THE CRITICAL ROLE OF HOUSE DUST IN UNDERSTANDING THE HAZARDS POSED BY CONTAMINATED SOILS

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The health risks posed by soil pollutants are generally thought to be due to soil ingestion and have often resulted in massive regulatory efforts to remedy such contamination. The contribution of this route to the actual human health hazard has been questioned, however, as soil removal alone seems to have little influence on the body burdens of soil contaminants in exposed individuals. Ongoing research also has repeatedly and substantially reduced the estimates of soil ingested daily. Because comparatively little time is spent outdoors by most individuals, exposure to soil brought indoors, present as house dust, is now thought to be nearly as important as the direct ingestion of soil. Exposure via house dust has not been studied specifically, but several observations suggest that it may be important. Dust is largely composed of fine particles of tracked-in soil. The smaller dust particles cling to surfaces better than soil, and contaminant concentrations are often higher in house dust. Fine particles are likely to be more bioavailable, and degradation is slower indoors. Contaminants thus may be concentrated and more readily available in the areas most frequented. In some studies, contaminant levels in dust are correlated more closely with body burdens of contaminants than other sources, suggesting that this route should be considered when assessing risks from soil. Until more research addressing exposure to dust is conducted, recommendations for assessing potential health risks from this pathway are provided.

Keywords contaminated soil, environmental risk, exposure assessment, house dust, soil ingestion

Not long ago, the annual meeting of the International Society of Risk Assessors was held in the same city as the annual meeting of the International Society of Risk Managers. Because the meetings were

held in different hotels, each group remained blissfully unaware of the other's presence in the city. After many learned presentations detailing their respective successes at protecting society from the risks confronting it, both groups elected to spend the final day of their meetings in relaxation. The risk assessors settled on a day at the beach whereas the risk managers decided on an ascent in a hot air balloon.

Shortly after take-off, the risk managers found themselves engulfed by fog and buffeted this way and that by treacherous winds until they were hopelessly lost. After what seemed hours aloft, they chanced on a break in the clouds. Below them, they spied the risk assessors at play on the beach. Attracting the attention of the risk assessors, they shouted down, "Where are we?" The risk assessors huddled together, considering their answer, and after a short time, they replied, "You are in a hot air balloon about 200 meters in the air." With that the fog closed in around the balloon and the wind drove it out to sea. As the risk managers were being swept to their doom, one frustrated risk manager asked another, "Why don't risk assessors ever tell us anything useful?" At the same instant on the ground, a risk assessor remarked to his colleagues, "Isn't it curious that risk managers never ask any really important questions?"

While this is an apocryphal tale, it has bearing on the issue of contaminated soils, house dust, and the risks both may pose to individuals exposed to them. In the early 1980s, at places such as Love Canal and Times Beach, questions were raised regarding the significance of contaminated soil regarding human health. Because the literature contained reports describing the hand-to-mouth activity by young children and ingestion of nonfood items and anecdotal reports of pica and geophagia in both children and adults, risk assessors concluded that ingestion of soil-bound contaminants might be an important pathway of human exposure to environmental contaminants. Since then, numerous papers have been published that discuss soil ingestion or dermal contact as it pertains to environmental exposure and risk assessment. Few have discussed, however, the significance of house dust and its relative contribution to the total human uptake of environmental contaminants.

EVOLUTION OF SOIL INGESTION ESTIMATES

The first estimates of the soil ingestion rates by children were little more than informed, albeit conservative, guesses (Kimbrough et al., 1984). The results of using these values in risk assessments made it clear that this exposure pathway would dominate most exposure assessments and risk management decisions when soil contaminants were present. For example, as shown by Paustenbach (1987), direct ingestion of contaminated soil represented 75% to 90% of the predicted

risk associated with most hazardous waste sites. In the absence of actual measures of soil ingestion, these estimates drove remedial actions involving contaminated soil throughout the 1980s and 1990s (Paustenbach, 1995).

Questions were raised about the accuracy of these soil ingestion estimates almost immediately, and applied research was undertaken to develop and refine quantitative estimates of soil ingestion rates, especially in children (Paustenbach, 1987). These studies relied on a mass balance approach in which multiple, poorly absorbed tracer elements were used as surrogates of soil ingestion (Binder et al., 1986; Clausing et al., 1987; Van Wijnen et al., 1989; Calabrese et al., 1989; Davis et al., 1990). The difference in the levels of tracers in materials ingested daily by the subjects (e.g., food, medicines, vitamins) compared with the levels detected in stool and urine samples collected daily was assumed to be due to ingested soil. By analyzing the tracer content of the associated soil and dust, an estimate of the daily soil ingestion was developed for each subject for each tracer used. Such studies (Calabrese et al., 1989) indicated that for the most reliable tracers soil ingestion rates for children were between 9 and 40 mg/day. Continued evaluation and improvement of these techniques and the results convincingly established the accuracy of these techniques and extended their application (Stanek et al., 1988; Calabrese et al., 1990; Stanek and Calabrese, 1991; Calabrese and Stanek, 1991; Calabrese et al., 1991; Calabrese and Stanek, 1992a,b; Stanek and Calabrese, 1992; Calabrese et al., 1993a,b; Stanek and Calabrese, 1995a,b; Calabrese and Stanek, 1995a,b). Appling (personal communication, 1996), using lead as a tracer, estimated a daily soil intake of approximately 10 mg/day, similar to the 4-mg/day, estimate of de Silva (1994) for the same element. The regulatory response in the United States to these studies has been to lower the default values for childhood soil ingestion rates from a high of 10,000 mg/day in 1982 to a reasonable maximum exposure of 200 mg/day for children (United States Environmental Protection Agency [U.S. EPA], 1995a).

Despite a reduction of nearly three orders of magnitude in what is believed to be the accurate childhood soil ingestion rate (Table 1), the soil pathway still dominates many risk assessments, mostly due to the continued use of the EPA's relatively high default soil ingestion rate (200 mg/day) and the unrealistic exposure scenarios often required by Federal and state regulatory agencies. Such exposure scenarios typically assess a child's risk due to ingestion of their entire daily ration of soil from the area of highest contamination without regard to the amount of time actually spent outdoors on site, the horizontal and vertical distribution of contaminants in the soil column, issues of contaminant half-life, or bioavailability (Paustenbach et al., 1992a,b;

Table 1. Source of historical estimates of the soil ingestion rates for children typically used in health risk assessment (1984 to 1996)

Study	Soil Ingested (mg/day)
Kimbrough et al., 1984[a]	10,000
Hawley, 1985	200
Paustenbach, 1986[b]	100
Binder et al., 1986[c]	100–600
Clausing et al., 1987[c]	100–1,000
Van Wijnen et al., 1989[c]	100–150
Calabrese et al., 1989[c]	10–40
Davis et al., 1990[c]	25–80
de Silva, 1994	4
Appling (personal communication, 1996)	10

[a]Used to assess Times Beach, MO.
[b]Used in an alternative assessment of Times Beach, MO.
[c]Quantitative tracer studies.

Paustenbach et al., 1996). The result has been the expenditure of tens of billions of dollars in the United States in the past 20 years to implement extensive soil remediation programs.

POSSIBLE IMPORTANCE OF HOUSE DUST IN LEAD EXPOSURE

Unfortunately, the money spent to remediate contaminated sites often has not produced measurable declines in the body burdens of the contaminant in exposed residents or improvement in the public health. One reason may be that soil ingestion is not the predominant way that most individuals are exposed to xenobiotics in their environment. If, for example, house dust is a major source of exposure, it is not surprising that removal of contaminated soil has produced little or no change in body burdens of the contaminant of concern (Kimbrough, 1995; Kimbrough et al., 1995; U.S. EPA, 1995b; Weitzman et al., 1993). There is growing evidence that in-home exposure is as important as exposure to exterior soils (Paustenbach, 1995). This evidence is largely derived from experience with inorganic contaminants such as lead (Sayre et al., 1974; Vostal et al., 1974; Lepow et al., 1975; Charney et al., 1980; Duggan, 1980; Roels et al., 1980; Bornschien et al., 1985), cadmium (Buchet et al., 1980), and arsenic (Polissar et al., 1990; Buchet et al., 1980), although similar findings have been reported for some pesticides (Davies et al., 1975) and polychlorinated biphenyls (PCBs) (McClanahan, personal communication, 1996). A model discussed by Clark et al. (1991) identified dust on children's hands as the primary source of lead exposure. Exterior soil and house dust both contribute to

hand dust in this model, but the relative contribution of each compartment is still unclear.

These observations, together with experimental evidence suggesting that 50% or more of the soil ingested by children on a daily basis is composed of house dust (Stanek and Calabrese, 1992), raise concern that dust may be a more important source of exposure to many, and perhaps most, long-lived (persistent) pollutants than exterior surface soil. Surface soil, therefore, may primarily be important as it influences the pollutant content of house dust, and soil removal or treatment as a remedial alternative may be ineffective at reducing human exposure if the house dust remains unaddressed. Consistent with the moral of our cautionary opening tale, the risk assessors may have provided the correct answer to the wrong question. We may have focused at many sites on the wrong medium in seeking to reduce the health risks posed by soil-bound contaminants.

In the absence of specific studies designed to test hypotheses regarding the role of house dust in exposure to soil-bound contaminants, the significance of house dust as an exposure vehicle remains somewhat conjectural. However, information from the literature is suggestive regarding the importance of house dust. The literature about sites contaminated with lead is the most persuasive. For example, during a study on lead exposure resulting from the operations of a former secondary smelter in Illinois that had contaminated the surrounding residential neighborhoods, Kimbrough et al. (1994) reported the results of educating the parents of children whose blood lead levels exceeded the Center for Disease Control (CDC)'s acceptable level of 10 μg/dl about sources of lead in the home and simple techniques to reduce exposure (e.g., hand washing, dust control). An average drop of blood lead levels of 50% was achieved immediately in these children, and the reduction persisted for at least 1 year. Because this reduction was achieved in the presence of elevated soil lead levels and lead paint in many of the homes, the authors concluded that house dust is a critical factor in predicting blood lead levels and that control of this medium had a significant effect on the body burden. Similarly, Clark and coworkers reported a decline in blood lead levels after dust control measures were implemented in Cincinnati homes (Clark et al., 1988; Clark et al., 1991; U.S. EPA, 1995b), although the decrease was not as pronounced as that reported by Kimbrough et al. (1994).

The Cincinnati portion of the U.S. EPA's Urban Soil Lead Abatement Demonstration Project (or Three City Study; U.S. EPA, 1995b) is the only study specifically designed to evaluate the effect of lead abatement of exterior soil and interior dust as an intervention technique for lead exposure. This study evaluated three different intervention strategies in neighborhood areas: exterior soil and dust removal (area A); interior

dust abatement (area B); and no action (area C). Soil lead abatement had little effect on blood lead levels, but a positive influence on blood lead reduction was noted in the neighborhoods in which interior dust abatement was performed. The difference, however, was not statistically significant, perhaps because of issues of sample size or the relatively low lead concentrations in dust, soil, or blood. Ten months post-abatement, the decline in blood lead levels was 2% in area A, 18% in area B, and 6% in area C. Interestingly, one of the main conclusions from each of the cities in the study (Boston, Baltimore, and Cincinnati) was that soil abatement was ineffective at reducing the lead burden of resident children. This suggests that the critical determinant of blood lead levels in children (and arguably adults) lies somewhere other than via direct soil ingestion.

Subsequent analysis of the Illinois data indicated that soil lead contributed only approximately 3% to the variance in the blood lead levels, whereas interior conditions explained four or more times the variance in body burdens (Kimbrough et al., 1995). Similar to findings from other studies (Angle and McIntire, 1979), inclusion of all environmental parameters in the analysis explained only approximately 40% of the variance again, indicating that behavioral and other factors may be as important or more important determinants of body burden than environmental parameters. Milar and Mushak (1982) and Charney et al. (1983) also have reported significant reductions in the lead burden of children after the reduction of interior dusts in their homes (even without addressing other potential sources).

ROLE OF HOUSE DUST IN EXPOSURE TO OTHER CHEMICALS

Lest one believe that, because of its past indoor use, lead is a unique case, other studies have suggested similar relationships between exposure to house dust and the body burdens of other persistent contaminants. For example, Buchet et al. (1985) reported a relationship between hand dust and urinary levels of arsenic and cadmium in a population near a Belgian smelter. Likewise, in the United States, Polissar et al. (1990) found a similar relationship between arsenic in hand dust and urinary arsenic levels. Davies et al. (1985) related the high levels of organochlorine pesticides in serum of a West Indies population, with the high levels of the pesticides found in house dust. Warnick (1972) also reported a positive, although not statistically significant, association between pesticides in house dust and serum in Utah residents, and Burns and Miller (1975) reported an association with hexachlorobenzene in house dust and serum in a Louisiana population. McClanahan (personal communication, 1996) reported that serum PCB levels in a Mississippi population were better correlated with interior surface

wipe samples than with soil concentrations or other environmental media. Although Stern et al. (1992) reported an association between chromium levels in house dust and urine, subsequent reduction in chromium levels in the house dust did not produce a reduction in urinary chromium levels (Freeman et al., 1995b). This may have been due to the marginal increase in the reported urinary chromium levels compared with background concentrations in urine.

Although such evidence is suggestive of a link between exposure to dust and body burdens, few studies designed to test this hypothesis specifically have been performed to date. There are, however, numerous studies that have reported a lack of a relationship between exterior soil levels and body burdens. It is our view that models that rely primarily on soil contact and ingestion as the sole or primary exposure pathway for soil-bound contaminants may be erroneous. In addition to the results of the Three City Study cited previously for lead (Weitzman et al., 1993; U.S. EPA, 1995b), the CDC failed to find elevated PCB body burdens in a dozen populations exposed to soil PCB levels as high as 13% (Kimbrough, 1995). A similar lack of elevation in average PCB serum concentrations has been reported for gardeners working with PCB-contaminated sewage sludge (Baker et al., 1980) or for other exposures involving contaminated soils (Miller et al., 1991; Yaffe and Reeder, 1989). Although the picture is far from complete, the data suggest that remediation of exterior surface soil may not eliminate exposure and that, in many cases, cleaning homes of dust may produce excellent results at a fraction of the cost of remediating the site.

COMPOSITION OF HOUSE DUST

House dust is a heterogenous mixture. A number of sources contribute to this mix, including tracked-in or resuspended soil particles, clothing, atmospheric deposition of particulates, hair, fibers (artificial and natural), molds, pollen, allergens, bacteria, viruses, arthropods, ash, soot, animal fur and dander, smoke, skin particles, cooking and heating residues, and building components among others (Lioy et al., 1993). Various researchers have examined the contribution of exterior soil to interior dusts (Stanek and Calabrese, 1992; Fergusson and Kim, 1991; Camann and Harding, 1989; Thornton et al., 1985; Hawley, 1985). These studies suggest that approximately 50% of house dust originates from exterior soil (Table 2), although a good deal of additional data are needed to gain confidence in this value.

The importance of exterior soil to the creation of interior dust is illusted by the work of Roberts et al. (1990), in which the amount of lead found in carpet was reduced by 90% or more by the simple expedient of removing shoes or using walk-off mats. Similarly, Thatcher and Layton (1995) demonstrated that the dust mass in a home declines as

Table 2. Estimates of the relative contribution of exterior soil to house dust

Investigator	% Dust from Soil
Hawley (1985)	>80
Thornton et al. (1985)	20
Camann and Harding (1989)	50
Fergusson and Kim (1991)	30–50
Calabrese and Stanek (1992)	20–78

one moves from the entry way through the home in relation to the traffic patterns. These researchers also concluded that the walls of a house present little barrier to the penetration of soil particles resuspended in outside air. It is unclear whether this was due to open doors, open windows, or simply miscellaneous leaks. Nonetheless, the exterior soil appears to serve in many cases as a reservoir from which contamination may be carried into the house and to provide on-going exposure to residents through dust.

CHARACTERISTICS OF HOUSE DUST THAT INFLUENCE EXPOSURE POTENTIAL

Several characteristics suggest that contaminated house dust may present a greater hazard to residents than contaminated soil. The key issues appears to be (1) particle size distribution, (2) the concentration of contaminants in dust relative to exterior soils, (3) fine particle enrichment, and (4) bioavailability of the fine versus the larger particles.

Dust Particle Size Distribution

House dust is composed of smaller particles than soil. For example, Bornschien et al. (1988) reported that 82% of exterior soil in their study area was larger than 150 μm in diameter, whereas Roberts et al. (1991) found that only 50% of house dust exceeded this diameter. Most important, Duggan et al. (1985) reported that greater than 99% of dust on the hands of children was less than 150 μm in diameter, and, in fact, 98% was less than 10 μm in diameter. Similar findings of the size distribution of hand dust were reported by Bornschien et al. (1988), Duggan and Inskip (1985), and Que Hee et al. (1985). In addition to being more mobile, as suggested by the influence of size on deposition rates (Thatcher and Layton, 1995), the evidence suggests that fine particles adhere to skin more effectively thus increasing exposure potential (Kissel et al., 1996; Finley et al., 1994).

Dust/Soil Contaminant Ratios

In general, the concentration of contaminants appears to be greater in house dust relative to exterior soil. For example, the dust/soil ratio for lead ranges from 0.3 to 9.2 in studies conducted both in Europe and North America (ATSDR, 1995; U.S. EPA, 1995b, Kimbrough et al., 1995; MDH, 1995; Cambra and Alonso, 1995; Bornschien et al., 1991a,b; Howells and Thornton, 1991; CDH, 1990; Thornton et al., 1990; Davies et al., 1990; Moffat, 1989; Bornschien et al., 1988; Schilling and Bain, 1988; Davies and Thornton, 1987; Harper et al., 1987; Thornton, 1986; Bornschien et al., 1986; Panhandle District, 1986; Duggan and Inskip, 1985; Angle et al., 1984; Culbard and Johnson, 1984; Culbard et al., 1983; Stark et al., 1982; Diemel et al., 1981; Angle and McIntire, 1979; Yankel et al., 1977; Lepow et al., 1975; Angle et al., 1974; Roberts et al., 1974). The lead levels in house dust are typically greater than those in exterior soils in most studies in which samples from both compartments were collected and analyzed (Table 3). Mining or smelting areas were more likely to have an elevated concentration of contaminants in soil compared with the concentration in dusts. However, in such cases, the concentrations of lead found in house dust were usually much higher than the levels found in homes from non–source dominated areas.

A similar relationship between dust and soil concentrations have been reported for other contaminants including cadmium (MDH, 1995; ATSDR, 1995; Bornschien et al., 1991a; CDH, 1990; Harper et al., 1987; Culbard and Johnson, 1984; Culbard et al., 1983; Moorcroft et al., 1982; Thornton et al., 1980), copper (Harper et al., 1987; Culbard and Johnson, 1984), zinc (Harper et al., 1987; Culbard and Johnson, 1984; Moorcroft et al., 1982; Thornton et al., 1980), pesticides (Fortmann et al., 1991; Budd et al., 1990; Yeh et al., 1977; Davies et al., 1975), and polynuclear aromatic hydrocarbons (Roberts et al., 1993). Arsenic, on the other hand, typically displays a dust/soil ratio of less than 1.0, although a few studies have reported higher arsenic levels in house dust than soil (Freeman et al., 1995a; Bornschien et al., 1991a,b; CDH, 1990; Culbard and Johnson, 1984). Finally, Calabrese et al. (1989) reported the following dust/soil ratios for various crustal elements in Massachusetts: aluminum (0.7), titanium (1.4), barium (1.4), manganese (0.7), vanadium (0.8), yttrium (0.7), and zirconium (1.0). From the available data, it is clear that the concentration of contaminants in house dust often may be greater than exterior soil levels by a factor of 1.5 to 6.0 (Thornton et al., 1985) and occasionally can exceed the concentrations found in exterior soils by factors of up to 10. Such differences, in many cases, could significantly influence the outcome of exposure assessments when all relevant factors are taken into account.

Table 3. Survey of published values that present the ratio of the concentrations of lead in house dust compared to the concentration in exterior soil

Location	Dust (ppm)	Soil (ppm)	Ratio	Source
UK	507	230	2.2	Harper et al., 1987
UK	561	289	1.9	Thornton et al., 1990
UK	1,263	486	2.6	Culbard et al., 1983
UK (London)	1,010	430	2.3	Thornton et al., 1990
UK (Derbyshire)	1,870	4,390	0.4	Thornton et al., 1990
UK (Shipham)	1,185	3,829	0.3	Thornton et al., 1990
Netherlands	957	240	3.9	Diemel et al., 1981
Canada	713	99	7.2	Roberts et al., 1974
Canada	1,550	1,715	0.9	Roberts et al., 1974
Spain	595	136	4.4	Cambra and Alonso, 1995
USA (CT)	11,000	1,200	9.2	Lepow et al., 1975
USA (IL)	1,283	450	2.9	Kimbrough et al., 1995
USA (MA)	1,094	707	1.5	U.S. EPA, 1995b
USA (MD)	1,334	231	5.8	U.S. EPA, 1995b
USA (MO)	608	599	1.0	MDH, 1995

Fine Particle Enrichment

The data are fairly convincing that house dust is composed of a higher percentage of fine particles than is exterior soil. In addition to influences on mobility and adherence, this also may be important in terms of the absolute concentration of contaminant found in the house dust. For example, various studies (Duggan et al., 1995; Sturges and Harrison, 1985; Que Hee et al., 1985; Rundle, 1984; Biggins and Harrison, 1980) have reported that the lead levels in dust from different sources (e.g., streets, playgrounds, homes) increased as particle diameter decreased from 500 to greater than 63 μm, although the inverse relationship between lead concentration and particle size was not seen in data collected in New Jersey homes (Wang et al., 1996). Other researchers have reported similar observations for other metals, including lead, arsenic, manganese, copper, chromium, and cadmium (Calabrese, personal communication, 1996; Mullins and Norman, 1994; Van Borm et al., 1988; Spittler and Feder, 1979) and pesticides (Calabrese, personal communication) in dust and soil. Although additional research is needed, it appears prudent to assume that the smaller particles in house dust may contain a higher concentration of contaminants than the coarser exterior soil particles (per gram of particles).

Bioavailability of Fine Particles

Nearly all available data indicate that fine particles of house dust are more bioavailable than soil particles, although the increase depends on

many factors. For example, Healy et al. (1982) showed that as the particle size of lead sulfide increases from 30 to 100 μm, the length of time required to dissolve the particles in gastric fluid doubles. Similarly, Barltrop and Meeks (1979) reported that blood lead levels in rats declined as the lead particle size increased, indicating that large particles are less bioavailable than small ones. This phenomenon has been reported for other chemicals on soil (Paustenbach et al., 1992a, 1997). This observation (alone) suggests that house dust, because of size alone, often may be a more important contributor to absorbed dose than exterior soils.

There are characteristics of dust that make this exposure pathway of interest in conducting risk assessments. First, 50% (or more) of house dust originates from exterior soil, so the presence of contaminated soil will result in contaminated house dust and an additional, generally unassessed exposure pathway. The dust contains a higher proportion of small particles and, as a consequence, is more mobile, more easily ingested or respired, and possesses better skin adherence properties than exterior soils. Hence, dust particles come in contact with humans more easily (and often) than soil particles. The contaminant levels in house dust are often higher than those in the exterior soil, and these concentrations may be higher in the finest particles, so the assumption that contaminant levels in house dust are similar in concentration or behavior to those in soil usually will underestimate exposure, depending on the quality of housekeeping. As the contaminants on fine particles are more easily absorbed, exposure to contaminated house dust may result in an increased absorbed dose relative to exposure to contaminated soils. One might assume, in light of this, that soil removal or steps to prevent direct contact with soil (e.g., vegetative cover or physical barriers) should eliminate the dust problem altogether, particularly if the source of the contamination is the soil alone. There are reasons, however, why this might not be so.

INDOOR ENVIRONMENTAL FACTORS

Pollutant behavior is different in the indoor environment compared with the outdoor and this, in turn, influences the exposure potential to contaminants in house dust and soil. For example, contaminants in exterior soils are subject to both biotic (i.e., microbial) and abiotic (i.e., photolysis, hydrolysis) degradation, aging, or dispersive influences (e.g., wind, rain) that contribute to reduction in contaminant concentrations and loss of availability. It has been demonstrated that bioavailability varies with the chemical form of inorganics (Barltrop and Meeks, 1975; Barltrop and Meeks, 1979) and that the chemical form of soil contaminants changes over time, usually in terms of decreasing availability (Davis et al., 1993; Davis et al., 1992). Numerous

researchers have demonstrated that metals in soil are less bioavailable than the metal salts (Davis et al., 1993; Ruby et al., 1992; Davis et al., 1992; Freeman et al., 1992; Chaney et al., 1989). For example, Chaney et al. (1984) reported that lead acetate freshly added to soil was less bioavailable than lead acetate alone, and the bioavailability further decreased with time. The addition of organic and inorganic materials to soil has been suggested as a soil treatment to reduce the bioavailability of lead to plants and grazing animals, and there is some evidence that it is effective (Zimdahl and Foster, 1976; Edwards and Clay, 1977). In a recent review, Alexander (1995) found that similar processes influence the availability of organics. Even though the absolute concentrations may remain relatively constant, organic chemicals become increasingly difficult to extract chemically from soil over time (often called *weathering*). Their availability to soil micro-organisms also decreases as does their toxicity to various species.

Because the indoor environment is characterized by limited sunlight, relatively constant temperature and low humidity, no wind or rain dispersion, a comparatively low microbial population, and a lack of reactive surfaces, the same degradative or aging processes that are effective at removal or reduction of contaminants outside do not operate or they operate at a much reduced efficiency indoors. This can be demonstrated through the observation that pesticide residues from simultaneous indoor and outdoor application can be found indoors long after they have vanished from the outside (Lewis et al., 1991; Long, unpublished observation, 1995).

Contaminants indoors also are protected from degradation by the existence of numerous "traps" within the home, particularly carpets, drapes, and upholstery (Roberts et al., 1995). Davies et al. (1985) reported that the lead dust loading in carpet was the single best predictor of children's blood lead levels, and rug lead concentration and loading are typically correlated with lead levels in foundation soil, yard soil, and indoor paint (Fortmann et al., 1991; Roberts et al., 1990). In results reported by Lewis et al. (1991), 20 of 31 target pesticides, including some out of use for more than 20 years, were detected in carpet dust, often in the part per million level. The older the home and rug, the more pesticides were found. Normal cleaning does not markedly or quickly reduced these levels (Hilts et al., 1995; Wang et al., 1995). Roberts et al. (1993) report that a year or more of normal cleaning is necessary to remove the dust reservoir that accumulates in rugs, although Ewers et al. (1994) found that cleaning was generally ineffective in reducing the lead levels in chronically contaminated carpet and recommended removal as a means to reduce exposure. The importance of carpet dust as a source of exposure is further supported by the work of Milar and Mushak (1982) who were able to effect marked reductions in blood lead levels of children through rug cleaning.

The exposure potential from these traps is increased by normal household activities. Kamens et al. (1991) reported significant increases in the level of suspended particles greater than 2.5 μm as the result of indoor activity, and Thatcher and Layton (1995) found that walking across a room increases the mass of suspended particles by 100% or more. The size of the most readily affected particles is between 5 and 25 μm, which composed 82% of the indoor particle mass in their study. Most cleaning activities ironically increase the likelihood of exposure to dust and any associated contaminants. Most vacuum cleaners do not trap small (<20 μm) particles and will simply re-entrain them into the air (Ewers et al., 1994; Kamens et al., 1991; Lefcoe and Inculet, 1975) from which they will then settle to exposed contact surfaces due to their high rate of deposition (Thatcher and Layton, 1995; Ewers et al., 1994). Gulson et al. (1995) reported a fourfold increase in surface dust loading after the first cleaning, as embedded dust is pulled from within the carpet and redeposited on various surfaces. Similar results were reported by Ewers et al. (1994). As studies have linked the rate of touching objects and surfaces as significantly related to elevation of blood lead levels among children (Roberts et al., 1993; Davies et al., 1990), such contaminant cycling may produce a significant contribution to the total exposure among residents.

The indoor environment also may contribute to increased exposure because the low humidity that exists in most indoor environments prevents particle coagulation and condensation, making it easier to resuspend particles and increase contact (Thatcher and Layton, 1995) than it would be outdoors. At the same time, low humidity promotes increased adherence of particles to the skin or clothing through electrostatic attraction (Schneider et al., 1994; Knutson, 1992; Que Hee et al., 1985) and an increased potential for absorption of chemicals through the cracked, dry skin produced by low humidity environments (Shu et al., 1988).

There is thus ample evidence to suggest that house dust can be more important than exposure to contaminated exterior soils. First, most people spend most of their time indoors. In the United States, 90% or more of time is spent indoors and little of the time outdoors is spent in direct (or indirect) contact with soil (U.S. EPA, 1995a). Second, the potential exposure to interior dust is much greater than for exterior soils. This is especially true for children during the ages of highest likely dust or soil ingestion (1–4 years of age). For instance, the blood lead levels of most children typically peak between ages 2 and 3 years (Duggan, 1983) and return to background by age 5. Unsupervised outdoor play is also generally the lowest during this age period, and mouthing of nonfood items is actively discouraged by caregivers. Third, house dust consists of finer particles than soil and these fine particles are more mobile, adhere better to the skin or clothing, and are more

easily ingested or respired. Fourth, contaminant levels may be higher in house dust than in the associated soils and may be higher in the finer, more available fraction of dust. Fifth, the indoor environment also protects residues from the degradation, aging, or dispersion that serves to reduce the levels or availability of contaminants outdoors. Finally, the normal activity that occurs in residues causes constant resuspension and settling of contaminants from dust traps, resulting in a higher frequency of contact with contaminated surfaces and a longer duration of exposure to dust-borne contaminants as a result. The protection and cycling afforded by dwellings probably explain the higher levels and persistence of contaminants found in house dust compared with those in exterior soil. This explains why soil remediation alone may be insufficient to eliminate exposure to contaminants.

ASSESSING EXPOSURE TO HOUSE DUST

Having demonstrated that house dust is a potentially important source of exposure, the problem becomes how best to assess and quantify exposure. Exposure to contaminants contained in house dust can occur through several paths, including inhalation or swallowing and absorption of suspended dusts; dermal contact with dust and dusty surfaces and subsequent absorption across the skin; ingestion of dusts as the result of hand-to-mouth activity; or ingestion as the result of dust transferred from hands or surfaces to food, drink, or other items placed in the mouth. Unfortunately, to the best of our knowledge, no studies have been conducted to date or are under way to determine specifically the extent of contaminant uptake via any of these pathways. The opportunities for identifying an appropriate population for studies are few and decreasing. Further, no validated methods for assessing contaminant uptake from house dust currently exist nor are there any accepted exposure default parameters.

Among the issues that must be resolved before any substantial progress can be made in understanding the significance of this exposure pathway is the answer to the question, "What constitutes an appropriate dust sample?" Dust sampling generally has relied on two measures to assess contamination and exposure potential: contaminant loading and contaminant concentration. The contaminant concentration, typically expressed as milligrams of chemical per gram of dust, is the more useful dosimetric for assessing whether contamination actually exists. The contaminant loading, typically expressed as milligrams of chemical per square centimeter of surface area, is a function of the contaminant concentration (mg chemical/g dust) and the amount of dust on a contact surface (g dust/cm² surface), and this is probably the most appropriate measure of potential exposure (Lioy et al., 1993). Any dust sampling program, however, should evaluate both.

Dust sampling methodologies have been in use since the 1940s but are crude, unstandardized, and unvalidated. Samples have been collected using a variety of techniques, including wiping surfaces with treated paper, paper towels, filter paper, moist cloth, or diaper wipes; collecting dust using tape, microscope slides, or Petri dishes; vacuuming surfaces; or simply emptying the houshold vacuum cleaner and analyzing the contents (Watt et al., 1983). There is no agreement on where or how many samples should be collected. A high degree of sampling variability exists between individuals collecting samples, even those using the same technique. Protocols for intramethod comparisons are poor or lacking altogether. Little information exists on the collection efficiency of the various methods and the influence of particle size or other physical characteristics of house dust, and appropriate quality assurance steps are lacking (Caplan, 1993; Lanphear et al., 1995).

Opportunities for research into these questions abound and some progress has been made. For example, Lioy et al. (1993) have developed a dust sampler for smooth surfaces that addresses and reduces many of the sources of variability that affect other methods including area sampled, number of wipes, and pressure applied while sampling. It has undergone limited laboratory and field trials but it appears promising (Freeman et al., 1996). Additional validation and protocol development for dust sampling is required. Roberts et al. (1991) have developed methodologies and specialized vacuums equipped with cyclones to collect dust from carpet. It is unclear, however, how much of the dust collected in this manner actually would have been available for contact and how much was embedded in the carpet away from exposure. Side-by-side comparisons of the wipe and cyclone collection methods have been conducted (Farfel et al., 1994a,b). Additional research is required to address a number of questions before such techniques gain wide acceptance for use in exposure assessment (Millson et al., 1994).

In the interim, a few suggestions are offered for how to estimate exposure through house dust: (1) assume that house dust contains more (2–5 times) of a contaminant than exterior soil in the absence of site-specific data; (2) assume that dust contributes a substantial portion of the daily soil ingestion rate (50% based on the contribution of soil to dust or perhaps 90% based on the relative proportion of time spent indoors and out); (3) assume a skin adherence rate of 0.5 to 1.0 mg/cm^2 of skin or higher based on the fine particle size and electrostatic attraction (Kissel et al., 1996; Finley et al., 1994); and (4) assume an airborne indoor dust level of 0.05 mg/m^3 in the absence of site-specific information. The dosemetrics of wipe sampling are important in the exposure assessment process (Adgate et al., 1995).

To understand and assess the significance of exposure to house dust, a research agenda must focus on a validated house dust sampling methodology that provides both loading and concentration measures or

a method for conservatively estimating house dust contaminant levels on the basis of exterior soil concentrations. Additionally, specific quantitative estimates are needed for dust ingestion rates by age group (Stanek and Calabrese, 1992), dust adherence factors for skin (Finley et al., 1994), dermal and oral bioavailability of contaminants from house dust (Paustenbach et al., 1996; Shu et al., 1988), contaminant and particle distribution, respirable fractions, and the influence of activity on resuspension and exposure potential. Much work similar to that recently published by Kissel et al. (1996) is needed to understand more precisely the contribution of house dust to the total uptake of a chemical.

CONCLUSION

Although there is much suggestive evidence, little is currently known about the quantitative significance of house dust when assessing the relative uptake of environmental contaminants. Research to date has focused almost exclusively on the role of soil in contributing to exposure. Although definitive studies have yet to be done, the contaminants in and the physical characteristics of house dust leave little doubt that this environmental compartment probably represents a substantial source of exposure to the public. In some if not most cases, house dust may be a more important source of exposure than the contaminated soil from which it is in large part derived. Thus, it should now be recognized that models in which direct soil contact is estimated to be responsible for most of the uptake of contaminants are likely to be in error. For instance, the U.S. EPA's (1994) Integrated Exposure Uptake Biokinetic Model for childhood lead exposure attributes 100% of blood lead levels to environmental sources, mostly soil. This is despite the fact that the contribution of all environmental compartments to lead body burdens rarely accounts for more than 40% of the variance in the blood lead (Kimbrough et al., 1995). Other routes and behavior, therefore, must play a more significant role in exposure than can be accounted for in such models. Soil remediation by itself, therefore, may not protect the public to the extent once thought to be true.

Research needs for quantifying house dust exposure are significant but could be resolved with 5 to 10 years of dedicated research for approximately 3 to 6 million dollars (the same amount spent to understand soil ingestion). As more than 50 billion dollars have been spent to date on soil cleanup in the United States alone, the investment in better understanding the contribution of house dust to environmental exposure seems appropriate.

REFERENCES

Adgate, J. L., Weisel, C., Wang, Y., Rhoads, G. G., and Lioy, P. J. 1995. Lead in house dust: Relationships between exposure metrics. *Environ. Res.* 70:134–147.

Alexander, M. 1995. How toxic are chemicals in soil? *Environ. Sci. Technol.* 29:2713–2717.

Angle, C. A., and McIntire, M. S. 1979. Environmental lead and children: The Omaha Study. *J. Toxicol. Environ. Health* 5:855–870.

Angle, C. R., McIntire, M. S., and Colucci, A. V. 1974. Lead in air, dustfall, soil, housedust, milk and water: correlation with blood lead of urban and suburban school children. In *Proc. 8th Ann. Conf. Trace Sub. Environ. Health*, ed. D. Hemphill, 23–29. Columbia, MO: University of Missouri.

Angle, C. A., Marcus, A., Cheng, I., and McIntire, M. S. 1984. Omaha childhood blood lead and environmental lead: a linear total exposure model. *Environ. Res.* 35:160–170.

Agency for Toxic Substances and Disease Registry. 1995. *Multisite Lead and Cadmium Study*. Atlanta, GA: ATSDR, Department of Health and Human Services, U.S. Public Health Service. NTIS PB95-199188.

Baker, E. L. J., Landrigan, P. J., Glucek, C. J., et al. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. *Am. J. Epidemiol.* 112:553–560.

Barltrop, D., and Meeks, F. 1975. Absorption of different lead compounds. *Postgrad. Med. J.* 51:805–809.

Barltrop, D., and Meeks, F. 1979. Effect of particle size on lead absorption from the gut. *Arch. Environ. Health* 34:280–285.

Biggins, P. D. E., and Harrison, R. M. 1980. Chemical speciation of lead compounds in street dusts. *Environ. Sci. Technol.* 14:336–338.

Binder, S., Sokal, D., and Maughan, D. 1986. Estimating the amount of soil ingested by young children through tracer elements. *Arch. Environ. Health* 41:341–345.

Bornschien, R. L., Succop, P., Dietrich, K. N., Clark, C. S., Que Hee, S., and Hammond, P. B. 1985. The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. *Environ. Res.* 38:108–118.

Bornschien, R. L., Succop, P. A., Krafft, K. M., Clark, C. S., Peace, B., and Hammond, P. B. 1986. Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In *Proc. 20th Ann. Conf. Trace Sub. Environ. Health*, ed. D. Hemphill, 322–332. Columbia, MO: University of Missouri.

Bornschien, R. L., Clark, C. S., Grote, J., Roda, S., Peace, B., and Succop, P. 1988. Soil lead-blood lead relationships in an urban community and in a mining community. In *Lead in Soil: Issues and Guidelines Environ. Geochem. Health Monograph*, eds. B. E. Davies and B. G. Wixson, 9:149–160.

Bornschien, R. L., Clark, C. S., Pan, W., and Succop, P. 1991a. Midvale community lead study. *Chem. Speciation Bioavailability* 3:149–162.

Bornschien, R. L., Clark, C. S., Pan, W., and Succop, P. 1991b. *The Butte-Silver Bow Environmental Health Lead Study*. Butte, MT: Butte-Silver Bow Department of Health.

Buchet, J. P., Roels, H., Lauwerys, R., Bruaux, P., Claeys-Thoreau, F., LaFontaine, A., and Verduyn, G. 1980. Repeated surveillance of exposure to cadmium, manganese, and arsenic in school age children living in rural, urban, and non-ferrous smelter areas in Belgium. *Environ. Res.* 22:95–108.

Budd, W. T., Roberts, J. W., and Ruby, M. G. 1990. *Field Evaluation of a High Volume Surface Sampler for Pesticides in House Dust*. Washington, DC: United States Environmental Protection Agency. EPA/600/3/90/030.

Burns, J. E., and Miller, F. M. 1975. Hexachlorobenzene contamination: its effects in a Louisiana population. *Arch. Environ. Health* 30:44–48.

Calabrese, E. J., Barnes, R., Stanek, E. J., et al. 1989. How much soil do young children ingest?: an epidemiological study. *Reg. Toxicol. Pharmacol.* 10:123–137.

Calabrese, E. J., and Stanek, E. J. 1991. A guide to interpreting soil ingestion studies, II: qualitative and quantitative evidence of soil ingestion. *Reg. Toxicol. Pharmacol.* 13:278–292.

Calabrese, E. J., and Stanek, E. J. 1992a. What proportion of household dust is derived from outdoor soil? *J. Soil Contam.* 1:253–263.

Calabrese, E. J., and Stanek, E. J. 1992b. Distinguishing outdoor soil ingestion from indoor dust ingestion in a soil pica child. *Reg. Toxicol. Pharmacol.* 15:83–85.

Calabrese, E. J., and Stanek, E. J. 1995a. A dog's tale: soil ingestion by a canine. *Ecotoxicol. Environ. Safety* 32:93–95.

Calabrese, E. J., and Stanek, E. J. 1995b. Resolving inter-tracer inconsistencies in soil ingestion estimation. *Environ. Health Perspect.* 103:454–457.

Calabrese, E. J., Stanek, E. J., Gilbert, C. E., and Barnes, R. M. 1990. Preliminary adult soil ingestion estimates: results of a pilot study. *Reg. Toxicol. Pharmacol.* 12:88–95.

Calabrese, E. J., Stanek, E. J., and Gilbert, C. E. 1991. Evidence of soil-pica behaviour and quantification of soil ingestion. *Human Exper. Toxicol.* 10:245–249.

Calabrese, E. J., Stanek, E. J., and Gilbert, C. E. 1993a. Lead exposure in a soil pica child. *J. Environ. Sci. Health* A28:353–362.

Calabrese, E. J., Stanek, E. J., and Gilbert, C. E. 1993b. A preliminary decision framework for deriving soil ingestion rates. In *Principles and Practices for Petroleum Contaminated Soil*, ed. E. J. Calabrese and P. T. Kostecki, 613–624. Chelsea, MI: Lewis.

Camann, D. E., and Harding, H. J. 1989. Evaluation of trapping of particle-associated pesticides on air by polyurethane foam. Research Triangle Park, NC: United States Environmental Protection Agency 68-02-4127, Research Triangle Institute.

Cambra, K., and Alonso, E. 1995. Blood lead levels in 2 to 3 year old children in the greater Bilbao area (Basque County, Spain): relation to dust and water lead levels. *Arch. Environ. Health* 50:362–366.

Caplan, K. J. 1993. The significance of wipe sampling. *Am. Ind. Hyg. Assoc. J.* 54:70–75.

Colorado Department of Health. 1990. *Leadville Metals Exposure Study.* Denver, CO: CDH.

Chaney, R. L., Sterrett, S. B., and Mielke, H. W. 1984. The potential for heavy metal exposure from urban gardens and soils. In *Proc. Symp. Heavy Metals in Urban Gardens*, ed. J. R. Preer, 37–44. Washington, DC: Agric. Exper. State Univ. Dist. Columbia.

Chaney, R. L., Mielke, H. W., and Sterrett, S. B. 1989. Speciation, mobility, and bioavailability of soil lead. In *Lead in Soil: Issues and Guidelines. Environ. Geochem. Health Monograph*, ed. B.E. Davies and B. G. Wixson, 9:105–130.

Charney, E., Sayre, J., and Coulter, M. 1980. Increased lead absorption in inner city children: where does the lead come from? *Pediatrics* 65:226–231.

Charney, E., Kessler, B., Farfel, M., and Jackson, D. 1983. Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. *N. Engl. J. Med.* 309:1089–1093.

Clark, S., Bornschien, R., Ryan, J., Kochanowski, A., Succop, P., and Peace, B. 1988. The Cincinnati soil-lead abatement demonstration project. In *Lead in*

Soil: Issues and Guidelines. Environ. Geochem. Health Monograph, ed. B. E. Davies and B. G. Wixson, 9:287–300.

Clark, S., Bornschien, R., Succop, P., Roda, S., and Peace, B. 1991. Urban lead exposures of children in Cincinnati, Ohio. *Chem. Speciation Bioavailability* 3:163–171.

Clausing, P., Brunekreef, B., and Van Wijnen, J. H. 1987. A method for estimating soil ingestion in children. *Int. Arch. Occup. Environ. Health* 59:73–82.

Culbard, E. B., and Johnson, L. R. 1984. Elevated arsenic concentration in house dusts located in a mineralized area of southwest England: implication for human health. In *Proc. 18th Ann. Conf. Trace Sub. Environ. Health*, ed. D. Hemphill, 311–319. Columbia, MO: University of Missouri.

Culbard, E. B., Thornton, I., Watt, J., Moorcroft, S., Brooks, K., and Thompson, M. 1983. Metal contamination of dusts and soils in urban and rural households in the United Kingdom, 2: a nationwide reconnaissance survey of the United Kingdom to determine metal concentrations in urban dusts and soils. In *Proc. 17th Ann. Conf. Trace Sub. Environ. Health*, ed. D. Hemphill, 236–241. Columbia, MO: University of Missouri.

Davies, B. E., Elwood, P. C., Gallacher, J., and Ginnever, R. C. 1985. The relationships between heavy metals in garden and house dusts in an old lead mining area of North Wales, Great Britain. *Environ. Pollut. B.* 9:255–266.

Davies, D. J. A., and Thornton, I. 1987. The influence of house age on lead levels in dust and soils in Brighton, England. *Environ. Geochem. Health* 9:65–67.

Davies, D. J. A., Thornton, I., Watt, J. M., *et al.* 1990. Lead intake and blood lead in two year old U.K. urban children. *Sci. Total Environ.* 90:13–29.

Davies, J. E., Edmundson, W. F., and Raffonelli, A. 1975. The role of house dust in human DDT pollution. *Am. J. Public Health* 65:53–57.

Davis, A., Ruby, M. V., and Bergstrom, P. D. 1992. Bioavailability of arsenic and lead from the Butte, Montana, mining district. *Environ. Sci. Technol.* 26:461–468.

Davis, A., Drexter, J. W., Ruby, M. V., and Nicholson, A. 1993. Micromineralogy of mine waste in relation to lead bioavailability, Butte, Montana. *Environ. Sci. Technol.* 27:1415–1425.

Davis, S., Waller, P., Buschom, R., Ballou, J., and White, P. 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: population-based estimates using aluminum, silicon, and titanium as soil tracer elements. *Arch. Environ. Health* 45:112–122.

de Silva, P. E. 1994. How much soil do children ingest?: a new approach. *Appl. Occup. Environ. Hyg.* 9:40–43.

Diemel, J. A. L., Brunekreef, B., Boleij, J. S. M., Biersteker, K., and Veenstra, S. J. 1981. The Arnhein lead study II: indoor pollution and indoor/outdoor relationships. *Environ. Res.* 25:449–456.

Duggan, M. J. 1980. Lead in urban dust: an assessment. *Water Air Soil Pollut.* 14:309–321.

Duggan, M. J. 1983. Contribution of lead in dust to children's blood lead. *Environ. Health Perspect.* 50:371–381.

Duggan, M. J., and Inskip, M. J. 1985. Childhood lead exposure to lead in surface dust and soils: a community health problem. *Public Health Rev.* 13:1–54.

Duggan, M. J., Inskip, M. J., Rundle, S. A., and Moorcroft, J. S. 1985. Lead in playground dust and on the hands of children. *Sci. Total Environ.* 44:65–79.

Edwards, W. C., and Clay, B. R. 1977. Reclamation of rangeland following a lead poisoning incident in livestock from industrial airborne contamination of forage. *Vet. Human. Toxicol.* 19:247–249.

Ewers, L., Clark, S., Menrath, W., Succop, P., and Bornschien, R. 1994. Clean-up of lead in household carpet and floor dust. *Am. Ind. Hyg. Assoc. J.* 55:650–657.

Farfel, M. R., Lees, P. S. J., Rohde, C. A., Lim, B. S., and Bannon, D. 1994a. Comparison of wipe and cyclone methods for the determination of lead in residential dusts. *Appl. Occup. Environ. Hyg.* 9:1006–1012.

Farfel, M. R., Lees, P. S. J., Rohde, C. A., Lim, B. S., Bannon, D., and Chisolm, J. J., Jr. 1994b. Comparison of wipe and vacuum collection method for the determination of lead in residential dusts. *Environ. Res.* 65:291–301.

Fergusson, J. E., and Kim, D. 1991. Trace elements in street and house dust: sources and speciation. *Sci. Total Environ.* 100:125–150.

Finley, B. L., Scott, P. K., and Mayhill, D. A. 1994. Development of a standard soil-to-skin adherence probability density function for use in Monte Carlo analyses of dermal exposure. *Risk Anal.* 14:555–569.

Fortmann, R. C., Sheldon, L. S., Smith, D., and Martin-Goldberg, M. 1991. *House Dust/Infant Pesticide Exposure Study (HIPES), Final Report.* Research Triangle Park, NC: United States Environmental Protection Agency-RTI/4657-75/00FR. Research Triangle Institute.

Freeman, G. B., Johnson, J. D., Killinger, J. M., et al. 1992. Relative bioavailability of lead from mining waste soils in rats. *Fund. Appl. Toxicol.* 19:388–398.

Freeman, G. B., Schoof, R. A., Ruby, M. V., et al. 1995a. Bioavailability of arsenic in soil and house dust impacted by smelter activities following oral administration in cynomolgus monkeys. *Fund. Appl. Toxicol.* 28:215–222.

Freeman, N. C. G., Wainmann, T., and Lioy, P. 1996. Field testing of the LWW sampler and association of observed household factors with dust loading. *Appl. Occup. Environ. Hyg.* 11:476–483.

Freeman, N. C. G., Wainman, T., Lioy, P. J., Stern, A. H., and Shupack, S. I. 1995b. The effect of remediation of chromium waste sites on chromium levels in urine of children living in the surrounding neighborhood. *J. Air Waste Management Assoc.* 45:604–614.

Gulson, B. L., Davis, J. J., Mizon, K. J., Korsch, M. J., and Bawden-Smith, J. 1995. Sources of lead in soil and dust and the use of dust fallout as a sampling medium. *Sci. Total Environ.* 166:245–262.

Harper, M., Sullivan, K. R., and Quinn, M. J. 1987. Wind dispersal of metals from smelter tips and their contribution to environmental contamination. *Environ. Sci. Technol.* 21:481–484.

Hawley, J. K. 1985. Assessment of health risk from exposure to contaminated soil. *Risk Anal.* 5:289–302.

Healy, M. A., Harrison, P. G., Aslam, M., Davis, S. S., and Wilson, C. G. 1982. Lead sulphide and traditional preparations: routes for ingestion, and solubility and reactions in gastric fluid. *J. Clin. Hosp. Pharm.* 7:169–173.

Hilts, S. R., Hertzman, C., and Marion, S. A. 1995. A controlled trial of the effect of HEPA vacuuming on childhood lead exposure. *Can. J. Public Health.* Sept–Oct:345–350.

Howells, J. C., and Thornton, I. 1991. Sources and pathways of environmental lead to children in a derbyshire mining village. *Environ. Geochem. Health* 13:127–135.

Kamens, R., Lee, C., Wiener, R., and Leith, D. 1991. A study to characterize indoor particles in three non-smoking homes. *Atmos. Environ.* 25A:939–948.

Kimbrough, R. 1995. Polychlorinated biphenyls (PCBs) and human health: an update. *Crit. Rev. Toxicol.* 25:133–163.

Kimbrough, R., Falk, H., Stehr, P., and Fries, G. 1984. Health implications of 2,3,7,8-TCDD contamination of residential soil. *J. Toxicol. Environ. Health* 14:47–93.

Kimbrough, R., LeVois, M., and Webb, D. 1994. Management of children with slightly elevated blood lead levels. *Pediatrics* 93:188–191.

Kimbrough, R., LeVois, M., and Webb, D. 1995. Survey of lead exposure around a closed lead smelter. *Pediatrics* 95:550–554.

Kissel, J. C., Richter, K. Y., and Fenske, R. A. 1996. Field measurement of dermal soil loading attributable to various activities: implications for exposure assessment. *Risk Anal.* 16:115–125.

Knutson, E. O. 1992. Measurments of radon progeny activity on typical indoor surfaces. *Rad. Protect. Dosimetry* 45:313–317.

Lanphear, B. P., Emond, M., Jacobs, D. E., Weitzman, M., Tanner, M., Winter, N. L., Yakir, B., and Eberly, S. 1995. A side-by-side comparison of dust collection methods for sampling lead-contaminated house dust. *Environ. Res.* 68:114–123.

Lefcoe, N. M., and Inculet, I. I. 1975. Particulates in domestic premises II: ambient levels and indoor-outdoor relationships. *Arch. Environ. Health* 30:565–570.

Lewis, R. G., Bond, A. E., Camann, D. E., Fortmann, R. C., and Sheldon, L. S. 1991. Determination of routes of exposure of infants and toddlers to household pesticides: a pilot study of test methods. *Proc. Ann. Meeting Air Waste Management Assoc.* 62:1. Vancouver, BC.

Lepow, M. L., Bruckman, L., Gillette, M., Markowitz, S., Robino, R., and Kapish, J. 1975. Investigations into sources of lead in the environment of urban children. *Environ. Res.* 10:415–426.

Lioy, P. J., Wainman, T., and Weisel, C. 1993. A wipe sampler for the quantitative measurement of dust on smooth surfaces: laboratory performance studies. *J. Exp. Anal. Environ. Epidemiol.* 3:315–320.

Lioy, P. J., Yiin, L. M., Adgate, J., Weisel, C., and Rhodes, G. 1997. The effectiveness of a home cleaning intervention strategy in reducing potential dust and lead exposures. *J. Exp. Anal. Environ. Epidem.*, in press.

Missouri Department of Health. 1995. *Jasper County Missouri Superfund Site Lead and Cadmium Exposure Study.* Jefferson City, MO: MDH.

Milar, C. R., and Mushak, P. 1982. Lead contaminated housedust: hazard, measurement and decontamination. In *Lead Absorption in Children: Management, Clinical, and Environmental Aspects*, ed. J. J. Chisolm and D. M. O'Hara, 143–152. Baltimore, MD: Urban and Schwarzenbert.

Miller, D. T., Condon, S. K., Kutzner, S., *et al.* 1991. Human exposure to polychlorinated biphenyls in greater New Bedford, Massachusetts: a prevalence study. *Arch. Environ. Contam. Toxicol.* 20:410–416.

Millson, M., Eller, P. M., Ashley, K. 1994. Evaluation of wipe sampling materials for lead in surface dust. *Am. Indust. Hyg. Assoc. J.* 55:339–342.

Moffat, W. E. 1989. Blood lead determinants of a population living in a former lead mining area in southern Scotland. *Environ. Geochem. Health* 11:3–9.

Moorcroft, S., Watt, J., Thornton, I., Wells, J., Strehlow, C. D., and Barltrop, D. 1982. Composition of dusts and soils in an apparently uncontaminated rural village in southwest England: implications to human health. In *Proc. 16th Ann. Conf. Trace Sub. Environ. Health.* ed. D. Hemphill, 153–163. Columbia, MO: University of Missouri.

Mullins, M. J. P., and Norman, J. B. 1994. Solubility of metals in windblown dust from mine waste dump sites. *Appl. Occup. Environ. Hyg.* 9:218–222.

Panhandle District Health Department. 1986. *Kellogg Revisited–Childhood Blood Lead Levels and Environmental Status Report.* ID: Panhandle District Health Department.

Paustenbach, D. J. 1987. Assessing the potential environment and human health risks of contaminated soils. *Comments Toxicol.* 1:185–221.

Paustenbach, D. J. 1995. The practice of health risk assessment in the United States (1975–1995): how the U.S. and other countries can benefit from that experience. *Hum. Ecol. Risk Assess.* 1:29–79.

Paustenbach, D. J., Wenning, R. J., Lau, V., Harrington, N. W., Rennix, D. K., and Parsons, A. H. 1992a. Recent development on the hazards posed by 2,3,7,8-tetrachlorodibenzo-p-dioxin in soil: implications for setting risk-based cleanup levels at residential and industrial sites. *J. Toxicol. Environ. Health* 36: 103–149.

Paustenbach, D. J., Jernigan, J. D., Bass, R. D., Kalmes, R., and Scott, P. 1992b. A proposed approach to regulating contaminated soil: identify safe concentrations for seven of the most frequently encountered exposure scenarios. *Regul. Toxicol. Pharm.* 16:21–56.

Paustenbach, D. J., Bruce, G. M., and Chrostowski, P. 1997. Current views on the oral bioavailability of mercury in soil: the impact on health risk assessments. *Risk Anal.*, in press.

Paustenbach, D. J., Shu, H. P., and Murray, F. J. 1986. A critical examination of assumptions used in risk assessment of dioxin contaminated soil. *Regul. Toxicol. Pharm.* 6:284–307.

Polissar, L., Lowry-Coble, K., Kalman, D. A., et al. 1990. Pathways of human exposure to arsenic in a community surrounding a copper smelter. *Environ. Res.* 53:29–47.

Que Hee, S., Shane, S., Peace, B., Clark, C. S., Boule, J. R., Bornschien, R. L., and Hammond, P. B. 1985. Evolution of efficient methods to sample lead sources, such as house dust, in the homes of children. *Environ. Res.* 38:77–95.

Roberts, J. W., Camann, D. E., and Spittler, T. M. 1990. Monitoring and controlling lead in house dust in older homes. Toronto, Quebec, Canada: *Fifth International Conference on Indoor Air.*

Roberts, J. W., Budd, W. T., Ruby, M. G., Bond, A. E., Lewis, R. G., Wiener, R. W., and Camann, D. E. 1991. Development and field testing of a high volume sampler for pesticides and toxics in dust. *J. Exp. Anal. Environ. Epidemiol.* 1:143–155.

Roberts, J. W., Budd, W. T., Chuang, J., Lewis, R. G. 1993. *Chemical Contaminants in House Dust: Occurrences and Sources.* Washington, DC: United States Environmental Protection Agency. EPA/600/A-93/215.

Roberts, J. W., Glass, G. L., and Spittler, T. M. 1995. Measurement of deep dust and lead in old carpets. In *Measurement of Toxic and Related Pollutants*, Proceedings of the Air and Waste Management Association, San Antonio, TX.

Roberts, T. M., Gizyn, W., and Hutchinson, T. C. 1974. Lead contamination of air, soil, vegetation, and people in the vicinity of secondary lead smelters. In *Proc. 8th Ann. Conf. Trace Sub. Environ. Health*, ed. D. Hemphill 155–166. Columbia, MO: University of Missouri.

Roels, H. A., Buchet, J. P., Lauwerys, R. R., Bruaux, P., Claeys-Thoreau, F., LaFontaine, A., and Verduyn, G. 1980. Exposure to lead by the oral and the

pulmonary routes of children living in the vicinity of a primary lead smelter. *Environ. Res.* 22:81–94.

Ruby, M. V., Davis, A., Kempton, J. H., Drexter, J. W., and Bergstrom, P. D. 1992. Lead bioavailability under simulated gastric conditions. *Environ. Sci. Technol.* 26:1242–1248.

Rundle, S. A. 1984. *The Variation of the Concentration of Lead in Dust with Particle Size.* Report DG/SSB/ESD/R159. London: Scientific Services Branch, Greater London Council.

Sayre, J. W., Charney, E., Vostal, J., and Pless, I. B. 1974. House and hand dust as a potential source of childhood lead exposure. *Am. J. Dis. Child.* 127:167–170.

Schneider, T., Bohgard, M., and Gudmundsson, A. 1994. A semiempirical model for particle deposition onto facial skin and eyes: role of air currents and electric fields. *J. Aerosol Sci.* 25:583–593.

Schilling, R. J., and Bain, R. P. 1988. Prediction of children's blood lead levels on the basis of household specific soil lead levels. *Am. J. Epidemiol.* 128:197–205.

Shu, H. P., Teitelbaum, P., Webb, A. S., Marple, L., Brunck, B., DeiRossi, D., Murray, F. J., and Paustenbach, D. J. 1988. Bioavailability of soil-bound TCDD: dermal bioavailability in the rat. *Fund. Appl. Toxicol.* 10:335–343.

Spittler, T. M., and Feder, W. A. 1979. A study of soil contamination and plant lead uptake in Boston urban gardens. *Com. Soil Sci. Plant Anal.* 10:1195.

Stanek, E. J., Calabrese, E. J. 1991. A guide to interpreting soil ingestion studies, I: development of a model to estimate the soil ingestion detection level of soil ingestion studies. *Reg. Toxicol. Pharmacol.* 13:263–277.

Stanek, E. J., and Calabrese, E. J. 1992. Soil ingestion in children: outdoor soil or indoor dust. *J. Soil Contam.* 1:1–28.

Stanek, E. J., and Calabrese, E. J. 1995a. Improved soil ingestion estimates for use in site evaluations using the best tracer method. *Human Ecol. Risk Assess.* 1:133–157.

Stanek, E. J., and Calabrese, E. J. 1995b. Daily estimates of soil ingestion in children. *Environ. Health Perspect.* 103:276–285.

Stanek, E. J., Calabrese, E. J., Barnes, R., *et al.* 1988. Ingestion of trace elements from food among preschool children: Al, Ba, Mn, Ti, V, Y, and Zr. *Trace Elements Exper. Med.* 1:179–190.

Stark, A. D., Quah, R. F., Meigs, J. W., and DeLouise, E. R. 1982. The relationship of environmental lead to blood lead levels in children. *Environ. Res.* 27:372–383.

Stern, A. H., Freeman, N. C. G., Pleban, P., Boesch, R. R., Wainman, T., Howell, T., Shupack, S. I., Johnson, B. B., and Lioy, P. J. 1992. Residential exposure to chromium waste-urine biological monitoring in conjunction with environmental exposure monitoring. *Environ. Res.* 58:147–162.

Sturges, W. T., and Harrison, R. M. 1985. An assessment of the contribution from paint flakes to the lead content of some street and household dusts. *Sci. Total Environ.* 44:225–234.

Thatcher, T. L., and Layton, D. W. 1995. Deposition, resuspension, and penetration of particles within a residence. *Atmos. Environ.* 29:1487–1497.

Thornton, I. 1986. Lead in United Kingdom soils and dusts in relation to environmental standards and guidelines. In *Proc. 20th Ann. Conf. Trace Sub.*

Environ. Health, ed. D. Hemphill, 298–307. Columbia, MO: University of Missouri.

Thornton, I., John, S., Moorcroft, S., and Watt, J. 1980. Cadmium at Shipham: a unique example of environmental geochemistry and health. In *Proc. 14th Ann. Conf. Trace Sub. Environ. Health,* ed. D. Hemphill, 27–37. Columbia, MO: University of Missouri.

Thornton, I., Culbard, E., Moorcroft, S., Watt, J., Wheatley, M., Thompson, M., and Thomas, J. F. A. 1985. Metals in urban dusts and soils. *Environ. Technol. Lett.* 6:137–144.

Thornton, I., Davies, D. J. A., Watt, J. M., and Quinn, M. J. 1990. Lead exposure in young children from dust and soil in the United Kingdom. *Environ. Health Perspect.* 89:55–60.

U.S. Environmental Protection Agency [U.S. EPA]. 1995a. *Exposure Factors Handbook* (Draft). Washington, DC: Exposure Assessment Group, Office of Health and Environmental Assessment, United States Environmental Protection Agency. EPA/600/P-95/002A.

U.S. EPA. 1995b. *Urban Soil Lead Abatement Demonstration Project* (Draft). Research Triangle Park, NC: United States Environmental Protection Agency. EPA/600/R-95/139. National Center for Environmental Assessment.

U. S. EPA. 1994. *Integrated uptake biokinetic model for children.* Washington, DC: EPA.

Van Borm, W., Keersmaekers, T., and Adams, F. 1988. Characteristics of resuspended soil particles with high concentrations of Cu, Zn, Cd, and Pb as a function of particle size. *Aerosol Sci.* 19:1287–1289.

Van Wijnen, J. H., Clausing, P., and Brunekreef, B. 1989. Estimated soil ingestion by children. *Environ. Res.* 51:147–162.

Vostal, J., Taves, E., Sayre, J. W., and Charney, E. 1974. Lead analysis of housedust: a method for detection of another source of lead exposure in inner city children. *Environ. Health Perspect.* 7:99–102.

Wang, E. Y., Willis, R. D., Buckley, T. J., Rhoads, G. G., and Lioy, P. J. 1996. The relationship between dust lead concentrations and the particle sizes of household dusts collected in Jersey City residences. *Appl. Occup. Environ. Hyg.* 11:199–206.

Warnick, S. L. 1972. Human and environmental exposure to pesticides in Utah. *Proc. Utah Acad. Sci. Arts Lett.* 49:11–20.

Watt, J., Moorcroft, S., Brooks, K., Culbard, E. B., and Thornton, I. 1983. Metal contamination of dust and soils in urban and rural households in the United Kingdom: I. Sampling and analytical techniques for household and external dust. In *Trace Substances in Environmental Health,* XVII, edited by D. D. Hemphill, pp. 229–235. Columbia, MO: University of Missouri.

Weitzman, M., Aschengrau, A., Bellinger, D., Jones, R., Hamlin, J. S., and Beiser, A. 1993. Lead-contamined soil abatement and urban children's blood lead levels. *JAMA* 269:1647–1654.

Yaffe, B. A., and Reeder, B. 1989. An epidemiologic assessment of exposure of children to polychlorinated biphenyls (PCBs) in a Toronto community. *Can. J. Public Health* 80:325–329.

Yankel, A. J., von Linden, I. H., and Walters, S. D. 1977. The Silver Valley Lead Study: the relationship between childhood blood lead levels and environmental exposure. *J. Air Pollut. Control Assoc.* 27:763–767.

Yeh, C. Y., Wang, G. Y., Kuo, P. H., and Tsai, S. T. 1977. House dust and pesticide residues pollution. *J. Formosan Med. Assoc.* 76:324–327.

Zimdahl, R. L., and Foster, J. M. 1976. The influence of applied phosphorus, manure, or lime on uptake of lead from soil. *J. Environ. Qual.* 5:31–33.