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REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD:

ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION

OAQPS DRAFT STAFF PAPER

Strategies and Air Standards Division
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency
Research Triangle Park, N.C. 27711

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REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD: ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION

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I. PURPOSE

This paper evaluates and interprets the most relevant scientific and technical information reviewed in the draft EPA document "Air Quality Criteria for Lead" (EPA, 1984) in order to better specify the critical elements that EPA staff believes should be considered in the possible revision of the primary and secondary National Ambient Air Quality Standards (NAAQS) for lead. This assessment is intended to help bridge the gap between the scientific review contained in the criteria document and the judgments required of the Administrator in setting ambient standards for lead. As such, particular emphasis is placed on identifying those conclusions and uncertainties in the available scientific literature that the staff believes should be considered in selecting the averaging times, forms, and levels for the primary and secondary standards. While the paper should be of use to all parties interested in the standards review, it is written for those decision makers, scientists, and staff who have some familiarity with the technical discussions contained in the criteria document (hereafter referenced as "CD").

II. BACKGROUND

Since 1970 the Clean Air Act, as amended, has provided authority and guidance for the listing of certain ambient air pollutants that may endanger public health or welfare and the setting and revising of NAAQS for those pollutants. Primary standards must be based on health effects

criteria and provide an adequate margin of safety to ensure protection of public health. As several recent judicial decisions have made clear, the economic and technological feasibility of attaining primary standards are not to be considered in setting them, although such factors may be considered to a degree in the development of state plans to implement the standards (D.C. Cir., 1980, 1981). Further guidance provided in the legislative history of the Act indicates that the standards should be set at "the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population." Also, margins of safety are to be provided such that the standards will afford "a reasonable degree of protection . . . against hazards which research has not yet identified" (Committee on Public Works, 1974). In the final analysis, the EPA Administrator must make a policy decision in setting the primary standard, based on his judgment regarding the implications of all the health effects evidence and on the requirement that an adequate margin of safety be provided.

Secondary ambient air quality standards must be adequate to protect the public welfare from any known or anticipated adverse effects associated with the presence of a listed ambient air pollutant. Welfare effects, which are defined in section 302(h) of the Act, include effects on vegetation, visibility, water, crops, man-made materials, animals, economic values and personal comfort and well-being. In specifying a level or levels for secondary standards the Administrator must determine at which point the effects become "adverse" and base his judgment on the welfare effects criteria.

The current primary standard for lead (to protect public health) is 1.5 micrograms per cubic meter ($\mu g/m^3$), maximum arithmetic mean

averaged over a calendar quarter. The current secondary standard for lead (to protect public welfare) is identical to the primary standard. For both primary and secondary standards, lead and its compounds are measured as elemental lead and collected using a high volume air sampler.

In addition to the lead NAAQS, several other federal regulations have been adopted in order to control human exposures to lead. These include EPA's schedule for the phased reduction of the allowable lead content in gasoline and the national primary drinking water standard for lead (50 µg/liter), as well as the Department of Housing and Urban Development's goals to eliminate lead-based paint hazards in federally-funded housing, the Food and Drug Administration's regulations on the lead content of foods and ceramic products, the Consumer Product Safety Commission's limit on lead content in paints, toys, and furniture, and the Occupational Safety and Health Administration's standards for occupational exposure to lead. In addition, the Centers for Disease Control have established criteria for health classification of children screened by lead poisoning prevention programs.

III. APPROACH

The approach used in this paper is to assess and integrate information derived from the criteria review in the context of those critical elements that the staff believes should be considered in the review of the primary and secondary standards. Particular attention is drawn to those judgments that must be based on the careful interpretation of incomplete or uncertain evidence. In such instances, the paper states the staff's evaluation of the evidence as it relates to a specific judgment, sets forth appropriate alternatives that should be considered, and recommends a course of action.

The principal focus of this paper is on the effects of inorganic lead either airborne or deposited from the air onto dusts, soils, vegetation, food, and water. The impact that lead from non-atmospheric sources, such as paint and solder, has on human exposure is also considered. The effects of organic lead vapors which are not commonly found in the atmosphere (see Section IV.A) will not be addressed.

Section IV presents relevant features of human exposure to atmospheric and non-atmospheric sources of lead through various pathways. Section V presents different approaches to estimate the impact of alternative air lead levels on lead body burdens as indicated by blood lead levels. Section VI addresses other essential elements with regard to the primary standards; these include the following:

1) identification of possible mechanisms of toxicity;

2) description of health effects and their relation to exposure levels; and

3) identification of the most sensitive population groups;

Drawing from the analyses in Sections IV, V, and VI of the criteria document information, and the associated appendices, Section VII identifies and assesses the factors the staff believes should be considered in selecting an averaging time, form, and level of the primary standard. Selecting an appropriate standard level includes a determination of an acceptable blood lead level and the impact a given air standard would have on the distribution of blood lead levels in the population of concern. Preliminary staff conclusions regarding alternative policy options in each of these areas are also presented.

Section VIII contains an examination of information in the criteria document the staff believes is most relevant with respect to the secondary standard. This discussion includes:

1) identification of the levels at which lead-induced changes in terrestrial and aquatic ecosystems have been demonstrated;

2) assessment of the available data base (laboratory and field studies) to identify quantitative relationships necessary for the prediction of environmental impact; and

3) preliminary staff recommendations concerning a secondary standard.

IV. LEAD EXPOSURE: MULTIMEDIA CONSIDERATIONS

In order to assess the risks associated with alternate lead NAAQS, it is necessary to understand the influence exerted by atmospheric lead on the total lead exposure of the population(s) of concern through the various exposure pathways. Relevant data on these pathways and the relationships between air lead and lead in other media are summarized in this section. Section V presents methodologies to estimate blood lead levels associated with alternative air lead levels.

The sources and pathways of human lead exposure are diagrammed in Figure 4-1 and typical levels of lead in different media to which U.S. populations are exposed are presented in Table 4-1. Between 85-90% of airborne lead in the U.S. originates from gasoline combustion with the remainder from stationary industrial processes such as primary and secondary lead smelting, battery plants, lead alkyl production, iron and steel production, ore pulverizing, copper smelting, and combustion of oil, coal, and waste oil. Atmospheric emissions can influence human exposure through direct inhalation of lead-containing particles and through ingestion of lead which deposits onto soil, dusts, vegetation, and other environmental surfaces. This deposition can occur by either or both of two mechanisms: 1) continuous dry deposition due to various mechanisms which may include gravitational settling or wind-related deposition; and 2) episodic wet depostion due to either washout of coarse mode particles (>2.5 micrometers in diameter) which are found mainly near emission sources, or the "rainout" (precipitation scavenging) of smaller particles which are more widely dispersed. Human exposure to lead can also be traced to lead in paint pigments and solder in canned foods and plumbing.

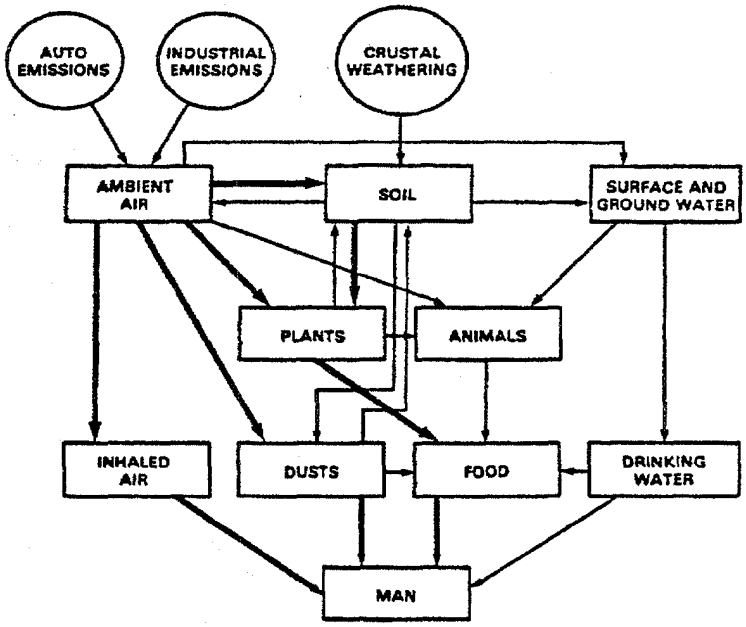

Figure 4-1. Principal pathways of human exposure to lead. Heavy arrows are those pathways discussed in greatest detail. From CD, Figure 7-1.

Table 4-1. Typical Lead Concentrations in Various Exposure Media

Medium	Rural Area	Urban Area	Near Point Source(s)[a]	References
Ambient Air ($\mu g/m^3$)[b]	0.01 - 0.3	0.03 - 4.4	0.2 - 10.2	Tyler, 1984; Yarn, 1984
Indoor Air ($\mu g/m^3$)[c]	0.003 - 0.2	0.01 - 0.4	0.07 - 3.1	Yocum, 1982; Cohen and Cohen, 1980; Rabinowitz et al., 1984b
Soil (ppm)	5 - 30	30 - 4500	150 - 15,000	CD, Table 7-11; Mielke et al.; 1983; See Tables B-1 and B-2
Street Dust (ppm)[d]	80 - 130 (90)	100 - 5,000 (1500)	(25,000)	Nriagu, 1978; CD, Table 7-26
House Dust (ppm)[d]	50 - 500 (300)	50 - 3,000 (1000)	70 - 100,000 (25,000)	U.S. EPA, 1977; Landrigan et al., 1975 Angle and McIntire (1979)
Typical Foods (ppm)	0.002 - 0.98	Same	Same	CD, Table 70-1
Water ($\mu g/l$)	<1 - 180 $\mu g/l$	Same	Same	Sharrett et al., 1982; EPA, 1985a
Paint[e]	<1 - >5 mg/cm^2	Same	Same	Billick and Gray, 1978

[a]Within 2-5 km of sources including primary and secondary lead smelters, battery plants.

[b]Represents quarterly averages monitored between July 1982 and July 1984.

[c]Range of indoor/outdoor ratios used (0.3 - 0.8) from Yocum, 1982 except near point sources where large particles predominate and infiltration into homes is low, ratio appears to be closer to 0.3 (Cohen and Cohen, 1980).

[d]Values in parentheses represent estimates provided in CD (Tables 7-24 and 7-26) as typical averages.

[e]Since there may be several layers of lead-based paint on a given surface, absolute concentration of lead is less useful than mg/cm^2. Surveys by HUD in Pittsburgh showed that more than 70% of pre-1940 dwelling units and 20% of post-1960 units had at least one surface with more than 1.5 mg/cm^2 lead paint (NAS, 1980).

A. Airborne Lead

1. Physical, Chemical and Spatial Characteristics

In general, about 50% of automotive lead emissions deposits within a few hundred meters of major roadways (Daines et al., 1970; Huntzicker et al., 1975; Ingalls and Garbe, 1982) while the remaining particles are small enough to remain airborne and travel hundreds or thousands of kilometers. This likely accounts for the surface contamination of polar glaciers, oceans, and other remote locations around the globe (Murozomi et al., 1969; Duce et al., 1975; Davidson et al., 1981b,c, 1982; Settle and Patterson, 1982). In general, U.S. urban and rural airborne lead particles have a mass median aerodynamic diameter* (MMAD) consistently between 0.3 and 0.7 micrometers (μm) with most of the mass associated with submicron particles, although a distinct peak is seen in the upper end of many of the particle size distributions between 5 and 10 μm (Davidson and Osborn, 1984).

Inorganic lead is emitted from automobiles as lead halides, hydroxides, and oxides and reacts with atmospheric ammonia and acid sulfates to form principally lead sulfate (e.g., $[NH_4]_2\ SO_4 \cdot PbSO_4$) with minor amounts of lead carbonates and halides remaining (Habibi, 1970; Ter Haar and Bayard, 1971; Dzubay and Stevens, 1973; Biggins and Harrison, 1978, 1979). Small amounts of lead additives used in gasoline (tetraethyl- and tetramethyl-lead) may escape to the atmosphere by evaporation from fuel systems or storage facilities. Relatively low concentrations of these organic lead compounds have been typically found in atmospheric samples (1 - 6% of total lead) except in special situations such as gasoline stations or garages (Skogerboe, 1975; Harrison et al., 1979). Because these organic lead compounds decompose by

*MMAD is used as an indicator of particle size of a lognormally distributed aerosol such that half the mass lies on either side of the MMAD.

photoreaction (Huntzicker et al., 1975) and are readily adsorbed onto atmospheric particles (Edwards et al., 1975), their presence in the atmosphere is transitory. Therefore, any health hazards associated with organic lead exposure are most likely to occur in an occupational setting and will not be assessed here.

In contrast to automobile exhaust, atmospheric lead emissions from industrial plants processing lead and its products contribute little to the overall pollution load across large, regional areas although fallout from these sources can be severe on a local scale. The high concentration levels which are found around lead smelters in particular result mainly from fugitive emissions predominately made up of large (>7 μm) lead particles resulting from materials handling, furnace upsets, and furnace charging and tapping operations (Landrigan et al., 1975; Jennett et al., 1977; GCA, 1984). Beyond the immediate area (0.25-1 km) of lead stationary sources, process emissions from stacks, predominately as lead sulfates and oxides with a size range between 2 and 10 μm, become the major source of lead in soils and air near these sources (Dorn et al., 1976; Roels et al., 1980; Davidson and Osborn, 1984).

2. Ambient Concentrations

As indicated in the CD (Section 7.2.1), lead levels in urban areas and near point sources have been markedly reduced since 1977 by the use of unleaded gasoline in new cars equipped with catalytic converters, the lead-in-gasoline phasedown program, and steady reductions in emissions from all types of industrial and commercial sources in compliance with the 1978 lead NAAQS. Recent (1980-1983) air quality data for 414 stationary source sites, micro-scale roadside sites, middle scale roadside sites, neighborhood scale roadside sites, and other non-classified sites are summarized in Table 4-2 (Battye, 1985). The monitoring sites located near stationary sources

Table 4-2. FREQUENCY DISTRIBUTIONS OF MAXIMUM QUARTERLY LEAD CONCENTRATIONS BY TYPE OF SITE[a]

Site type/time frame	Percent of site years in concentration ranges[b] <0.5	0.5-1.0	1.0-1.5	1.5-2.0	>2.0	Mean	Total site-years	Total number of sites
1980 through 1983								
All monitors[c]	69	22	5	2	2	0.53	1189	414
Stationary source	62	16	8	3	11	0.95	159	56
Microscale roadside[d]	17	40	27	13	3	0.99	30	18
Middle scale	39	42	18	0	0	0.68	33	15
Neighborhood scale	61	32	5	0	2	0.51	62	29
1983 only								
All monitors[c]	71	19	5	1	3	0.51	360	360
Stationary source	53	24	9	2	11	0.88	45	45
Microscale roadside[d]	16	47	32	5	0	0.83	19	19
Middle scale	53	33	13	0	0	0.60	15	15
Neighborhood scale	72	28	0	0	0	0.40	29	29

[a]Data are from the SAROAD system and represent the numbers of site-years for which the maximum quarterly concentrations fall within the designated concentration ranges. To be included, a site-year must have four valid quarters of data.

[b]Concentration ranges are in units of $\mu g/m^3$.

[c]Includes sites previously classified into categories (e.g., urban) that do not meet any of the current definitions.

[d]Microscale sites are within 5-15 meters from a major roadway and 2-7 meters above the ground.

Source:Battye, 1984

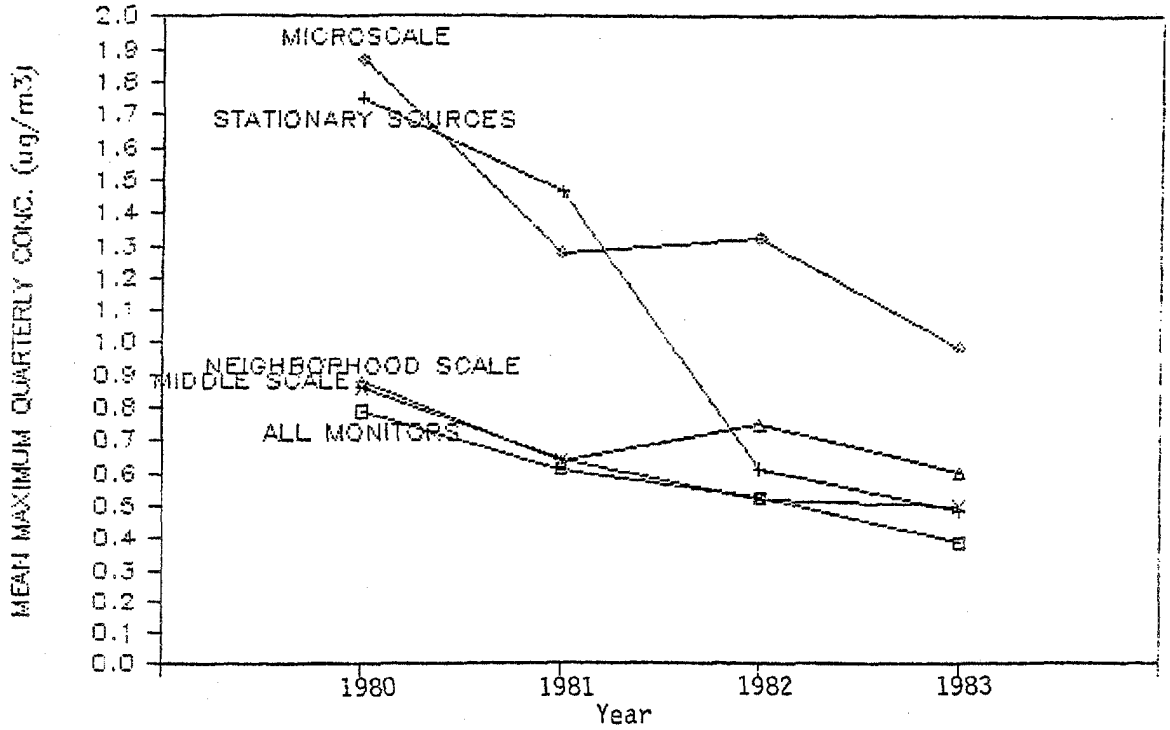

Figure 4-2. Trends in maximum quarterly lead concentration for various monitor types. Source: Battye, 1984

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and major roadways (i.e., microscale) reported the majority of concentrations over 1.5 $\mu g/m^3$. Only one population-oriented or neighborhood site had a quarterly average greater than 1.5 $\mu g/m^3$ between 1980 and 1983, whereas 14% and 16% of quarterly averages in source-oriented and microscale site-years, respectively, exceeded this value.

Trends in maximum quarterly averages between 1980 and 1983 are illustrated in Figure 4-2. Because of recent changes in lead monitor siting guidelines, the lead monitor network currently is being expanded and changed. In order to avoid biases relating to changes in the monitoring network, the trends analysis included only sites where valid data were available for three of the four years studied (Battye, 1985a). It should be noted, therefore, that these trends data, with the exception of those for "all" monitoring sites which include currently unclassified site-types, are based on a limited number of available monitoring sites that remained in service since 1980 (e.g., 27 stationary source sites, 9 neighborhood scale sites). Despite the new siting guidelines implemented in 1982 that added sites closer to traffic emissions, microscale readings have been declining, corresponding to the phasedown of the lead content in gasoline and reductions in leaded gasoline usage due to the gradual phase-out of older lead-burning cars. Similarly, annual and maximum quarterly average lead levels near point sources have also decreased since 1980, partly due to increased controls by lead-emitting sources in compliance with state implementation plans as well as to decreased industrial production at lead-emitting facilities (Silvasi, 1985).

B. Lead in Soil

The natural occurrence of lead in the earth's crust averages 5-50 µg lead/g soil [one µg/g is equivalent to one part per million or 1 ppm] lead in various soils (Shacklette et al., 1971; McKeague and Wolynetz, 1980). Much of the lead in the atmosphere deposits on terrestrial surfaces

where it is retained in organic complexes near the soil surface (CD, p. 7-28). The ability of soil to immobilize lead largely depends on soil pH and organic content (i.e., fulvic and humic substances). Many U.S. soils appear to have a large capacity to bind lead with only a small fraction dissolved in soil moisture where it is available for plant uptake (see Section VIII). The environmental impact of lead on the biota of terrestrial and aquatic ecosystems is discussed in Section VIII.

The upper layer of roadside soils may contain atmospheric lead from 30 to 2000 ppm in excess of natural levels within 25 meters of the roadbed beyond which concentrations decline abruptly in relation to traffic density and vehicle speed (Page and Ganje, 1970; Quarles et al, 1974; Wheeler and Rolfe, 1979; Pierson and Brachaczek, 1976). In contrast, soil lead concentrations around various lead point sources generally decrease exponentially within a 3-10 km radius from 100-60,000 ppm down to background levels (CD, Appendix 7-C). However, elevated soil lead levels have even been found at distances exceeding 20-25 km from some smelters (Wixson, 1978). Limited data indicate that lead in soil near primary and secondary lead smelters occurs as lead oxides, sulfide, sulfate, and elemental lead (Olson and Skogerboe, 1975; Corrin and Natusch, 1977).

Urban soils are contaminated by lead from a combination of automotive and point source deposition as well as from paint chips from indoor and outdoor surfaces. Elevated soil concentrations (as high as 2000 ppm) have been found within 10 feet of wood frame houses painted with lead-based paint (Ter Haar and Aronow, 1974). Accumulations of lead in soils (and dusts) around brick or stone structures have also been found, and can be partially attributed to washoff lead collected on roofs, ledges, and exterior walls (Wheeler and Rolfe, 1979). Vegetable garden soil samples collected

in a 30-mile radius of downtown Baltimore had lead levels up to 10,900 ppm with a median value of 100 ppm (Mielke et al., 1983). Much of this lead was attributed to automotive exhaust and industrial emissions because of the near absence of lead painted houses and the clustered pattern of lead levels around downtown. Soil lead concentrations in a sample of city parks have also been reported to be quite high, ranging from 200 to 3300 ppm (Chow et al., 1975; Zimdahl and Hassett, 1977).

The contribution that lead in soil can make to total lead exposure under alternative air lead levels is addressed in the modeling approaches presented in Section V.

C. Lead in Dust

Dust is a normal component of the home (i.e., "house dust") as well as the outdoor environment where it can be found on sidewalks, playgrounds, driveways, and other hard surfaces. Anthropogenic materials deposited on these outdoor surfaces will be referred to as "street dusts". In addition, the very top layer of soils (including leaf litter) to which people, particlarly children, come in direct contact, are considered in the criteria document to be "soil dusts".

Both house dust and street/soil dust contain lead from atmospheric deposition, "natural" soil, and paint chips. As with roadside soil, significant correlations between lead concentrations in street dust and the proximity and density of traffic have been found (Rolfe et al., 1977; Lau and Wong, 1982). As summarized by Nriagu (1978), street dusts from different U.S. cities contained between 300 and 18,000 ppm lead. A survey of street dusts in 77 midwestern cities showed an average lead content of 1,636 ppm in residential neighborhoods and 2,413 ppm in commercial and industrial areas (NAS, 1980). Limited chemical analyses of roadside soils and dusts from

U.S. cities found lead predominately as sulfate, along with minor amounts of oxide and halide salts (Olson and Skogerboe, 1975; Corrin and Natusch, 1977). Gutter debris in Dearborn, Michigan contained mostly large lead particles ranging between 40 and 1000 μm (Pierson and Brachaczek, 1976).

Airborne lead deposited on streets, sidewalks and driveways is subject to further distribution by wind and water. Windblown particles associated with dust are apt to be redeposited within the urban environment because of the complex wind currents caused by buildings and street canyons. There is somewhat conflicting evidence on the persistence of lead in street dust. Laxen and Harrison (1977) found that only a light rainfall (2 to 3 mm) is sufficient to remove 90 percent of the lead from the road surface, mainly to surrounding soil and to waterways. A survey of rainwashed areas in the U.K. and New Zealand concluded, however, that the acidity of rain (pH between 4 and 5) was insufficient to completely dissolve and transport lead particles, and that residue near streets pose a health hazard to children who are prone to ingest street dusts (Day et al., 1979). The rate of removal of lead from dusts is also dependent on the frequency and efficiency of street cleaning operations. In the absence of empirical data regarding this mechanism, it is reasonable to assume that lead concentrations in urban street, curb, and sidewalk dust will rise between precipitation and street cleaning episodes, and drop thereafter.

Lead levels in house dust can be expected to vary considerably depending on house cleaning practices, the presence and condition of lead-based paint, the presence of cigarette smoke, the amount of dust and soil blown into or carried into the house on clothing and shoes, (especially on those occupationally exposed to lead), indoor sources of lead other than paint (e.g., soldering), the permeability of the home to outdoor air (which can

vary with season), and the outdoor concentrations of air lead. Surveys of a diverse set of homes indicate a wide range of house dust lead levels between 18 and 16,000 ppm (EPA, 1977; Harrison, 1979; Angle and McIntire, 1979) and as high as 100,000 ppm within 2 km of smelter (Landrigan et al., 1975).

It is well established that children who play in dust and soil, especially in urban areas and on sites polluted by long-term fallout from industrial emissions, ingest lead in these media through normal mouthing behavior. Significant correlations have been found between blood lead levels and lead in soil, household and street dusts, and on children's hands (Lepow et al., 1975; Sayre et al., 1974; Angle and McIntire, 1982; Quah et al., 1982; Brunekreef et al., 1983).

In order to assess the impact of atmospheric lead on children's total exposure, it is necessary to estimate the contributions of different air lead levels to outdoor and indoor soil/dust lead levels as well as the amount of dirt a child may ingest, both inadvertantly and deliberately. These issues are addressed in the modeling approaches presented in Section V.

D. Lead in the Diet

The ingestion of food appears to be a major component of most individuals' total lead uptake, although the exact amount is a function of the size and type of diet. The occurrence of lead in the diet may be a result of a) natural sources of lead; b) deposition of airborne lead particles onto vegetation, soils, and water; and c) the harvesting, processing, transportation, packaging, preparation, and storage of food during which lead can be introduced at every stage either by atmospheric deposition or through metallic contamination, particularly from solder.

As would be expected from the differences in the handling of various

foods, lead contents vary considerably, ranging from 0.002 to 0.98 ppm (CD, Table 7D-1). Lead in unprocessed foods such as fresh fruits and vegetables comes from atmospheric deposition on vegetation surfaces and uptake from soil through roots (Schuck and Locke, 1970; Motto et al., 1970). The potential for lead uptake through the roots is determined primarily by the soil's organic content and pH; at normal soil pH levels (4.5 to 8) and with sufficient organic content, lead is bound to organic complexes in preference to other metals that are plant nutrients, e.g., zinc, calcium, manganese, magnesium (CD, p. 6-32). With changes in soil properties such as reduced organic content and lower pH, a greater fraction of total soil lead may become mobilized and its potential for uptake by plant roots enhanced (CD, p. 6-38).

Several studies have shown that lead on the surface of leaves (and bark) is closely related to traffic density and distance from the highway, (or more specifically to air lead concentration and particle size distribution), and that not all of the lead particles deposited on plant surfaces can be washed off (Motto et al., 1970; Schuck and Locke, 1970; Lagerwerff et al., 1973; Pilegaard, 1978; Garty and Fuchs, 1982; Tanaka and Ichikuni, 1982). Leafy aboveground vegetables whose edible portions are exposed to atmospheric lead (e.g., spinach, lettuce) tend to have the highest lead levels (Spittler and Feder, 1978) while for many crops, the edible internal portions (e.g., corn and wheat kernels) have considerably less lead than the outer exposed parts such as stems, leaves, and husks (Ter Haar, 1970). Belowground crops such as potatoes and onions are only partially protected from atmospheric deposition since their roots accumulate lead from the soil (CD, p. 7-34). Meat products that enter our food chain may also be affected by atmospheric lead via the deposition and accumulation of lead on forage

subsequently eaten by livestock (Graham and Kalman, 1974; Crump and Barlow, 1982).

Recent studies on lead content of various foods exposed to and protected from atmospheric lead and lead solder, and on dietary patterns in the U.S., provide information on dietary lead intakes for different populations (Wolnik et al., 1983; National Food Processors Associations, 1982; Pennington, 1983; U.S. FDA, 1983, 1984). Based on these data, the CD has apportioned lead in "typical" child and adult diets to four sources: natural lead, atmospheric lead, lead in solder, and lead whose origin cannot be determined at present. These estimates and their associated uncertainties are discussed in terms of their application to estimating lead exposure discussed in in Section V.

E. Lead in Water

Lead is a natural, usually minor, constituent of surface and ground waters. Atmospheric lead can enter the aquatic system through direct fallout or in surface runoff as suspended particles or adsorbed to soil particles. Under most conditions (pH, temperature, alkalinity), lead forms insoluble salts and precipitates to sediments, which probably accounts for the low lead content of U.S. source water supplies; the average concentration ranging between 3 and 4 micrograms Pb per liter water (µg/l).

In contrast, lead levels in household drinking water can be much higher due to plumbing corrosion and subsequent leaching of lead, ranging between 10 and 30 µg/l on average. The combination of corrosive (i.e., soft or acidic) water and lead pipes or soldered joints in distribution systems create localized zones of high lead concentrations (Worth et al., 1981) as high as 380 µg/l. In general, levels are highest in samples of hot and/or stagnant "first draw" water.

Drinking water is a major source of lead exposure among many infants while they are dependent on baby formulas during their first year. EPA's Office of Drinking Water is currently reviewing the degree of protection afforded by the existing lead standard of 50 µg/l against health risks in this sensitive population.

The CD's estimates of the contributions that atmospheric and solder lead make to young children's total lead exposure via drinking water are addressed in the modeling approaches presented in Section V.

V. ESTIMATING LEAD EXPOSURES AND BLOOD LEAD LEVELS

In order to assess the health risks associated with alternative air lead levels, it is necessary to estimate the blood lead (PbB) levels that would be distributed in the population(s) of concern under various air lead concentrations. [The amount of lead measured in whole blood is an index of lead absorption and is generally used as the dosage, or index of exposure, in investigating the various human health effects associated with lead.] Three approaches are examined that can be used to estimate or predict the impact of inhaled and ingested lead aerosols and compounds on the body burden of lead as indexed by blood lead. These approaches are presented for consideration in possibly using one, two, or all three (with any necessary modifications using improved data) in assessing the protection afforded by alternative lead NAAQS.

The first approach is to use measured rates of "uptake" of lead through different pathways (e.g., inhalation, ingestion) from experimental studies together with available mathematical models from lead balance studies to project either total body burden or the amount of lead in any of the presumed "physiological" kinetic compartments (e.g., blood, soft tissue, bone) at any time (Hammond et al., 1981). This "uptake/biokinetic" modeling approach attempts to account for the following: a) the amount of lead in the body at any one time is the product of dynamic interactions of partially offsetting processes of absorption, distribution, storage, mobilization, and excretion; b) these processes vary with the route and rate of exposure, a person's age, nutritional and health status, and baseline exposure; and c) uptake from all sources by all absorption routes can be modeled, thus providing an estimate of the relative importance of atmospheric lead exposure, either directly or indirectly, to total body burden.

The level of lead in one tissue (e.g., blood) and its relationship to levels of lead in other tissues responds to many external and internal factors. A discussion of lead's absorption, excretion, retention, and distribution within a child's body under different exposure and physiological conditions is provided in Appendix A. Any application of this model's outputs, should however, recognize the limitations of the data and the significant variability among populations in their behavioral, exposure, and physiological characteristics.

The second modeling approach is to estimate separate empirical relationships between average levels of lead in air, food, water, dust, soil and in blood, which are available from experimental exposure and observational, or epidemiological, studies of different populations (i.e., "disaggregate" approach) (CD, Section 11.4). The third is referred to as the "aggregate" approach whereby a relationship between blood lead and air lead is derived that reflects both direct inhalation exposure and indirect exposures via secondary deposition processes. The validity of these latter two approaches, which rely primarily on epidemiological data, requires that the input of lead from all sources must have been reasonably constant long enough for virtual equilibration to occur between blood lead and environmental lead levels (Hammond et al., 1981). The nature of the relationships detected between lead concentrations in blood and various environmental media, and the merits and limitations in using empirical relationships to represent lead exposure through multiple pathways, will be discussed in Section V.B.

A. Integrated Lead Uptake/Biokinetic Model

There have been several studies measuring the intake, uptake, and metabolism of lead in volunteers, from which balance schemes have been constructed (Kehoe, 1961; Chamberlain et al., 1978, Rabinowitz et al.,

1976, 1977). These balance schemes are derived from limited data obtained under experimental conditions and their application thus far has been restricted to adult males. It is also possible to construct balance schemes by making estimates about lead intakes and various metabolic factors. This approach suffers from the fact that the data and assumptions employed are based on the results of many separate pieces of research, and therefore important variables may be omitted. However, provided the data exist, balance schemes can be constructed for different groups with different exposures to lead, and for assessing the importance of specific exposure factors under variable conditions (e.g., alternative lead NAAQS, phaseout of lead in soldered cans and in gasoline).

The staff has attempted to devise model balance schemes for U.S. children at and under 6 years of age under alternative air lead levels. Because the analysis assumes that the various air lead levels are maintained constantly, averaging time (considered in Section VII) will not affect the uptake estimates. The reasons for specifying this age group are discussed in Section VI.C. The results summarized in Table 5-1 give a broad outline of the actual and relative magnitudes of average intakes and uptakes of lead by different pathways and from different sources. It is important to recognize the limitations of this exercise, which involves many uncertainties and assumptions due to a lack of sufficient data. These limitations along with details of the available data and calculations are provided in Appendix B. The groups of children considered in the model do not include the whole U.S. population; they comprise groups with different exposures to lead, some of them extreme, in order to illustrate the variations in lead exposure and absorption in different situations. Section V.A.2 presents methodologies to relate estimates of average lead uptake under alternative air lead levels

Table 5-1. Model Balance Schemes for Average Lead Intake and Uptake in Children Under Alternative Air Lead Levels[1]

	0.5 μg/m³		0.75 μg/m³		1.0 μg/m³	
	General[2]	Point Source[3]	General	Point Source	General	Point Source
1. Outdoor air lead (μg/m³)	0.5	0.5	0.75	0.75	1.0	1.0
2. Indoor air lead (μg/m³)	0.15-0.4	0.15	0.23-0.6	0.23	0.3-0.8	0.3
3. Time spent outdoors (hours/day)	2-4	2-4	2-4	2-4	2-4	2-4
4. Time weighted average (μg/m³)	.18-.42	.18-.21	.27-.63	.27-.32	.36-.83	.36-.42
5. Volume of air respired (m³/day)	5-10	5-10	5-10	5-10	5-10	5-10
6. Lead Intake from air (μg/day)	0.9-4.2	0.9-2.1	1.4-6.3	1.4-3.2	1.8-8.3	1.8-4.2
7. % deposition/absorption in lungs	25-50	45-75	25-50	45-75	25-50	45-75
8. Total lead uptake from lungs (μg/day)	0.23-2.1	0.41-1.6	0.35-3.2	0.63-2.4	0.45-.42	0.81-3.1
9. Dietary Lead Consumption (μg/day)						
a) from solder, undetermined sources	10.1	10.1	10.1	10.1	10.1	10.1
b) atmospheric lead	1.0	1.0	1.0	1.0	1.0	1.0
10. % absorption in gut	42-53	42-53	42-53	42-53	42-53	42-53
11. Dietary lead uptake (μg/day)	2.7-5.9	2.7-5.9	2.7-5.9	2.7-5.9	2.7-5.9	2.7-5.9
12. Street dust/soil lead (μg/g)	100-450	350-800	275-800	600-1250	500-1150	750-1450
13. Indoor dust lead (μg/g)	220-300	450-700	375-650	625-850	525-875	800-1150
14. Time weighted average (μg/g)	210-325	442-716	366-675	623-917	523-921	796-1200
15. Amount of dirt ingested (g/day)	0.1	0.1	0.1	0.1	0.1	0.1
16. Lead intake from dirt (μg/day)	21.0-32.5	44.2-71.6	36.6-67.5	62.3-91.7	52.3-92.1	79.6-120.0
17. % dirt lead absorption in gut	30	30	30	30	30	30
18. Lead uptake from dirt (μg/day)	6.3-9.8	13.2-21.5	11.0-20.3	18.7-27.5	15.7-27.6	23.9-36.0
19. Total lead uptake from lung and gut (μg/day)	9.2-17.8	16.3-29.0	14.1-29.4	22.0-35.8	18.9-33.9	27.4-45.0

[1]Assumptions and calculations are described in Appendix B by row number.
[2]Refers to children living in typical urban or rural environments.
[3]Refers to children living near one or more lead point sources.

Table 5-1. Model Balance Schemes for Average Lead Intake and Uptake in Children Under Alternative Air Lead Levels[1] (Continued)

	1.25 µg/m^3		1.5 µg/m^3		1.75 µg/m^3	
	General	Point Source	General	Point Source	General	Point Source
1. Outdoor air lead (µg/m^3)	1.25	1.25	1.5	1.5	1.75	1.75
2. Indoor air lead (µg/m^3)	0.38-1.0	0.38	.45-1.2	0.45	.53-1.4	0.53
3. Time spent outdoors (hours/day)	2-4	2-4	2-4	2-4	2-4	2-4
4. Time weighted average (µg/m^3)	0.45-1.0	.45-.54	0.54-1.25	.54-.63	0.63-1.46	.63-.73
5. Volume of air respired (m^3/day)	5-10	5-10	5-10	5-10	5-10	5-10
6. Lead Intake from air (µg/m^3)	2.3-10.0	2.3-5.3	2.7-12.5	2.7-6.3	3.2-14.6	3.2-7.3
7. % deposition/absorption in lungs	25-50	45-75	25-50	45-75	25-50	45-75
8. Total lead uptake from lungs (µg/day)	0.58-5.2	1.0-3.9	0.68-6.3	1.2-4.7	0.8-7.3	1.4-5.5
9. Dietary Lead Consumption (µg/day)						
a) from solder, undetermined sources	10.1	10.1	10.1	10.1	10.1	10.1
b) atmospheric lead	2.0	2.0	2.0	2.0	2.0	2.0
10. % absorption in gut	42-53	42-53	42-53	42-53	42-53	42-53
11. Dietary lead uptake (µg/day)	5.1-6.4	5.1-6.4	5.1-6.4	5.1-6.4	5.1-6.4	5.1-6.4
12. Street dust/soil lead (µg/g)	600-1250	850-1000	700-1400	1000-1750	775-1450	1075-1950
13. Indoor dust lead (µg/g)	625-1000	1050-1400	750-1150	1200-1700	800-1200	1350-1900
14. Time weighted average (µg/g)	623-1041	1033-1333	746-1192	1183-1708	798-1241	1327-1908
15. Amount of dirt ingested (g/day)	0.1	0.1	0.1	0.1	0.1	0.1
16. Lead intake from dirt (µg/day)	62.3-104.1	103.3-133.3	74.6-119.2	118.3-170.8	79.8-124.1	132.7-190.8
17. % dirt lead absorption in gut	30	30	30	30	30	30
18. Lead uptake from dirt (µg/day)	18.7-31.2	31.0-40.0	22.4-35.7	35.5-51.2	23.9-37.2	39.8-57.2
19. Total lead uptake from lung and gut (µg/day)	24.4-42.8	37.1-50.3	28.2-48.4	41.8-62.3	29.8-50.9	46.3-69.1

[1]Assumptions and calculations are described in Appendix B by row number.
[2]Refers to children living in typical urban or rural environments.
[3]Refers to children living near one or more lead point sources.

to blood lead levels. Although this approach predicts hypothetical outcomes and the absolute numbers should not be used uncritically, the model does strive to use the available data, with all its limitations, to the fullest extent possible and to provide a useful tool in eventually distinguishing the health impacts of alternative lead NAAQS.

1. Estimates of Lead Uptake

The method employed to estimate the degree to which each environmental source of lead contributes to a child's total daily lead uptake is based on the maximum ambient air lead level allowable for each level considered, probable exposure conditions with respect to other exposure media such as food and dust, and average biological absorption rates for each exposure route. The method consists of a four-step process:

1) definition of ambient concentrations of lead for major exposure sources (i.e., air, food, soil, dust);

2) determination of daily lead intake according to the relationship:

$$I_i = C_i \cdot [Pb]_i$$

where I_i is the daily lead intake from source i, C_i is the consumption per day of each lead source i and $[Pb]_i$ is the concentration of lead in each source i_i;

3) calculation of the amount of lead absorbed from each exposure source i:

$$U_i = I_i \cdot A_i$$

where U_i is lead uptake for each exposure source i, I_i is the daily lead intake from each source i, and A_i is the percent absorption of lead, via the appropriate exposure route for the particular source; and

4) calculation of the total lead uptake from all sources, U_μ:

$$U_\mu = \Sigma (I_i \cdot A_i)$$

Certain assumptions are required to calculate the average daily intake and uptake of lead for children living in the two general areas specified -- urban/rural and near (within 2-5 km) one or more point sources. The assumptions and estimates used in the calculations presented in Table 5-1 are discussed in Appendix B.

2. Uptake and Blood Lead Concentration

To estimate children's blood lead (PbB) levels under different exposure scenarios, several different kinds of studies can be used to derive a relationship between absorbed lead (or lead uptake) and blood lead. Available studies include population surveys in which the blood lead concentration of individuals or groups is correlated with measured lead concentrations in air, food, water, soil, or dust; experiments in which volunteers are exposed to controlled air lead concentrations and their PbB levels measured; and lead balance studies of individuals with measured lead intakes. The most relevant of these studies are discussed in Appendix C along with descriptions of the analyses used to derive uptake/PbB relationships. Data on children from population surveys are discussed in Section V.B. Because of significant metabolic differences, experimental data on adults are used for comparative purposes only.

Figure 5-1 compares the relationships derived from the Ryu et al. (1983) dietary intake study on infants, the Chamberlain and Heard (1981) analysis of the adult epidemiological data, and the metabolic lead balance/ compartmental models of Rabinowitz et al. (1976) and Harley and Kneip (1985). Despite the diverse nature of the populations, study designs, and methodologies, there is a fair degree of consistency in the relationships. Each study found that a linear function provided as good a fit, if not better, than other non-linear forms at the relatively low exposure levels investigated. Some experimental and epidemiological evidence suggests however, that the

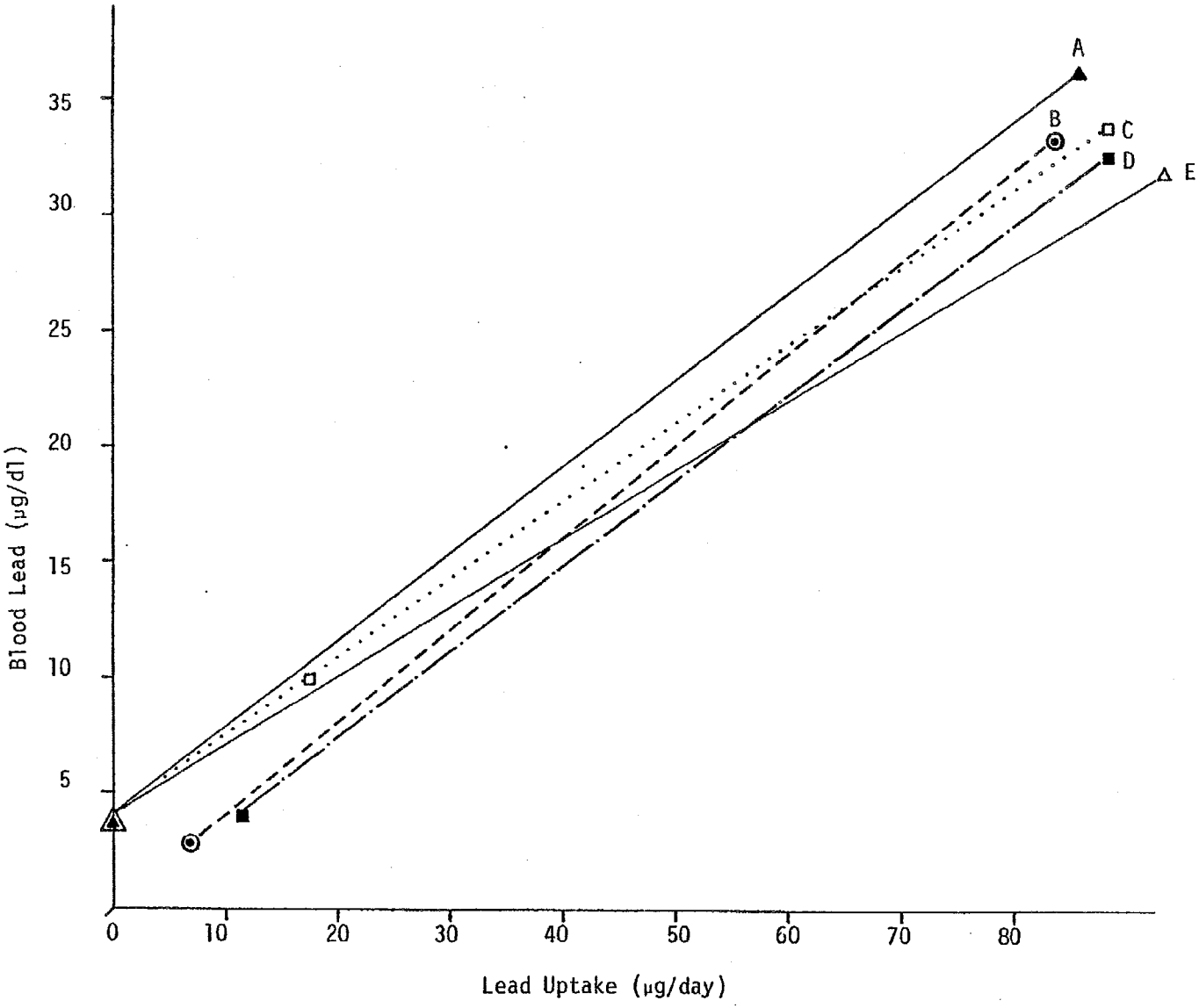

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Figure 5-1. Summary of relationships between daily lead uptake and blood lead, derived from: a) Ryu et al. (1983)--curves A (▲) and E (Δ) represent estimates using 0.42 and 0.53 for lead absorption rates through the gut; b) Harley and Kneip (1985)--curve B (◉) represents estimates for 2 year old children; c) Chamberlain and Heard's (1981) analysis of epidemiological data on adult men (curve C); and d) Rabinowitz et al.'s (1976) metabolic study on adult men (curve D;■).