1.645, is the value of the standard normal deviate used to calculate the 95th percentile from a lognormal distribution of blood lead concentration.

$R_{\text{fetal/maternal}}$ = Constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration (dimensionless).

The soil lead concentration associated with a given exposure scenario and $PbB_{\text{adult, central, goal}}$ can be calculated by rearranging Equation 1 and substituting $PbB_{\text{adult, central, goal}}$ for $PbB_{\text{adult, central}}$:

$$RBRG = PbS = \frac{(PbB_{\text{adult, central, goal}} - PbB_{\text{adult, 0}}) \cdot AT}{(BKSF \cdot IR_s \cdot AF_s \cdot EF_s)} \quad (\text{Equation 4})$$

It is this form of the algorithm that can be used to calculate a RBRG where the RBRG represents the soil lead concentration (PbS) that would be expected to result in a specified adult blood lead concentration ($PbB_{\text{adult, central, goal}}$) and corresponding 95th percentile fetal blood lead concentration ($PbB_{\text{fetal, 0.95, goal}}$).

Equations 1-4 are based on the following assumptions:

1. Blood lead concentrations for exposed adults can be estimated as the sum of an expected starting blood lead concentration in the absence of site exposure ($PbB_{\text{adult, 0}}$) and an expected site-related increase.
2. The site-related increase in blood lead concentrations can be estimated using a linear biokinetic slope factor (BKSF) which is multiplied by the estimated lead uptake.
3. Lead uptake can be related to soil lead levels using the estimated soil lead concentration (PbS), the overall rate of daily soil ingestion (IR_s), and the estimated fractional absorption of ingested lead (AF_s). The term "soil" is used throughout this document to refer to that portion of the soil to which adults are most likely to be exposed. In most cases, exposure is assumed to be predominantly to the top layers of the soil which gives rise to transportable soil-derived dust. Exposure to soil-derived dust occurs both in outdoor and indoor environments, the latter occurring where soil-derived dust has been transported indoors. Other types of dust, in addition to soil-derived dust, can contribute to adult lead exposure and may even predominate in the occupational setting; these include dust generated from manufacturing processes (e.g., grinding, milling, packaging of lead-containing material), road dust, pavement dust, and paint dust. This methodology, as represented in Equations 1 and 4, does not specifically account for site exposure to dusts that are not derived from soil. However, the methodology can be modified to include separate variables that represent exposure to lead in various types of dust. This approach is discussed in greater detail in the Appendix.

4. As noted above, exposure to lead in soil may occur by ingesting soil-derived dust in the outdoor and/or indoor environments. The default value recommended for IR_S (0.05 g/day) is intended for occupational exposures that occur predominantly indoors. More intensive soil contact would be expected for predominantly outdoor activities such as construction, excavation, yard work, and gardening.
5. A lognormal model can be used to estimate the inter-individual variability in blood lead concentrations (i.e., the distribution of blood lead concentrations in a population of individuals who contact similar environmental lead levels).
6. Expected fetal blood lead concentrations are proportional to maternal blood lead concentrations.

The primary basis for using Equation 4 to calculate a RBRG is that fetuses and neonates are a highly sensitive population with respect to the adverse effects of lead on development and that 10 $\mu\text{g}/\text{dL}$ is considered to be a blood lead level of concern from the standpoint of protecting the health of sensitive populations (U.S. EPA, 1986, 1990; NRC, 1993). Therefore, risk to the fetus can be estimated from the probability distribution of fetal blood lead concentrations (i.e., the probability of exceeding 10 $\mu\text{g}/\text{dL}$), as has been the approach taken for estimating risks to children (U.S. EPA, 1994a,c). Equation 4 can be used to estimate the soil lead concentration at which the probability of blood lead concentrations exceeding a given value (e.g., 10 $\mu\text{g}/\text{dL}$) in fetuses of women exposed to environmental lead is no greater than a specified value (e.g., 0.05).

The methodology can be modified to accommodate different assumptions or to estimate RBRGs for different risk categories. For example, a RBRG could be estimated for risks to adults (e.g., hypertension) by substituting an appropriate adult blood lead concentration benchmark. Similarly, other exposure scenarios can be incorporated into the assessment. Alternative methods for estimating soil lead risk by partitioning soil into outdoor soil and indoor dust components are discussed in the Appendix.

Recommended default values for each of the parameters in Equations 1 - 4 are presented in Table 1. These defaults should not be casually replaced with other values unless the alternatives are supported by high quality site-specific data to which appropriate statistical analyses have been applied and that have undergone thorough scientific review. Examples of the output from the methodology are presented in Figures 1 and 2, which show plots of the calculated $PbB_{\text{fetal}, 0.95}$ as a function of PbS when different combinations of default parameter values are used. The rationale for each default value listed in Table 1 is summarized in the Appendix.

Table 1. Summary of Default Parameter Values for the Risk Estimation Algorithm (Equations 1 - 4)

Parameter	Unit	Value	Comment
$PbB_{fetal, 0.95, goal}$	$\mu\text{g/dL}$	10	For estimating RBRGs based on risk to the developing fetus.
$GSD_{i, adult}$	--	1.8 2.1	Value of 1.8 is recommended for a homogeneous population while 2.1 is recommended for a more heterogeneous population.
$R_{fetal/maternal}$	--	0.9	Based on Goyer (1990) and Graziano et al. (1990).
$PbB_{adult, 0}$	$\mu\text{g/dL}$	1.7-2.2	Plausible range based on NHANES III phase 1 for Mexican American and non-Hispanic black, and white women of child bearing age (Brody et al. 1994). Point estimate should be selected based on site-specific demographics.
BKSF	$\mu\text{g/dL}$ per $\mu\text{g/day}$	0.4	Based on analysis of Pocock et al. (1983) and Sherlock et al. (1984) data.
IR_s	g/day	0.05	Predominantly occupational exposures to indoor soil-derived dust rather than outdoor soil; (0.05 g/day = 50 mg/day).
EF_s	day/yr	219	Based on U.S. EPA (1993) guidance for average time spent at work by both full-time and part-time workers (see Appendix for recommendations on minimum exposure frequency and duration).
AF_s	--	0.12	Based on an absorption factor for soluble lead of 0.20 and a relative bioavailability of 0.6 (soil/soluble).

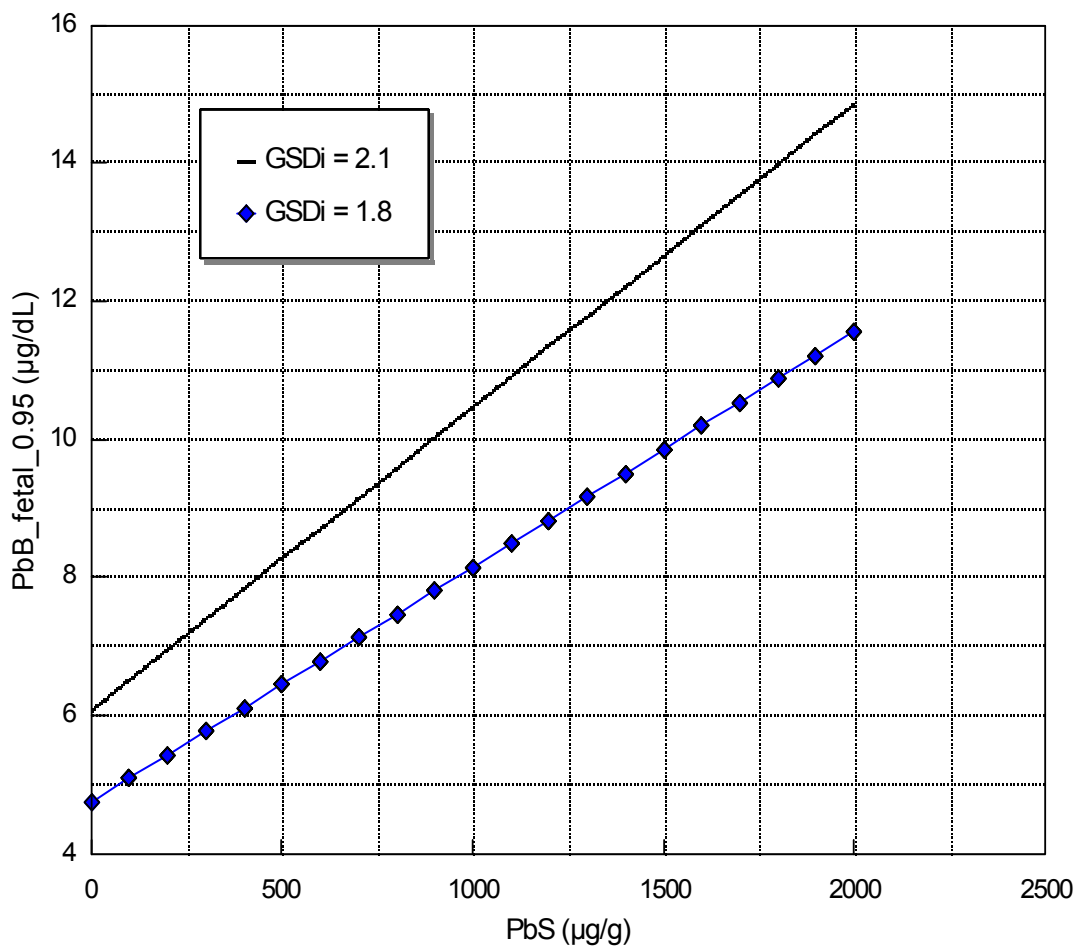


Figure 1. Example output of risk estimation algorithm (Equation 4) assuming a $PbB_{adult,0}$ of 2.0 $\mu\text{g/dL}$ (mixed racial) and a $GSD_{i,adult}$ of either 1.8 (homogeneous population) or 2.1 (heterogeneous urban population).

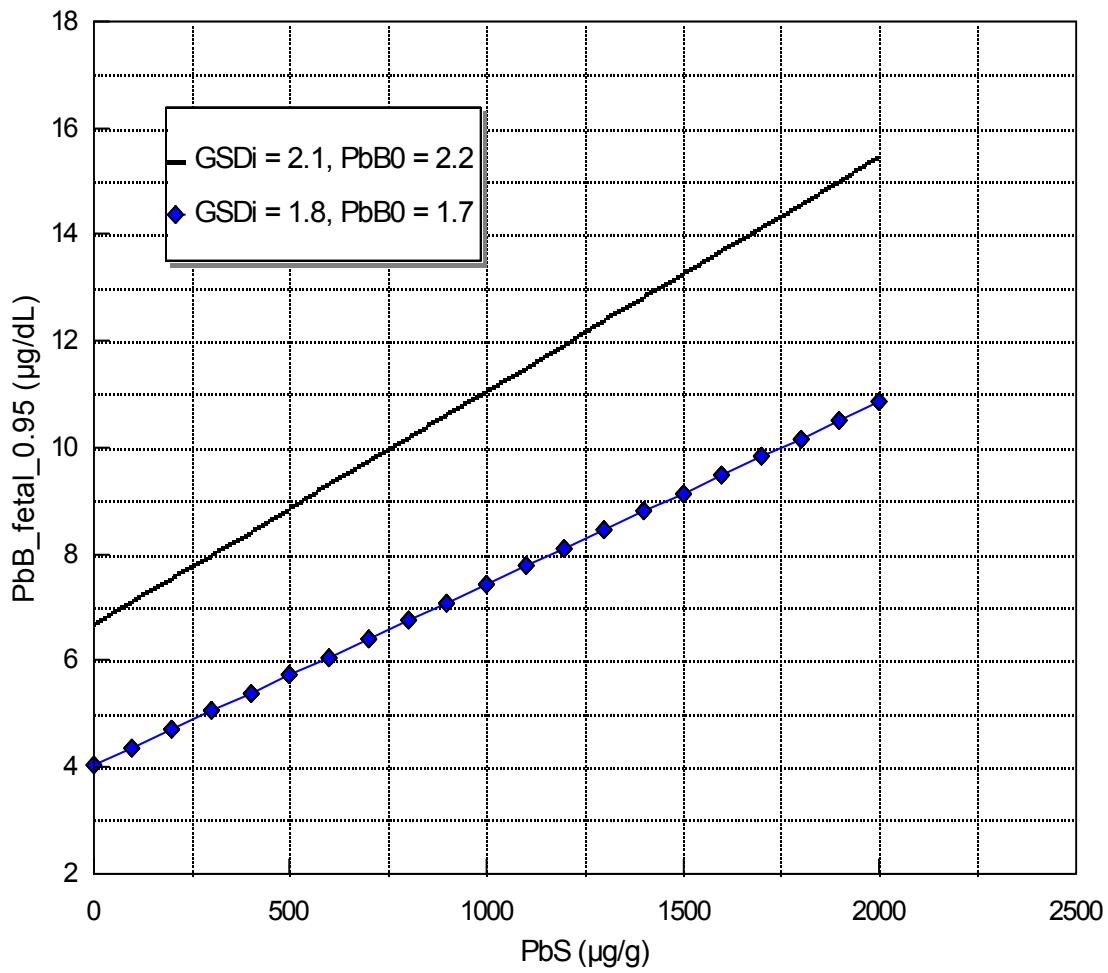


Figure 2. Example output of risk estimation algorithm (Equation 4) assuming plausible default minimum and maximum values of $PbB_{adult,0}$ (1.7 and 2.2 $\mu\text{g/dL}$) and $GSD_{i,adult}$ (1.8 and 2.1).

3. REFERENCES

- Balbus-Kornfeld, J. 1994. Comments and Recommendations on the Draft Interim Guidance for Screening Levels of Lead in Soil for Non-Residential Sites. Letter from John Balbus-Kornfeld to Bruce Means. November 17, 1994.
- Bowers, T.S., B.D. Beck and H.S. Karam. 1994. Assessing the relationship between environmental lead concentrations and adult blood lead levels. *Risk Analysis*. 14(2): 183-189.
- Brody, D.J., J.L. Pirkle, R.A. Kramer, K.M. Flegal, T.D. Matte, E.W. Gunter and D.C. Paschal. 1994. Blood lead levels in the U.S. population. Phase 1 of the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA*. 272(4): 277-283.
- Goyer, R.A. 1990. Transplacental transport of lead. *Environ. Health Perspect.* 89: 101-105.
- Graziano, J.H., D. Popovac, P. Factor-Litvak, P. ShROUT, J. Kline, M.J. Murphy, Y. Zhao, A. Mehmeti, X. Ahmedi, B. Rajovic, Z. Zvicar, D. Nenezic, N. Lolacono and Z. Stein. 1990. Determinants of elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. *Environ. Health Perspect.* 89: 95-100.
- NRC. 1993. *Measuring Lead Exposure in Infants, Children and Other Sensitive Populations*. National Academy Press. Washington, DC. ISBN 0-309-04927-X.
- Pocock, S.J., A.G. Shaper, M. Walker, C.J. Wale, B. Clayton, T. Delves, R.F. Lacey, R.F. Packham and P. Powell. 1983. Effects of tap water lead, water hardness, alcohol, and cigarettes on blood lead concentrations. *J. Epi. Comm. Health.* 37: 1-7.
- Sherlock, J.C., D. Ashby, H.T. Delves, G.I. Forbes, M.R. Moore, W.J. Patterson, S.J. Pocock, M.J. Quinn, W.N. Richards and T.S. Wilson. 1984. Reduction in exposure to lead from drinking water and its effect on blood lead concentrations. *Human Toxicol.* 3: 383-392.
- U.S. EPA. 1986. *Air Quality Criteria for Lead Volumes I - IV*. Environmental Criteria and Assessment Office, Office of Research and Development, RTP, NC. EPA 600/8-83-028 a-d.
- U.S. EPA. 1990. *Supplement to the 1986 EPA Air Quality Criteria Document for Lead - Volume 1 Addendum*. Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC. EPA-600/8-89/049A.
- U.S. EPA. 1993. *Superfund's Standard Default Exposure Factors for the Central Tendency and RME-Draft*. Working Draft, November 1993.
- U.S. EPA. 1994a. *Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities*. OSWER Directive No. 9355.4-12. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/F-94/043, PB94-963282.

U.S. EPA. 1994b. Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children (v. 0.99d). Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-94/040, PB94-963505.

U.S. EPA. 1994c. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-93/081, PB93-963510.

U.S. EPA. 1995. A TRW Report: Review of a Methodology for Establishing Risk-Based Soil Remediation Goals for the Commercial Areas of the California Gulch Site. Technical Review Workgroup for Lead, October, 1995.

APPENDIX A

Equations and Rationale for Default Values Assigned to Parameters in the Slope Factor Approach and Exposure Model for Assessing Risk Associated with Adult Exposures to Lead in Soil

Equations and Rationale for Default Values Assigned to Parameters in the Slope Factor Approach and Exposure Model for Assessing Risk Associated with Adult Exposures to Lead in Soil

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1. Equations for the Adult Lead Model

The format of the equations used in the adult lead methodology follows the approach used in the IEUBK Model for Lead in Children (IEUBK Model). Note that the equations may consist of variables that include superscripts and/or subscripts. The convention adopted in this report is to use superscripts as exponents (i.e., a mathematical operation), whereas subscripts represent key words that provide additional information to distinguish between similar variables. The term "soil" refers to that portion of the soil to which adults are most likely to be exposed. In most cases, exposure is assumed to be predominantly to the top layers of the soil which gives rise to transportable soil-derived dust. Exposure to soil-derived dust occurs both in outdoor and indoor environments, the latter occurring where soil-derived dust has been transported indoors. Other types of dust, in addition to soil-derived dust, can contribute to adult lead exposure and may even predominate in some occupational settings; these include dust generated from manufacturing processes (e.g., grinding, milling, packaging of lead-containing material), road dust, pavement dust, and paint dust.

Exposure to lead from soil (direct and through indoor soil-derived dust) and lead intake:

$$INTAKE = \frac{PbS \cdot IR_s \cdot EF_s}{AT} \quad \text{(Equation A-1)}$$

INTAKE = Daily average intake (ingestion) of lead from soil taken over averaging time AT ($\mu\text{g}/\text{day}$).

PbS = Soil lead concentration ($\mu\text{g}/\text{g}$) (appropriate average concentration for individual).

IR_s = Intake rate of soil, including outdoor soil and indoor soil-derived dust (g/day).

EF_s = Exposure frequency for contact with assessed soils and/or dust derived in part from these soils (days of exposure during the averaging period); may be taken as days per year for continuing, long term exposures.

AT = Averaging time; the total period during which soil contact may occur; 365 days/year for continuing long term exposures.

Lead uptake:

$$UPTAKE = AF_s \cdot INTAKE \quad \text{(Equation A-2)}$$

UPTAKE = Daily average uptake of lead from the gastrointestinal tract into the systemic circulation ($\mu\text{g}/\text{day}$).

AF_s = Absolute gastrointestinal absorption fraction for ingested lead in soil and lead in dust derived from soil (dimensionless).

Central estimate of adult blood lead concentration:

$$PbB_{adult,central} = PbB_{adult,0} + BKSF \cdot UPTAKE \quad (\text{Equation A-3})$$

$PbB_{adult,central}$ = Central estimate of blood lead concentrations ($\mu\text{g}/\text{dL}$) in adults (i.e., women of child-bearing age) that have site exposures to soil lead at concentration, PbS .

$PbB_{adult,0}$ = Typical blood lead concentration ($\mu\text{g}/\text{dL}$) in adults (i.e., women of child-bearing age) in the absence of exposures to the site that is being assessed.

BKSF = Biokinetic slope factor relating (quasi-steady state) increase in typical adult blood lead concentration to average daily lead uptake ($\mu\text{g}/\text{dL}$ blood lead increase per $\mu\text{g}/\text{day}$ lead uptake).

Distributional model for adult blood lead:

In this methodology, variability in blood lead concentrations among a population is mathematically described by a lognormal distribution defined by two parameters, the geometric mean (GM) and the geometric standard deviation (GSD):

$$PbB_{adult} \sim \text{Lognormal}(GM, GSD)$$

PbB_{adult} = Adult blood lead concentration (which is a variable quantity having the specified probability distribution).

GM = Geometric mean blood lead concentration ($\mu\text{g}/\text{dL}$) for adults having site exposure. The central estimate of adult blood lead, $PbB_{adult,central}$, constructed in Equation A-3 is treated as a plausible estimate of the geometric mean.

GSD = Geometric standard deviation for blood lead concentrations among adults having exposures to similar on-site lead concentrations, but having non-uniform response (intake, biokinetics) to site lead and non-uniform off-site lead exposures. The individual blood lead concentration geometric standard deviation, GSD_i , is

substituted for GSD. As described below (Section 2 of the Appendix), GSD_i is assumed to address sources of variability in blood lead concentrations among the exposed population.

Parameter estimates for the geometric mean (GM) and geometric standard deviation (GSD) of the lognormal distribution are described below. Note that blood lead concentrations for site exposures can be quantified at any percentile of the population using these parameters. For example, the 95th percentile blood lead concentration can be calculated by Equation A-4:

$$PbB_{adult,0.95} = PbB_{adult,central} \cdot GSD_i^{1.645} \quad (\text{Equation A-4})$$

$PbB_{adult, 0.95}$ = 95th percentile blood lead concentration ($\mu\text{g/dL}$) among individuals having exposures to the specified site soil lead concentrations. This is interpreted to mean that there is a 95% likelihood that an adult exposed to the specified soil lead concentrations would have a blood lead concentration less than or equal to $PbB_{adult,0.95}$.

Distributional model for fetal blood lead:

$$PbB_{fetal} = R_{fetal/maternal} \cdot PbB_{adult} \quad (\text{Equation A-5})$$

PbB_{fetal} = Fetal blood lead concentration ($\mu\text{g/dL}$) (which, like PbB_{adult} , is a variable quantity having the specified probability distribution).

$R_{fetal/maternal}$ = Constant of proportionality between fetal and maternal blood lead concentrations.

PbB_{adult} = Adult blood lead concentration ($\mu\text{g/dL}$), estimated with parameters appropriate to women of child bearing age.

Note that this relationship implies a deterministic (non-random) relationship between maternal and fetal blood lead concentrations. This assumption omits a source of variability (varying individual-specific ratios of fetal to maternal blood lead) that would tend to increase the variance of fetal blood lead concentrations. The assumption of proportionality implies that fetal blood lead concentrations also are lognormally distributed:

$$PbB_{fetal} \sim \text{Lognormal}(GM, GSD)$$

GM = Geometric mean blood lead concentration ($\mu\text{g/dL}$) for fetuses, equal to $R_{\text{fetal/maternal}}$ multiplied by $PbB_{\text{adult,central}}$.

GSD = Geometric standard deviation of blood lead concentration among adults, GSD_i (Section 2 of the Appendix).

Similarly, percentiles of the fetal blood lead distribution can be estimated (for fetuses carried by women exposed to the specified concentration of lead at the assessed site). For example:

$$PbB_{\text{fetal},0.95} = R_{\text{fetal/maternal}} \cdot PbB_{\text{adult,central}} \cdot GSD_{i,\text{adult}}^{1.645} \quad (\text{Equation A-6})$$

$PbB_{\text{fetal},0.95}$ = 95th percentile blood lead concentration ($\mu\text{g/dL}$) among fetuses born to women having exposures to the specified site soil lead concentrations. This is interpreted to mean that there is a 95% likelihood that a fetus born, in a woman who experiences such exposures, would have a blood lead concentration no greater than $PbB_{\text{fetal},0.95}$.

Note that when the expressions for $PbB_{\text{adult,central}}$, INTAKE, and UPTAKE (Equations A-1, A-2 and A-3) are substituted into Equation A-6, we obtain the complete expression for $PbB_{\text{fetal},0.95}$ that is presented in the fact sheet (Overview of the Approach, Equations 1 and 2):

$$PbB_{\text{fetal},0.95} = R_{\text{fetal/maternal}} \cdot GSD_i^{1.645} \cdot \left[\frac{(PbS \cdot BKSF \cdot IR_s \cdot AF_s \cdot EF_s)}{AT} + PbB_{\text{adult},0} \right] \quad (\text{Equation A-7})$$

Equation A-7 represents variability in blood lead concentration arising from two main factors: 1) exposure variables, including inter-individual variability in activity-weighted ingestion rates, and 2) inter-individual variability in physiology, including factors affecting lead biokinetics.

2. Individual Blood Lead Geometric Standard Deviation (GSD_i)

The GSD_i is a measure of the inter-individual variability in blood lead concentrations in a population whose members are exposed to the same nonresidential environmental lead levels. Ideally, the value(s) for GSD_i used in the methodology should be estimated in the population of concern at the site. This requires data on blood lead concentration and exposure in a representative sample of sufficient size to yield statistically meaningful estimates of GSD in subsamples stratified by nonresidential exposure level. In the absence of high quality data for the site, GSD_i may be extrapolated from estimates for other surrogate populations. In making such extrapolations, factors that might contribute to higher or lower variability in the surrogate population than among similarly exposed individuals in the population of concern, should be evaluated. These factors include variability in exposure (level and pathways), and biokinetics (see Section 6 of Appendix), socioeconomic and ethnic characteristics, degree of urbanization and geographical location. Such

extrapolations, therefore, are site-specific and are a potentially important source of uncertainty in the methodology.

GSD values measured in populations (GSD_p) reflect the combined effect of 1) variability in environmental concentration levels; and 2) activity-weighted exposures and lead biokinetics. Thus, estimates of GSD_p can be considered a surrogate for estimating the GSD_i . Site data on blood lead concentrations collected from populations of varying homogeneity may be useful for establishing a plausible range of values of GSD_i , provided that the data are of adequate quality and can be stratified by nonresidential exposure level. The lowest values of GSD_p are expected among homogeneous populations (e.g., individuals with similar socioeconomic and ethnic characteristics living within a relatively small geographic area) exposed to a single, dominant source of lead (e.g., lead mining or smelter sites). For example, a GSD_p of 1.8 was recently calculated among adult women living in Leadville, CO (U.S. EPA, 1995). This relatively low GSD is consistent with an analysis of blood lead concentration data in mining communities in the United States and Canada, which suggest that GSD_p ranges from 1.6 - 1.8 at active mining sites where blood lead concentrations are less than 15 $\mu\text{g/dL}$ (U.S. EPA, 1992). By contrast, higher values of GSD_p might be expected from a national survey. Although lead exposures among the general population are likely to be more greatly impacted by diet than soil (e.g., compared with populations exposed at a waste site), the national population is very heterogeneous, in that it includes individuals with different socioeconomic and ethnic characteristics living in distinct geographic areas.

The TRW has conducted a preliminary analysis of blood lead concentration data collected in NHANES III Phase 1 from 1988 to 1991 and found that the GSD_p for women ages 17 to 45 years may range from 1.9 - 2.1 (Table A-1). Because of the complex survey design used in NHANES III (e.g., large oversampling of young children, older persons, black persons, and Mexican-Americans), this analysis used sampling weights included in the NHANES III Phase 1 data file to produce population estimates for blood lead concentration. The weighting factor "WTPEXMH1" was used to reflect the non-random sampling of individuals in both the mobile examination units (MEC) and the home examinations. The analysis did not account for the design effects associated with the selection of strata and primary sampling units (PSUs), which may result in an underestimation of sampling variance. Since this bias is not likely to greatly impact the GSD_p (Brody, personal communication), the amount of underestimation of the GSD_p by the values given in Table A-1 is likely to be small. Geometric mean blood lead concentrations listed in Table A-1 are within 0.2 $\mu\text{g/dL}$ of those reported in Brody et al. (1994).

The TRW estimates that 1.8 - 2.1 is a plausible range for GSD_i , based on an evaluation of available blood lead concentration data for different types of populations. In cases where site-specific data are not available, a value within this range should be selected based on an assessment as to whether the population at the site would be expected to be more or less heterogeneous than the U.S. population with respect to racial, ethnic, cultural and socioeconomic factors that may affect exposure.

Table A-1. NHANES III Phase 1 Summary Statistics for Blood Lead Concentration Among U.S. Women by Age and Ethnic/Racial Characteristics^a.

Age Group (years)	Non-Hispanic White			Non-Hispanic Black			Mexican American		
	No.	GM	GSD	No.	GM	GSD	No.	GM	GSD
20 - 49	728	1.9	1.90	622	2.3	2.01	729	2.1	2.10
50 - 69	476	3.2	1.88	256	4.2	1.80	255	3.3	2.12
> 69	562	3.5	1.82	135	4.1	1.86	75	2.9	2.03
20 +	1,766	2.4	2.01	1,013	2.7	2.07	1,059	2.3	2.14
17 - 45	742	1.7	1.89	658	2.1	1.98	763	2.0	2.10

^aAnalysis of data weighted by MEC and home weighting factor (WTPEXMH1), excluding samples missing data on blood lead concentration or age. GM PbB ($\mu\text{g/dL}$) = $\exp(\mu_{\ln})$; GSD PbB = $\exp(\sigma_{\ln})$.

3. Fetal/Maternal Blood Lead Concentration Ratio ($R_{\text{fetal/maternal}}$)

The TRW recommends a default value of 0.9 based on studies that have explored the relationship between umbilical cord and maternal blood lead concentrations (Goyer, 1990; Graziano et al., 1990). The Goyer (1990) estimate of an average fetal/maternal blood lead concentration ratio of 0.9 is supported by a large body of data that has been summarized in Agency documents (U.S. EPA, 1986, 1990). Graziano et al. (1990) compared maternal and umbilical cord blood lead concentrations at delivery in 888 mother-infant pairs who were between 28 and 44 weeks of gestation. The relationship was linear with a slope of 0.93 $\mu\text{g/dL}$ cord blood per $\mu\text{g/dL}$ maternal blood; the correlation coefficient was 0.92. The slope of 0.93 from the Graziano et al. (1990) study supports 0.9 as a point estimate for $R_{\text{fetal/maternal}}$.

Although average fetal/maternal blood lead concentration ratios, as reflected in cord blood, tend to show consistent trends (Goyer, 1990; Graziano et al., 1990), the trends may not reflect significant inter-individual variability in maternal and possibly fetal blood lead concentrations due to physiological changes associated with pregnancy. For example, mobilization of bone lead stores during pregnancy may be more substantial in some women, and iron and calcium deficiency associated with poor nutritional status, as well as pregnancy, may enhance gastrointestinal absorption of lead (U.S. EPA, 1990; Franklin et al., 1995). Conversely, maternal blood lead concentration may decrease during the later stages of pregnancy because of the dilution effect associated with a 30% rise in plasma volume, as well as an increased rate of transfer of lead to the placenta or to fetal tissues (Alexander and Delves, 1981). These changes may give rise to fetal/maternal blood lead concentration ratios that are different from 0.9.

4. Baseline Blood Lead Concentration ($\text{PbB}_{\text{adult},0}$)

The baseline blood lead concentration ($\text{PbB}_{\text{adult},0}$) is intended to represent the best estimate of a reasonable central value of blood lead concentration in women of child-bearing age who are not exposed to lead-contaminated nonresidential soil or dust at the site. In this analysis, geometric mean blood lead concentrations are used for this purpose. Ideally, the value(s) for $\text{PbB}_{\text{adult},0}$ used in the

methodology should be estimated in the population of concern at the site. This requires data on blood lead concentrations in a representative sample of adult women who are not exposed to nonresidential soil or soil-derived dust at the site, but who may experience exposures to other environmental sources of lead that are similar in magnitude to exposures experienced by the population of concern. This would include exposure to lead in food and drinking water as well as residential soil and dust (dust derived from soil and all other non-site related sources). The sample must be of sufficient size to yield statistically meaningful estimates of $PbB_{adult,0}$.

In the absence of high quality data for the site, $PbB_{adult,0}$ may be extrapolated from estimates for other surrogate populations that would be expected to have a similar $PbB_{adult,0}$ distribution as that of the population of concern. In making such extrapolations, factors that might contribute to differences between the geometric mean $PbB_{adult,0}$ in the surrogate population and population of concern should be evaluated. These factors include differences in the residential exposure (level and pathways), socioeconomic, ethnic and racial demographics, housing stock, degree of urbanization, and geographical location. Such extrapolations, therefore, are site-specific.

In cases where site-specific extrapolations from surrogate populations are not feasible, the TRW recommends 1.7 - 2.2 $\mu\text{g}/\text{dL}$ as a plausible range, based on the results of Phase 1 of the NHANES III as reported by Brody et al. (1994). Table A-2 summarizes the analysis of blood lead concentrations from a sample of 2,083 women ages 20 - 49, and stratified into the three ethnic and racial categories.

Table A-2. NHANES III Phase 1 Summary Statistics for Blood Lead Concentration Among Different Populations of U.S. Women Ages 20 - 49 (Brody et al., 1994).

Population	No.	GM (95% CI)
Mexican American women	732	2.0 (1.7 - 2.5)
non-Hispanic black women	623	2.2 (2.0 - 2.5)
non-Hispanic white women	728	1.7 (1.6 - 1.9)
Total	2,083	

The TRW recommends that the estimates from Table A-2 be used in combination with data on the ethnic and racial demographics of the population of concern to select the most appropriate point estimate from within the plausible range of 1.7 - 2.2 $\mu\text{g}/\text{dL}$. For example, if the population at the site was predominantly Mexican American, 2.0 $\mu\text{g}/\text{dL}$ might be selected as the point estimate. The plausible range is based on surveys of large samples of the national population and may not encompass central tendencies estimated from smaller regional or site-specific surveys, either because of bias associated with the smaller sample or because of real differences between the surveyed population and the national population. This needs to be evaluated in deciding whether or not to use data from small surveys that yield point estimates for $PbB_{adult,0}$ that fall outside of the plausible range.

5. Biokinetic Slope Factor (BKSF)

The BKSF parameter relates the blood lead concentration ($\mu\text{g Pb/dL}$) to lead uptake ($\mu\text{g Pb/day}$). The TRW recommends a default value of $0.4 \mu\text{g Pb/dL blood per } \mu\text{g Pb absorbed/day}$ for the BKSF parameter based on data reported by Pocock et al. (1983) on the relationship between tap water lead concentrations and blood lead concentrations for a sample of adult males, and on estimates of the bioavailability of lead in tap water (see Section 6 of the Appendix).

Pocock et al. (1983) analyzed data on lead concentrations in first draw tap water and blood lead concentrations in a population of 910 adult males. A linear model imposed on the data yielded a slope of $0.06 (\mu\text{g/dL per } \mu\text{g/L first draw water})$ for water lead concentrations equal to or less than $100 \mu\text{g/L}$ (a lower slope was applied to the data for higher water concentrations). Pocock et al. (1983) also obtained data on lead concentrations in flushed water (and "random daytime") samples, in addition to first draw samples. Given the following assumptions, it is possible to derive a slope factor for ingested water lead (INGSF) from the Pocock et al. (1983) data:

- The lead concentration of flushed water was 25% of the concentration of first draw water ($C_{f/1st} = 0.25$) (U.S. EPA, 1995).
- Daily water intake consisted of 30% first draw and 70% flushed ($F_{1st} = 0.3, F_f = 0.7$) (U.S. EPA, 1992).
- Daily water ingestion (including tap water and beverages made with tap water) was 1.4 L/day ($IR_w = 1.4$) (U.S. EPA, 1989).

Based on the above assumptions, a INGSF of $0.09 \mu\text{g/dL per } \mu\text{g intake/day}$ is estimated as follows:

$$INGSF = \frac{0.06}{IR_w \cdot (F_{1st} + (C_{f/1st} \cdot F_f))} \quad (\text{Equation A-8})$$

$$INGSF = \frac{0.06}{1.4 \cdot (0.3 + (0.25 \cdot 0.7))}$$

$$INGSF = 0.09$$

This suggests that the product of the BKSF, reflecting the slope for absorbed rather than ingested lead, and the absorption factor for lead in drinking water (AF_w) should be approximately 0.09 if it is to match the estimate of INGSF based on the Pocock et al. (1983) study:

$$INGSF = BKSF \cdot AF_w \quad (\text{Equation A-9})$$

Values of AF_w within the range 0.20 - 0.25 would correspond to a range for BKSF of 0.36 - 0.45, or approximately 0.4 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ (rounded to one significant figure). A range of 0.20 - 0.25 for AF_w is supported by data from numerous lead bioavailability studies (see Section 6 of the Appendix for a more detailed discussion of these studies).

The above estimate of 0.4 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ for the BKSF can be compared with the approach described by Bowers et al. (1994), who used the same data set along with different assumptions and arrived at essentially the same estimate of the BKSF, 0.375 or approximately 0.4 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$. Bowers et al. (1994) assumed a daily tap water intake of 2 L/day and 8% absorption of lead ingested in tap water; and did not make adjustments for a mixture of first draw and flushed water intake in the Pocock et al. (1983) study.

Several uncertainties should be considered in applying the default value of 0.4 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ to any specific population. Since it is based on the Pocock et al. (1983) data, it represents an extrapolation from adult men to women of child bearing age. Physiological changes associated with pregnancy may affect the value of the BKSF (see Section 6 of the Appendix); therefore, some uncertainty is associated with applying the default value to populations of pregnant women.

An additional uncertainty concerns the assumption of linearity of the relationship between lead intake and blood lead concentration. The Pocock et al. (1983) study provides data on a large sample population of adult men whose members were exposed to relatively low drinking water lead levels; 898 subjects (97%) were exposed to first draw water lead concentrations less than 100 $\mu\text{g}/\text{L}$ and 473 (52%) to 6 $\mu\text{g}/\text{L}$ or less. A smaller study of adult women exposed to higher concentrations was reported by Sherlock et al. (1982, 1984); out of 114 subjects, 32 (28%) had flush drinking water lead concentrations less than 100 $\mu\text{g}/\text{L}$ and only 13 (11%) less than 10 $\mu\text{g}/\text{L}$. Sherlock et al. (1982, 1984) used a cube root regression model, rather than a linear model, to describe the relationship between drinking water and blood lead concentration. Given the much larger sample size in the Pocock et al. (1983) study, particularly towards the low end of the distribution for water lead concentration, greater confidence can be placed in the estimated slope of the linear regression model from the Pocock et al. (1983) study than in the cube root regression model of Sherlock et al. (1982, 1984). Nevertheless, it is useful to compare the output of the two models because they were applied to the different sexes and because they differ so fundamentally in the treatment of the blood lead - water lead slope; the slope is constant in the linear model and decreases in the cube root model as water lead concentration increases. Figure A-1 compares the output of the two models and shows the output of a linear regression of the unweighted output of the Sherlock et al. (1984) model. Three observations can be made from this comparison that are relevant to the BKSF:

1. Both the Pocock et al. (1983) and Sherlock et al. (1984) models predict higher blood lead concentrations than would be expected in the average U.S. population today as suggested from NHANES III. This is indicative of higher lead intakes in the study populations which may have contributed to the apparent nonlinearities observed (e.g. above 100 $\mu\text{g}/\text{L}$ in Pocock et al.(1983) and at lower concentrations in Sherlock et al. (1984).
2. The cube root regression model of Sherlock et al. (1984) predicts lower blood lead concentrations than the linear model of Pocock et al. (1983). This may reflect

greater lead intakes from sources other than drinking water in the Pocock et al. (1983) population (see Section 6 of the Appendix for further discussion).

3. The linear approximation of the Sherlock et al. (1984) and the linear model from Pocock et al. (1983) have similar slopes; 0.08 and 0.06 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{L}$, respectively. Thus, although the Sherlock et al. (1984) study casts some degree of uncertainty on the assumption of linearity of the blood lead - drinking water lead relationship both at low ($<10 \mu\text{g}/\text{L}$) and high ($> 100 \mu\text{g}/\text{L}$) tap water lead concentrations, a linear model with a constant slope of 0.06 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{L}$ appears to approximate the output of the nonlinear model of Sherlock et al. (1984) reasonably well for water lead concentrations less than $100 \mu\text{g}/\text{L}$.

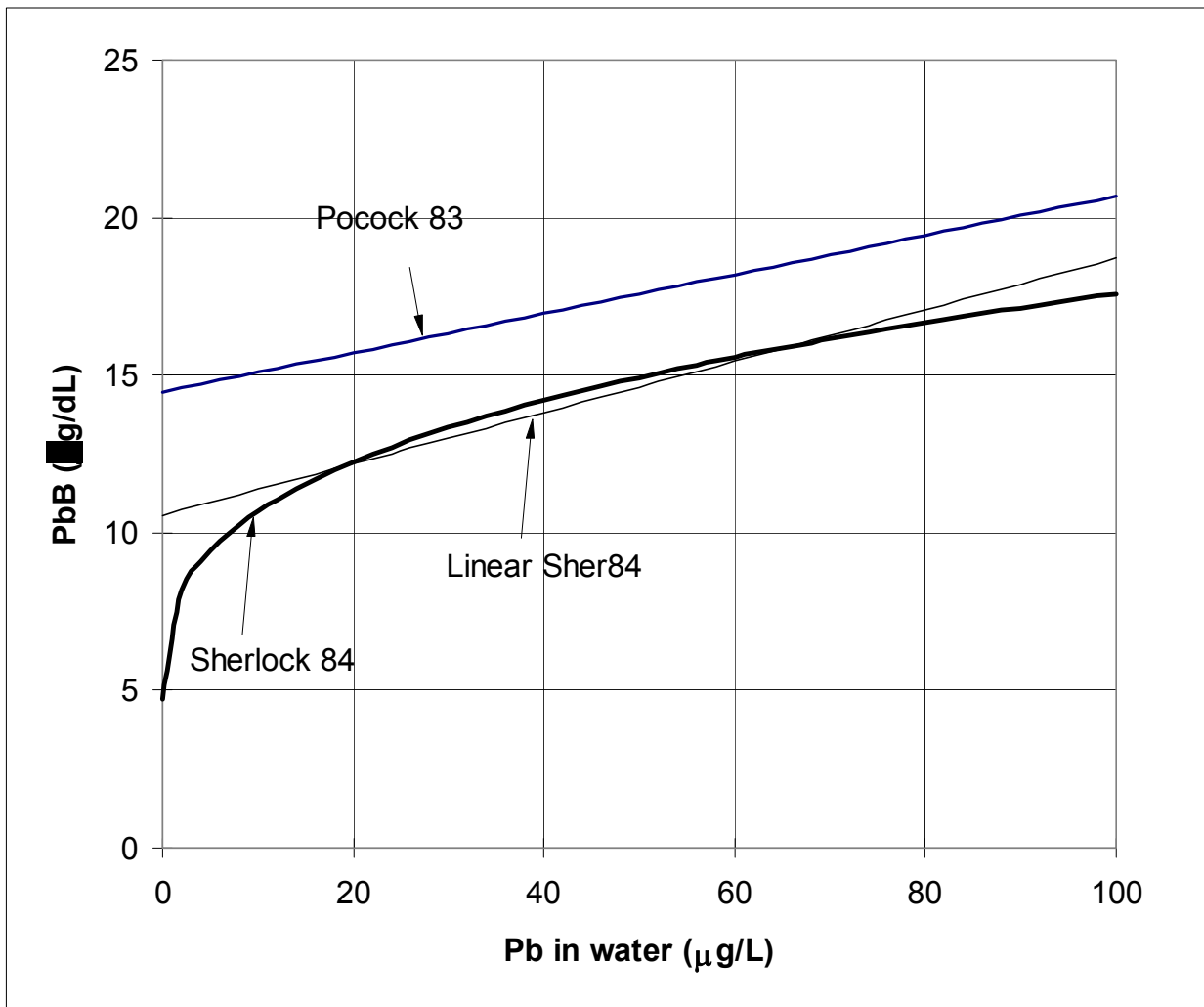


Figure A-1. Comparison of linear model of Pocock et al. (1983) with cube root model of Sherlock et al. (1984) and a linear model imposed on the unweighted output of the Sherlock model over the water lead range 0 - 100 µg/L (linear Sher84). The slope of the linear Sher84 model is 0.08 µg/dL per µg/L. The slope of the Pocock et al. (1983) model is 0.06 µg/dL per µg/L.

Experimental data on the pharmacokinetics of lead in adult humans support the default value of 0.4 ($\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ absorbed lead) for BKSF estimated from Pocock et al. (1983). Several distinct kinetic pools of lead are evident from observations of the rate of change of blood lead isotope with time after a period of daily dosing in which lead is abruptly terminated (Rabinowitz et al., 1976). A rapid exchange pool, denoted pool 1, includes the blood and a portion of the extracellular fluid, and is the physiological pool from which urinary and hepatobiliary excretion of blood lead occurs. Several estimates of the size of pool 1 (V_1) and the residence times for lead in pool 1 (T_1) have been derived from experiments in which human subjects were administered tracer doses of stable isotopes of lead from which pool 1 clearances (C_1) have been estimated; these estimates are summarized in Table A-3.

Table A-3. Summary of Experimental Studies with Humans to Assess Clearance Rates of Lead from Blood and Extracellular Fluid.

Subject	V_1^a (dL)	T_1^b (day)	$T_{1/2}^c$ (day)	C_1^d (dL/day)	Reference
A	77	34	24	2.3	Rabinowitz et al., 1974
B	115	50	35	2.3	
A	74	34	24	2.2	Rabinowitz et al., 1976
B	100	40	28	2.5	
C	101	37	26	2.7	
D	99	40	28	2.5	
E	113	27	19	4.2	
ACC	70 ^e	29	20	2.4	Chamberlain et al., 1978
DN	94 ^e	39	27	2.4	
PL	85 ^e	40	28	2.1	
ACW	94 ^e	48	33	2.0	
MJH	97 ^e	41	28	2.4	
ANB	95 ^e	40	28	2.4	
Mean \pm SD	93 \pm 14	38 \pm 6	27 \pm 4	2.5 \pm 0.5	

^aThe reported volume of pool 1, which refers to blood and rapidly exchangeable extracellular fluid compartment.

^bThe reported residence time for lead in pool 1.

^cThe half life of lead in pool 1; $T_{1/2} = (T_1) \times \ln(2)$.

^dClearance of lead from pool 1; $C_1 = V_1/T_1$.

^eEstimated assuming $V_1 = V_{\text{blood}} \times 1.7$ (Rabinowitz et al., 1976).

The above experiments support a value for C_1 of 2.5 dL/day. At steady state, the clearance is equivalent to the rate of uptake of lead into pool 1 per unit of blood lead concentration ($\mu\text{g}/\text{day}$ per $\mu\text{g}/\text{dL}$). Theoretically, this should correspond to a slope factor of 0.40 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ absorbed lead (i.e., the reciprocal of the clearance estimate). Thus, the default value for the BKSF parameter of 0.4 $\mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{day}$ absorbed lead derived from the population survey data of Pocock et al. (1983) is consistent with the clearance estimates from experimental studies.

6. Soil Lead Absorption Factor (AF_S)

The AF_S parameter is the fraction of lead in soil ingested daily that is absorbed from the gastrointestinal tract. The TRW recommends a default value of 0.12 based on the assumption that the absorption factor for soluble lead (AF_{soluble}) is 0.2 and that the relative bioavailability of lead in soil compared to soluble lead ($RBF_{\text{soil/soluble}}$) is 0.6:

$$AF_S = AF_{\text{soluble}} \cdot RBF_{\text{soil/soluble}} \quad (\text{Equation A-10})$$

$$AF_S = 0.2 \cdot 0.6 = 0.12$$

The default value of 0.2 for AF_{soluble} in adults represents a weight of evidence determination based on experimental estimates of the bioavailability of ingested lead in adult humans with consideration of three major sources of variability that are likely to be present in populations, but are not always represented in experimental studies; these are variability in food intake, lead intake, and lead form and particle size.

Effect of food on lead bioavailability. The bioavailability of ingested soluble lead in adults has been found to vary from less than 10% when ingested with a meal to 60 - 80% when ingested after a fast (Blake, 1976; Blake et al., 1983; Blake and Mann, 1983; Graziano et al., 1995; Heard and Chamberlain, 1982; James et al., 1985; Rabinowitz et al., 1976, 1980). The general consensus is that constituents of food in the gastrointestinal tract decrease absorption of ingested lead, although the exact mechanisms by which this occurs are not entirely understood. Lead intake within a population would be expected to occur at various times with respect to meals. Therefore, the central tendency for lead absorption would be expected to reflect, in part, meal patterns within the population and to have a value between the experimentally determined estimate for fasted and fed subjects.

An estimate of a "meal-weighted" AF_{soluble} can be obtained from the data reported by James et al. (1985) and certain simplifying assumptions. James et al. (1985) assessed the effects of food on lead bioavailability by measuring the fraction retained in the whole body of adult subjects 7 days after they ingested a dose of radioactive lead either after a fast or at various times before or after a meal. The total lead dose was approximately 50 μg (fasted) - 100 μg (with food). Lead retention was 61 ± 8.2 (SD)% when lead was ingested on the 12th hour of a 19-hour fast and decreased to 4% - 16% when lead was ingested between 0 and 3 hours after a meal; retention was further reduced ($3.5 \pm 2.9\%$) when lead was ingested with a meal (breakfast) (the bioavailability may have been more than these retention estimates since some absorbed lead would have been excreted during the 7 day

interval between dosing and measurement of whole-body lead). Since ingested material may be retained in the human stomach or at least 1 hour (Hunt and Spurrel, 1951; Davenport, 1971), lead bioavailability also may be reduced when lead is ingested 1 hour before a meal. The average “meal-weighted” bioavailability can be estimated based on the average number of waking hours during the day, the number of meals eaten, the bioavailability of lead ingested within 1 hour before a meal, the bioavailability of lead ingested within 0 to 3 hours after a meal, and the bioavailability of lead at other times during the day. For example, if it is assumed that people eat three meals each day and, based on the James et al. (1985) study, the bioavailability of lead ingested within 1 hour before a meal or 0 to 3 hours after a meal is approximately 0.1, and the bioavailability of lead ingested at all other times in a 16 hour day is 0.6, then the average “meal-weighted” bioavailability during a 16 hour day is approximately 0.2:

$$\frac{(0.1 \cdot 12 \text{ hrs}) + (0.6 \cdot 4 \text{ hrs})}{16 \text{ hrs}} = 0.23$$

This example suggests that the use of 0.2 as a default value for AF_{soluble} is plausible for populations in which soil lead intake occurs throughout the day, interspersed with meals. This may not apply to all members of a population. For example, the average bioavailability would be higher if less than three meals were consumed each day (e.g., using a similar calculation it can be shown that the average bioavailability for one meal each day would be 0.5). Average bioavailability also may be greater than 0.2 if lead intake was to occur predominantly in the early morning, before the first meal of the day.

Although lead bioavailability may be lower in individuals whose soil lead ingestion coincides with meals, the TRW cautions against the use of a value less than 0.2 for several reasons. Iron and calcium deficiency associated with poor nutritional status may enhance absorption (U.S. EPA, 1990). In addition, numerous factors may affect the absorption, distribution, excretion, and mobilization of lead during pregnancy: increased plasma volume (i.e., hemodilution); decreased hematocrit; previous exposure history of the mother (i.e., bone lead sequestration); changes in nutritional status; significant loss of body weight or depletion of fat stores; hormonal modulation; age; race; administration of drugs; and illness (Silbergeld, 1991). There is likely to be significant inter-individual variability in these factors, and studies of women at different stages of pregnancy have not shown clear trends in effects on blood lead concentration (Gershanik et al., 1974; Alexander and Delves, 1981; Baghurst et al., 1987; Silbergeld, 1991). While there is evidence to support 0.2 as a reasonable estimate of AF_{soluble} for women of child-bearing age, there is still some basis for concern regarding potentially elevated absorption during pregnancy. However, a potential increase in lead absorption during pregnancy would be expected to occur dynamically with changes in bone mobilization, blood volume and glomerular filtration rate. Thus, the TRW cautions against adjusting the value for AF_{soluble} (or BKSF) based on assumptions regarding the effects of pregnancy on blood lead concentration.

Nonlinearity in blood lead concentration. Another reason for caution in adopting values for AF_{soluble} less than 0.2 derives from uncertainty about the relationship between blood lead concentration, lead intake, and lead absorption. Several studies have shown that the relationship between environmental lead levels (e.g., drinking water lead concentration) and blood lead

concentration is nonlinear and suggest the possibility that fractional absorption of ingested lead is dose-dependent, and decreases as lead intake (and blood lead concentration) increases. Pocock et al. (1983) reported a nonlinear relationship between blood lead concentration and water lead that could be approximated by two linear equations: a slope of 0.06 µg/dL per µg/L was estimated for water lead concentrations equal to or less than 100 µg/L and a slope of 0.01 was estimated for water lead concentrations above 100 µg/L. Sherlock et al. (1982, 1984) used a cube root regression model to relate blood and water lead concentrations; however, over the range of water lead concentrations of 100 µg/L or less, the slope of 0.06 µg/dL per µg/L water lead from Pocock et al. (1983) approximates the relationship observed in the Sherlock et al. (1982, 1984) study (Figure A-1). The linear relationship between water lead and blood lead in the Pocock et al. (1983) study extends from a blood lead concentration range of 14 to 20 µg/dL. Based on these data, the value of AF_{soluble} of 0.2 may be considered a reasonable default estimate if applied to exposure scenarios in which the estimates of blood lead concentration do not exceed 20 µg/dL. At blood lead concentrations greater than this, absorption of soluble lead may be less than the default value.

An appropriate value of AF_{soluble} also can be supported by estimating the range of daily lead intake that is likely to result in a linear relationship between intake and blood lead concentration. Data represented in Figure A-1 suggest that if water lead concentrations are less than 100 µg/L, the blood lead - water lead relationship is approximately linear. If assumptions regarding the magnitude of first draw and flushed water intakes and lead concentrations are applied (see Equations A-8 and A-9 and discussion of BKSF), a first draw water lead concentration of 100 µg/L in the Pocock et al. (1983) study represents a water lead intake of approximately 70 µg/day:

$$100 \cdot 1.4 \cdot (0.3 + (0.25 \cdot 0.7)) \approx 70$$

We do not know with certainty the total lead intake in the Pocock et al. (1983) population, although we can be certain that it exceeded the above estimated intake from drinking water since intake from diet and other sources, including occupational, would have occurred; this is consistent with the higher blood lead concentrations that were observed in the male population. Sherlock et al. (1982) estimated that, in their study population of adult women, the dietary contribution to total lead intake was equal to that from drinking water when the water lead concentration was 100 µg/L, and that the contribution of lead from sources other than diet and water was very small. If the same assumption is applied to the Pocock et al. (1983) study, it is likely that total lead intake in the male population was at least 140 µg/day (70 µg/day from drinking water and 70 µg/day from diet; the Pocock et al., 1983 study included 40 households from the Sherlock et al., 1982 study site), and may have been higher because of occupational exposure in the male population. A crude estimate of the relative magnitudes of the non-water lead intakes in the two studies can be obtained by comparing the predicted water lead concentration required to achieve the same blood lead concentration in the two populations. For example, a water lead concentration of 100 µg/L corresponded to a predicted blood lead concentration of approximately 18 µg/dL in the female population (Sherlock et al., 1984); the same blood lead concentration corresponded to a water lead concentration of 50 µg/L in the male population (Pocock et al., 1983). Therefore, the non-water lead intakes in the male population may have been twice that in the female population. If it is assumed that drinking water and diet contributed equally to lead intake in both studies, then a drinking water lead concentration of 100 µg/L in the Pocock et al. (1983) study translates to a total lead intake of approximately 300 µg/day:

$$I_{total} = I_{water} + I_{diet} + I_{other} \quad (\text{Equation A-11})$$

$$I_{total} = 70 + 70 + 140 \approx 300 \mu\text{g/day}$$

Thus, the departure from linearity observed in the Pocock et al. (1983) study may have occurred at lead intakes at or above 300 $\mu\text{g/day}$. In the various experimental assessments of lead bioavailability, subjects ingested lead in amounts that varied among the studies but were all within the range 100 - 300 μg (Blake, 1976; Blake et al., 1983; Blake and Mann, 1983; Graziano et al., 1995; Heard and Chamberlain, 1982; James et al., 1985; Rabinowitz et al., 1976, 1980), which is within the approximate linear range, if the extrapolation from the Pocock et al. (1983) and Sherlock et al. (1982) studies is reasonable. Based on these considerations, the value of $AF_{soluble}$ of 0.2 is considered to be a reasonable default value if applied to exposure scenarios in which lead intakes are less than 300 $\mu\text{g/day}$. At intakes greater than this, absorption of soluble lead may be less than the default value; however, it can be similarly argued that, based on the Sherlock et al. (1984) regression model, the default $AF_{soluble}$ may underestimate absorption by some degree at low exposures.

Effect of lead form and particle size on lead bioavailability. The default value of 0.2 for $AF_{soluble}$ applies to soluble forms of lead in drinking water and food and would be expected to overestimate absorption of less soluble forms of lead in soil. Experimental studies have shown that the bioavailability of lead in soil tends to be less than that of soluble lead. Weis et al. (1994) assessed the relative bioavailability of lead in soil compared to water soluble lead (acetate) in immature swine and estimated that the relative bioavailability of lead in soil from Leadville, CO was 0.6 to 0.8. Ruby et al. (1996) reported estimates of the relative bioavailability of lead in a variety of soils from mining sites and smelters as assessed in the Sprague-Dawley rat; the estimates ranged from 0.09 to 0.4. Maddaloni et al. (1996) reported preliminary data from a study in which 6 fasted human subjects were administered a single dose of lead-contaminated soil. The dose was 250 μg lead normalized to a 70 kg body weight; the concentration of lead in the soil was 2850 $\mu\text{g/g}$ and the amount of soil administered to each subject was generally a little less than 100 mg. The average estimate of lead absorption in the six subjects was 26%. If the absorption factor for soluble lead in fasted adults is assumed to be 0.6 (James et al., 1985), then the Maddaloni et al. (1996) estimate suggests a relative bioavailability of 0.5 (i.e., 0.3/0.6) for lead in soil.

Based on the above evidence, the TRW considers 0.6 to be a plausible default point estimate for the relative bioavailability of lead in soil compared to soluble lead ($RBF_{soil/soluble}$) when site-specific data are not available. Such data are highly desirable as variation in relative bioavailability is expected for different species of lead and different particle sizes (Barltrop and Meek, 1975, 1979), both of which may vary from site to site. For example, the bioavailability of metallic lead has been shown to decrease with increasing particle size (Barltrop and Meek, 1979), therefore, the default value for $RBF_{soil/soluble}$ may overestimate absorption of lead if applied to soils contaminated with large lead particles such as firing range debris or mine tailings. Here again, the TRW cautions against the use of a lower value for the $RBF_{soil/soluble}$, unless it can be supported by experimental assessments of relative bioavailability.

The default value of 0.6 for $RBF_{\text{soil/soluble}}$, coupled with the default value of 0.2 for AF_{soluble} , yields a default value of 0.12 for AF_S ($0.6 \cdot 0.2$). The TRW considers 0.12 to be a plausible point estimate for the absorbed fraction of ingested soil lead for use in assessments in which site-specific data on lead bioavailability are not available. The default value of 0.12 takes into account uncertainties regarding the possible nonlinearity in the relationship between lead intake and absorption and should be adequately protective in scenarios in which predicted blood lead concentrations are less than 20 $\mu\text{g}/\text{dL}$. The use of the default value for populations that have substantially higher blood lead concentrations may result in an overestimate of lead uptake, and conversely, lead uptake may be underestimated at lower exposures.

7. Daily Soil Ingestion Rate (IR_S)

The TRW recommends a default value of 0.05 g/day as a plausible point estimate of the central tendency for daily soil intake from all occupational sources, including soil in indoor dust, resulting from non-contact intensive activities. This would include exposures that are predominantly indoors. More intensive soil contact would be expected for predominantly outdoor activities such as construction, excavation, yard work, and gardening (Hawley, 1985). Site-specific data on soil contact intensity, including potential seasonal variations, should be considered in evaluating whether or not the default value is applicable to the population of concern and, if not, activity-weighted estimates of IR_S that more accurately reflect the site can be developed.

In adopting the single IR_S parameter to describe all sources of ingested soil, the methodology remains consistent with recommendations of the Superfund program and their implementation for risk assessment; specifically, the 0.05 g/day value used for adult soil ingestion addresses all occupational soil intake by the individual, whether directly from soil or indirectly through contact with dust (U.S. EPA, 1993). This value specifically applies to the assessment of soil lead risk, and not risks associated with non-soil sources of lead in dust. In making soil ingestion exposure estimates under the Risk Assessment Guidelines for Superfund (RAGS) framework, no specific assumptions are needed about the fraction of soil intake that occurs through dust.

An alternative approach was needed in the IEUBK Model because childhood lead exposures are often strongly influenced by indoor sources of lead in dust (e.g., indoor paint) (U.S. EPA, 1994b). In a situation where indoor sources of dust contamination are important, an exposure estimate that addresses only soil exposures (including the soil component of dust) would be incomplete. The IEUBK Model assigns separate values to outdoor soil and total indoor dust ingestion and partitions the indoor dust into soil-derived and non-soil-derived sources. At a minimum, paired soil and indoor dust samples should be collected to adequately characterize exposure to lead where indoor sources of dust lead may be significant.

Alternate method for calculating soil and dust ingestion as separate exposure pathways. In this alternate approach, separate estimates are made of lead intake from the direct ingestion of outdoor soil and from the ingestion of indoor dust (which may contain lead from soil and as well as from indoor sources such as deteriorated lead based paint). Exposure to lead from soil (outdoor

contact) can be calculated using Equation A-12, while exposure to lead from indoor dust can be calculated using Equation A-13.

$$INTAKE_{S, outdoors} = \frac{PbS \cdot IR_{S, outdoors} \cdot EF_{Site}}{AT} \quad (\text{Equation A-12})$$

$$INTAKE_{D, indoors} = \frac{PbD \cdot IR_{D, indoors} \cdot EF_{Site}}{AT} \quad (\text{Equation A-13})$$

$INTAKE_{S, outdoors}$	=	Daily average intake (ingestion) of lead from soil ingested outdoors ($\mu\text{g}/\text{day}$).
$INTAKE_{D, indoors}$	=	Daily average intake (ingestion) of lead from dust ingested indoors ($\mu\text{g}/\text{day}$).
PbS	=	Soil lead concentration ($\mu\text{g}/\text{g}$) (average concentration in assessed individual exposure area).
PbD	=	Indoor dust lead concentration ($\mu\text{g}/\text{g}$).
$IR_{S, outdoors}$	=	Intake rate (ingestion) of outdoor soil (g/day).
$IR_{D, indoors}$	=	Intake rate (ingestion) of indoor dust (g/day).
EF_{Site}	=	Exposure frequency at site (days of exposure during the averaging period); may be taken as days per year for continuing, long term exposures.
AT	=	Averaging time, the total period during which the assessed exposures (from all sources) occur (days). May be taken as 365 days per year for continuing, long term exposures.

Note that, in Equations A-12 and A-13, exposure frequency refers to the number of days that an individual is present at the site and does not partition between periods of indoor and outdoor exposures. The intake rate is a long term average value appropriate for that media and is influenced by both the duration of outdoor (or indoor) exposures and the intensity of those exposures.

Calculation of $IR_{S, outdoors}$ and $IR_{D, indoors}$ from total intake of soil and dust (IR_{S+D}).

Intermediary calculations may be needed to generate estimates of the parameters in the intake equations. An estimate of the total intake of soil and dust materials (IR_{S+D}) serves as a starting point. Note that IR_{S+D} differs from IR_S , which was discussed above, because IR_{S+D} includes not only the total mass of soil ingested (both directly and as a component of indoor dust), but also the ingested

mass of non-soil derived dust components including various materials of indoor origin. Since a substantial fraction of the mass of indoor dust comes from sources other than outdoor soils, an estimate of IR_{S+D} will be higher than the corresponding estimate of IR_S . Secondly, an estimate of the fraction the total soil and dust intake that is ingested directly as soil is needed ($Weighting_{soil}$). This estimate needs to take into account the intensity and duration of the outdoor soil intake and the indoor dust intake. Equations A-14 and A-15 can be used to derive media-specific ingestion rates from IR_{S+D} and $Weighting_{soil}$.

$$IR_{S,outdoors} = Weighting_{soil} \cdot IR_{S+D} \quad (\text{Equation A-14})$$

$$IR_{D,indoors} = (1 - Weighting_{soil}) \cdot IR_{S+D} \quad (\text{Equation A-15})$$

$Weighting_{soil}$ = Fraction of total soil and dust intake that is directly ingested as soil (dimensionless).

IR_{S+D} = Total daily average intake of outdoor soil and indoor dust (all dust components) (g/day).

Data are needed to generate separate estimates of the concentrations of lead in outdoor soil and in indoor dust. A site assessment using this alternate methodology would generally be based on direct measurement data for both soil and dust at the facilities of concern. For comparison with exposure estimates based on total soil ingestion (the primary approach presented in this paper), Equation A-16 may be utilized to estimate the ratio of dust lead concentration to soil lead concentration.

$$PbD = PbS \cdot K_{SD} \quad (\text{Equation A-16})$$

K_{SD} = Ratio of indoor dust lead concentration to soil lead concentration (dimensionless).

Assuming that the same absorption fraction is applicable to both soil and dust, Equation A-17 may be used to estimate the uptake of lead from these two sources.

$$UPTAKE = AF_{S,D} \cdot (INTAKE_{S,outdoors} + INTAKE_{D,indoors}) \quad (\text{Equation A-17})$$

UPTAKE = Daily average uptake of lead from the gastrointestinal tract into the systemic circulation; soil and dust sources ($\mu\text{g}/\text{day}$).

$AF_{S,D}$ = Absolute gastrointestinal absorption fraction for ingested lead in soil and dust (dimensionless).

Comparison of lead intake estimated from principal and alternate approaches. It is helpful to compare exposure estimates derived using our principal approach based on total soil intake (including soil present in ingested dust) with the results of the disaggregated pathway analysis for soil and dust. We will consider the case in which there are not important indoor sources of lead in dust. We can then compare the total lead intake estimates from the two approaches.

Under the model based on total soil ingestion (which we re-label as $IR_{S,total}$ for clarity):

$$INTAKE = \frac{PbS \cdot IR_{S,total} \cdot EF_{Site}}{AT} \quad (\text{Equation A-18})$$

By contrast, using the disaggregated soil and dust model, Equations A-14, A-15, A-16, and A-18 may be combined to give Equation A-19:

$$INTAKE = \frac{PbS \cdot IR_{S+D} \cdot (Weighting_{soil} + K_{SD} \cdot (1 - Weighting_{soil})) \cdot EF_{Site}}{AT} \quad (\text{Equation A-19})$$

When applied to the same exposure assessment problem, the two approaches should give equivalent estimates of lead intake. The estimates will be equivalent when:

$$IR_{S+D} \cdot (Weighting_{soil} + K_{SD} \cdot (1 - Weighting_{soil})) = IR_{S,total}$$

8. Exposure Frequency (EF_S)

The TRW recommends a default value of 219 days/year. This is the same as the central tendency occupational exposure frequency recommended by U.S. EPA (1993) Superfund guidance, which is based on 1991 data from the Bureau of Labor Statistics. This estimate corresponds to the average time spent at work by both full-time and part-time workers engaged in non-contact intensive activities (U.S. EPA, 1993). Site-specific data on exposure frequency should be considered in evaluating whether or not the default value is applicable to the population of concern.

In evaluating site-specific data, it should be kept in mind that exposure frequency and daily soil ingestion rate (IR_S) may be interdependent variables, particularly in contact-intensive scenarios; therefore, the assignment of a site-specific value to EF_S should prompt an evaluation of the applicability of the default value for IR_S to the population of concern (see Section 7 of the Appendix for further discussion).

Nonresidential exposure scenarios in which exposure frequency would be substantially less than 219 days/year are frequently encountered. Examples include trespassing and recreational use of a site. Important methodology constraints on exposure frequency and duration must be considered in assigning values to EF_s that would represent infrequent contact with the site; these constraints relate to the steady state assumptions that underlie the BKSF. The BKSF derived from the Pocock et al. (1983) data applies to exposures that result in a quasi-steady state for blood lead concentration; that is, an intake over a sufficient duration for the blood lead concentration to become nearly constant over time. Based on estimates of the first order elimination half-time for lead in blood of approximately 30 days for adults (Rabinowitz, et al., 1974, 1976; Chamberlain et al., 1978), a constant lead intake rate over a duration of 90 days would be expected to achieve a blood lead concentration that is sufficiently close the quasi-steady state. This is the minimum exposure duration to which this methodology should be applied.

Infrequent exposures (i.e., less than 1 day per week) over a minimum duration of 90 days would be expected to produce oscillations in blood lead concentrations associated with the absorption and subsequent clearance of lead from the blood between each exposure event. Based on the above assumptions about the elimination half-time lead in blood, the TRW recommends that this methodology should not be applied to scenarios in which EF_s is less than 1 day/week.

9. Applying Monte Carlo Analysis to the Adult Lead Methodology

Recent EPA guidance (Browner, 1995) recommends that risk assessments include a clear and transparent discussion of variability and uncertainty. The lead risk assessment methodology presented here develops explicit estimates of the variability of blood lead levels among adults who are exposed to specified concentrations of environmental lead. This analysis relies on data from a large number of studies (baseline blood lead levels, variability of blood lead levels, contact rates with environmental media, lead bioavailability, and lead biokinetics) to support a predictive probabilistic (lognormal) model for adult and fetal blood lead concentrations. Important issues regarding the uncertainty in parameter inputs and the mathematical form of the model are discussed in the sections of this Appendix. The TRW recognizes that there is considerable scientific interest in the different analytical approaches that may be applied to aid in the analysis of variability and uncertainty in risk assessments. In particular, under appropriate circumstances, Monte Carlo methods may provide a useful approach for developing quantitative estimates of the variability, uncertainty (or both) in risk predictions.

The TRW chose not to pursue application of Monte Carlo or other stochastic simulation methods in this effort addressing adult lead risk assessment. Several factors went into this decision. First, the TRW understood the needs of EPA Regions for a risk model that could be developed relatively rapidly and which Regional lead risk assessors could apply easily with limited need for additional study or training. These considerations made it advantageous to focus on models that are conceptually similar to the IEUBK model for children in terms of applying a parametric lognormal modeling approach to address distributions for blood lead levels. Secondly, the TRW recognized that there would be substantial scientific issues associated with developing widely applicable stochastic simulation models for adult lead risk assessment. These difficulties primarily relate to the absence of reliable distributional data for a variety of important variables in the assessment. As

one example, very limited data are available on soil ingestion rates in adults and a distributional choice for this key parameter would depend heavily on individual judgement with little Agency precedent for support. Additionally, in a stochastic assessment, a greater complexity would arise due to likely correlations among the variables in the adult lead risk assessment. Stochastic analyses need to explicitly account for important correlations among variables if the simulations are to provide realistic distributions of risk. As an example, dependence is likely to exist between the starting (non-site related) blood lead concentrations for individuals and their site-related increases in blood lead. This dependence may result from individual patterns of behavior and from biological factors associated with lead pharmacokinetics. However, data on this dependence are sparse or absent, and the necessary statistical estimates of the correlation strength would depend heavily on personal judgement.

The TRW does encourage further efforts to better define the distributional data on which stochastic simulations of lead risks might rest. Further attention to these data can provide useful insights for lead risk assessment. The TRW also recognizes that Regions may be presented with lead risk assessments based on Monte Carlo modeling. In order to facilitate review of Monte Carlo analyses, some EPA Regions have found it important to establish requirements for the orderly development and review of these assessments. Borrowing on this approach, the TRW recommends that:

- A plan for the use of Monte Carlo analysis in a lead risk assessment should be submitted to responsible Regional personnel and accepted by them before the Monte Carlo analysis is undertaken.
- In general, it is expected that site-specific exposure related parameters that are supported with site-specific information will provide the basis for proposed Monte Carlo simulations.
- Scientific review is needed to determine that the risk assessment conformed to the plan and to evaluate the reliability of the results.

These recommendations are designed to ensure that assessments can provide meaningful results that can be understood and evaluated. If analyses are submitted in a format that is difficult to understand, the utility of the analysis will be diminished. We recommend that Regional staff seek advice from the TRW as a resource in this process.

10. References

- Alexander, F.W. and H.T. Delves. 1981. Blood lead levels during pregnancy. *Int. Arch. Occup. Environ. Health.* 48: 35-39.
- Baghurst, P.A., A.J. McMichael, G.V. Vimpani, E.F. Robertson, P.D. Clark, and N.R. Wigg. 1987. Determinants of blood lead concentrations of pregnant women living in Port Pirie and surrounding areas. *Medical J. of Australia.* 146: 69-73.
- Balbus-Kornfeld, J. 1994. Comments and Recommendations on the Draft Interim Guidance for Screening Levels of Lead in Soil for Non-Residential Sites. Letter from John Balbus-Kornfeld to Bruce Means. November 17, 1994.
- Barltrop, D. and F. Meek. 1975. Absorption of different lead compounds. *Postgrad. Med. J.* 51: 805-809.
- Barltrop, D. and F. Meek. 1979. Effect of particle size on lead absorption from the gut. *Arch. Environ. Health.* 34: 280-285.
- Blake, K.C.H. 1976. Absorption of ^{203}Pb from gastrointestinal tract of man. *Environ. Res.* 11: 1-4.
- Blake, K.C.H. and M. Mann. 1983. Effect of calcium and phosphorus on the gastrointestinal absorption of ^{203}Pb in man. *Environ. Res.* 30: 188-194.
- Blake, K.C.H., G.O. Barbezat and M. Mann. 1983. Effect of dietary constituents on the gastrointestinal absorption of ^{203}Pb in man. *Environ. Res.* 30: 182-187.
- Bowers, T.S., B.D. Beck and H.S. Karam. 1994. Assessing the relationship between environmental lead concentrations and adult blood lead levels. *Risk Analysis.* 14(2): 183-189.
- Brody, D.J. Personal communication on October 24, 1996 and October 29, 1996.
- Brody, D.J., J.L. Pirkle, R.A. Kramer, K.M. Flegal, T.D. Matte, E.W. Gunter and D.C. Paschal. 1994. Blood lead levels in the U.S. population. Phase 1 of the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA.* 272(4): 277-283.
- Browner, C.M. 1995. Policy for Risk Characterization at the U.S. EPA. Memorandum from U.S. EPA Administrator dated March 21, 1995.
- Chamberlain, A.C., M.J. Heard, P. Little, D. Newton, A.C. Wells and R.D. Wiffen. 1978. Investigations into lead from motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority, Report No. AERE-R9198.
- Davenport, H.W. 1971. Gastric digestion and emptying; absorption. *In: Physiology of the Digestive Tract*, 3rd ed. Year Book Medical Publishers Inc., Chicago. pp. 165-168.

- Franklin, C.A., M.J. Inskip, C.L. Baccanale, E.J. O'Flaherty, W.I. Manton, D.L. Schanzer, J. Blenkinsop and C.M. Edwards. 1995. Transplacental transfer of lead in non-human primates (*Macaca fascicularis*): use of serially administered stable isotope tracers of lead to elicit contribution of maternal bone lead to blood lead and the fetus. Poster presented at the 1995 meeting of the Society of Toxicology, Baltimore, MD. *The Toxicologist*. 15: 194.
- Gershanik, J.J., G.G. Brooks, and J.A. Little. 1974. Blood lead values in pregnant women and their offspring. *Amer. J. Obstet. Gynecol.* 4: 508-511.
- Goyer, R.A. 1990. Transplacental transport of lead. *Environ. Health Perspect.* 89: 101-105.
- Graziano, J.H., D. Popovac, P. Factor-Litvak, P. Shrout, J. Kline, M.J. Murphy, Y. Zhao, A. Mehmeti, X. Ahmedi, B. Rajovic, Z. Zvicer, D. Nenezic, N. Lolocono and Z. Stein. 1990. Determinants of elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. *Environ. Health Perspect.* 89: 95-100.
- Graziano, J.H., W.I. Manton, C.B. Blum and N.J. Lolocono. 1995. Bioavailability of lead in wine, by stable isotope dilution. Poster presented at the 1995 meeting of the Society of Toxicology, Baltimore, MD. *The Toxicologist*. 15: 135 (abst).
- Hawley, J.D. 1985. Assessment of health risk from exposure to contaminated soil. *Risk Analysis*. 5: 289-302.
- Heard, M.J. and A.C. Chamberlain. 1982. Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. *Human Toxicol.* 1: 411-415.
- Hunt, J.N. and W.R. Spurrell. 1951. The pattern of emptying of the human stomach. *J. Physiol.* 113: 157-168.
- James, H.M., M.E. Milburn and J.A. Blair. 1985. Effects of meals and meal times on uptake of lead from the gastrointestinal tract of humans. *Human Toxicol.* 4: 401-407.
- Maddaloni, M., W. Manton, C. Blum, N. Lolocono and J. Graziano. 1996. Bioavailability of soil-borne lead in adults, by stable isotope dilution. *The Toxicologist*. 30: 15 (abst.)
- NRC. 1993. *Measuring Lead Exposure in Infants, Children and Other Sensitive Populations*. National Academy Press. Washington, DC. ISBN 0-309-04927-X.
- Pocock, S.J., A.G. Shaper, M. Walker, C.J. Wale, B. Clayton, T. Delves, R.F. Lacey, R.F. Packham and P. Powell. 1983. Effects of tap water lead, water hardness, alcohol, and cigarettes on blood lead concentrations. *J. Epidemiol. Commun. Health.* 37: 1-7.
- Rabinowitz, M.B., G.W. Wetherill and J.D. Koppel. 1974. Studies of human lead metabolism by use of stable isotope tracers. *Environ. Health Perspect.* 7: 145-153.

- Rabinowitz, M.B., G.W. Wetherill and J.D. Koppel. 1976. Kinetic analysis of lead metabolism in health humans. *J. Clin. Invest.* 58: 260-270.
- Rabinowitz, M.B., J.D. Koppel and G.W. Wetherill. 1980. Effect of food intake on fasting gastrointestinal lead absorption in humans. *Am. J. Clin. Nutr.* 33: 1784-1788.
- Ruby, M.V., A. Davis, R. Schoof, S. Eberle and C. M. Sellstone. 1996. Estimation of lead and arsenic bioavailability using a physiologically based extraction test. *Environ. Sci. Technol.* 30: 422-430.
- Sherlock, J., G. Smart, G.I. Forbes, M.R. Moore, W.J. Patterson, W.N. Richards and T.S. Wilson. 1982. Assessment of lead intakes and dose-response for a population in Ayr exposed to a plumbosolvent water supply. *Human Toxicol.* 1: 115-122.
- Sherlock, J.C., D. Ashby, H.T. Delves, G.I. Forbes, M.R. Moore, W.J. Patterson, S.J. Pocock, M.J. Quinn, W.N. Richards and T.S. Wilson. 1984. Reduction in exposure to lead from drinking water and its effect on blood lead concentrations. *Human Toxicol.* 3: 383-392.
- Silbergeld, E.K. 1991. Lead in bone: Implications for toxicology during pregnancy and lactation. *Environ. Health Perspect.* 91: 63-70.
- U.S. EPA. 1986. Air Quality Criteria for Lead Volumes I - IV. Environmental Criteria and Assessment Office, Office of Research and Development, RTP, NC. EPA 600/8-83-028 a-d.
- U.S. EPA. 1989. Exposure Factors Handbook. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/043.
- U.S. EPA. 1990. Supplement to the 1986 EPA Air Quality Criteria Document for Lead - Volume 1 Addendum. Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC. EPA-600/8-89/049A.
- U.S. EPA. 1992. A TRW Report: Review of the EPA Uptake Biokinetic Model for Lead at the Butte NPL Site. Technical Review Workgroup for Lead, October, 1992.
- U.S. EPA. 1993. Superfund's Standard Default Exposure Factors for the Central Tendency and RME-Draft. Working Draft, November 1993.
- U.S. EPA. 1994a. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive No. 9355.4-12. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/F-94/043, PB94-963282.
- U.S. EPA. 1994b. Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children (v. 0.99d). Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-94/040, PB94-963505.

U.S. EPA. 1994c. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-93/081, PB93-963510.

U.S. EPA. 1995. A TRW Report: Review of a Methodology for Establishing Risk-Based Soil Remediation Goals for the Commercial Areas of the California Gulch Site. Technical Review Workgroup for Lead, October, 1995.

Weis, C.P., G.M. Henningsen, R.L. Poppenga, B.J. Thacker, A. Curtis, R. Jolly and T. Harpstead. 1994. Use of an immature swine model to sensitively differentiate lead absorption from soluble and mineralogical matrices. Presented at the Society for Environmental Geochemistry and Health, Salt Lake City, UT, July 18-19, 1994.

APPENDIX B

Calculations of Preliminary Remediation Goals (PRGs)

Calculations of Preliminary Remediation Goals (PRGs)
U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee

Version date 8/14/01

Exposure Variable	PRG Equation ¹		Description of Exposure Variable	Units	Values for Non-Residential Exposure Scenario			
	1*	2**			Using Equation 1		Using Equation 2	
					GSDi = 1.9	GSDi = 2.3	GSDi = 1.9	GSDi = 2.3
PbB _{fetal,0.95}	X	X	95th percentile PbB in fetus	ug/dL	10	10	10	10
R _{fetal/maternal}	X	X	Fetal/maternal PbB ratio	--	0.9	0.9	0.9	0.9
BKSF	X	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4	0.4	0.4
GSD _i	X	X	Geometric standard deviation PbB	--	1.9	2.3	1.9	2.3
PbB ₀	X	X	Baseline PbB	ug/dL	1.4	1.8	1.4	1.8
IR _s	X		Soil ingestion rate (including soil-derived indoor dust)	g/day	0.050	0.050	--	--
IR _{s+d}		X	Total ingestion rate of outdoor soil and indoor dust	g/day	--	--	0.050	0.050
W _s		X	Weighting factor; fraction of IR _{s+d} ingested as outdoor soil	--	--	--	1.0	1.0
K _{SD}		X	Mass fraction of soil in dust	--	--	--	0.7	0.7
AF _{s,d}	X	X	Absorption fraction (same for soil and dust)	--	0.12	0.12	0.12	0.12
EF _{s,d}	X	X	Exposure frequency (same for soil and dust)	days/yr	219	219	219	219
AT _{s,d}	X	X	Averaging time (same for soil and dust)	days/yr	365	365	365	365
PRG	Preliminary Remediation Goal			ppm	1,712	710	1,712	710

¹ Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_s, K_{SD}). When IR_s = IR_{s+d} and W_s = 1.0, the equations yield the same PRG.

***Equation 1, based on Eq. 4 in U.S. EPA (1996)**

PRG =	$\frac{([PbB_{95\text{ fetal}}/(R*(GSD_i^{1.645}))]-PbB_0)*AT_{s,d}}{BKSF*(IR_{s+d}*AF_{s,d}*EF_{s,d})}$

****Equation 2, alternate approach based on Eq. 4 and Eq. A-19 in U.S. EPA (1996)**

PRG =	$\frac{([PbB_{\text{fetal},0.95}/(R*(GSD_i^{1.645}))]-PbB_0)*AT_{s,d}}{BKSF*([(IR_{s+d})*AF_s*EF_s*W_s]+[K_{SD}*(IR_{s+d})*(1-W_s)*AF_d*EF_d])}$

Source: U.S. EPA (1996). Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil

APPENDIX C

Memorandum April 7, 1999

MEMORANDUM

DATE: April 7, 1999

SUBJECT: Use of the TRW Interim Adult Lead Methodology in Risk Assessment

TO: Mark Maddaloni, Chair
TRW Adult Lead Subgroup

FROM: Pat Van Leeuwen
Region 5 Superfund Program

Paul White
ORD/NCEA

The December 1996 TRW report "Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil" presents tools that can be used in a risk assessment to provide an evaluation of risk relevant to the adult population. This report, referred to as the interim Adult Lead Methodology (ALM), focuses on the estimation of the blood lead concentrations in fetuses carried by women exposed to lead contaminated soils. However, the presentation in that document emphasizes the calculation of cleanup goals for soil and it has become apparent that there is some confusion among risk assessors regarding how to apply this methodology in a "forward" manner to predict baseline risks resulting from measured soil concentrations. This memorandum presents equations for calculation of fetal risks from adult exposures to specified levels of soil lead contamination. This approach will support EPA's goal of limiting the risk of elevated fetal blood lead concentrations due to lead exposures to women of child-bearing age. We have prepared this memorandum so that it may be included as an Appendix to the interim ALM to provide the needed clarification to risk assessors and others who use the ALM to support an evaluation of risk.

The risk assessment methodology in the ALM is based on a lognormal probability model for blood levels in adult women exposed to lead contaminated soils, coupled with an estimated constant of proportionality between fetal and maternal blood lead levels. These relationships specify that the distribution of fetal blood lead levels also follows a lognormal distribution:

$$PbB_{fetal} \sim \text{Lognormal}(GM, GSD)$$

Estimation of the probability that fetal blood lead levels will exceed the EPA blood lead level of concern of 10 ug/dL is a two step process:

(1) Calculate the geometric mean (central) fetal blood lead concentration ($PbB_{fetal,GM}$). Equation A-3 in the ALM Appendix provides an estimate of the central tendency adult blood lead level which is used to provide a plausible estimate of the geometric mean in the lognormal model for blood lead. When the expressions for lead UPTAKE (ALM Equations A-1 and A-2) are substituted into ALM Equation A-3 the following relationship is obtained:

$$PbB_{adult,central} = PbB_{adult,0} + \frac{PbS \cdot BKSF \cdot IR_S \cdot AF_S \cdot EF_S}{AT} \quad (\text{ALM Equation 1})$$

A second equation provides the constant of proportionality between fetal and adult blood lead levels:

$$PbB_{fetal} = R_{fetal/maternal} \cdot PbB_{adult} \quad (\text{ALM Equation A-5})$$

Combining these two relationships, the resulting equation for the fetal geometric mean blood lead level has the following form:

$$PbB_{fetal,GM} = R_{fetal/maternal} \cdot [PbB_{adult,0} + \frac{PbS \cdot BKSF \cdot IR_S \cdot AF_S \cdot EF_S}{AT}] \quad (\text{Equation 1})$$

Where:

- $PbB_{fetal,GM}$ = Central estimate of blood lead concentrations ($\mu\text{g/dL}$) for fetuses carried by women who have site exposures to soil lead at concentration, PbS.
- $R_{fetal/maternal}$ = Constant of proportionality between fetal and maternal blood lead concentrations.
- $PbB_{adult,0}$ = Typical blood lead concentration ($\mu\text{g/dL}$) in adults (i.e., women of child-bearing age) in the absence of exposures to the site that is being assessed.
- PbS = Soil lead concentration ($\mu\text{g/g}$) (appropriate average concentration for individual).
- BKSF = Biokinetic slope factor relating the (quasi-steady state) increase in typical adult blood lead concentration to average daily lead uptake ($\mu\text{g/dL}$ blood lead increase per $\mu\text{g/day}$ lead uptake).

IR_s	=	Intake rate of soil, including both outdoor soil and the soil-derived component of indoor dust (g/day).
AF_s	=	Absolute gastrointestinal absorption fraction for ingested lead in soil and lead in dust derived from soil (dimensionless).
EF_s	=	Exposure frequency for contact with assessed soils and/or dust derived in part from these soils (days of exposure during the averaging period); may be taken as days per year for continuing, long term exposures.
AT	=	Averaging time; the total period during which soil contact may occur; 365 days/year for continuing long term exposures.

(2) Determine the probability that the blood lead level for a fetus carried by a woman exposed to lead at a site exceeds 10 ug/dL. This calculation uses the fetal geometric mean (GM) blood lead from Equation 1 and the geometric standard deviation (GSD) value appropriate for the risk assessment. Note that because of the assumption of proportionality between fetal and maternal blood lead levels, the adult GSD and the fetal GSD are equal. If the assessor is using a spreadsheet or statistical program that provides a function to calculate lognormal probabilities, the GM and GSD values may directly used to calculate the exceedence probabilities. (Care must be taken to determine the exact form of the inputs needed by the statistical function, e. g., whether log scale inputs are required.) Alternatively, the following formula and table provide the needed tools for the probability calculation.

Recall that the logarithm of a lognormal variable follows a normal probability distribution. Exceedence probabilities for the lognormal model can be determined from standard normal model statistical tables after the GM, GSD, and exceedence criterion are converted to log scale values and a “standard normal deviate” or “z-value” is calculated:

$$z = \frac{\ln(10) - \ln(GM)}{\ln(GSD)} \quad (\text{Equation 2})$$

In this equation, $\ln()$ represents the natural logarithm function (log base e) which is applied in the definition of the lognormal distribution. Note, however, that calculations using base 10 logarithms would also yield the same numerical result.

A statistical program or a normal probability table can then be used to determine the exceedence probability. The attached standard normal table displays both positive and negative values of z for ease of reference. The table gives the probability, p, that a standard normal variable has a value less than z. The probability that the fetal blood lead level exceeds 10 ug/dL is obtained by from the expression 1-p.

EXAMPLE:

Assume that the risk calculation (Equation 1) gives a GM fetal blood lead level of 7.0 ug/dL, and the appropriate GSD is 1.8.

Then:

$$z = \frac{\ln(10) - \ln(7.0)}{\ln(1.8)} = \frac{2.303 - 1.946}{0.588} = 0.607$$

Under the normal distribution, the probability that z is less than 0.607 is $p = 0.728$ (obtained from a statistical program). From the attached normal table the probability may be adequately approximated by rounding 0.607 to 0.61 to get a probability of 0.729, or approximately 0.73.¹

The probability that the fetal blood lead level exceeds 10 ug/dL is estimated as $1 - p = 1 - 0.73 = 0.27$, or approximately 27%.

¹The precision of values obtained from the table can be increased, when necessary, by using linear interpolation between the table entries. For this example, interpolation using the values of $z = 0.60$ and $z = 0.61$ can be applied to calculate a probability, $p = 0.728$, as shown in the following equation:

$$0.72575 + (0.72907 - 0.72575) \cdot \frac{(0.607 - 0.600)}{(0.610 - 0.600)} = 0.728. \text{ In general, to interpolate the probability, } p, \text{ associated with}$$

z , find the z -values bracketing z in the normal table; i. e., find the z_1 and z_2 in adjoining rows of the table so that $z_1 < z < z_2$. Next find the values p_1 and p_2 in the table corresponding to z_1 and z_2 , respectively. The linearly interpolated

value for p is then: $p = p_1 + (p_2 - p_1) \cdot \frac{(z - z_1)}{(z_2 - z_1)}$.

Normal Probability Table

z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)
-3.50	0.00023	-3.00	.00135	-2.50	0.00621	-2.00	0.02275	-1.50	0.06681
-3.49	0.00024	-2.99	0.00139	-2.49	0.00639	-1.99	0.02330	-1.49	0.06811
-3.48	0.00025	-2.98	0.00144	-2.48	0.00657	-1.98	0.02385	-1.48	0.06944
-3.47	0.00026	-2.97	0.00149	-2.47	0.00676	-1.97	0.02442	-1.47	0.07078
-3.46	0.00027	-2.96	0.00154	-2.46	0.00695	-1.96	0.02500	-1.46	0.07215
-3.45	0.00028	-2.95	0.00159	-2.45	0.00714	-1.95	0.02559	-1.45	0.07353
-3.44	0.00029	-2.94	0.00164	-2.44	0.00734	-1.94	0.02619	-1.44	0.07493
-3.43	0.00030	-2.93	0.00169	-2.43	0.00755	-1.93	0.02680	-1.43	0.07636
-3.42	0.00031	-2.92	0.00175	-2.42	0.00776	-1.92	0.02743	-1.42	0.07780
-3.41	0.00032	-2.91	0.00181	-2.41	0.00798	-1.91	0.02807	-1.41	0.07927
-3.40	0.00034	-2.90	0.00187	-2.40	0.00820	-1.90	0.02872	-1.40	0.08076
-3.39	0.00035	-2.89	0.00193	-2.39	0.00842	-1.89	0.02938	-1.39	0.08226
-3.38	0.00036	-2.88	0.00199	-2.38	0.00866	-1.88	0.03005	-1.38	0.08379
-3.37	0.00038	-2.87	0.00205	-2.37	0.00889	-1.87	0.03074	-1.37	0.08534
-3.36	0.00039	-2.86	0.00212	-2.36	0.00914	-1.86	0.03144	-1.36	0.08691
-3.35	0.00040	-2.85	0.00219	-2.35	0.00939	-1.85	0.03216	-1.35	0.08851
-3.34	0.00042	-2.84	0.00226	-2.34	0.00964	-1.84	0.03288	-1.34	0.09012
-3.33	0.00043	-2.83	0.00233	-2.33	0.00990	-1.83	0.03362	-1.33	0.09176
-3.32	0.00045	-2.82	0.00240	-2.32	0.01017	-1.82	0.03438	-1.32	0.09342
-3.31	0.00047	-2.81	0.00248	-2.31	0.01044	-1.81	0.03515	-1.31	0.09510
-3.30	0.00048	-2.80	0.00256	-2.30	0.01072	-1.80	0.03593	-1.30	0.09680
-3.29	0.00050	-2.79	0.00264	-2.29	0.01101	-1.79	0.03673	-1.29	0.09853
-3.28	0.00052	-2.78	0.00272	-2.28	0.01130	-1.78	0.03754	-1.28	0.10027
-3.27	0.00054	-2.77	0.00280	-2.27	0.01160	-1.77	0.03836	-1.27	0.10204
-3.26	0.00056	-2.76	0.00289	-2.26	0.01191	-1.76	0.03920	-1.26	0.10383
-3.25	0.00058	-2.75	0.00298	-2.25	0.01222	-1.75	0.04006	-1.25	0.10565
-3.24	0.00060	-2.74	0.00307	-2.24	0.01255	-1.74	0.04093	-1.24	0.10749
-3.23	0.00062	-2.73	0.00317	-2.23	0.01287	-1.73	0.04182	-1.23	0.10935
-3.22	0.00064	-2.72	0.00326	-2.22	0.01321	-1.72	0.04272	-1.22	0.11123
-3.21	0.00066	-2.71	0.00336	-2.21	0.01355	-1.71	0.04363	-1.21	0.11314
-3.20	0.00069	-2.70	0.00347	-2.20	0.01390	-1.70	0.04457	-1.20	0.11507
-3.19	0.00071	-2.69	0.00357	-2.19	0.01426	-1.69	0.04551	-1.19	0.11702
-3.18	0.00074	-2.68	0.00368	-2.18	0.01463	-1.68	0.04648	-1.18	0.11900
-3.17	0.00076	-2.67	0.00379	-2.17	0.01500	-1.67	0.04746	-1.17	0.12100
-3.16	0.00079	-2.66	0.00391	-2.16	0.01539	-1.66	0.04846	-1.16	0.12302
-3.15	0.00082	-2.65	0.00402	-2.15	0.01578	-1.65	0.04947	-1.15	0.12507
-3.14	0.00084	-2.64	0.00415	-2.14	0.01618	-1.64	0.05050	-1.14	0.12714
-3.13	0.00087	-2.63	0.00427	-2.13	0.01659	-1.63	0.05155	-1.13	0.12924
-3.12	0.00090	-2.62	0.00440	-2.12	0.01700	-1.62	0.05262	-1.12	0.13136
-3.11	0.00094	-2.61	0.00453	-2.11	0.01743	-1.61	0.05370	-1.11	0.13350
-3.10	0.00097	-2.60	0.00466	-2.10	0.01786	-1.60	0.05480	-1.10	0.13567
-3.09	0.00100	-2.59	0.00480	-2.09	0.01831	-1.59	0.05592	-1.09	0.13786
-3.08	0.00104	-2.58	0.00494	-2.08	0.01876	-1.58	0.05705	-1.08	0.14007
-3.07	0.00107	-2.57	0.00508	-2.07	0.01923	-1.57	0.05821	-1.07	0.14231
-3.06	0.00111	-2.56	0.00523	-2.06	0.01970	-1.56	0.05938	-1.06	0.14457
-3.05	0.00114	-2.55	0.00539	-2.05	0.02018	-1.55	0.06057	-1.05	0.14686
-3.04	0.00118	-2.54	0.00554	-2.04	0.02068	-1.54	0.06178	-1.04	0.14917
-3.03	0.00122	-2.53	0.00570	-2.03	0.02118	-1.53	0.06301	-1.03	0.15151
-3.02	0.00126	-2.52	0.00587	-2.02	0.02169	-1.52	0.06426	-1.02	0.15386
-3.01	0.00131	-2.51	0.00604	-2.01	0.02222	-1.51	0.06552	-1.01	0.15625

Normal Probability Table

z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)
-1.00	0.15866	-0.50	0.30854	0.00	0.50000	0.50	0.69146	1.00	0.84134
-0.99	0.16109	-0.49	0.31207	0.01	0.50399	0.51	0.69497	1.01	0.84375
-0.98	0.16354	-0.48	0.31561	0.02	0.50798	0.52	0.69847	1.02	0.84614
-0.97	0.16602	-0.47	0.31918	0.03	0.51197	0.53	0.70194	1.03	0.84849
-0.96	0.16853	-0.46	0.32276	0.04	0.51595	0.54	0.70540	1.04	0.85083
-0.95	0.17106	-0.45	0.32636	0.05	0.51994	0.55	0.70884	1.05	0.85314
-0.94	0.17361	-0.44	0.32997	0.06	0.52392	0.56	0.71226	1.06	0.85543
-0.93	0.17619	-0.43	0.33360	0.07	0.52790	0.57	0.71566	1.07	0.85769
-0.92	0.17879	-0.42	0.33724	0.08	0.53188	0.58	0.71904	1.08	0.85993
-0.91	0.18141	-0.41	0.34090	0.09	0.53586	0.59	0.72240	1.09	0.86214
-0.90	0.18406	-0.40	0.34458	0.10	0.53983	0.60	0.72575	1.10	0.86433
-0.89	0.18673	-0.39	0.34827	0.11	0.54380	0.61	0.72907	1.11	0.86650
-0.88	0.18943	-0.38	0.35197	0.12	0.54776	0.62	0.73237	1.12	0.86864
-0.87	0.19215	-0.37	0.35569	0.13	0.55172	0.63	0.73565	1.13	0.87076
-0.86	0.19489	-0.36	0.35942	0.14	0.55567	0.64	0.73891	1.14	0.87286
-0.85	0.19766	-0.35	0.36317	0.15	0.55962	0.65	0.74215	1.15	0.87493
-0.84	0.20045	-0.34	0.36693	0.16	0.56356	0.66	0.74537	1.16	0.87698
-0.83	0.20327	-0.33	0.37070	0.17	0.56749	0.67	0.74857	1.17	0.87900
-0.82	0.20611	-0.32	0.37448	0.18	0.57142	0.68	0.75175	1.18	0.88100
-0.81	0.20897	-0.31	0.37828	0.19	0.57535	0.69	0.75490	1.19	0.88298
-0.80	0.21186	-0.30	0.38209	0.20	0.57926	0.70	0.75804	1.20	0.88493
-0.79	0.21476	-0.29	0.38591	0.21	0.58317	0.71	0.76115	1.21	0.88686
-0.78	0.21770	-0.28	0.38974	0.22	0.58706	0.72	0.76424	1.22	0.88877
-0.77	0.22065	-0.27	0.39358	0.23	0.59095	0.73	0.76730	1.23	0.89065
-0.76	0.22363	-0.26	0.39743	0.24	0.59483	0.74	0.77035	1.24	0.89251
-0.75	0.22663	-0.25	0.40129	0.25	0.59871	0.75	0.77337	1.25	0.89435
-0.74	0.22965	-0.24	0.40517	0.26	0.60257	0.76	0.77637	1.26	0.89617
-0.73	0.23270	-0.23	0.40905	0.27	0.60642	0.77	0.77935	1.27	0.89796
-0.72	0.23576	-0.22	0.41294	0.28	0.61026	0.78	0.78230	1.28	0.89973
-0.71	0.23885	-0.21	0.41683	0.29	0.61409	0.79	0.78524	1.29	0.90147
-0.70	0.24196	-0.20	0.42074	0.30	0.61791	0.80	0.78814	1.30	0.90320
-0.69	0.24510	-0.19	0.42465	0.31	0.62172	0.81	0.79103	1.31	0.90490
-0.68	0.24825	-0.18	0.42858	0.32	0.62552	0.82	0.79389	1.32	0.90658
-0.67	0.25143	-0.17	0.43251	0.33	0.62930	0.83	0.79673	1.33	0.90824
-0.66	0.25463	-0.16	0.43644	0.34	0.63307	0.84	0.79955	1.34	0.90988
-0.65	0.25785	-0.15	0.44038	0.35	0.63683	0.85	0.80234	1.35	0.91149
-0.64	0.26109	-0.14	0.44433	0.36	0.64058	0.86	0.80511	1.36	0.91309
-0.63	0.26435	-0.13	0.44828	0.37	0.64431	0.87	0.80785	1.37	0.91466
-0.62	0.26763	-0.12	0.45224	0.38	0.64803	0.88	0.81057	1.38	0.91621
-0.61	0.27093	-0.11	0.45620	0.39	0.65173	0.89	0.81327	1.39	0.91774
-0.60	0.27425	-0.10	0.46017	0.40	0.65542	0.90	0.81594	1.40	0.91924
-0.59	0.27760	-0.09	0.46414	0.41	0.65910	0.91	0.81859	1.41	0.92073
-0.58	0.28096	-0.08	0.46812	0.42	0.66276	0.92	0.82121	1.42	0.92220
-0.57	0.28434	-0.07	0.47210	0.43	0.66640	0.93	0.82381	1.43	0.92364
-0.56	0.28774	-0.06	0.47608	0.44	0.67003	0.94	0.82639	1.44	0.92507
-0.55	0.29116	-0.05	0.48006	0.45	0.67364	0.95	0.82894	1.45	0.92647
-0.54	0.29460	-0.04	0.48405	0.46	0.67724	0.96	0.83147	1.46	0.92785
-0.53	0.29806	-0.03	0.48803	0.47	0.68082	0.97	0.83398	1.47	0.92922
-0.52	0.30153	-0.02	0.49202	0.48	0.68439	0.98	0.83646	1.48	0.93056
-0.51	0.30503	-0.01	0.49601	0.49	0.68793	0.99	0.83891	1.49	0.93189

Normal Probability Table

z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)	z-value	p (prob. of lesser value)
1.50	0.93319	2.00	0.97725	2.50	0.99379	3.00	0.99865		
1.51	0.93448	2.01	0.97778	2.51	0.99396	3.01	0.99869		
1.52	0.93574	2.02	0.97831	2.52	0.99413	3.02	0.99874		
1.53	0.93699	2.03	0.97882	2.53	0.99430	3.03	0.99878		
1.54	0.93822	2.04	0.97932	2.54	0.99446	3.04	0.99882		
1.55	0.93943	2.05	0.97982	2.55	0.99461	3.05	0.99886		
1.56	0.94062	2.06	0.98030	2.56	0.99477	3.06	0.99889		
1.57	0.94179	2.07	0.98077	2.57	0.99492	3.07	0.99893		
1.58	0.94295	2.08	0.98124	2.58	0.99506	3.08	0.99896		
1.59	0.94408	2.09	0.98169	2.59	0.99520	3.09	0.99900		
1.60	0.94520	2.10	0.98214	2.60	0.99534	3.10	0.99903		
1.61	0.94630	2.11	0.98257	2.61	0.99547	3.11	0.99906		
1.62	0.94738	2.12	0.98300	2.62	0.99560	3.12	0.99910		
1.63	0.94845	2.13	0.98341	2.63	0.99573	3.13	0.99913		
1.64	0.94950	2.14	0.98382	2.64	0.99585	3.14	0.99916		
1.65	0.95053	2.15	0.98422	2.65	0.99598	3.15	0.99918		
1.66	0.95154	2.16	0.98461	2.66	0.99609	3.16	0.99921		
1.67	0.95254	2.17	0.98500	2.67	0.99621	3.17	0.99924		
1.68	0.95352	2.18	0.98537	2.68	0.99632	3.18	0.99926		
1.69	0.95449	2.19	0.98574	2.69	0.99643	3.19	0.99929		
1.70	0.95543	2.20	0.98610	2.70	0.99653	3.20	0.99931		
1.71	0.95637	2.21	0.98645	2.71	0.99664	3.21	0.99934		
1.72	0.95728	2.22	0.98679	2.72	0.99674	3.22	0.99936		
1.73	0.95818	2.23	0.98713	2.73	0.99683	3.23	0.99938		
1.74	0.95907	2.24	0.98745	2.74	0.99693	3.24	0.99940		
1.75	0.95994	2.25	0.98778	2.75	0.99702	3.25	0.99942		
1.76	0.96080	2.26	0.98809	2.76	0.99711	3.26	0.99944		
1.77	0.96164	2.27	0.98840	2.77	0.99720	3.27	0.99946		
1.78	0.96246	2.28	0.98870	2.78	0.99728	3.28	0.99948		
1.79	0.96327	2.29	0.98899	2.79	0.99736	3.29	0.99950		
1.80	0.96407	2.30	0.98928	2.80	0.99744	3.30	0.99952		
1.81	0.96485	2.31	0.98956	2.81	0.99752	3.31	0.99953		
1.82	0.96562	2.32	0.98983	2.82	0.99760	3.32	0.99955		
1.83	0.96638	2.33	0.99010	2.83	0.99767	3.33	0.99957		
1.84	0.96712	2.34	0.99036	2.84	0.99774	3.34	0.99958		
1.85	0.96784	2.35	0.99061	2.85	0.99781	3.35	0.99960		
1.86	0.96856	2.36	0.99086	2.86	0.99788	3.36	0.99961		
1.87	0.96926	2.37	0.99111	2.87	0.99795	3.37	0.99962		
1.88	0.96995	2.38	0.99134	2.88	0.99801	3.38	0.99964		
1.89	0.97062	2.39	0.99158	2.89	0.99807	3.39	0.99965		
1.90	0.97128	2.40	0.99180	2.90	0.99813	3.40	0.99966		
1.91	0.97193	2.41	0.99202	2.91	0.99819	3.41	0.99968		
1.92	0.97257	2.42	0.99224	2.92	0.99825	3.42	0.99969		
1.93	0.97320	2.43	0.99245	2.93	0.99831	3.43	0.99970		
1.94	0.97381	2.44	0.99266	2.94	0.99836	3.44	0.99971		
1.95	0.97441	2.45	0.99286	2.95	0.99841	3.45	0.99972		
1.96	0.97500	2.46	0.99305	2.96	0.99846	3.46	0.99973		
1.97	0.97558	2.47	0.99324	2.97	0.99851	3.47	0.99974		
1.98	0.97615	2.48	0.99343	2.98	0.99856	3.48	0.99975		
1.99	0.97670	2.49	0.99361	2.99	0.99861	3.49	0.99976		

