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ANALYTICAL PERFORMANCE CRITERIA FOR LEAD TEST KITS AND OTHER ANALYTICAL METHODS

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SECTION 1
INTRODUCTION

1.1 BACKGROUND

The adverse health effects resulting from exposure of young children to environmental lead has received increasing attention in recent years. Studies have shown that chronic exposure even to low levels of lead can result in impairment of the central nervous system, mental retardation, and behavioral disorders. Although young children are at the greatest risk, adults may suffer harmful effects as well.

The major sources of exposure to lead in housing units are thought to be paint, dust and soil. Food, water and airborne lead are also potential sources, but are considered to be less significant avenues of exposure. Currently, lead-based paint is receiving emphasis as a critical area of concern and a principal medium for lead contamination and exposure. It is particularly significant when painted walls, woodwork and furniture are low enough for children to touch and to chew. Although less consideration has been given to soil and dust, they are also important routes of exposure. Soil, which is often contaminated with lead from petroleum additives or from the leaching of exterior paint (near driplines, etc.), may be tracked into homes. Like dust, it becomes collected on hands, toys and food and is ingested. Concentrations in paint, dust and soil must be determined if a comprehensive approach to the problem of lead exposure from housing sources is to be established.

There are two ranges of concentration which are of concern. The first includes the level of lead in paint that necessitates abatement and levels in dust and/or soil that necessitate removal. The second includes the levels of lead in the paint and dust after abatement, and levels in soil and dust after removal that indicate acceptable levels of cleanliness, i.e., that the dwelling site meets "clearance" requirements. Both ranges are driven by health effects, though the abatement level of lead in paint has also been driven by the ability to measure lead in paint using the portable X-ray fluorescence spectrometer (XRF). These levels are also being measured by atomic spectroscopic methods in the laboratory and in the dwelling unit using chemical spot tests. The spot test and XRF methods are being developed and/or

improved at a rapid pace in response to the tremendous interest in lead exposure. Also, the atomic spectroscopic methods are being evaluated for their accuracy and precision, and especially for analysis of old hardened paints. There is clearly a need for some analytical performance criteria to be established which are in accord with health effects, abatement, clearance and other driving forces such as regulations. The intent of this document is to propose such analytical performance criteria as targets for the development of test kits and other analytical methods.

1.2 DOCUMENT DESIGN

Development of the analytical performance criteria has been performed in stages. First to be investigated were the health effects. Numerous papers were located and read, and many personal contacts were made to identify the most recent data regarding the relationships between levels of lead in various matrices and health effects. Next to be investigated were Federal and State regulations for lead exposure. Third, current performance capabilities for methods of measurement were reviewed. All those data were then brought together to arrive at proposed analytical performance standards for lead test kits and other analytical methods.

SECTION 2
IMPACT OF LOW LEVEL LEAD EXPOSURE

Any analytical performance criteria should be based on the ultimate use of the data to be collected, which is to decide whether levels of lead present will or will not cause adverse health effects and/or whether governmental regulations or standards have been met. Accordingly three areas have been investigated - health effects of lead, sources of lead, and Federal and State regulations. First to be considered are the health, i.e., biological, effects of lead.

2.1 ABSORPTION AND DISTRIBUTION

The major route of absorption of lead is through the gastrointestinal tract. Ziegler et al. (1983) have estimated that the absorption rate in adults is approximately 5 percent, whereas children absorb lead at a rate of 40 - 50 percent, and retain about 30 percent of ingested lead. Once lead is absorbed, it is distributed to the blood, the soft tissues and the bones. About 95 percent of absorbed lead is bound to erythrocytes for approximately 4 to 6 weeks, and then accumulated in calcified tissues, particularly in the bone marrow, for years. The skeleton system acts as a mineral reservoir by releasing lead into the blood when blood levels fall and facilitating deposition when ingestion exceeds excretion.

Blood lead levels are the most widely used indicator of lead exposure. Determination of dentine lead in deciduous teeth offers the potential of an appropriate biological marker for chronic lead exposure (Biddle, 1982).

2.2 TOXICOLOGICAL EFFECTS

Chronic lead exposure at high levels usually occurs only in occupational settings, such as lead smelters, battery plants, house painting or scraping. Exposure to hazardous levels has been shown to cause peripheral neuropathy in adults and encephalopathy in children (Goyer, 1986).

There has been increasing concern about the hazards associated with low level exposure, especially in vulnerable population groups, such as infants, children, women of child-bearing age and the elderly. Findings in a study by

the Agency for Toxic Substances and Disease Registry (ATSDR) (Agency for Toxic Substances and Disease Registry, 1988) identified toxicological effects of lead as brain or central nervous system (CNS) dysfunctions, impairment in the heme-forming and vitamin D regulatory systems, cardiovascular effects, and reproductive disorders.

The Centers for Disease Control (CDC) has defined an "elevated" blood lead level as 25 μg/dL for children (Centers for Disease Control, 1985), but this value is currently undergoing revision. Exposure levels resulting in adverse effects also vary with susceptibility. It is believed that levels of 10 - 15 μg/dL are significant enough to affect development of the fetus (Marbury, 1990). The lowest blood level associated with adverse biological effects has been observed to be 10 μg/dL (Minnesota Department of Health, 1984), but a "safe" lead level has not been established. Individuals may show similar effects at different blood lead concentrations. The current "action level" for occupational exposure in the U.S. is 50 μg/dL (Quinn and Sherlock, 1990).

Results from the Second National Health and Nutrition Examination Survey (NHANES-II, 1984) indicated that during the survey period (1976-1980), 8.5 million children in the United States had blood lead levels of $\geq$15.0 μg/dL (Houk et al., 1989). Children are particularly vulnerable to the toxic effects of lead, likely from an ingestion/absorption route, rather than inhalation. Reasons for this vulnerability are as follows:

- the increased intestinal efficiency of absorption in children, approximately 40 - 50 percent of the ingested amount as opposed to 10 percent in adults,
- increased absorption associated with nutritional deficiencies in iron, calcium and zinc, often relatively common in childhood,
- hand-to-mouth activities and pica habits,
- increased metabolic rate in children,
- immature enzymatic systems and blood brain barrier, and
- the percentage of compact bone for final absorption of the body burden of lead is lower in children than in adults, leading to a greater possibility for lead to reach dangerous concentrations in target organs in children.

Several of the specific toxic effects of lead are described in the following sections.

2.2.1 Interference with Heme Biosynthesis

Lead has been shown to interfere with heme biosynthesis by inhibiting δ-aminolevulinic acid dehydratase (ALA-D), the enzyme that catalyzes the condensation of two molecules of δ-aminolevulinic acid (ALA) to yield one molecule of porphobilinogen. Lead decreases the activity of ALA-D in erythrocytes, and thereby inhibits the formation of porphobilinogen. Chisolm investigated blood lead levels and ALA-D activity in children and determined that a blood lead (PbB) level of 5 μg/dL was a no-effect level for ALA-D activity (Chisolm et al., 1985). Roels found a threshold level of 19.9 μg/dL for inhibition of this activity in children (Roels et al., 1976). Animal studies (Azar et al., 1973) have shown that ALA-D activity in rats dosed (feed) with lead acetate for 2 years decreased at blood lead levels of 18.5 μg/dL. Significant decreases in hemoglobin and hematocrit were noted at blood lead levels of 98.6 μg/dL.

Lead also inhibits ferrochelatase, the enzyme that catalyzes the incorporation of iron into the porphyrin ring, the last step of heme formation. Failure to insert iron into protoporphyrin results in depressed heme formation. The excess protoporphyrin takes the place of heme in the circulating red blood cells. Free erythrocyte protoporphyrin (EP) levels are increased with increasing blood concentrations of lead. EP levels have become a biological indicator of lead exposure, although not as commonly used as blood lead levels. Piomelli estimated the threshold for no adverse health effects from elevated EP levels in study children to be between 16 and 20 μg/dL (Piomelli et al., 1982). Roels found a no-effect limit of 19.9 μg/dL (Roels et al., 1976).

2.2.2 Impairment of Vitamin D Biosynthesis

Adverse renal effects have been shown to be caused by lowered levels of the hormonal form of Vitamin D, 1,25-dihydroxycholecalciferol (1,25-CC), in the blood. The synthesis of 1,25-CC is carried out in the renal mitochondria, known to be a target organ for carcinogenic effects (see Section 2.3). Rosen found that blood lead levels above 55 μg/dL interfered with vitamin D metabolism (Rosen et al., 1980).

2.2.3 Neurotoxic Effects

The most significant effects of lead on human health and performance are on the central nervous system (Needleman, 1980). Peripheral neuropathy results from exposure to toxic amounts of lead in adults. Symptoms such as footdrop and wristdrop were associated with exposure to high levels of lead in occupational settings more than half a century ago (Thomas, 1904). Epidemiological studies have shown a reduction in IQ scores for children with blood lead levels of 30 μg/dL (Grant and Davis, 1989). These children showed no other signs of lead toxicity. Other studies have shown deficiencies in cognitive development at blood lead levels of 10 - 15 μg/dL (USEPA, 1986; USEPA, 1986a; Davis and Svendsgaard, 1987). In general these deficiencies have been related to prenatal lead exposure, but there are also indications that correlations between neurological deficiencies and blood lead levels may involve a lag of months or years (USEPA, 1986a).

At blood lead levels of 80 - 100 μg/dL, acute lead encephalopathy has been observed (Boeckx, 1986). The lowest observed adverse effects levels (LOAELs) for significant behavioral alterations have been detected in primates whose maximum blood lead was 15 μg/dL (Rice, 1985). There have not been extensive studies for comparing human and animal dose-response relationships for lead exposure, but studies have suggested that rats, and possibly monkeys, may tolerate a higher exposure level than humans to achieve equivalent blood levels (Hammond et al., 1985). The greatest similarities between human and animal effects involve cognitive and complex behavioral processes. The question of comparability in blood levels across species is unanswered. The lowest levels at which neurobehavioral effects have been observed are:

- children - 10 - 15 μg/dL
- primates - 15 μg/dL
- rodents - 20 μg/dL

Table 2-1 gives a summary of some toxicological effects of lead and blood lead levels associated with these effects in adults.

Table 2-1. Minimum Blood Lead Levels
Associated with Toxic Effects in Adults

Blood Lead (μg/dL)	Toxic Effect
5 - 10	ALA-D inhibition
15 - 20 (women) 20 - 25 (men)	Increased Erythrocyte Protoporphyrin (EP)
40	Increased urinary ALA excretion
40	Peripheral neuropathy
50	Minimal brain dysfunction
50	Lowered hemoglobin
>80	Encephalopathy

Sources: Grandjean, 1978; Quinn and Sherlock, 1990

2.3 CARCINOGENIC EFFECTS

The carcinogenic activity of lead has been investigated in animals and in humans. Tumorigenicity studies in rats and mice revealed a direct relationship between the incidence of renal tumors and increasing dosages of lead in both sexes. These findings are sufficient to establish carcinogenicity in experimental animals by criteria given in EPA Guidelines for Carcinogen Risk Assessment (USEPA, 1986b). Data for cancer mortality in human exposure groups is equivocal. Carcinogenic potential is suggested, but has not be quantitatively established. Therefore, lead is considered a Group 2B carcinogen by the International Agency for Research on Cancer (IARC) and is classified by EPA criteria as a B2 carcinogen, a probable carcinogen in humans.

2.4 CONTRIBUTION TO BODY BURDEN FROM ENVIRONMENTAL SOURCES

In order to effectively reduce exposure, there has been an increasing interest in the potential environmental sources of lead exposure (air, drinking water, soil, dust, paint, and food) and in differentiating the contribution from each source to the total concentration of lead in the body, usually expressed as a blood lead level (PbB, μg/dL), and its relationship to overall health effects.

The most significant sources of lead in childhood exposure are lead in paint, dust, soil and drinking water. Children in an urban environment are exposed to lead by the air they breathe, the water they drink, and the food and non-food ingested. Exposure from these sources may be intercorrelated as shown schematically by a source/intake model given in Figure 2-1.

It is currently believed that children should not be exposed to more than 100 - 150 μg Pb/day (Boeckx, 1986), but many children ingest up to 175 μg Pb/day in food, water and air alone. Typical exposure source levels are given in Table 2-2.

Boeckx (1986) has estimated that a child exposed to lead in water, air and food may ingest 160 μg Pb/day and absorb 74 μg. Pica activities (eating paint chips or soil) can increase the total intake to 2600 μg Pb/day and the absorption to 550 μg Pb. Lead based paint has the potential to be the most concentrated source of exposure in children. According to Sayre, "The

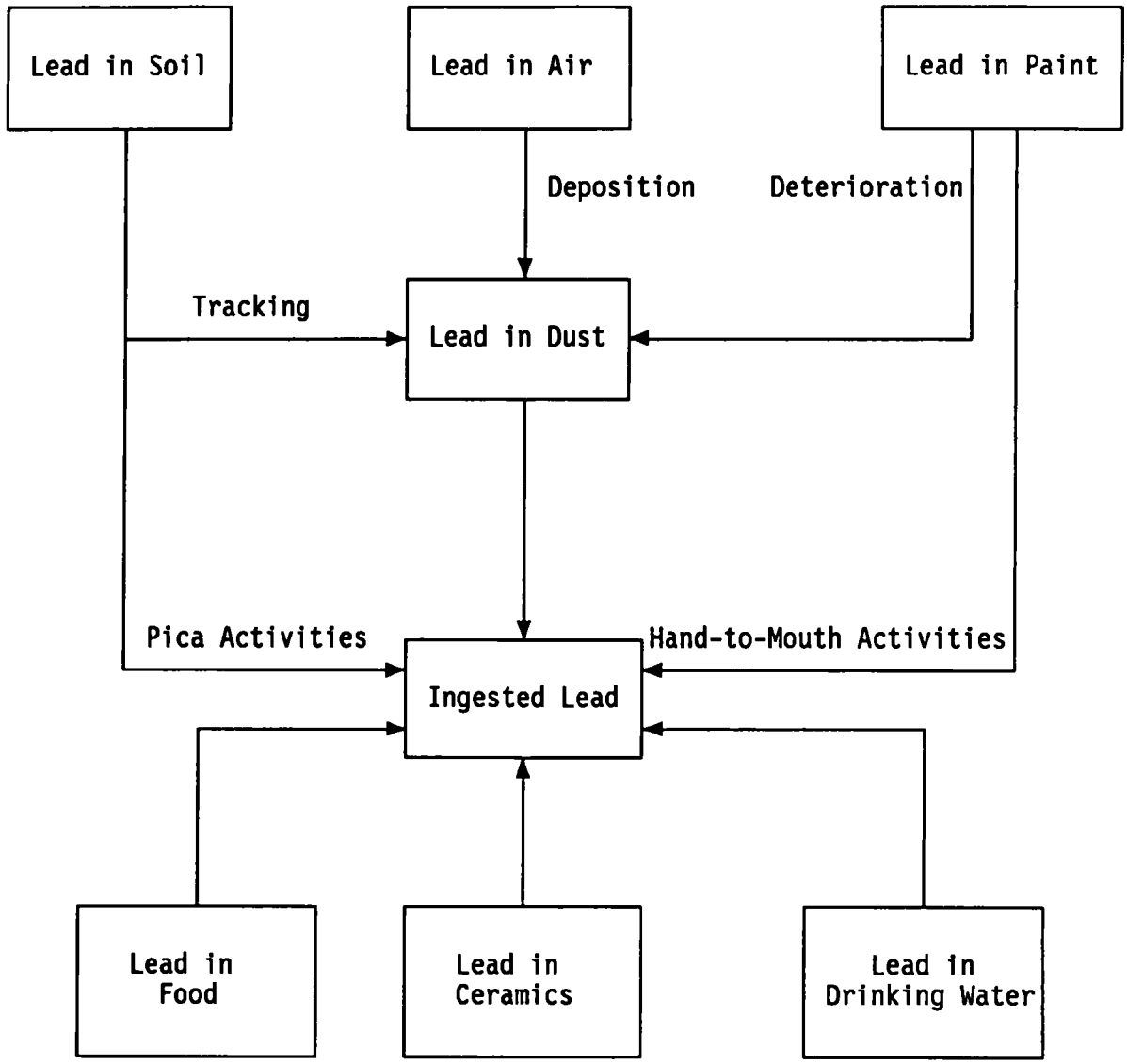

Figure 2-1. Environmental Sources for Lead Uptake

Table 2-2. Typical Lead Concentrations from Environmental Sources

Medium	Range of Typical Lead Concentrations
Air (ambient)	0.5 - 2 $\mu g/m^3$
Air (near heavy traffic)	5 - 10 $\mu g/m^3$
Water	<1 - 20 $\mu g/L$
Typical foods	0.1 - 0.5 $\mu g/g$
Soil (upper few centimeters)	<100 - >10,000 $\mu g/g$
Street dust	206 - 20,000 $\mu g/g$
House dust	18 - 11,000 $\mu g/g$
Paint	<1 - >5 mg/cm^2

Source: Boeckx, 1986

ingestion of leaded paint chips has been implicated clearly as the major mechanism leading to overtly symptomatic childhood lead poisoning and lead encephalopathy in particular," (Sayre et al., 1974). Contributions of lead from various sources are described in the following sections.

2.4.1 Air

Airborne lead is a primary source for lead found in food and dusts. The EPA has estimated that blood lead levels will rise 1 $\mu g/dL$ for every increase of 1 $\mu g/m^3$ of lead in air inhaled (Snee, 1981). The relative contribution to body burden is approximately 5 to 10 percent of adult blood lead levels (10 - 20 $\mu g/dL$) because ambient air levels rarely exceed 2 $\mu g/m^3$. At a mean air lead concentration of 0.75 $\mu g/m^3$ with an estimation of 40 percent absorption, Boeckx suggests that a child would absorb 3 μg/day directly from air. It is important to realize that the ultimate fate of airborne lead is deposition as street and house dust, providing a pathway for more a concentrated intake of lead.

2.4.2 Drinking Water

Lacey showed a linear relationship between blood lead and water lead level (Lacey et al., 1985). Houk found that drinking water contributes 22 percent to blood lead at 50 ppb and the mean blood lead increases by 1 $\mu g/dL$ as the concentration of lead in water increases by 50 ppb (Houk et al., 1989). Mushak suggested that consumption of 1-2 liters of tap water with lead levels of 20-40 ppb results in an increase in blood lead of 3.2 - 6.4 $\mu g/dL$ in children (Mushak and Crocetti, 1989). The EPA estimated (USEPA, 1979) that a lead-in-water concentration of 50 ppb would result in an average blood level concentration in children of 15 $\mu g/dL$.

2.4.3 Paint

Approximately 52 percent of all U.S. housing stock has lead levels in paint that exceed the concentration considered "positive" for lead by the CDC, 0.7 mg/cm^2 (Mushak and Crocetti, 1989). Lin-Fu (1980) studied high lead concentrations in peeling paint and blood lead levels in children. The study showed that 50 percent of the homes of children having high blood lead levels (40 - 70 $\mu g/dL$) had peeling paint with high concentrations of lead. In the

control group (blood lead levels of $\leq$30 μg/dL), 25 percent of the homes had paint with high lead levels. Mushak and Crocetti (1989) have estimated that 1.2 million children have sufficient paint lead-based exposure to raise their lead levels above 15 μg/dL. For comparison, the authors have estimated that 3.8 million children are exposed to lead in tap water at levels that may cause an increase in blood lead levels.

2.4.4 Soil

Boeckx (1986) has indicated that lead from soil and dust are absorbed with an efficiency of 30 percent, as opposed to an absorption of 50 percent for dietary lead (food and drinking water). Although the CDC (1985) has indicated that "the blood lead level in children in general does not begin to increase until soil lead levels are in the 500 to 1000 ppm range," Albritti et al. (1989) suggest that for each increase of 100 ppm (μg Pb/g) in the lead content of surface soil, above a level of 500 ppm, there is a mean increase in blood lead of 1 to 2 μg/dL. The U.S. EPA estimates the blood lead/soil slope to be 0.6 to 0.8 μg/dL per 1000 ppm of soil lead (USEPA, 1983). Duggan and Williams (1977) estimated a 5 μg/dL increase in blood lead level in children for every 1000 ppm increase of lead in soil, and indicated that determinations of a "safe" soil level were precluded by the variation (100-fold) in the amount of soil ingested by children.

2.4.5 Dust

Paint and airborne lead from automobile exhaust and other sources are considered to be the primary contributors to dust and soil lead levels. The primary pathway of exposure in children is believed to be dust, but the contribution of paint deterioration to dust lead is uncertain. Dr. Robert Elias of the U.S. Environmental Protection Agency, has stated that studies are now underway to estimate this value. Mechanisms for lead-based paint turning to dust include abrasion, and simple shedding of particles at the surface of the paint. This shedding would occur from degradation of the paint through oxidation and/or photodecomposition and expansion and contraction of the paint with temperature variations.

Data for blood lead levels from dust are inconsistent. As a rule, studies rely on data for exposure of children to high lead concentrations, such as around smelters. EPA estimates from a summary of studies that the contribution from dust to the body burden is 1.8 μg Pb/dL blood per 1000 μg Pb/g dust (Elwood EPA, 1986). Duggan (1980) estimates a higher contribution of 5 μg/dL per 1000 μg Pb/g dust. Laxen (1987) estimates an increase of 1.9 μg/dL for every 1000 ppm increase in dust lead concentration.

2.4.6 Food

The uptake of lead in food is difficult to measure. It is believed to be associated with dietary uptake, as well as with the use of canned food with soldered seals, ceramic glazes, crystal, and by absorption from water during cooking. The effects of lead levels in soil are also a consideration. Gallacher et al. (1984) estimated the contribution to blood lead from vegetables grown in soil contaminated by lead mining operations to be 3 μg/dL.

Studies have indicated that an increase in gastrointestinal absorption of lead is associated with deficiencies of calcium, iron and zinc (Mahaffey, 1983). Moreau et al. (1982) found a direct relationship between alcohol consumption and increases in blood lead. Studies on the synergistic effects of alcohol and smoking have been carried out by Shaper who showed that blood lead levels for men who smoked and consumed alcohol were as much as 44 percent higher than levels observed for participants who did not smoke or drink (Shaper et al., 1982).

Although there appears to be a decrease in dietary uptake of lead in recent years (Solgaard et al., 1979), possibly from the use of fresh and frozen foods, a nationwide survey of preschool children in the U.S. (Bander et al., 1983) indicated an average uptake from food of 56 μg Pb/day. If an average body weight of 10 kg is assumed, this value exceeds the provisional tolerable weekly intake of 25 μg/kg of body weight established by the Joint FAO/WHO Expert Committee of Food Additives (WHO, 1987). Most studies have revealed a dietary uptake of 200 to 300 μg/day for adults which would lead to an absorption of 20 to 30 μg/day (WHO, 1977).

Increase in blood lead levels resulting from exposure to lead from air, drinking water, paint, dust, soil and food are summarized in Table 2-3. The minimum concentrations of lead in environmental sources believed to result in increases in blood lead level are given in Table 2-4.

Table 2-3. Increments in Blood Lead Level as a Function of Exposure Concentration

Medium	Incremental Exposure Concentration	Resultant Incremental Blood Lead Level	References
Air	1 $\mu g/m^3$	1 $\mu g/dL$	USEPA, 1972
	1 $\mu g/m^3$	1 $\mu g/dL$	Snee, 1981
Drinking Water	50 ppb	1 $\mu g/dL$	Houk et al., 1989
	1 ppb	0.06 $\mu g/dL$	Pocock et al., 1983 USEPA, 1986
	1 ppb	0.05 $\mu g/dL$	Elwood, 1984
Paint	16.8 $\mu g/Kg$ Body Weight	20 - 54 $\mu g/dL$	National Academy of Sciences, 1976
Soil	1000 $\mu g/g$	0.6 - 0.8 $\mu g/dL$	USEPA, 1983
	1000 $\mu g/g$	2 $\mu g/dL$ (adults) 4 $\mu g/dL$ (children)	Gallacher et al., 1984
	5000 $\mu g/g$	3 $\mu g/dL$	Elwood, 1986
	600 $\mu g/g$	$\leq$ 5 $\mu g/dL$	Madhavan et al., 1989
	1000 $\mu g/g$	0.6 $\mu g/dL$	Barltrop et al., 1975
Dust	1000 $\mu g/g$	1.9 $\mu g/dL$	Laxen et al., 1987
	1000 $\mu g/g$	1.8 $\mu g/dL$ (overall)	USEPA, Elwood, 1986
	1000 $\mu g/g$	5 $\mu g/dL$	Duggan, 1980
	1000 $\mu g/g$	4.0 $\mu g/dL$	Barltrop et al., 1975
Food	Vegetables from contaminated soil	3 $\mu g/dL$	Gallacher et al., 1984

Table 2-4. Minimum Concentration of Lead Causing Elevations in Blood Lead Level

Medium	Minimum Concentration	Reference
Air	2 $\mu g/m^3$	Yankel et al., 1977
Drinking Water	50 ppb	WHO (1984) USEPA (1977)
Paint	0.06% = 600 ppm*	Consumer Product Safety Commission (CPSC)
Soil	1000 ppm 600 ppm	Yankel et al., 1977 Madhavan et al., 1989
Dust	123 $\mu g/ft^2$ 1000 - 2000 $\mu g/g$ 114 $\mu g/ft^2$	Charney et al., 1980 Laxen et al., 1987 Sayre et al., 1974 Farfel and Chisolm, 1990

*The concentration of lead in paint associated with an elevation in blood lead level has not been established. The value of 0.06% was recommended by the CPSC as the highest allowable concentration of lead in paint formulations.

2.5 GOVERNMENTAL RECOMMENDATIONS

There is not a concentration for lead that is considered "safe" from an environmental standpoint. OSHA has regulations in place for lead in air and medical action levels, and the Centers for Disease Control (CDC) has defined an "elevated" blood lead level of 25 μg/dL for children. This level is currently under review. A number of states have regulations in place and/or have adopted "recommended levels" of lead in environmental media. For the most part, states are establishing recommended levels based on the Maryland and HUD guidelines, and to some extent on CDC findings.

2.5.1 Paint

Guidelines for threshold levels of lead-based paint have been established by HUD and by a number of states. These guidelines have been based primarily upon measurement limitations, a level of lead that is detectable by X-ray fluorescence. HUD has recommended that a level of 1.0 mg/cm^2 be considered "positive" for lead; the Centers for Disease Control has suggested a level of 0.7 mg/cm^2 as "positive." States have adopted guidelines accordingly. Federal and State permissible levels are presented in Tables 2-5 and 2-6.

Currently, the Consumer Product Safety Commission has established a limit of 0.06 percent by weight for lead in paint, compared to a previous limit of 0.5 percent. These levels correlate with concentrations of 600 and 5000 ppm for atomic absorption spectrometric (AAS) measurements. A number of states have accepted these values as limiting AAS concentrations. (See Table 2-6.)

Spot tests are recognized as a confirmation of lead in paint by Massachusetts. Massachusetts regulations establish a dangerous level of lead in paint as "a positive reaction with 6 to 8 percent sodium sulfide solution indicative of more that 0.5 percent lead by dry weight (Commonwealth of Massachusetts, 1990)."

2.5.2 Soil

At the present time there are no Federal regulations for hazardous levels of lead in soil. A number of states have adopted a level that CDC has considered protective, 500 - 1000 ppm. According to CDC (1985), "In general, lead in soil and dust appears to be responsible for blood lead levels in chil-

Table 2-5. Federal Guidelines for Lead Hazards

Concentration of Lead									
Agency	Paint		Soil	Dust			Drinking Water	Air	Blood
				Floor	Window Sill	Window Well			
ACGIH	NE	NE	NE	NE	NE	NE	NE	150 $\mu g/m^3$	NE
CDC	NE	NE	NE	NE	NE	NE	NE	NE	Children: 25 $\mu g/dL$
HUD	0.5% (w/w)	1.0 mg/cm^2	NE	200 $\mu g/ft^2$	500 $\mu g/ft^2$	800 $\mu g/ft^2$	NE	NE	NE
NIOSH	NE	NE	NE	NE	NE	NE	NE	100 $\mu g/m^3$	60 $\mu g/dL$
OSHA	NE	NE	NE	NE	NE	NE	NE	50 $\mu g/m^3$	50 $\mu g/dL$
USEPA	NE	NE	NE	NE	NE	NE	50 ppb	NE	NE
USCPSC	0.06% (w/w)	NE	NE	NE	NE	NE	NE	NE	NE

NE = Not Established

Table 2-6. State Guidelines for Lead Hazards

State	Concentration of Lead								
	Paint		Soil	Dust			Drinking Water	Air	Blood
				Floor	Window Sill	Window Well			
	AAS % w/w (dry)	XRF (mg/cm^2)	(ppm)	($\mu g/ft^2$)	($\mu g/ft^2$)	($\mu g/ft^2$)	(ppb)	($\mu g/m^3$)	($\mu g/dL$)
CA	0.5		1000	-----	-----	-----	50 ppb*		40 (tiered monitoring)
CT	0.5	revising	-----	-----	-----	-----			25
MA	0.06	1.2	1000	200	500	800	50		25 (PbB) w/EP = 35 $\mu g/dL$
MD	0.5	0.7 Confirm: 0.5 - 2.0	500	200	500	800			
MN	Before 1/1/90: 0.5 After 1/1/90: 0.06	1.0	500	———	500	———	50		
NC	0.5	1.0	500	200	500	800	50		Elevated: 25
NJ	1.0	1.0	250 (recommended)	200**	500**	800**			25
SC	0.06	0.7	2500 - 5000	———	1000	———			25
WI	0.5	1.0	-----	-----	-----	-----			Reported: 25

* Moving to 10 ppb
**Recommended only; not regulatory

dren increasing above background level when the concentration in the soil or dust exceeds 500-1000 ppm." Wisconsin and North Carolina evaluate each site individually with respect to location, proximity to children's play and other health hazards as an assessment for possible abatement.

The New Jersey Department of Health has established maximum permissible levels for lead in soil on the basis of the dose-response relationship of soil lead levels and blood lead levels in children (Madhavan et al., 1989). The guidelines are as follows:

1. A maximum permissible level of 250 ppm of lead in soil is recommended in areas without grass cover and repeatedly used by children below 5 years of age among whom mouthing objects is highly prevalent. This level may add at the most about 2 μg/dL to the blood level of children.

2. A maximum permissible level of 600 ppm of lead in soil is recommended in areas repeatedly used by children below 12 years of age. This level may add at the most 5 μg/dL to blood lead level of children.

3. A maximum permissible level of 1000 ppm of lead in soil is recommended in areas such as industrial parks or along streets and highways or in other areas infrequented by children. Although these areas are not expected to be places where children play, we do not feel that this can always be assured. Additionally, we are concerned about migration of lead off these sites on the footwear or clothes of adults.

EPA Superfund is developing a Biokinetic Uptake Model for lead in soil at Superfund sites. This model will allow sites to be evaluated for hazardous lead levels and subsequent abatement on a site-specific basis. Parameters for the model will include factors such as the geographic location of the site and the concentration of lead in the soil and water.

2.5.3 Dust

Studies (Sayre et al., 1974; Charney et al., 1980; Charney et al., 1983; Farfel and Chisolm, 1990) have shown that lead in dust in new, "lead-free," suburban homes to be in the range of 100 to 120 $\mu g/ft^2$. Maryland has used the data from these studies in conjunction with the practicality of abatement techniques to establish guidelines for clearance of surface dust after abatement. Massachusetts and North Carolina, as well as HUD, have adopted the Maryland guidelines of 200 $\mu g/ft^2$ for floor dust, 500 $\mu g/ft^2$ for window sill

dust, and 800 $\mu g/ft^2$ for dust in window wells. The differentiation between site specific dust levels is not only concentration dependent (window wells collect higher concentration of paint chips), but also a function of effectiveness of clearance. (Floor dust is easily cleared.)

As a rule, programs and regulations at the State level are at an early stage. A listing of guidelines for a number of states is given in Table 2-6.

SECTION 3
DETECTION METHODS FOR LEAD

3.1 INTRODUCTION

Even though the lead test kit is the primary focus of this document, the analytical performance criteria may also be used as guides in evaluation and/or development of other methods currently used for measurement including the atomic spectroscopic methods, atomic absorption spectroscopy and inductively coupled argon plasma emission spectrometry, and X-ray fluorescence.

3.2 ATOMIC ABSORPTION SPECTROSCOPY (AAS) AND INDUCTIVELY COUPLED ARGON PLASMA EMISSION SPECTROMETRY (ICP)

Lead in all sample types including paint, dust, soil, water, food and air can be measured in digested or dissolved form using AAS or ICP. Typical solution detection limits for lead are 0.5 μg/mL using flame atomic absorption (FAAS), 0.05 μg/mL using plasma emission spectrometry (ICP) and 0.001 μg/mL using graphite furnace atomic absorption spectrophotometry (GFAA). The lower levels of concern in paint (0.06%, 600 ppm) will not present a detection problem for any of these measurement techniques once the sample is solubilized. That is, a 100 mg paint sample at 600 ppm dissolved in 25 mL of solution will yield a >2 μg/mL solution, which is well above the detection limit for ICP and GFAA. Precision at these levels is better than $\pm$ 10%. The same is true for soil, but it is not true for dust because of the small quantities of dust that are typically collected. High-volume dust samplers are being tested by EPA at this time and, thus collection of sufficient dust for analysis should not be a problem in the future. The difficulty encountered is getting the sample into solution such that a representative solution is obtained for the measurement step. Acid digestion methods are recommended in the HUD Guidelines, though the efficacy of these with old paints has not been fully substantiated. It has been determined in the RTI laboratories that these acid extraction procedures leave some undissolved lead in the digestion residue. Digestion efficiencies for the recommended methods are being determined at this time.

3.3 X-RAY FLUORESCENCE

X-ray fluorescence is being used in the field and in the laboratory for analysis.

3.3.1 Portable XRF for Field Analysis

The portable XRF allows for measurement of lead in paint in the field. These devices utilize a radioactive source to provide the excitation X-rays; the lead fluorescent X-rays from the sample are detected with a solid-state, room temperature detector. The direct reading, lead-specific instruments such as the Warrington and Princeton Gamma-Tech have detection limits in the range of 0.3 - 0.5 mg/cm^2. The SCITEC instrument, which utilizes actual spectrum analysis and software-driven matrix correction, has a detection limit of approximately 0.1 mg/cm^2. The best estimate of precision of a measurement made with this latter instrument over wood or plaster is 0.3 mg/cm^2. (McKnight et al., 1990). The estimated systematic error of the procedure is 0.1 mg/cm^2 which indicates an overall confidence limit of $\pm$ 0.7 mg/cm^2. The precision of a result obtained using the SCITEC spectrum analyzer is expected to be about twice as good as that of the direct-reading Warrington or Princeton Gamma-Tech instruments, though it is expected that all of these instruments will be improved with time.

3.3.2 Laboratory XRF

The laboratory XRF is different from the portable XRF in several ways. The excitation X-rays are a result of bombardment of a metal cathode by a high voltage electron beam; the lead fluorescent X-rays produced by the sample are detected using a cryogenically-cooled solid state detector. The high intensity of the X-ray excitation beam and the sensitivity of the detector allow measurement of lead in dust and soil at concentrations as low as approximately 20 $\mu g/g$ (ppm).

3.4 SPOT TESTS

The spot test has great potential to serve as a complement to these other laboratory and field methods currently in use and described above. Spot tests are presently being used as a qualitative test for the "presence" of lead,

though the accuracy and reliability of the tests remain uncertain. Spot tests of adequate and known accuracy and reliability could be used for direct determination of lead in paint, either to confirm portable XRF results or to replace the XRF totally. Appropriately designed spot tests could also be used to measure lead in soil and dust and to measure lead in these same media after abatement or cleanup. Spot tests are especially attractive because they offer the potential of providing a means of performing the tens of thousands of onsite analyses required in the near future. These analyses cannot be performed by XRF because of the limited availability of such devices. Finally spot tests offer the potential of providing inexpensive, safe and reliable detection of lead by consumers. In general, limitations of the spot tests include the following:

- The technique is qualitative; therefore no standards for accuracy and precision are available.
- The presence of ions other than lead, for example, barium, can give rise to positive results, and thus, there is a "built in" false positive factor.
- Detection is by visual comparison of color changes; results are subjective, and may be inconsistent.
- Results for colored paints may be difficult to interpret. An observed darkening may be the result of wetting with the solution, rather than the formation of a colored precipitate.
- Detection in layers below the surface may be affected by the briskness of application and thus extraction. This effort may not be reproducible.
- Interpretation is often a function of available lighting.

These are two principle chemistries currently used for lead spot tests, reaction with sodium sulfide to form the dark colored or black lead sulfide precipitate, and reaction with rhodizonate to form a pink complex.

3.4.1 Sodium Sulfide

3.4.1.1 Detection --

In the sodium sulfide test, a drop of sodium sulfide is placed on exposed layers of paint. Layers that contain lead will turn gray or black as a lead

sulfide precipitate is formed. In a test of this method at the Civil Engineering Laboratory (Vind and Mathews, 1976) positive results for lead were observed at a minimum concentration of 0.5% (w/w). Interference from a few biocides and driers was noted (see 3.4.1.2 Selectivity) which gave rise to some "false positive" results.

The authors noted that even though the detection limit of the test was approximately the regulatory limit, 0.5% (w/w), detection of lead at this level in darker paints would not be possible.

Studies by McKnight et al. (1989) and Blackburn (1990) have shown inconsistencies in the detection limit of the spot test. Blackburn tested 377 paired paint chips. The concentration of lead for one chip in each pair was determined by atomic absorption spectroscopy (FAAS) and converted to mg/cm^2. The concentration of the other chip in the pair was determined using the spot test method. The author found variations in the color of the precipitates: black, gray, green, blue, brown, copper and orange. The observation of black of gray precipitates was correlated with 96 percent of the "positive results." Blackburn observed positive results (black coloration) to increase with lead concentration from 28.3 percent at a concentration of 0.7 - 0.9 mg/cm^2 to 80.4 percent at FAAS concentrations of $\geq$10.0 mg/cm^2. The frequency of negative results was found to be technician dependent. On wood substrates only, negative test results at 0.7 mg/cm^2 - 0.9 mg/cm^2 were 51.1 percent; whereas negative results at concentrations of $\geq$10.0 mg/cm^2 decreased to 20.5 percent.

Blackburn (1990) concluded that the overall false negative results on wood were 25.0 percent. This was inconsistent with the findings of McKnight et al. (1989) who estimated the false negative results of sodium sulfide spot tests to be about 10 percent. Blackburn questioned the statistical validity of the McKnight results on the basis of the the magnitude of the 95 percent confidence interval (0 - 23 percent) reported, and the background and training of the testers in the NIST study.

3.4.1.2 Selectivity --

A number of inorganic compounds contain metals whose sulfides are dark. A list is given in Table 3-1. Vind and Mathews (1976) and MRI studies (Midwest Research Institute, 1990) evaluated the formation of colored precipitates with sodium sulfide solution for inorganic materials having potential uses in

Table 3-1. Metallic Elements Having at Least One Black Sulfide.

Element	Colors of Sulfides	Some Uses of Compounds in Paints
Actinium	Black	None
Antimony	Black, Red	Pigment
Bismuth	Black, Brown, Gray	Pigment
Chromium	Black, Brown, Gray	Pigment, Corrosion Inhibitor
Cobalt	Black, Gray, Red	Pigment, Drier
Copper	Black	Biocidal Pigment
Iron	Black, Green, Yellow	Pigment
Lead	Black	Pigment, Drier, Corrosion Inhibitor
Manganese	Black, Green, Pink	Pigment, Drier
Mercury	Black, Red	Pigment, Biocide
Molybdenum	Black, Brown, Gray	Pigment, Corrosion Inhibitor
Nickel	Black, Gray, Yellow	Pigment
Silver	Black, Gray	None
Tin	Black, Gold, Gray	Gilding Agent

Source: Vind and Mathews (1976)

paint formulations (biocides or pigments). The authors observed positive results for mercuric oxide, mercuric iodide and phenylmercuric oleate, all used as biocides. Cobalt napthenate and manganese naphthenate, used as curing agents, also turned black with the application of sodium sulfide solution. Bismuth trioxide changed from greenish-white to light brown in the presence of the sodium sulfide solution.

3.4.2 Sodium Rhodizonate

Sodium rhodizonate forms a pink complex with lead in acidic solutions (Feigl and Suter, 1942). It may be used to detect lead in:

- paint,
- dust,
- soil,
- dilute solutions,
- the presence of interferent ions,
- ores and minerals,
- alloys, and
- pigments and glass.

The test is rapid and sensitive. In evaluation studies now being performed at the Research Triangle Institute, four commercially available rhodizonate-based kits have a positive reaction to lead ranging from about 0.5 μg Pb (absolute in solution) to 5 μg Pb, with the reproducibility being $\pm$ 0.05 μg to $\pm$ 0.5 μg, respectively. A final report of these and other evaluation results will be available in early 1991.

SECTION 4
PERFORMANCE CRITERIA FOR TEST KITS

As stated in Section 3, there is a great need for spot tests which may complement and/or take the place of measurements now performed with a portable XRF, and also measure lead in soil and dust before and after removal or cleanup. Because, as also noted in Section 3, there are many uncertainties in the performance of test kits currently available, criteria are needed which will serve as guides for improvement of existing kits and development of new kits. First to be considered are these performance criteria.

4.1 RELEVANT TEST KIT PERFORMANCE CRITERIA

There are a number of criteria which must be considered. These include accuracy, precision, selectivity, sensitivity, response time, safety, appearance, reproducibility, and stability.

4.1.1 Accuracy

As a rule, accuracy (bias) and precision are expressed for quantitative determinations. Accuracy is a comparison of an observed value to a "true" value usually determined from a reference standard. Because results of test kit determinations are qualitative, calculation of bias is not possible. In this case, an approximate expression of accuracy would be the ratio of a number of "correct" determinations, n', to the total number of measurements, using a standard whose concentration was detectable at a level previously determined by a quantitative method, i.e., AAS. For example, if a spot test was checked for N samples known to have a concentration of 1200 ppm, the accuracy, A, might be expressed as:

$$A = n'/N \times 100.$$

Another method for estimating accuracy of the method is by using duplicate real world samples. The concentration of one sample is determined by a quantitative method and lead in the second sample detected by the spot test. A negative or positive spot test result would be evaluated relative to the concentration determined quantitatively, with concentrations above the

abatement concentration considered as positive spot test results. Blackburn (1990) has used this approach to estimate the accuracy of the sodium sulfide method for a series of paint samples. His findings show that the accuracy of the method varies with the analyst and with the concentration of the lead in paint. He indicates that an average of approximately 25 percent of the results of the spot tests were inconsistent with the concentration of lead determined by AAS.

4.1.2 Precision

Precision for a quantitative method is usually expressed as the relative standard deviation for replicate analyses. For qualitative analyses, a mean value cannot be determined. Therefore, precision cannot be expressed in this way.

4.1.3 Selectivity

Selectivity is a measure of the responsiveness of the test kits to lead relative to their responsiveness to other materials present in paint, soil and dust. These have been investigated for both the sulfide and rhodizonate-based test kits.

Sodium Sulfide -- According to Vind and Mathews (1976) and MRI findings (Midwest Research Institute, 1990), a number of elements other than lead have a least one dark sulfide. These elements are potential interferents in the spot test results and would contribute to false positives. Copper, iron and zinc pigments in paint formulations are common interferents. Titanium dioxide, another common pigment, acts as an interferent in test results by masking color changes.

Sodium Rhodizonate -- A number of metals used as pigments and biocides in paint also produce complexes with sodium rhodizonate under neutral conditions. Examples are barium, mercury, copper, bismuth, and zinc. The presence of these ions would result in "false positive" results in neutral solution, but should have no effect under acidic conditions, the pH of choice for test kits. Chloride (Cl^-) and sulfate (SO_4^{2-}) anions will interfere with accurate detection of lead by competing with the rhodizonate for complexation of the lead. As a result of the competitive reactions, the detectable lead level appears lower than the actual lead level.

4.1.4 Sensitivity

Detection limits are critically important and should be specific to the intended purpose of the test kit, for example, testing for the need for abatement. The optimum criteria for spot test sensitivity is to establish a detection limit pertinent to the health effects associated with hazardous levels of lead in different media. While governmental agencies have addressed guidelines, recommendations, and regulations for abatement and clearance, the correlation between these recommended levels and NOAELs are unclear. For example, the HUD guidelines for abatement of lead in paint as determined by XRF (1.0 mg/cm^2) are based upon the instrumental sensitivity, i.e., confidence of detectability of lead by the technique. (CDC considers a value of 0.7 mg/cm^2 as positive for lead.)

Because there are risks associated with environmental exposure and exposure during abatement, it is desirable to detect lead at levels that are indicative of hazards. Optimally, detection criteria for all media - paint, soil, dust, water and ceramics - would ensure a 95 percent confidence level at concentrations determined to produce adverse health effects in vulnerable population groups.

In the case of soil lead, a Biokinetic Uptake Model is being developed to evaluate the lead levels at Superfund sites in order to determine levels requiring abatement at specific sites. The State of Maryland has developed guidelines for post-abatement clearance of dust lead by determining a concentration in dust that is both relevant to health effects and achievable to clear from a practical standpoint.

4.1.5 Response Time

The response time required for detection using the test kit and the time stability of the response are important to accuracy and reproducibility. These times should be well-suited to normal work operations and consistent from test to test for a particular kit.

4.1.6 Safety

Both consumers and trained technicians are potential users of test kits. Safety criteria are important considerations in both cases. In the case of

consumers, safety of use, child safety and disposal must be addressed, whereas for trained technicians, only handling and disposal criteria must be met.

4.1.7 Appearance

Statements should be made in the package insert about physical properties, such as appearance, which may or may not have any effect on the accuracy of detection. Variations in appearance may include the following:

- suspensions or discolorations in reagent solutions, and
- discoloration of test strips.

4.1.8 Reproducibility

Because the detection of lead using qualitative test kits is based upon color changes, the reproducibility of color is essential to the effectiveness of the method. It would be desirable to include references so that the user could compare color changes. Options for references include the following:

- standard solutions for lead at varying concentrations,
- blank solutions, or
- color chart for correlations between color intensity and lead concentration.

Reproducibility in color changes should be determined for variations in production lots. An option is to require the manufacturer to ensure colorimetric precision of $\pm$ 10 percent for solutions and test papers by quality assurance checks of production lots with standard reference materials.

4.1.9 Stability

Consideration must be given to stability of the detection kits. In the short term, the effects of temperature changes, exposure to UV light and air should be evaluated and minimized. Long term stability of the test kit materials may be designated as an expiration date.

4.2 PROPOSED TEST KIT PERFORMANCE CRITERIA

Consideration has been given to the levels of lead that yield adverse health effects, Federal and State regulations, and desired performance

criteria for test kits. On the basis of these considerations the following are proposed.

4.2.1 Sensitivity

The optimum criteria for test kit sensitivity is the detection of lead at the lowest concentration associated with adverse health effects; i.e., increases in blood lead levels. Criteria are proposed in Tables 4-1A, 4-1B and 4-1C for lead in soil, dust and paint.

The proposed test kit sensitivity for lead in soil and dust is more conservative than levels given in Table 2-4. Because of the expected lowering of the CDC "protective" levels from 500-1000 ppm to 300-500 ppm for lead in soil, a concentration range of 150 - 450 ppm is proposed for positive detection of lead in soil. Selection of sensitivity criteria for lead in dust is also based upon health effect findings. Because a concentration of 300 ppm in dust is considered to be clearly unacceptable (Chaney, 1990), detection at levels greater than 150 ppm, with 95% of results positive at 450 ppm is proposed. Dust loading levels greater than 75 $\mu g/ft^2$ are believed to be appropriate for detection with 95% of results positive at 225 $\mu g/ft^2$. The criteria are proposed on the basis of findings of loading in clean dwellings in the range of 4 to 130 $\mu g/ft^2$.

Because a quantitative relationship between lead-based paint and elevation of blood lead levels has not been established, criteria for paint are proposed for both abatement and clearance on the basis of guidelines already in existence. For abatement, levels considered positive from an instrumental standpoint have been used to propose measurement criteria. Concentrations of 0.7 mg/cm^2 (positive by CDC) and 1.0 mg/cm^2 (positive by HUD) are considered unacceptable (i.e., necessitate abatement) and should result in positive detection. A minimum level of approximately 1/5 of the CDC "positive" concentration, i.e., 0.1 mg/cm^2, is proposed as negative for lead.

Clearance standards were proposed on the basis of the following:

- the current ceiling level for lead in new paint, 600 ppm, regulated by the CPSC, and
- consideration of cleared paint as a dust source, i.e., a clearly positive concentration of 450 ppm.

Table 4-1A

PROPOSED ANALYSIS PERFORMANCE CRITERIA FOR
LEAD-IN-SOIL

Normal and/or Acceptable Lead Levels

Reference	Concentration
Madhaven et al., 1989	For Children <5 years: <250 ppm For Children >5, <12 years: <600 ppm
CDC	500 - 1000 ppm
Charney et al., 1980. (Rochester study)	mean: 1000 ppm medium: 633 ppm

Unacceptable Lead Levels

Yankel et al., 1977.	1000 ppm

Proposed Performance Criteria

Concentration Level of Concern: 300 ppm

95% of results positive $\geq$ 450 ppm
95% of results negative $\leq$ 150 ppm

Comments:

- CDC presently considers 500 - 1000 ppm "protective."
- CDC will change "protective" level to 300 - 500 ppm sometime in the future according to Chaney.
- EPA developing BioKinetic Uptake Model for lead in soil.

Table 4-1B

PROPOSED ANALYSIS PERFORMANCE CRITERIA
FOR LEAD-IN-DUST

Normal (clean) Lead Levels

Reference	Technique	Loading	Concentration
Clark et al., 1985 (Univ. of Cincinnati)	vacuuming	mean: 19 $\mu g/ft^2$ range: 4 - 111 $\mu g/ft^2$	350 ppm 192 - 1160 ppm
Chisolm, personal communication (Baltimore study)	wipe	mean: 20 - 30 $\mu g/ft^2$ max: 120 - 130 $\mu g/ft^2$	---
Sayre et al., 1974 (Rochester study)	wipe	mean: 27 $\mu g/ft^2$ max: 114 $\mu g/ft^2$	---
Charney et al., 1980 (Rochester study	wipe	mean: 123 $\mu g/ft^2$ medium: 55 $\mu g/ft^2$	---
Elias, personal communication	---	---	100 - 200 ppm
Chaney, personal communication	---	---	Rural: 30 - 100 ppm Urban: $\leq$ 300 ppm (acceptable)

Unacceptable Lead Levels

Reference	Technique	Loading	Concentration
Farfel, personal communication (Baltimore study)	wipe	$\geq$ 150 $\mu g/ft^2$	---
State of Maryland Guidelines	wipe	$\geq$ 200 $\mu g/ft^2$	---
Chaney, personal communication	---	---	$>$ 300 ppm, 1000 ppm is clearly unacceptable

Table 4-1B (continued)

Loading Level of Concern: 150 $\mu g/ft^2$

95% of results positive $\geq$ 225 $\mu g/ft^2$
95% of results negative $\leq$ 75 $\mu g/ft^2$

Concentration Level of Concern: 300 ppm

95% of results positive $\geq$ 450 ppm
95% of results negative $\leq$ 150 ppm

Comments:

- Loading (area concentration) is determined by wipe type kits or area vacuuming onto filter for direct analysis.
- Concentration (gravimetric) is determined by vacuuming techniques and extraction of weighed bulk or weighed filter samples.
- "The correlation of blood lead concentrations with lead loading (r=0.46) was much higher than for lead concentrations (r=0.21). For a given loading, the concentration could range from being high where there was very little dust and hence very little lead) to, conversely, low where there was a larger volume of dust (and hence much available lead)." Davies et al., 1990.
- Loading is a more appropriate means of indicating the presence of lead. Reductions in amounts of dust, i.e., improved housekeeping, result in decreases in loading, yet concentration of lead in dust may remain unchanged: J. Chisolm, personal communication.
- "In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background level when the concentration in the soil or dust exceeds 500 - 1000 ppm." CDC, 1985.
- CDC "Levels of Concern" may be lowered in the future: R. Chaney, personal communication.
- "Dust lead concentration is a more useful predictor of blood lead than lead loading...Lead concentration is thus the more useful measure of exposure, since there is no standardized way to measure lead loading, preventing comparison between studies." Laxen et al., 1987.

Table 4-1C

PROPOSED ANALYSIS PERFORMANCE CRITERIA
FOR LEAD-IN-PAINT

Standards for New/Replacement Paint

Reference	Concentrations
CPSC, FDA	600 ppm, 0.12 mg/cm^2

Proposed Performance Criteria

Concentration Level of Concern: 0.06% (w/w), 600 ppm

95% of results positive $\geq$ 0.045% (w/w), 450 ppm
95% of results negative $\leq$ 0.015% (w/w), 150 ppm

Standards of Abatement

Reference	Concentration
HUD	1.0 mg/cm^2
State of Maryland	0.7 mg/cm^2

Proposed Performance Criteria

Concentration Level of Concern: 0.7 mg/cm^2

95% of results positive $\geq$ 1.0 mg/cm^2
95% of results negative $\leq$ 0.1 mg/cm^2

Comments:

- No quantitative relationship between lead level in paint and health effects has been established.
- With pica activities, difficulty arises in transforming XRF values to average daily intake.
- HUD considers 1.0 mg/cm^2 (5000 ppm) a "positive" XRF measurement for lead and requires abatement at this concentration.
- CDC considers 0.7 mg Pb/cm^2 paint a "positive" XRF measurement for lead.
- The CPSC level of concern for new paint is 0.06% (600 ppm). This level may be decreased to 100 ppm in the future.

Accordingly, clearance performance criteria are proposed to show 95% positive results at 450 ppm and 95% negative results at 150 ppm.

Results of evaluations of test kits have shown that a three-fold range from clearly negative to clearly positive results is achievable for total lead in solution (Research Triangle Institute, 1990). Test kit sensitivity is limited by the ability to extract lead from the medium.

4.2.2 Selectivity

The test kits shall be selective for lead over potential interferences. Through selection of the primary color-forming reagent, use of chemical agents to mask interferences and other chemical parameters such as pH, the selectivity ratio for lead to any other potential interferences shall be 100 to 1.

4.2.3 Accuracy

Test kits on the market were shown to have poor accuracy. Results were found to depend on the ability to extract lead from the matrix, a function of the lead species and the physical form of the matrix, rather than the concentration of lead in the matrix.

Criteria for accuracy, 95 percent of the results positive at a specified sensitivity, are believed to be achievable for concentrations proposed if test kit solutions extract lead quantitatively.

4.2.4 Response Time

The test kits shall develop full color or change within 30 seconds and be stable for a minimum of one (1) hour.

4.2.5 Safety

Hazard evaluations of materials, i.e., sodium rhodizonate, should be carried out. Information on dermal effects, toxicity, etc. shall be indicated, if necessary, on enclosures similar to package inserts for medications or Material Safety Data Sheets for chemicals. Precautions and personal protection, i.e., gloves, shall be included if special handling needs are required.

The use of fracture- or splatter-resistant containers is important. The design of containers is particularly important when kits contain solutions. Special considerations for child safety, such as child-proof containers and vials, must be given to kits used by homeowners. Testing solutions, strips, etc. shall be sealed so that they are inaccessible to children.

Disposal instructions for solutions, paper strips, test ware (vials, cups, wands, etc.) shall be included in the test kit. Options, including flushing into the sanitary sewer or wrapping in newspaper for disposal in a landfill, shall be specified.

4.2.6 Appearance

Warnings shall be included in the test kit about physical properties which may affect accuracy and reproducibility of the test kit, including change in color or reagents, precipitates, etc.

4.2.7 Reproducibility

The test kits shall include some reference device or material to assure the reproducibility of the test kit. Options for this materials include:

- A standard test solution or lead-impregnated strip
- A color chart or wheel

Reproducibility shall be $\pm$ 10 percent between individual test kits and between production lots.

4.2.8 Stability

Test kits shall be labeled with a production lot number and an expiration date. Test kits shall have a shelf life of a minimum of six (6) months.

SECTION 5
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SECTION 6
LIST OF CONTACTS

The following list includes names of professionals involved in environmental lead programs. Names of persons who were contacted and responded are given in Table 6-1; attempts were made to reach those listed in Table 6-2. Potential contacts are given in Table 6-3.

Table 6-1. Telephone Contacts

Name	Address	Telephone Number
Beale, Allison Environmental Technology and Water Advisor	University of CA Cooperative Extension	(916) 366-2013
Berg, Marlene USEPA	Toxics Integration Branch U.S. EPA, OS-230 401 M. Street, SW Washington, DC 20460	(202) 475-9493
Binder, Suzanne, MD CDC	Centers for Disease Control Atlanta, GA 30333	(404) 488-4880
Bolden, Verdell Program Manager State of CT	Department of Health Services Maternal and Child Health Section 150 Washington Street Hartford, CT 06106	(203) 566-3186
Chisolm, Julian, MD Kennedy Institute	Kennedy Institute for Handicapped Children Johns Hopkins University 707 N. Broadway Baltimore, MD 21205	(301) 550-9035
Eberle, Sandra Program Manager Chemical Hazards	U.S. Consumer Product Safety Commission Bethesda, MD 20207	(301) 492-6550

Table 6-1. (continued)

Name	Address	Telephone Number
Farfel, Mark Sc.D. Kennedy Institute	Kennedy Institute for Handicapped Children 707 N. Broadway Baltimore, MD 21205	(301) 955-3864
Goldman, Lynn MD State of CA	Department of Health Services 2151 Berkeley Way, Room 515 Berkeley, CA 94704	(415) 526-6693
Guyaux, Susan State of MD	Coordinator, Enviromental Program Lead Poisoning Prevention Division 2500 Broening Highway Baltimore, MD 21224	(301) 631-3859
Hunter, Paul State of MA	Department of Public Health Childhood Lead Poisoning Prevention Program State Laboratory Institute 305 South Street Jamaica Plain, MA 02130	(617) 522-3700 Ext. 187
Marcus, Allan, PhD Battelle	Battelle Applied Statistics P. O. Box 13758 Research Triangle Park, NC 27709	(919) 549-8970
Jesneck, Charlotte State of NC	Department of Enviromental Health and Natural Resources Divison of Solid Waste Mangement Superfund Section 401 Oberlin Road, P. O. Box 27687 Raleigh, NC 27611	(919) 733-2801

Table 6-1. (continued)

Name	Address	Telephone Number
McCreary, Charlotte RN, MPH State of SC	Division of Children's Health Department of Health and Environmental Control 2600 Bull Street Columbia, SC 29201	(803) 737-4054
McNutt, Sam State of SC	Department of Health and Environmental Control Bureau of Environmental Health 2600 Bull Street Columbia, SC 29201	(803) 737-5072
Miller, Colleen MT, ASCP State of NC	Department of Environmental Health and Natural Resources Enviromental Epidemiology Section P. O. Box 27687 Raleigh, NC 27611	(919) 733-3410
Murphy, Nancy, RN State of NJ	Department of Health Accident Prevention and Poison Control Program CN 363 Trenton, NJ 08625	(609) 292-5666
Papanek, Paul MD State of CA	Toxics Epidemiology Program 2615 South Grand Avenue, Sixth Floor Los Angeles, CA 90007	(213) 744-3235

Table 6-1. (continued)

Name	Address	Telephone Number
Schiffman, Carole USFDA	Consumer Affairs US Food and Drug Administration	(202) 245-1317
Schirmer, Joe State of WI	Department of Health and Social Services Division of Health ECDE-DOH P. O. Box 309 Madison, WI 53701	(608) 266-2670
Sides, Steve Director of Health and Safety	National Paint and Coatings Assoc. 1500 Rhode Island AVe., NW Washington, DC 20005	(202) 462-6272
Van Benthysen, Gene State of NJ	Department of Health Accident Prevention and Poison Control Program CN 363 Trenton, NJ 08625	(609) 292-5666

Table 6-2. Contacts Attempted

Name	Address	Telephone Number
Miele, Mary State of NY	Health Department Childhood Lead Poisoning Prevention Program Corning Tower, Room 7880 Albany, NY 12237	(518) 474-2749
Turner, Martha State of NH	Department of Health and Human Services Childhood Lead Poisoning Prevention Program Division of Public Health Services Six Hazen Drive Concord, NH 03301	(603) 271-4507

Table 6-3. Potential Contacts

Name	Address	Telephone Number
Crosby, Lee State of NC	Department of Enviromental Health and Natural Resources Division of Solid Waste Management Section Chief, Superfund Section 401 Oberlin Road P. O. Box 27687 Raleigh, NC 27611	
McClanahan, Mark ATSDR	Agency for Toxic Substances and Disease Registry (ATSDR)	(404) 488-4100
Petrosivich, Chuck ATSDR	ATSDR	(404) 488-4100
Simpson, Jim CDC	Centers for Disease Control Center for Enviromental Health Blood Lead Proficiency Testing Childhood Lead Program Koger Center F-37 1600 Clifton Road Atlanta, GA 30338	(404) 488-4780