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Current uses of the EPA lead model to assess health risk and action levels for soil

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The EPA lead model predicts mean blood lead levels and risk of elevated blood lead levels in children based on lead uptake from multiple sources. In the latest model versions, environmental data from individual homes within a community can be used to predict the overall blood lead distribution and percent risk of exceeding a specific blood lead level (*i.e.* 10 μg dl^{-1}). Recent criteria used by the EPA to evaluate this information include no more than 5% of houses with a greater than 5% lead risk, and a community weighted-average risk below 5%. Environmental (primarily soil) and blood lead data from a residential community near a smelter were used to illustrate recent uses of the model. Scheduled remediation in the community will remove soil for approximately 60% of the houses (*i.e.* those with lead levels > 1000 mg kg^{-1}). After remediation, the model results indicate a relatively low community risk (0.5–1.9%), although the percentage of houses with lead risks above 5% ranged from 3 to as high as 13%, depending on the variation in blood lead and assuming the model's 7 μg dl^{-1} increase in blood lead with each 1000 mg kg^{-1} increase in soil lead level. A comparison of the limited blood lead data with soil lead levels below 1000 mg kg^{-1}, however, indicated no apparent relationship. Given these uncertainties, less invasive actions than additional soil removal (*e.g.* exposure intervention, monitoring conditions, and follow-up as necessary) may be appropriate under the new EPA guidance for lead in soil.

Keywords: Lead, soil, EPA lead model, blood lead level, lead smelter

Introduction

The US Environmental Protection Agency's (EPA) current guidelines for assessing health risks and remedial action levels for lead in soil involves the use of the integrated exposure uptake/biokinetic lead model (EPA lead model; Laws, 1994). This mathematical model estimates blood lead levels in young children based on environmental exposures to lead from various sources including soil. The model was initially developed by the EPA Office of Air Quality Planning and Standards and New York University (US EPA, 1994), and has been reviewed by other agency work groups (US EPA SAB, 1992).

The latest versions of the EPA lead model can be used to predict several measures of health risk associated with lead in soil for a community and for a given individual. These results provide risk managers with more information about the nature of lead risk in a community. In addition, recent EPA guidance on remedial actions for lead in soil have taken a more holistic approach for managing exposure and health risks. The primary tool for developing preliminary remediation goals is the use of the EPA lead model; however, the type of remediation may involve a variety of risk management actions, many of which are less invasive than soil removal, but together have the potential to more completely manage lead exposures.

This paper uses data from a lead smelter site to illustrate some of the latest uses of the model by the EPA in assessing cleanup of lead in soil. This example also demonstrates some of the scientific aspects of the model which need to be resolved in order to reconcile the model predictions with statistical analyses of actual blood lead and environmental lead data.

Methodology

Background on the EPA lead model

Young children (*e.g.* age seven and under) are generally considered to be the age group of most concern for environmental exposures to lead (CDC, 1991) because of their mouthing behaviour, tendency to play on the ground, higher gastrointestinal absorption of lead, greater sensitivity to adverse effects of lead, and higher likelihood to have nutritional deficiencies that promote lead absorption. The EPA lead model thus attempts to take into account the many sources of lead in the environment in predicting blood lead levels of children. The concentration of lead in blood (usually expressed as micrograms of lead per decilitre of whole blood, μg dl^{-1}) is routinely used as an indicator of lead exposure and the potential for toxic effects (ATSDR, 1993).

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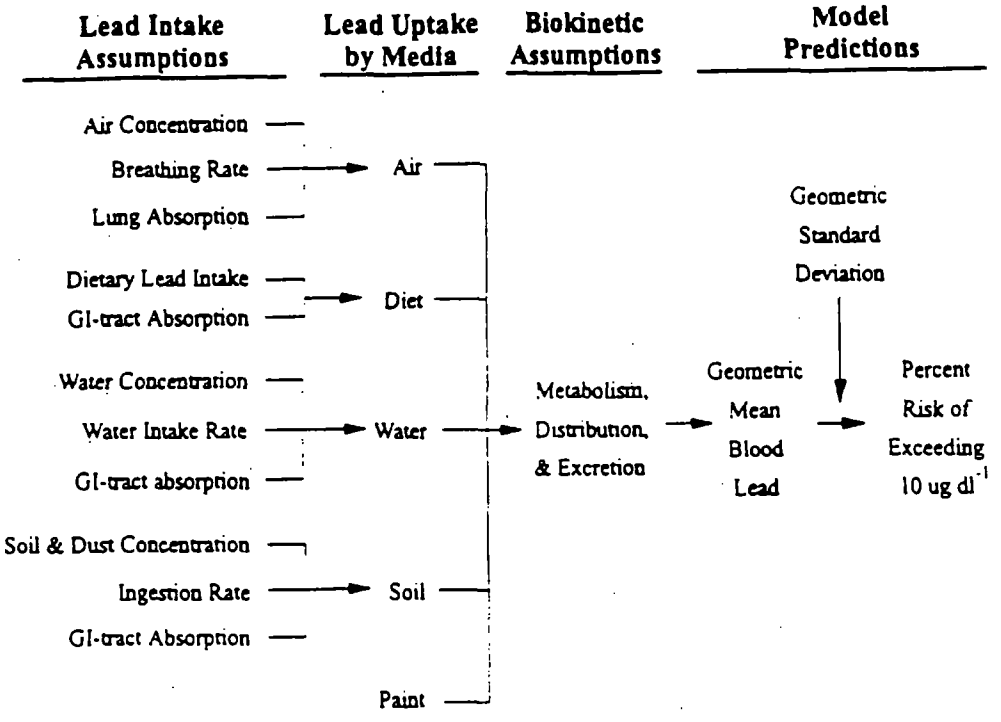

Figure 1 *EPA lead model diagram.*

The model combines input assumptions on lead uptake (*e.g.* lead concentrations, intake rates, and absorption rates for air, diet, water, soil/dust, and optionally lead intake from paint) with biokinetic assumptions on how lead behaves in the body (*e.g.* distribution and excretion; Figure 1). The user is able to specify uptake but not biokinetic assumptions. All input assumptions should be average or central tendency values (*e.g.* geometric mean for log normal distributions) in order to predict a geometric mean blood lead level (US EPA, 1994). Input of a geometric standard deviation (GSD) for the expected variation in blood lead results in a prediction of the log normal probability distribution for blood lead, from which the percent risk of exceeding a given blood lead level can be determined (Figure 2).

Current uses of the EPA lead model to assess lead risk

The EPA lead model has been used to address several assessment factors in evaluating blood lead risk due to soil lead levels in communities (Table 1). Past uses of the model have been to input a community average soil concentration to predict the community mean blood lead level and the percentage of children with blood lead levels in excess of a certain level (no longer recommended; US EPA, 1994). Alternatively, the soil concentration for a house can be used to estimate an individual child's mean blood lead level and percent risk of exceeding a given blood lead level. The EPA's criteria for assessing the model results have been for the average blood lead level to be less than 10 μg dl^{-1} and that the risk of exceeding a 10 μg dl^{-1} blood lead level (*alias* 'lead risk') for a "typical (or hypothetical) child or group of similarly exposed children" should not exceed 5% (Laws, 1994).

Versions 0.6 and higher of the lead model are able to incorporate the available data on soil, dust, water, *etc.*, from each house in the community using batch mode calculations that allow predictions of the distribution of mean blood lead levels

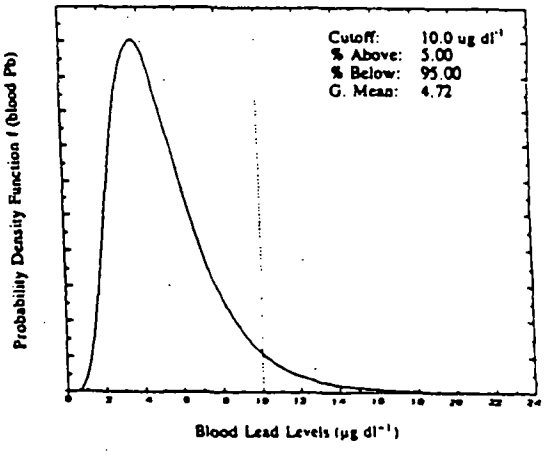

Figure 2 *Example of EPA lead model results: blood lead distribution or probability density function (G. Mean = geometric mean).*

Table 1 *Assessment of health risk depending on soil input data*

Mode	Type of soil data	Assessment factor	Criteria
Simple calculation	Community mean	Community mean blood lead level	$\leq 10\ \mu g\ dl^{-1}$
	Community mean	Probability of an average child in the community having a blood lead level $> 10\ \mu g\ dl^{-1}$	$\leq 5\%$
	Community mean	Percent of children with $\geq 10\ \mu g\ dl^{-1}$ blood lead level	$\leq 5\%$
	Single residence	Probability of a child having a blood lead level $> 10\ \mu g\ dl^{-1}$	$\leq 5\%$
Batch mode	All residences	Predicted mean blood lead levels	$\leq 10\ \mu g\ dl^{-1}$
	All residences	Percent of children with $> 5\%$ chance of exceeding $10\ \mu g\ dl^{-1}$	$\leq 5\%$
	All residences	Community wide-risk of exceeding $10\ \mu g\ dl^{-1}$	$\leq 5\%$

within a community. The percentage of houses associated with a greater than 5% lead risk, and the lead risk for the community based on a weighted average of the data from individual houses can also be calculated using external calculations to the batch mode. The number of children (or houses) with a greater than 5% risk of exceeding a 10 $\mu g\ dl^{-1}$ blood lead level within a community should not exceed 5% (US EPA Region VIII, 1993).

Smelter site example

Data from a residential community near an operating lead smelter in Montana are used as an example for presenting the results of the EPA lead model. Deposition of airborne lead and surface water runoff from the smelter site over the past 100 years have elevated lead levels in soil, although current operations and remediation have greatly reduced airborne emissions and controlled runoff to the surrounding area. The responsible party has agreed to remove and replace soil for all playgrounds and schools, residential properties within the zone closest to the smelter site, and all other residences with soil concentrations in excess of 1000 mg kg^{-1}. These actions will result in remediation for over 60% of houses in the community according to the recent sampling data. As of the end of 1993, soil concentrations were known for over 85% of homes in the community ($N = 772$). A risk assessment was conducted on post-soil remediation conditions to provide a basis for decisions on whether the soil remediation programme will be adequately protective of the community.

The risk assessment used version 0.6 of the EPA lead model as specified by EPA Region VIII. Although the recent version (0.99d; US EPA, 1994) of the model was unavailable at the time, EPA Region VIII provided default exposure assumptions of version 0.99d. Apart from the expo-

Table 2 *EPA lead model input values*

Exposure Parameter	Value
Outdoor air lead concentration ($\mu g\ m^{-3}$)	0.39 to 1.53
Indoor air lead concentration (% of outdoor)	30
Time spent outdoors ($h\ day^{-1}$)	1, 2, 3, 4, 4, 4*
Ventilation rates ($m^3\ day^{-1}$)	2, 3, 5, 5, 5, 7*
Lung absorption (%)	32
Dietary intake ($\mu g\ day^{-1}$)	3.19, 3.21, 4.42, 4.28, 4.14, 4.39*
Gastrointestinal absorption from diet and water (%)	50
Lead concentration in drinking water ($\mu g\ l^{-1}$)	4
Drinking water intake ($l\ day^{-1}$)	0.20, 0.50, 0.52, 0.53, 0.55, 0.58*
Soil lead concentrations ($mg\ kg^{-1}$)	Site data
Indoor dust lead levels (% of soil)	77
Ingestion weighting factor (% soil % dust)	45/55
Amount of soil and dust ingested daily ($mg\ day^{-1}$)	85, 135, 135, 135, 100, 90*
Gastrointestinal absorption from soil and dust (%)	30
Paint lead intake ($\mu g\ day^{-1}$)	0
Maternal contribution method	Infant model
Mother's blood lead level at birth of child ($\mu g\ dl^{-1}$)	3.8
Geometric standard deviation	1.35, 1.666

*Age-specific values: 0–12, 12–24, 24–36, 36–48, 48–60, 60–72 months.

sure assumptions, version 0.99d is similar to 0.6 except that changes in the biokinetic portion of the newer version generally result in slightly higher risk estimates.

Calculations of mean blood lead levels and lead risk used the batch mode feature with the assumption that a young child (age 0 to 72 months as specified by EPA Region VIII; current guidance includes ages up to 84 months; US EPA, 1994) lives in each sampled residence. The distribution of mean blood lead levels in the community was averaged over the age range. Because the model is unable to calculate the distribution of lead risk within the community, we estimated this distribution by ranking houses by soil concentration and calculating the lead risk at various concentration percentiles. In developing a weighted-average lead risk for the community, we multiplied the fraction of houses within 100 mg kg^{-1} soil concentration intervals from 0 to 900 mg kg^{-1} by the lead risk associated with the midpoint of each interval (or single point at 828 mg kg^{-1} within the 800 to 900 mg kg^{-1} interval).

Model input assumptions were primarily US EPA (1994) default values, with the exception of site-specific data for air concentrations, house dust, soil, maternal blood lead; recent FDA dietary data; and a range of potential GSDs (Table 2).

Air. The most recent air monitoring data for lead at the time of study included quarterly and annual averages for 1992 at two stations in the community immediately downwind from the smelter site. Annual average air concentrations were estimated for four areas of the community (CPP, 1993), ranging from 0.39 to 1.53 µg m^{-3}. Batch mode calculations of mean blood lead level incorporated the air concentration of the appropriate area for each house. Estimates of the lead risk conservatively used 1.53 µg m^{-3} for graphical display of the results.

House Dust. The lead concentration in indoor dust is assumed to be an average of all dust contacted by a child in the home, although sampling investigations typically focus on floor dust from rooms where a child spends the most time (*e.g.* Bornschein *et al.*, 1991). The default assumption is that the lead concentration in indoor dust is the same as that of outdoor soil or 70% of outdoor soil with additional contribution of lead deposited from air. The actual relationship between indoor and outdoor lead levels can vary considerably depending on the presence of other sources of lead in the home such as lead-based paint, hobbies, and airborne lead.

CDC *et al.* (1986) collected house dust data (from vacuum cleaner bags) at 396 houses in 1983. Vacuum bag samples may underestimate lead levels in fine surface dust, although the sieving of samples before analysis should have reduced this possibility. Comparison of lead levels measured in soil and house dust showed an average indoor dust level of 77% of outdoor soil lead, although the correlation was weak (r^2 = 0.27; Hydrometrics, 1993). Although this relationship assumes soil lead is the source of all house dust lead, the older homes which are closer to the smelter site may also have greater lead contributions to dust from paint and air emissions. Smelter emissions were also higher in 1983 than at present.

Soil. Soil data were collected by HRS (1993) as a part of the remedial investigation for the site. The soil concentration for each house was represented as the average of four quadrant samples, each a composite of five samples. Soil sampling targeted exposed areas of soil, play areas, gardens, drip lines near houses, or areas where concentrations might be higher due to deposition of sediment by water runoff. After scheduled remediation, lead concentrations for the residences are projected to range from 49.1 to 828 mg kg^{-1}, with 60% at 49.1 mg kg^{-1}, the estimated fill concentration for remediated lots. The geometric mean concentration is 94 mg kg^{-1}.

Maternal blood lead level. The maternal blood lead level at a child's birth was increased from the default value of 2.5 µg dl^{-1} (US EPA, 1994) to 3.8 µg dl^{-1}, the estimated mean blood lead of women in the community who were pregnant, nursing, or considering having children (1991 blood lead survey, see below; LCCCHD, 1991). Sixty percent of the blood lead levels were below the detection limit of 4 µg dl^{-1}.

Diet. US EPA default values for dietary lead intake are based on a preliminary analysis of the 1986 to 1988 data from the Food and Drug Administration (FDA) total diet study. Because the levels of lead in food have continued to decrease along with lead use in gasoline and other products, the FDA preliminary analysis of the 1989 to 1990 data from the total diet study were used to develop updated estimates of the dietary contribution of lead. The FDA calculated dietary lead intakes of 3.2 and 4.4 µg day^{-1} for 6 to 11 month-old and two-year-old children, respectively (Gunderson, 1993). The ratio between the revised estimates and the default values for these two age groups were used to calculate dietary intake rates for other age groups of children.

Geometric standard deviation. Depending on the use of the model, the GSD should be either an estimate of the community-wide variation in blood lead levels or the amount of variation expected for an individual child at a particular house. The former allows predictions of the proportion of children in a community that might exceed a

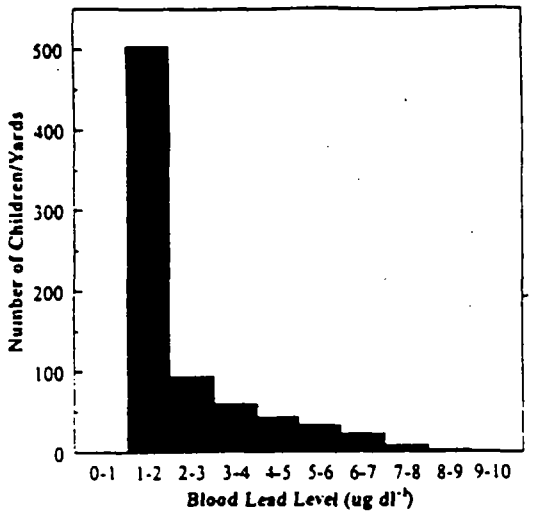

Figure 3 *Distribution of predicted mean blood lead levels in the community.*

specific blood lead level, whereas the latter permits estimation of an individual's lead risk and is appropriate for batch mode applications. A range of individual GSDs from 1.35 to 1.666 was used in evaluating the results. US EPA (1991) recommended 1.35 as an individual GSD based on an analysis of six sites using multiple regression to adjust for community-wide variation in environmental factors, as allowed by the available data. US EPA (1994) currently recommends 1.6 as the individual GSD. An adjusted GSD of 1.53 has been calculated for the community examined here based on the 1983 blood lead survey (Marcus, 1992). US EPA Region VIII specified the use of the 1983 community-wide GSD of 1.666 as the upper range of a possible individual GSD for this site.

Blood lead data. Model results were compared to the available blood lead data. Blood lead levels were most recently surveyed in 1991 by the local health department (LCCCHD, 1991; detection limit = 4 μg dl^{-1} ± 3 μg dl^{-1}). This voluntary survey attempted to recruit as many people as possible in the community. Participation rates of pre-school-age children were 56% in the zone closest to the smelter, 84% within one mile of the smelter, and 62% between 1 and 2.24 miles. To evaluate the post-remediation relationship between blood lead and yard soil (soil lead levels < 1,000 mg kg^{-1}), blood lead results of young children (age < 72 months) were regressed on soil data for houses with soil concentrations less than 1,000 mg kg^{-1} in 1991. Blood lead samples below the detection limit were assigned a value of 3 μg dl^{-1}, which was estimated to be closer to the average of these samples than half the detection limit, as is commonly used to represent non-detectable levels. Because of the limited number of samples (N = 59), this comparison is considered a screening of the potential relationship between blood lead and soil lead levels.

Results

Model results

Predicted mean blood lead levels for all 772 houses evaluated were below 10 μg dl^{-1}, with an average of 2.1 μg dl^{-1} (Figure 3). The community weighted-average risk (*i.e.* chance of exceeding 10 μg dl^{-1}) based on the batch mode calculations was low as

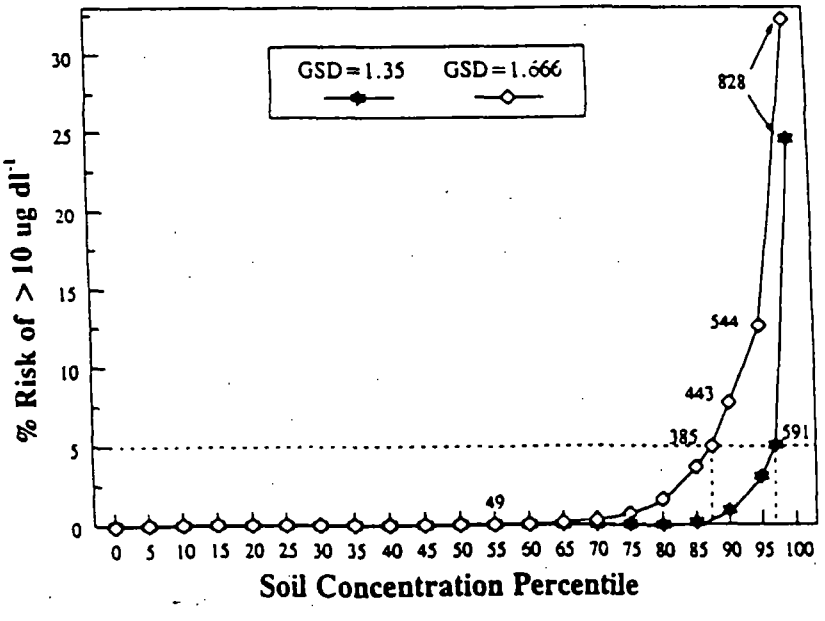

Figure 4 *Risk of exceeding a 10 μg dl^{-1} blood lead level at various soil concentration percentiles in the community (soil concentrations are displayed at specific percentiles in mg kg^{-1}).*

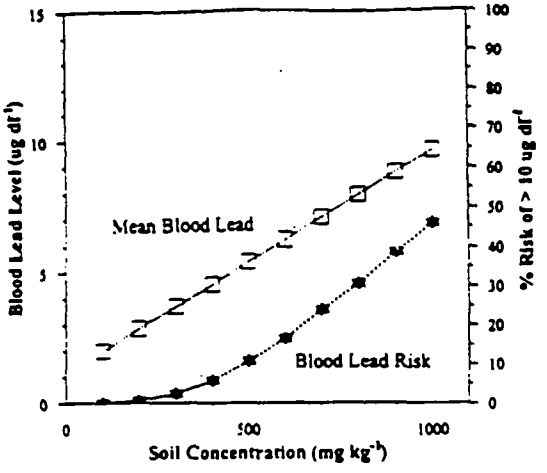

Figure 5 *EPA lead model predictions as a function of soil concentration.*

anticipated by cleanup of 60% of residences: 0.5 to 1.9% chance of exceeding 10 μg dl^{-1} for GSDs of 1.35 to 1.666, respectively (assuming midpoint air concentrations between the high and low estimates in the community).

The percentage of houses with lead risk of more than 5% was very sensitive to the value assumed for the individual GSD (Figure 4). The lower soil concentration for exceeding a 5% risk was about 400 mg kg^{-1} for the worst-case community-wide GSD of 1.666, and about 600 mg kg^{-1} for the recommended individual GSD of 1.35. If at worst-case the highest average air concentration (1.53 μg m^{-3}) existed throughout the community, GSDs of 1.35 and 1.666 result in 3% and as many as 13% of houses having a lead risk exceeding 5%. Consequently, lead risks were less than the EPA criterion (*i.e.* no more than 5% of residences with a greater than 5% lead risk) at the lower individual GSD, but not at the higher community GSD.

Comparison of model results to blood lead and soil data

The EPA lead model (version 0.6) predicts an approximate 7 μg dl^{-1} increase in mean blood lead level with every 1000 mg kg^{-1} increase in soil lead. The model's predicted risk of exceeding a 10 μg dl^{-1} blood lead level likewise increases strongly with soil lead (Figure 5). By contrast, the available blood lead and soil lead data show considerable variability and no obvious dependence of blood lead on soil lead for concentrations less than 1000 mg kg^{-1} ($r^2 = 0.0022$, $F = 0.126$, $P = 0.72$; Figure 6). Log transformation of the data had little effect on increasing the variance explained by soil lead or on improving the fit of the regression ($r^2 = 0.0029$, $F = 0.164$, $P = 0.69$).

Discussion and Conclusions

Model predictions

As with any estimation technique, the accuracy of the model would be improved with more house-

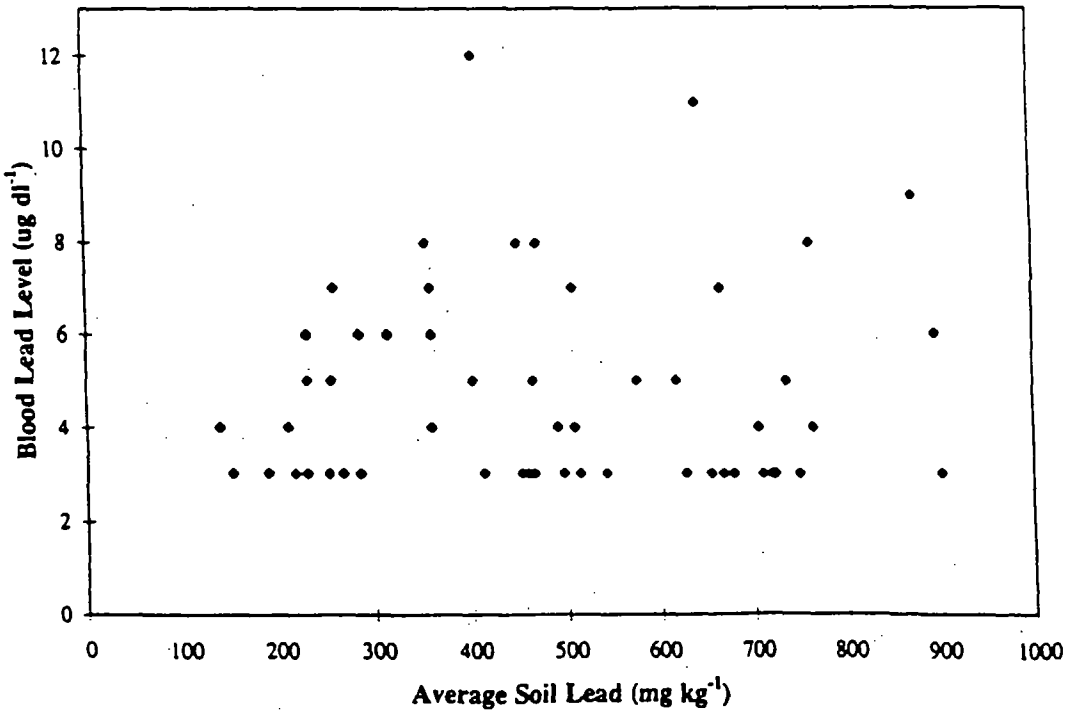

Figure 6 *Comparison of measured blood lead levels with soil concentrations below 1000 mg kg^{-1}.*

Table 3 *EPA draft guidance for lead in soil under section 403 of TSCA.*

Soil level (mg kg^{-1})	Action (children present)	Action (children rarely present)
> 5000	Permanent soil abatement	Permanent soil abatement
2000 – 5000	Interim controls Monitor conditions Public notice by local agency	Interim controls Monitor conditions Public notice by local agency
400 – 2000	Interim controls Monitor conditions Public notice by local agency	No further investigation generally required
< 400	No further investigation generally required	No further investigation generally required

specific information on indoor dust, paint, and air concentrations. In addition, any model has limitations in characterising actual environmental exposures for children. For example, this application of the model assumes exposure occurs only at a child's house and is unable to account for the lower overall lead levels in the environment due to the remediation of schools, play areas and much of the community. The batch mode calculations, however, improve on earlier versions of the model by providing perspective on the nature and extent of blood lead risk in the community rather than a single point estimate based on the soil concentration for the community or a single house.

The utility of the batch mode depends on the policy of the remedial decision makers at sites. If extensive remedial actions are triggered by a single worst-case soil concentration (*e.g.* based on a lead risk of 5% using a higher community-wide GSD), then the batch mode calculations are unnecessary. When all soil concentrations are below this trigger concentration, the batch mode results will similarly be below their target criteria. Nevertheless, the potential for elevated blood lead levels within a community, which may be reflected by the batch mode calculations, can vary greatly regardless of the soil concentration at the 5% risk level (see Smuggler Mountain Technical Advisory Committee, 1993).

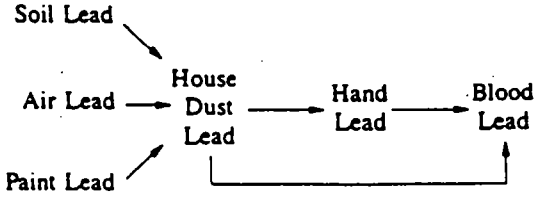

Figure 7 *Child lead exposure model based on structural equation analysis (arrows indicate significant correlations).*

Appropriate GSD

Another area for model refinement is the use of the GSD. Although the model predicts a blood lead probability distribution, the model is deterministic rather than stochastic. Because the distribution depends on single point assumptions and a specified GSD, the model tends to give biased results, especially if greater than central tendency values are used for many of the assumptions in an attempt to compensate for uncertainties. The model results are particularly sensitive to biases in the GSD.

Estimates of the community-wide GSD have increased over time with the decline in the mean blood lead levels (US EPA, 1994). This increase in community-wide variation may be the result of an overall decrease in background sources of lead for most individuals with a few still exposed at higher levels due to sporadic sources such as paint. By contrast, the individual GSD should be less affected by such changes in community-wide environmental variation.

The current default individual GSD of 1.6 is based on blood lead data of children grouped by similar soil and house dust exposures (US EPA, 1994). A GSD of 1.6 is the median of such weighted-average GSDs for 'grouped' data from Baltimore, Maryland (1.53); Butte, Colorado (1.60); and Midvale, Utah (1.69; US EPA, 1994). This method, however, is unable to account for all sources of community-wide variation (*e.g.* soil, house dust, air, water, paint, and house characteristics), and is potentially biased upward by small sample sizes within individual soil/dust groups. A recent modified method used by US EPA Region VIII (1995) attempts to maximise sample sizes within groups, thereby resulting in lower GSDs (*e.g.* approximately 1.4 for two sites in Utah).

Another method used to estimate an individual GSD statistically adjusts the community-wide GSD for the effect of environmental variation. Use of this regression method for six blood lead

data sets indicates that the current default GSD is near the upper end of the 1.3 to 1.63 range of adjusted GSDs (Marcus, 1992). These adjusted GSDs are also likely to overestimate the individual GSD because the data do not include all sources of variation among houses. For example, this smelter site example with an adjusted GSD of 1.53 (Marcus, 1992) had inadequate data for correction of variation in house dust and paint (CDC *et al.*, 1986).

A paradoxical result of the current GSD estimates is that the recommended individual GSD is similar to community-wide GSDs (see summaries by Marcus, 1992; US EPA, 1994). This result implies that variation in soil concentration throughout a community explains little of the variation in a child's blood lead level. The EPA blood lead model, however, predicts a strong dependence of mean blood lead and risk of exceeding 10 μg dl^{-1} on soil lead level.

Blood lead–soil lead relationship

Unlike the EPA lead model, this example showed no apparent correlation between blood lead levels and soil lead levels below 1000 mg kg^{-1}. Such an observation may be due to limited sample size ($N = 59$); variation in blood lead; accuracy of the soil lead average for predicting exposure; lack of adjustment for house dust, air, or paint contributions; other unaccounted for sources; or variation in individual characteristics of children. To a certain extent, house dust, air, and possibly paint may be indirectly correlated with soil due to their mutual correlation with proximity to the smelter site. The lack of adjustment for these factors would be expected to increase the apparent correlation of blood lead with soil, although relative differences in proximity to the site may be minor for houses with lead soil concentrations in the 0 to 1000 mg kg^{-1} range.

Despite the limitations of the available blood lead and environmental data, other studies with more robust data sets also depict wide variation in blood lead irrespective of soil concentration and a low amount of explained variation (Bornschein *et al.*, 1988; Bornschein *et al.*, 1991; Bornschein *et al.*, 1994; Marcus and Elias, 1994). Structural equation analyses from these studies have noted a different relationship of environmental lead and blood lead than assumed by the lead model. According to these statistical analyses, blood lead levels in children are indirectly rather than directly correlated with soil lead (Marcus and Elias, 1994). Blood lead is usually directly correlated with hand lead and house dust lead, whereas soil lead is correlated with house dust lead but not blood lead (Figure 7). The correlation coefficients in these relationships are also small. Accordingly, the EPA recognises that dust and paint are major contributors to elevated blood lead levels in children and 'any strategy to reduce overall lead risk at a site needs to consider not only soil, but these other sources and their potential exposure pathways' (Laws, 1994).

New EPA guidance for lead in soil

Perhaps because of the limitations and uncertainties in the model predictions, the latest US EPA guidance on lead in soil appears to take a more holistic approach for assessing and managing lead risks. The two separate programmes specifying this guidance allow considerably more flexibility for site managers than the previous directive for clean-up levels of 500 and possibly 1000 mg kg^{-1} (Clay, 1991). One set of guidance is for RCRA and CERCLA sites (Laws, 1994), the other is the section 403 interim hazard guidance under the Toxic Substances Control Act (Goldman, 1994) for management of lead-based paint and lead in soil and dust around residences, particularly public housing facilities. Both programmes specify a lower limit of 400 mg kg^{-1} (based on the EPA lead model) below which no further action is generally required. Above 400 mg kg^{-1}, further investigation is warranted, such that the type and extent of measures to address health risks should be proportional to the degree of risk. Both sets of guidance mention interim controls or intervention measures that change the use patterns and create barriers to reduce exposure. Such activities may include vegetative cover, fencing, removal of play equipment from areas, community education, and institutional controls. Abatement actions (*e.g.* soil removal or lead paint abatement) are reserved for higher risk situations.

The RCRA/CERCLA guidance specifies the use of the EPA lead model to assess health risks and define preliminary remediation goals, although soil levels defined using the EPA lead model do not necessarily require the excavation of soil.

The section 403 guidance differs from that for RCRA/CERCLA sites in that actions are recommended at specific soil concentrations above 400 mg kg^{-1} (Table 3). Above 5000 mg kg^{-1}, soil removal or capping is recommended. Below this level, less permanent or aggressive measures are specified for areas where children would regularly be exposed. For areas where children rarely frequent, such actions are generally not recommended until soil lead levels exceed 2000 mg kg^{-1}.

The new EPA guidance for lead in soil may allow site-specific alternatives to using one uniform remediation level and one action (*e.g.* soil removal) to manage lead exposures. For example, if the batch mode calculations and other site information indicate a relatively low risk throughout the com-

munity, a programme of education, intervention, and monitoring may be instituted with the option of removing the soil if warranted in specific cases. Given the wide variation in children's blood lead levels, irrespective of soil concentration and other sources of lead such as paint, this latter approach would more effectively manage lead risks than would widespread removal of soil.

In conclusion, areas for improvement in using the EPA lead model are for further refinement and calibration with data from a greater variety of sites, reconciliation of the individual GSD with the model assumptions on soil lead (or development of a more stochastic version of the model), and incorporation of all scientific information from the model and site blood lead and environmental data into remedial decisions. Ultimately, whether these new model developments and guidance change the management of lead risks depends on the ability of the risk managers to understand the information provided by the model, the model's limitations, and the available data on the environmental sources of lead and their relative impacts on blood lead levels.

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