NATIONAL AMBIENT AIR QUALITY STANDARD FOR LEAD

NOTICE OF PROPOSED RULEMAKING
[40 CFR PART 50]
[FRL 821-4]

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U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Air and Waste Management
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

ENVIRONMENTAL PROTECTION AGENCY

[40 CFR Part 50]

[FRL 821-4]

Docket Number OAQPS 77-1

PROPOSED NATIONAL AMBIENT AIR QUALITY STANDARD FOR LEAD

AGENCY: Environmental Protection Agency

ACTION: Proposed Rule

SUMMARY: In response to a court order to adopt a national ambient air quality standard for lead, EPA proposes to set a national standard for airborne lead of 1.5 micrograms lead per cubic meter ($\mu g\ Pb/m^3$), monthly average. Following promulgation of the standard, States will develop implementation plans for EPA approval which demonstrate how the standard will be attained by 1982, and maintained thereafter. The proposed standard for lead is based on EPA judgments about groups in the population that are at particular risk to lead, the lowest levels of lead exposure associated with adverse effects on health, and the relative importance of airborne lead as a source of lead exposure. EPA believes its proposal reflects the increasing concern from medical research about prolonged low level exposure to lead by young children. The air standard proposed b EPA is based on a goal for total lead exposure lower than previously advocated by other Federal agencies. There is, however, continuing controversy over key areas of research underlying the standard. EPA would welcome information and views pertaining to EPA's approach in developing the standard and to the factors discussed in this notice. EPA also believes that the analyses and judgments that will lead to setting the air standard for lead will have strong implications for other regulatory programs related to lead at the Federal and other levels of government. In the six-month period between proposal and final promulgation, EPA will continue its examination of these difficult issues related to setting the level of the

ambient air quality standard for lead and will seek to involve the public and other affected Federal agencies, both on the final decisions on this air standard as well as planning on ways to control population exposure to lead from non-air sources.

DATES: Comments must be received by February 17, 1978. There will be a public hearing at EPA, 401 M Street, S.W., Washington, D.C. 20460 on January 17, 1978. The standard will be promulgated by June, 1978.

FOR FURTHER INFORMATION AND SUBMISSION OF COMMENTS, CONTACT:

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AVAILABILITY OF SUPPORTING INFORMATION: A docket (Number OAQPS-77-1) containing information used by EPA in development of the proposed standard is available for public inspection between 8:00 a.m. and 4:30 p.m. Monday through Friday, at EPA's Public Information Reference Unit, Room 2922, Waterside Mall, 401 M Street, S.W., Washington, D.C. 20460.

The Federal Reference Method for collecting and measuring lead and its compounds in the ambient air is described in Appendix G to this proposal. Regulations for development of State implementation plans for lead are proposed under 40 CFR Part 51 elsewhere in this Federal Register. The environmental and economic impacts of implementing this standard are described in an Environmental Impact Statement and an Economic Impact Assessment available upon request from Mr. Joseph Padgett at the address shown above.

The documents "Air Quality Criteria for Lead" and "Control Techniques for Lead Air Emissions" are being issued simultaneously with this proposal. Both documents are available upon request from Mr. Joseph Padgett at the address shown above.

SUPPLEMENTARY INFORMATION:

BACKGROUND

Lead is emitted to the atmosphere by vehicles burning leaded fuel and by certain industries. Lead enters the human body principally through ingestion and inhalation with consequent absorption into the blood stream and distribution to all body tissues. Clinical, epidemiological, and toxicological studies have demonstrated that exposure to lead adversely affects human health.

EPA's initial approach to controlling lead in the air was to limit the lead emissions from automobiles, the principal source of lead air emissions. In January of 1972, EPA proposed regulations under Section 211 of the Clean Air Act for phase-down of the lead in gasoline. Subsequently, this action was divided into the promulgation of regulations for the availability of lead-free gasoline for catalyst-equipped cars and other vehicles certified for unleaded fuel and reproposal of the regulations for lead phase-down in leaded gasoline. The regulations for lead phase-down in the total gasoline pool were promulgated in 1973 and, following litigation, modified and put into effect in 1976.

In 1975, the Natural Resources Defense Council (NRDC) and others brought suit against EPA to list lead under Section 108 of the Clean Air Act as a pollutant for which air quality criteria would be developed and a National Ambient Air Quality Standard be established under Section 109 of the Act. The Court ruled in favor of NRDC. EPA listed lead on March 31, 1976, and proceeded to develop air quality criteria and the standard.

In proposing this air standard, EPA is concerned that there are reciprocal effects between the goals and actions taken to control the level of lead in the air, and the parallel judgments and actions taken under other Federal programs. These other programs include EPA's own responsibilities to set standards for lead in drinking water and for the disposal of hazardous waste, the authorities of the Food and Drug Administration to control lead in food, and the regulations adopted by the Consumer Product Safety Commission to control lead in paint. EPA has raised through the Interagency Regulatory Liaison Group the need to coordinate the programs of the Food and Drug Administration, Consumer Product Safety Commission, and the Occupational Safety and Health Administration. Where appropriate, EPA will continue to work with other Federal agencies in developing a general Federal approach to limiting other avenues of exposure to environmental lead.

In parallel with developing the proposed standard, EPA has used information available to assess the economic impact of technological controls necessary to reduce air emissions of lead from industrial facilities For primary copper smelters, primary and secondary lead smelters, gray iron foundries and battery plants, attaining the standard may require control of fugitive lead emissions, i.e., those emissions escaping from process steps, other than emissions from smoke stacks. Fugitive emissions are difficult to estimate, measure, or control, and it is also difficult

to predict their impact on air quality near the facility. From the information available to the Agency, it does appear that non-ferrous smelters may have great difficulty in achieving lead air quality levels consistent with the proposed standard in areas immediately adjacent to the smelter complex. While the possible impact of the standard on these facilities is of concern to EPA, and will be the subject of continuing studies and analysis, these impacts have not entered into determination of the level of the standard.

LEGISLATIVE REQUIREMENTS FOR NATIONAL AMBIENT AIR QUALITY STANDARDS

Two sections of the Clean Air Act govern the development of a National Ambient Air Quality Standard. Section 108 instructs EPA to document the scientific basis for the standard:

Sec. 108(a) "(2) The Administrator shall issue air quality criteria for an air pollutant within 12 months after he has included such pollutant in a list under paragraph (1). Air quality criteria for an air pollutant shall accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the ambient air, in varying quantities. The criteria for an air pollutant, to the extent practicable, shall include information on--

"(A) those variable factors (including atmospheric conditions) which of themselves or in combination with other factors may alter the effects on public health or welfare of such air pollutant;

"(B) the types of air pollutants which, when present in the atmosphere, may interact with such pollutant to produce an adverse effect on public health or welfare; and

"(C) any known or anticipated adverse effects on welfare."

Section 109 addresses the actual setting of the standard:

Sec. 109 "(b) (1) National primary ambient air quality standards, prescribed under subsection (a) shall be ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health. Such primary standards may be revised in the same manner as promulgated.

"(2) Any national secondary ambient air quality standard prescribed, under subsection (a) shall specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air. Such secondary standards may be revised in the same manner as promulgated."

EPA interprets these sections of the Act to mean that the level of the standard is to be determined from information covered in the Criteria Document pertaining to the health and welfare implications of lead air pollution. This is in contrast to other sections of the Act which allow EPA to consider costs of air pollution control and availability of technological controls in determining the level of a standard. Also, EPA should not attempt to place the standard at a level anticipated to represent the threshold for adverse effects, but should set a more stringent level which provides a margin of safety. EPA believes that the extent of margin of safety represents a judgment issue in which the Agency should consider the severity of adverse effects, the probability that the effects may occur, and uncertainties associated with scientific knowledge about the biological effects of lead.

DEVELOPMENT OF AIR QUALITY CRITERIA

Following the listing of lead, EPA proceeded with development of the document, "Air Quality Criteria for Lead". In the process of developing the Criteria Document, EPA has provided a number of opportunities for external review and comment. Three drafts of the Criteria Document have been made available for external review and EPA has received 60 to 80 written comments on each draft. The Criteria Document was the subject of three meetings of the Subcommittee on Scientific

Criteria for Environmental Lead of EPA's Science Advisory Board. Each of these meetings has been open to the public and a number of individuals have presented both critical review and new information for EPA's consideration.

Development of the Criteria Document indicated to EPA that there are a number of areas in which additional research could provide information useful to determining the level for the lead standard. It is also evident that scientific controversy exists about facts or interpretation of material included in the Criteria Document, including two areas critical to the setting of the standard: the health significance of abnormal biological effects associated with blood lead levels below traditional levels of concern, and the relative significance of lead air emissions as the direct or indirect source of lead exposure, compared to other sources of exposure.

However, the provisions of the Act requiring a deadline for proposal and promulgation of the standard, and the requirements for periodic future review of air quality criteria and standards, indicate that Congress intends for the Agency to proceed even where scientific knowledge is not complete or where there is an absence of full scientific consensus. EPA has, therefore, developed the proposed air standard on the basis of its best judgment as to what the Act requires, and what information the "Air Quality Criteria for Lead" provides. To arrive at the air standard, EPA has attempted to use numerical estimates of key factors. In several instances, factors which are not known precisely have a large effect on the level of the standard. EPA invites information, views and judgments both on its approach to setting a level for the standard and the numerical values used for key factors described in the following sections.

SUMMARY OF GENERAL FINDINGS FROM AIR QUALITY CRITERIA FOR LEAD

From the extensive review of scientific information presented in the Criteria Document, conclusions in several key areas have particular relevance for setting the lead standard.

1. There are multiple sources of lead exposure. In addition to air lead these sources include: lead from paint and inks, lead from water supplies and distribution systems, lead from pesticides, and lead in fresh and processed food. The relative contribution to population exposure from each source is difficult to quantify.

2. Exposure to air lead can occur directly by inhalation, or indirectly by ingestion of lead contaminated food, water, or non-food materials including dust and soil.

3. There is a significant variability in response to lead exposure. Certain subgroups within the population are more susceptible to the effects of lead or have a greater potential for exposure. Of these, young children represent a population of foremost concern. Even within a particular population, group response to lead exposure may vary widely from the average response.

4. Within the human body, three systems appear to be most sensitive to interference by lead--the blood-forming or hematopoietic system, the nervous system, and the renal system. In addition, lead has been shown to affect the normal functions of the reproductive, endocrine, hepatic, cardiovascular, immunologic, and gastrointestinal systems.

5. Effects reported in the Criteria Document range from impairment of biochemical systems (inhibition of aminolevulinic acid dehydratase (ALAD)) at a blood lead level of 10 micrograms lead per deciliter blood (μg Pb/dl) to encephalopathy at 80 to 100 μg Pb/dl.

6. From various studies of lead exposure, estimates can be made of the impact of exposure through inhalation and ingestion on blood lead level. Of particular importance, are the estimates of: air lead/blood lead ratios, the percentage of deposition and absorption of air lead, the percentage of absorption of ingested material, estimates of the variability of blood lead within a population exposed to uniform levels of lead, and estimates of the contribution of air lead to blood lead.

Determination of a proposed level for the lead standard requires the use and interpretation of specific information for each of these areas. The approach taken is described in the following sections.

GENERAL APPROACH TO SETTING THE LEAD STANDARD

Development of the National Ambient Air Quality Standard for lead requires certain judgments by EPA about the relationship between concentrations of lead in the air and possible adverse health effects experienced by the public. This relationship is greatly complicated by the fact that lead in the air is not the only source of lead exposure; that there is variability of response among individuals exposed to lead; and that there are numerous effects of lead on health, occurring at various levels of exposure which vary in public health significance.

In developing the standard, EPA has made judgments in five key areas:

1. Determining the critically sensitive population.
2. Determining the pivotal adverse health effect.
3. Determining the mean population blood lead level which would be consistent with protection of the sensitive population.
4. Determining the relationship between air lead exposure and resulting blood lead level.
5. Determining the allowable blood lead increment from air.

DETERMINING THE CRITICALLY SENSITIVE POPULATION

Certain subgroups within the general population differ in sensitivity to lead exposure. Protection of populations exhibiting the greatest sensitivity of response to lead is a major consideration in determining the level of the lead standard. From information presented in the Criteria Document, there are a number of populations for which lead exposure poses a greater risk: young children, pregnant women and the fetus; the occupationally exposed; and individuals suffering from dietary deficiencies or exhibiting the genetic inability to produce certain blood enzymes.

EPA believes that young children (ages 1-5 years) should be regarded as the foremost critically sensitive population for setting the lead standard. This is because hematologic and neurologic effects in children are shown to occur at lower thresholds than adults, and because children have a greater risk of exposure to non-food material containing lead, such as dust and soil, as the result of normal hand-to-mouth activity. The Criteria Document also states that children may be at greater risk than adults due to 1) greater intake of lead via inhalation and ingestion per unit body weight; 2) greater absorption and retention of ingested lead; 3) physiologic stresses due to rapid growth rate and dietary habits; 4) incomplete development of metabolic defense mechanisms; and 5) greater sensitivity of developing systems.

Pregnant women and the fetus are at risk because of transplacental movement of lead to the fetus and the possibility of maternal complications at delivery. Because there is a balance between maternal blood lead

levels and fetal blood lead levels, concern exists that development of the nervous system of the fetus may be impaired due to neurotoxicity of lead. Changes in fetal heme synthesis and premature births have been associated with prenatal exposure of the fetus to lead. However, available evidence does not indicate that pregnant women and the fetus would require a more stringent standard than young children.

Groups exposed to lead in the workplace also comprise a population at greater risk. Because members of such groups are generally healthy and do not have a greater physiological sensitivity to lead than young children, EPA believes that the protection of such groups does not require an air quality standard for lead more stringent than that for young children.

Other possible critically sensitive populations suggested in the Criteria Document include individuals with genetic conditions such as sickle cell disease. The Criteria Document cites a tentative association between the existence of sickle cell disease in children and increased risk of peripheral neuropathy due to lead exposure. Individuals suffering from iron deficiency or malnutrition may also be at greater risk from lead exposure. There is, however, insufficient data to determine the effects threshold for such groups or to accurately characterize such groups within the general population.

DETERMINING THE PIVOTAL ADVERSE HEALTH EFFECT

The toxic effects of lead resulting from high levels of exposure are well documented. Among the first effects noted historically were the severe and sometimes fatal consequences such as colic, palsy, and

encephalopathy which followed acute occupational exposure in the mining and smelting industries. Exposure to high concentrations of lead in paints, inks, pesticides, and plumbing have similarly been implicated in cases of severe poisoning.

Recent widespread increase of lead in the environment as a result of human activities has stimulated research on the possible effects of the longer-term, low level exposure characteristic of the general population. Clinical and epidemiological studies have revealed that lead accumulates in the body throughout life, to a large extent immobilized in bone, but with a significant mobile fraction in the blood and soft tissues. Blood lead concentrations respond predictably to changes in the level of environmental exposure and, as a result, are generally accepted as good indicators of that exposure as well as of the internal dose of lead to which all body tissues are exposed. The threshold for a particular health effect is considered to be the blood lead level at which the effect is first detected.

The Criteria Document provides a ranking by blood lead threshold of the health effects observed in children.

SUMMARY OF HEALTH EFFECTS IN CHILDREN

Blood Lead Threshold (µg Pb/dl)	Effect	Population Group
10	ALAD inhibition	Children and adults
15 - 20	Erythrocyte protoporphyrin elevation	Women and children
40	Increased urinary ALA excretion	Children and adults
40	Anemia	Children
40	Coproporphyrin elevation	Adults, children
50 - 60	Central Nervous System (CNS) deficits	Children
50 - 60	Peripheral neuropathies	Adults and children
80 - 100	Encephalopathic symptoms	Children

ALAD Inhibition

Inhibition of the enzyme aminolevulinic acid dehydratase (ALAD) represents the lowest level effect of lead that has been detected. The decreased activity of this enzyme, while observable, is not sufficient at blood leads at and below 10 µg Pb/dl to interfere with the step in heme synthesis which it mediates. Because no significant accumulation of precursors occurs at this level of exposure, ALAD inhibition of this degree is not regarded as a physiological impairment of the system. This effect becomes more significant at higher lead concentrations (40 µg Pb/dl) which reduce the activity of ALAD sufficiently to cause build-up of the precursor (ALA) in the urine.

Erythrocyte Protoporphyrin Elevation

Above 15-20 µg Pb/dl, the Criteria Document notes a correlation between blood lead levels in children and the elevation of protoporphyrin in red blood cells. Unlike ALAD inhibition at 10 µg Pb/dl, the accumulation of erythrocyte protoporphyrin (EP) indicates a functional impairment of the heme synthetic pathway.

In regard to the implications for health of EP elevation, the Criteria Document provides the following description:

> "Accumulation of protoporphyrin in the erythrocytes is the result of decreased efficiency of iron insertion into protoporphyrin, the final step in heme synthesis which takes place inside the mitochondria. When this step is blocked by the effect of lead, large amounts of protoporphyrin without iron accumulate in the erythrocyte, occupying the available heme pockets in hemoglobin."
>
> "The effect of lead on iron incorporation into protoporphyrin is not limited to the normoblast and/or to the hematopoietic system. Formation of the heme-containing protein, cytochrome-P450, which is an integral part of the liver mixed-function oxidase, may also be inhibited by lead. Accumulation of protoporphyrin in the presence of lead has been shown to occur also in cultured cells of chick dorsal root ganglion, indicating that inhibition of heme

synthesis takes place in the neural tissue as well. These observations, and the fact that lead is known to disrupt the mitochondrial structure and function, indicate that the lead effect on heme synthesis is exerted on all body cells, possibly with different dose/response curves holding for effects in different cell types. On the other hand, it must be noted that increased levels of protoporphyrin in the erythrocyte reflect an accumulation of substrate and therefore imply a functional alteration of mitochondrial function in the same way that the increased urinary excretion of urinary δ-ALA implies impairment. In other words, if a "reserve" activity of ferrochelatase exists, such as has been suggested for δ-ALAD, accumulation of protoporphyrin in the erthrocytes indicates that this has been hampered by the lead effect to the point that the substrate is accumulated. For these reasons, as well as for its implication of the impairment of mitochondrial function, accumulation of protoporphyrin has been taken to indicate physiological impairment relevant to human health."

The remaining effects listed in the table present progressively greater health risks to susceptible individuals including anemia, the possibility of irreversible learning deficits, and lead encephalopathy.

EPA is proposing that lead-induced elevation in children of EP should be accepted as the pivotal adverse effect of lead. Accordingly, the air lead standard should be designed to prevent the occurrence of EP elevation in children. EPA bases its determination that EP elevation due to lead should be regarded as an adverse health effect on the following points:

1. EP elevation indicates an abnormal impairment of various cell functions, which should not be allowed to persist as a chronic condition.
2. The impairment of cellular function indicated by EP elevation extends to all body cells, and may have particular implications for the functioning of neural and hepatic tissues.
3. The air lead standard is intended to establish a level of airborne lead which can be regarded as consistent with protecting the health over a lifetime of exposure. The pervasive

biological involvement of lead in the body, and its demonstrated impairment of biological functions are a strong impetus to the Agency in adopting the lowest threshold biological effect which can be considered adverse to health.

4. The Center for Disease Control has also used EP elevation as an indicator of undue lead exposure, although their guidelines published in 1975 are oriented to establishing an individual threshold for risk (30 μg Pb/dl) in populations of children exposed to high-dose lead sources such as lead-based paint rather than for establishing a safe mean population blood lead level with a margin of safety.

5. The Act intends that the air standard be precautionary. Taking the lowest adverse effect levels is compatible with the scientific uncertainty about the health consequences of prolonged low level lead exposure, and with the downward trend in levels of lead in the blood regarded as adverse to health by the public health community.

As an alternative to using elevation of EP as the pivotal health effect, EPA could take the position that EP elevation, while of concern to public health, is not sufficiently adverse to health, and that the standard should be based on the more severe effects such as anemia, or CNS deficits. EPA would welcome comments on whether what is known, or anticipated, about EP elevation or other subclinical effects has sufficient implications to warrant a role in determining the level of the standard.

DETERMINING A SAFE BLOOD LEAD LEVEL FOR PROTECTION OF THE SENSITIVE POPULATION

The third key area for judgment in the development of the proposed standard involves the determination of the mean population blood lead level for children at which EP elevation does not occur. EPA is proposing that this standard for lead be based on the judgment that the mean population blood lead for children not exceed 15 µg Pb/dl. This is the lowest value given in the Criteria Document as a threshold for the correlation of EP with blood lead level, based on studies by Roels (1976) and Piomelli (1977). On the basis of present knowledge, EPA believes that a population mean of 15 µg Pb/dl can be regarded as an indicator of a safe level of total lead exposure for children.

There are two reasons why the use of a blood lead target as an intermediate goal between air quality and EP levels is necessary. First, most of the scientific literature covered by the Criteria Document reports studies which link air lead with blood lead levels. Second, EP levels can be expected to respond to all sources of lead exposure; blood lead level serves as an indicator of total exposure.

In selecting 15 µg Pb/dl mean population blood lead as a target, EPA wishes to stress that it is proposing a statistical measure of population exposure. EPA is not suggesting that individual blood lead levels in excess of 15 µg Pb/dl necessarily constitute a significant risk to health. It can be expected that a population with a mean blood lead level of 15 µg Pb/dl will have individuals with higher and lower blood lead levels.

There will also be a variation of EP levels for individuals with a given blood lead level. It is also true that the absence of statistical correlation of EP levels with blood lead levels below 15 µg Pb/dl does not necessarily mean that these lower blood lead levels are known to be without risk. However, the threshold of 15 µg Pb/dl does represent a point below which the sensitive population as a group has not been seen to show an elevation in EP due to lead and above which EP elevation has been demonstrated to rise with increasing implications for health. While other thresholds for EP elevation have been found (Sassa, 1973), EPA is using the lowest level cited in the Criteria Document in order to establish a margin of safety.

Alternatively, EPA could attempt to judge the actual level of EP elevation which represents an adverse effect on health, and then apply an adjustment for margin of safety. For example, in 1975, the Center for Disease Control established as a guideline for undue or increased lead absorption in children a blood lead level of 30 µg Pb/dl or EP levels of 60 µg/dl. The level of 30 µg Pb/dl in the blood represents some degree of health risk, but it is difficult to know whether any intermediate levels between 30 µg Pb/dl and 15 µg Pb/dl safeguard the public health.

EPA believes that elevations in individual blood levels and corresponding changes in EP levels are reversible, and may not in a single cycle constitute a serious physiological impairment. However, taken as a population average, underlying an environmental standard describing the safe limits for a lifetime of exposure, EPA is proposing that no

elevation of EP associated with lead exposure should be seen as free from risk to the health of the sensitive population.

In establishing the target mean blood lead level for the sensitive population, EPA has used the lowest threshold for EP rather than attempt to use statistical techniques discussed in the Criteria Document in order to take into account the extent of individual variation in blood lead levels for a given level of exposure. The Criteria Document points out that data from epidemiological studies show that the log values of individual blood lead values in a uniformly-exposed population are normally distributed with a standard geometric deviation of 1.3 to 1.5. Using standard statistical techniques, it is possible to calculate the mean population blood lead level which would place a given percentage of the population below the level of an effects threshold. For example, a mean population blood lead level of 15 µg Pb/dl would place 99.5% of a population of children below the Center for Disease Control guideline of 30 µg Pb/dl.

EPA believes that variable response within the sensitive population should be taken into consideration in setting the level of the standard, but recognizes a number of problems in using the log-normal distribution in the case of the lead standard.

(1) The log-normal distribution describes the variable response of individuals' blood lead levels to air exposure. It can be expected that there is also a probability distribution associated with the elevation of EP among individuals with a given blood lead level. The parameters of this second probability distribution are not presented in the Criteria Document, but it is reasonable to expect that only a small

percentage of those individuals just above the threshold blood lead level will experience EP elevation beyond what could be expected from the normal scatter of EP values around blood lead levels just below the threshold. The effect of using blood lead as an intermediary between air lead exposure and EP levels is to combine two probability distributions, one known and one unknown, between population blood values and EP elevation.

(2) There are a number of sources of variability in blood lead levels other than individual differences of response within a population group. These include variability from possible non-uniform exposure to lead in the populations studied and from analytical and process techniques used in measuring blood lead.

For these reasons, EPA believes that use of a log-normal correction may overestimate the degree to which the population mean should be below the threshold blood lead level. This is particularly true in dealing with the threshold for EP where considerable margin of safety results from selection of the target blood lead level at which slight EP elevation is first detected, rather than a level at which lead has had a substantial impact on EP levels.

DETERMINING THE RELATIONSHIP BETWEEN AIR LEAD EXPOSURE AND RESULTING BLOOD LEAD LEVEL

On the basis of clinical and epidemiological studies evaluated, the Criteria Document concludes:

> "Evidence indicates that a positive relationship exists between blood and air lead levels, although the exact functional relationship has not yet been clarified. Available data indicate that in the range of air lead exposures generally encountered by the population, the ratio of the increase in blood lead per unit of air lead is from 1 to 2. It appears that the ratio for children is in the upper end of the range and that ratios for males may be higher than those for females."

The range of ratios for children's blood lead response to a one µg increase in air lead cited in the Criteria Document is from 1.2 to 2.3. The lower ratio comes from studies at Kellogg, Idaho, where dust levels of lead were separately correlated with blood lead. In view of the tendency of children to experience higher ratios due to greater intake and absorption of air lead, EPA has selected a ratio of 1:2 in calculating the impact of air lead levels on blood lead levels in children.

DETERMINING THE ALLOWABLE BLOOD LEAD INCREMENT FROM AIR

The fifth area of judgments made by EPA in developing the proposed standard for lead is related to an aspect of lead which has not characterized any pollutant previously addressed by EPA under Section 109 of the Clean Air Act: that significant amounts of the pollutant result from sources that are not subject to control by implementing an air quality standard.

Some studies reported in the Criteria Document clearly show that levels of lead in the blood derive from non-air sources. For example, studies in areas with minimal air lead levels still show significant levels of lead in the blood (Johnson, Tillery 1975). A study of children in Boston correlates blood lead levels with lead levels in water supplies (Worth, in press).

Other studies demonstrate a strong relationship of blood lead level with air lead. Clinical studies on adult volunteers in chamber studies demonstrate changes of blood lead with changes of the concentration of lead in the air (Griffin, et. al, 1975). Epidemiological studies show a general pattern of urban-rural difference where blood lead levels are higher in urban settings where air lead levels are also higher. Other epidemiological studies directly correlate air lead with blood lead. These include studies using personal dosimeters to accurately gauge lead

exposure (AZAR, 1975), and the extensive population studies conducted in the community around the smelter complex at Kellogg, Idaho (Yankel and von Lindern, 1977).

Implications of Multiple Sources of Lead in Setting an Air Standard

The implications of multiple sources of environmental lead are difficult to reconcile with the concept of a National Ambient Air Quality Standard. If the air were the only source of lead, it would be a reasonably straightforward matter to identify a safe level and to require that, regardless of what prevailing levels of air lead are today, the safe level be achieved. However, since non-air sources contribute lead as well, the level of an ambient air quality standard which will protect public health is affected by the contribution of these non-air sources. If their contribution is far below the allowable level of blood lead, the air contribution can be permitted to be relatively high. However, if they alone contribute more than the allowable blood lead level, even a zero ambient air quality standard would not prevent EP elevation in children.

EPA believes that it should assume some level of blood lead attributable to non-air sources in order to determine what the air lead contribution can be, and what the ambient air quality standard should be as a result. This calculation is complicated, however, by the fact that the non-air contribution to blood lead varies from time-to-time and place to place. As a result, the level selected as the basis for determining the allowable contribution from air and the resulting air quality standard becomes in part a policy choice reflecting how much of the lead pollution problem should be dealt with through control of air sources.

Because of the factors just discussed, no National Ambient Air Quality Standard can be assured of being protective in all locations. Regardless of what the non-air contribution is assumed to be, the air standard will

be overprotective in areas where lead from non-air sources is low and underprotective in areas where it is high. EPA does not believe, however, that it is given the latitude to set area specific air quality standards under Section 109. EPA has, therefore, undertaken to make a single judgment as to what contribution to population blood lead levels derives from non-air sources. This single numerical value represents, in fact, what EPA proposes should be taken as a goal in limiting lead exposures from non-air sources. The level for non-air contribution used in this proposal is EPA's best judgment as to the appropriate level based partly on what is known about non-air lead contribution from a limited number of studies and partly on what EPA believes is an appropriate goal for air pollution control, consistent with the Agency's responsibility to protect the public health. The specific derivation of the goal for non-air contribution to mean population blood lead levels is described in the next section.

Basis for EPA's Estimate of Contribution to Blood Lead Levels from Non-Air Sources

The level of the standard is very strongly influenced by judgments made regarding the size of non-air contribution to total exposure. EPA has encountered difficulties in attempting to estimate exposure from various lead sources in order to determine the contribution of such sources to blood lead levels:

(1) Studies reviewed in the Criteria Document do not provide detailed or widespread information about relative contribution of various sources to young children. Estimates can only be made by inference from other empirical or theoretical studies, usually involving adults.

(2) It can be expected that the contribution to blood lead levels from non-air sources can vary widely, is probably not in constant proportion to air lead contribution, and in some cases may alone exceed the target mean population blood lead level.

In spite of these difficulties, EPA has attempted to assess available information in order to estimate the general contribution to population blood lead levels from air and non-air sources. This has been done with evaluation of evidence from general epidemiological studies, studies showing decline of blood lead levels with decrease in air lead, studies of blood lead levels in areas with low air lead levels, and isotopic tracing studies.

Studies reviewed by the Criteria Document show that mean blood lead levels for children are frequently above 15 μg Pb/dl. In studies reported, the range of mean population blood lead levels for children was from 16.5 μg Pb/dl to 46.4 μg Pb/dl with most studies showing mean levels greater than 25 μg Pb/dl (Fine, 1972; Landrigan, 1975; von Lindern, 1975). EPA believes that for most of these populations, the contribution to blood lead levels from non-air sources exceeds the desired target mean blood lead level.

In a number of studies, it is apparent that reduction in air lead levels results in a decline in children's blood lead levels. A study of blood lead levels in children in New York City showed that children's mean blood lead levels fell from 30.5 μg Pb/dl to 21.0 μg Pb/dl from 1970 to 1976, while during the same period air lead levels at a single monitoring site fell from 2.0 μg Pb/dl to 0.9 μg/Pb, (Billick, 1977). Studies at Omaha, Nebraska (Angle, 1977) and Kellogg, Idaho (Yankel, von Lindern, 1977) also show a drop in mean blood lead levels with declines in air lead levels. However, as air lead levels decline there appears to be a rough limit to

the drop in blood lead levels. EPA has also examined epidemiological studies in the Criteria Document where air lead exposure is low, and can be assumed to be a minor contributor to blood lead. These studies provide an indication of blood lead levels resulting from a situation where non-air sources of lead are predominant.

Studies Reporting Blood Lead Levels in Children
Exposed to Moderate - Low Air Lead Levels

Investigator	Blood Lead μg Pb/dl	Air Lead μg Pb/m^3	Comment
Hammer, 1972	11.6	0.1	Children in Helena, Montana
Angle, 1974	14.4	0.14	Suburban children ages 1 to 4 in Omaha
Goldsmith, 1974	13.7	0.2 - 0.7	Children in Benecia, California
	13.8	0.3 - 0.6	Children in Crocket, California
Johnson, Tillery, 1975	10.2	0.6	Female children - mean age 9 in Lancaster, California

The range of mean blood lead levels in those studies is from 10.2 μg Pb/dl to 14.4 μg Pb/dl, with an average at 12.7 μg Pb/dl.

In addition to epidemiological investigations, EPA has reviewed studies that examine the source of blood lead by detecting characteristic lead isotopes. A study using isotopic tracing (Manton, 1977) suggests that for several adults in Houston, Texas, 7 to 41 percent of blood lead could be attributed to air lead sources. An earlier isotopic study (Rabinowitz, 1974) concluded that for two adult male subjects studied, approximately one-third of total daily intake of lead could be attributed to exposure to air lead levels of 1-2 μg Pb/m^3. While these results

cannot be directly related to children, it is reasonable to assume that children may exhibit the same or higher percentages of air lead contribution to blood lead level because of a greater potential for exposure to indirect air sources, soil and dust.

From reviewing these areas of evidence, EPA concludes that:

1. In studies showing mean blood lead levels above 15 µg Pb/dl, it is probable that both air and non-air sources of lead contribute significantly to blood lead with the possibility that contributions from non-air sources exceed 15 µg Pb/dl.

2. Studies showing a sustained drop in air lead levels show a corresponding drop in blood lead levels, down to an apparent limit in the range of 10.2 to 14.4 µg Pb/dl. These studies show the rough range of the lowest blood lead levels that can be attributed to non-air sources.

3. Isotopic tracing studies show air contribution to blood lead to be 7-41 percent in one study and about 33 percent in another study.

In considering this evidence, EPA notes that if, from the isotopic studies, approximately two-thirds of blood lead is typically derived from non-air sources, a mean blood lead target of 15 µg Pb/dl would attribute 10 µg Pb/dl to non-air sources. On the other hand, the average blood lead level from studies EPA believes to represent the least amount of blood lead attributable to non-air sources is 12.7 µg/Pb. In the absence of more precise information, EPA is proposing that the lead standard be based on the assumption that in general, 12 µg Pb/dl of the blood lead level in children is derived from lead sources unaffected by the lead air quality standard. EPA is aware that actual population blood lead

levels, either individually or as a population mean, may exceed this benchmark. However, if EPA were to use a larger estimate of non-air contribution to blood lead, the result would be an exceptionally stringent standard, which would not address the principle source of lead exposure. Conversely, EPA believes that it should not adopt an estimate of non-air contribution below the level shown in available studies to be the lowest mean blood lead level documented in the Criteria Document.

Because of the strong impact that adopting this goal for non-air sources has on the level of the standard, EPA welcomes information and judgments about the validity of the numerical value chosen for this factor, as well as views about alternative ways in which EPA could develop an air standard that takes into account other routes of exposure.

CALCULATION OF THE AIR STANDARD

EPA has calculated the proposed standard based on the conclusions reached in the previous sections:

1)	Sensitive Population:	children, ages 1-5
2)	Health Basis (lowest detectable adverse effect):	elevation of erythrocyte protoporphyrin (EP)
3)	Effect Threshold in Sensitive Population:	15 μg Pb/dl
4)	Assumed Goal for Contribution to Blood Lead from Non-Air Sources:	12 μg Pb/dl
5)	Allowable Contribution to Blood Lead from Air Sources: 15 μg Pb/dl - 12 μg Pb/dl =	3 μg Pb/dl
6)	Air Lead Concentration Consistent with Blood Lead Contribution from Air Sources:	

$$3\ \mu g\ Pb/dl \times 1\ \mu g/m^3\ air = 1.5\ \mu g\ Pb/m^3$$

$$2\ \mu g/dl\ blood$$

SELECTION OF THE AVERAGING PERIOD FOR THE STANDARD

To be protective of human health, the averaging period for the lead standard should be chosen such that variations in exposure which could result in adverse effects do not occur unless the standard is exceeded. The averaging period is the length of time over which measured concentrations of air lead are averaged to obtain an air quality level which is compared to the standard level to determine if a violation of the standard has occurred.

Moderate increases in air lead levels have been shown to produce increases in blood lead levels in adults after seven weeks of exposure (Griffin, 1975). Because of the slow response of blood lead levels to increases in air lead levels, it is not probable that short-term peaks in air lead levels will cause adverse effects.

Based on available information, EPA has concluded that the averaging period for the lead standard be a calendar month, based on the average of 24-hour measurements. This period is somewhat shorter than the time observed for the adjustment of blood lead levels in adults to changes in air lead concentration because of the greater risk of exposure of young children.

MARGIN OF SAFETY

EPA believes that the recommended standard incorporates a sufficient margin to protect the public health and welfare from the adverse effects of lead exposure deriving from lead in the air. Margin of safety considerations have entered into the development of the standard in several key areas:

1) The standard is based on protection of young children, a critically sensitive general subgroup within the population.

2) The standard is based on the lowest threshold for the first adverse effect occurring with increasing blood lead levels in children: elevation of protoporphyrin in red blood cells at a blood lead level of 15 μg Pb/dl.

3) In estimating the change in blood lead levels resulting from the change in air lead levels, EPA has selected a ratio at the protective end of the range provided in the Criteria Document.

IMPACT OF LEAD DUSTFALL ON BLOOD LEAD

The significance of dust and soil lead as indirect routes of exposure has been of particular concern in the case of young children. Play habits and mouthing behavior between the ages of one and five have led to the conclusion that greater potential may exist in these children for ingestion and inhalation of the lead available in dust and soil.

Studies reviewed in the Criteria Document indicate a correlation between soil and dust levels and childrens' blood lead levels in highly contaminated environments (Yankel and von Lindern, 1977; Barltrop, 1974; Galke, in press). The lead threshold for concern has been reported as 1,000 ppm in soil (Yankel and von Lindern, 1977); at exposures of 500 and 1,000 ppm soil the Document concludes that blood lead levels begin to increase. A two-fold increase in soil concentration in this range is predicted to result in a 3-6 percent rise in blood lead levels. Below 500 ppm soil, no correlation has been observed with blood lead levels.

The normal background for lead in soil is cited in the Criteria Document as 15 ppm. Due to human activities, the average levels in most areas of the U.S. are considerably higher. Soil studies conducted by EPA's Office of Pesticides Programs from 1974-1976 in 17 urban areas reported only 3 cities with arithmetic mean concentrations in excess of 200 ppm, with the highest value 537 ppm. Concentrations in the soils surrounding large point sources of lead emissions, or heavily-travelled roads, on the other hand, may reach several thousand ppm.

Because of the many factors involved, EPA is unable to predict the relationship between air lead levels, dustfall rates, and resulting soil accumulation. Complicating factors include: particle size distribution, rain-out, other meteorological factors, topographical features affecting deposition, and removal mechanisms.

EPA believes, however, that significant impacts on blood lead of soil and dust lead are mainly limited to areas of high soil concentration (in excess of 1,000 ppm) around large point sources and in major urban areas which also experience high air lead levels. Evidence suggests that soil lead levels in areas with air lead levels in the range of the proposed standard are well below the threshold for blood lead impact (Johnson, Tillery, 1975; Johanson, 1972; EPA, 1975 Air Quality Data and Soil Levels).

WELFARE EFFECTS

Available evidence cited in the Criteria Document indicates that animals do not appear to be more susceptible to adverse effects from lead than man nor do adverse effects in animals occur at lower levels of exposure than comparable effects in humans.

There is some evidence that atmospheric sources of lead may be injurious to plants. Lead is absorbed but not accumulated to any great extent by plants from soil. Lead is either unavailable to plants or is fixed in the roots and only small amounts are transported to the above ground portions. Lead may be deposited on the leaves of plants and present a hazard to grazing animals. Although some plants may be susceptible to lead in the natural environment, it is generally in a form that is largely nonavailable to them.

There is no evidence to indicate that ambient levels of lead result in significant damage to man-made materials. Effects of lead on visibility and climate are minimal.

Based on such data, EPA concludes that significant welfare effects associated with exposure to lead which would necessitate a secondary standard more restrictive than the primary standard have not been established. Therefore, the primary ambient air quality standard should protect against known and anticipated adverse effects on public welfare. A more restrictive secondary standard will not be established at this time.

ECONOMIC IMPACT ASSESSMENT

The Agency conducted a general analysis of the economic impact that might result from the implementation of lead emission control measures. This analysis pointed out that the categories of sources likely to be affected by control of lead emissions are primary lead and copper smelters, secondary lead smelters, gray iron foundries, gasoline lead additive manufacturers, and lead storage battery manufacturers. This analysis further indicates that primary and secondary lead smelters and copper smelters may be severely strained both technically and economically in achieving emission reductions that may be required in implementing the proposed air quality standard.

There are, however, uncertainties associated with evaluating the impact of attaining the standard. For smelters, foundries and battery plants, attaining the standard may require control of fugitive lead emissions, i.e., those emissions escaping from process steps, other than emissions from smoke stacks. Fugitive emissions are difficult to estimate, measure, or control and it is also difficult to predict their impact on air quality near the facility. From the information available to the Agency, it does appear that non-ferrous smelters may have great difficulty in achieving lead air quality levels consistent with the proposed standard in areas immediately adjacent to the smelter complex. While the possible impact of the standard on these facilities is of concern to EPA, and will be the subject of continuing studies and analysis, these impacts have not entered into determination of the level of the standard.

OTHER EPA REGULATIONS

In 1975, EPA promulgated the national interim primary drinking water regulation for lead. The standard was aimed at protecting children from undue lead exposure and limited lead to 0.05 milligrams per liter (mg/l) which was considered as low a level as practicable. In 1977,

the National Academy of Sciences evaluated the interim drinking water standards and concluded that a no-observed-adverse health effect for lead cannot be set with assurance at any value greater than 0.025 mg/l. The Office of Water Supply is currently reviewing the need to revise the interim drinking water standard for lead.

Based on its toxicity, EPA included lead on its 1977 list of priority pollutants for which effluent guidelines will be developed by early 1979. Effluent guidelines for non-ferrous smelters, the major stationary source emitters of airborne lead, are being developed based on achievement of best available technology.

EPA's Office of Pesticide Programs has promulgated regulations based on toxicity of lead which require the addition of coloring agents to the pesticide lead arsenate and specify disposal procedures for lead pesticides. Use of lead in pesticides is a small and decreasing proportion of total lead consumption in the U.S.

The Resource Conservation and Recovery Act of 1976 through which EPA is to establish standards on how to treat, dispose, or store hazardous wastes, provides a means for specifying how used crankcase oil and other waste streams containing lead should be recycled or safety disposed of. At the present time, no regulatory actions related to wastes containing lead have been proposed.

EPA has regulations for reducing the lead content in gasoline to 0.5 grams/gallon by October 1, 1979, and regulations providing for lead-free gasoline required for cars equipped with catalytic converters and other vehicles certified for use of unleaded fuel. The former regulations are based on reducing exposure to airborne lead to protect public health. Other EPA actions which result in the reduction of airborne lead levels include ambient standards and State implementation plans for other pollutants such as particulate matter and sulfur dioxide and new source

performance standards limiting emissions of such pollutants. Existing and new sources of particulate matter emissions generally use control techniques which reduce lead emissions as one component of particulate matter.

OTHER FEDERAL AGENCY REGULATIONS AND POSITIONS ON LEAD

The Occupational Safety and Health Administration proposed regulations in 1975 to limit occupational exposure to lead to 100 μg Pb/m^3, 8-hour time weighted average. The exposure limit was based on protecting against effects, clinical or subclinical, and the mild symptoms which may occur below 80 μg Pb/dl, providing an adequate margin of safety. The level of 100 μg Pb/m^3 is anticipated to limit blood lead levels in workers to a mean of 40 μg Pb/dl and a maximum of 60 μg Pb/dl. OSHA is presently reviewing the latest information on lead exposure and health effects in preparation for promulgation of the workplace standard for lead.

The Department of Housing and Urban Development (HUD) has requirements for reducing human exposure to lead through the prevention of lead poisoning from ingestion of paint from buildings, especially residential dwellings. Their activities include (1) prohibition of use of lead-based paints on structures constructed or rehabilitated through Federal funding and on all HUD-associated housing; (2) notification of purchasers of HUD-associated housing constructed prior to 1950 that such dwellings may contain lead-based paint; and (3) research activities to develop improved methods of detection and elimination of lead-based paint hazards.

The Consumer Product Safety Commission (CPSC) promulgated regulations in September 1977 which ban 1) paint and other surface coating materials containing more than 0.06 percent lead; 2) toys and other articles intended for use by children bearing paint or other similar surface coating material containing more than 0.06 percent lead; and 3) furniture coated with materials containing more than 0.06 percent lead. These regulations are based on

CPSC's conclusion that it is in the public interest to reduce the risk of lead poisoning to young children from ingestion of paint and other similar surface-coating materials.

The Food and Drug Administration adopted in 1974 a proposed tolerance for lead of 0.3 ppm in evaporated milk and evaporated skim milk. This tolerance is based on maintaining children's blood lead levels below 40 μg Pb/dl. FDA also has a proposed action level of 7 μg/ml for leachable lead in pottery and enamelware, although the exact contribution of such exposure to total human dietary intake has not been established.

The Center for Disease Control concluded in 1975 that undue or increased lead absorption exists when a child has confirmed blood lead levels 30-70 μg Pb/dl or an EP elevation of 60-189 μg Pb/dl except where the elevated EP level is caused by iron deficiency. This guideline is presently accepted by the scientific community but because of more recent data is being reevaluated.

STATE AIR QUALITY STANDARDS

Four states currently have lead air quality standards - California, Pennsylvania, Montana and Oregon. California has the lowest standard of 1.5 μg Pb/m^3, 30-day average, which is based on limiting the portion of blood lead that is air derived to 5 percent if individual values are held to 30 μg Pb/dl or less. California concludes that this standard is consistent with restricting mean blood lead levels to less than 15 μg Pb/dl. Pennsylvania based their standard of 5.0 μg Pb/m^3, 30-day average on the health effects of absorbed lead and concluded that 50 μg/day of lead can be safety absorbed from the air. Assuming a daily respiration volume of 20 m^3 and a 50 percent absorption rate, a maximum of 5 μg/m^3 is

allowed in the air. Montana's standard of 5.0 μg Pb/m^3, 30-day average, was adopted as a goal based on Pennsylvania's experience. Oregon has a standard of 3.0 μg Pb/m^3, 30-day average, which was based primarily on health effects data with some consideration of economic implications.

THE FEDERAL REFERENCE METHOD

The Federal Reference Method for Lead describes the appropriate techniques for determining the concentration of lead and its compounds measured as elemental lead in ambient air. The method is based on measuring the lead content of suspended particulate matter on glass fiber filters using high volume sampling. The lead is then extracted from the particulate matter using nitric acid with heat or ultrasonic energy; finally, the lead content is measured by atomic absorption spectrometry.

The method has received single laboratory evaluation using samples of airborne particulates collected at a number of locations. In addition, four other laboratories have conducted two abbreviated collaborative tests using particulate samples. All available precision and accuracy information from these tests is included in the proposed method. Additional methodological studies will be completed between this date and promulgation.

EPA does not anticipate changing the sampling method or analytical principle involved but may amend the final Federal Reference Method for Lead in any or all of the following ways:

1. Removal of some inherent judgment processes left to the individual analyst.

2. Inclusion of a third extraction procedure which uses aqua regia. This permits the analyst to extract more metals than just lead quantitatively thereby permitting him to analyze the same extract for more than one metal.

3. Although the atomic absorption principle was selected as the method of analysis, other analytical principles appear to be equally applicable and are currently being evaluated. These methods are flameless atomic absorption, optical emission spectrometry, and anodic stripping voltametry. These analytical principles may be included in the final method but probably will be handled via the "equivalent method" route.

PUBLIC PARTICIPATION

All interested persons are invited to comment on all aspects of the proposed standard and the Federal Reference Method. In particular, data, views and arguments are solicited on the level of the standard, and conclusions, assumptions, and calculations used by EPA in selecting that level. Comments should be submitted in duplicate to: Mr. Joseph Padgett, Strategies and Air Standards Division, MD-12, Research Triangle Park, North Carolina 27711.

December 8, 1977
Date

Administrator

The Agency proposes to amend 40 CFR Part 50 by adding the following:

§50.12 National primary and secondary ambient air quality standards for lead

The national ambient air quality standards for lead and its compounds measured as elemental lead by a reference method based on Appendix G to this part, or by an equivalent method, are: 1.5 micrograms per cubic meter--monthly arithmetic mean.

(Sections 109 and 301(a) of the Clean Air Act as Amended (42 U.S.C. 7409, 7601(a))).

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APPENDIX G

REFERENCE METHOD FOR THE DETERMINATION OF LEAD IN SUSPENDED PARTICULATE MATTER COLLECTED FROM AMBIENT AIR

1. Principle and Applicability

1.1 Ambient air suspended particulate matter is collected on a glass-fiber filter for 24-hours using a high volume air sampler.

1.2 Lead in the particulate matter is solubilized by extraction with nitric acid (HNO_3), facilitated by heat or ultrasonication.

1.3 The lead content of the sample is analyzed by atomic absorption spectrometry using an air-acetylene flame, the 283.3 or 217.0 nm lead absorption line, and the optimum instrumental conditions recommended by the manufacturer.

2. Range, Sensitivity and Lower Detectable Limit

The values given below are typical of the methods capabilities. Absolute values will vary for individual situations depending on the type of instrument used, the lead line, and operating conditions.

2.1 Range. The typical range of the method is 0.03 to 7.5 µg Pb/m^3 assuming an upper linear range of analysis of 15 µg/ml and an air volume of 2400 m^3.

2.2 Analytical sensitivity. Typical sensitivities for a 1% change in absorption (0.0044 absorbance units) are 0.2 and 0.5 µg Pb/ml for the 217.0 and 283.3 nm lines, respectively.

2.3 Lower Detectable Limit (LDL). A typical LDL is 0.03 μg Pb/m^3. This LDL is for the 217 nm line. The LDL for the 283.3 nm line will be somewhat higher. The above value was calculated by doubling the between laboratory standard deviation obtained for the lowest measurable lead concentration in a collaborative test of the method.[15] An air volume of 2400 m^3 was assumed.

3. Interferences

Two types of interferences are possible: chemical, and light scattering.

3.1 Chemical. Reports on the absence [1,2,3,4,5] of chemical interferences far outweigh those reporting their presence,[6] therefore, no correction for chemical interferences is given here. If the analyst suspects that the sample matrix is causing a chemical interference, the interference can be verified and corrected for by carrying out the analysis using the method of standard additions.[7]

3.2 Light Scattering. Non-atomic absorption or light scattering, produced by high concentrations of dissolved solids in the sample, can produce a significant interference, especially at low lead concentrations.[2] The interference is greater at the 217.0 nm line than at the 283.3 nm line. No interference was observed using the 283.3 nm line with a similar method.[1]

Light scattering interferences can, however, be corrected for instrumentally. Since the dissolved solids can vary depending on the origin of the sample, the correction may be necessary, especially when using the 217.0 nm line. Dual beam instruments with a continuum source give the most accurate correction. A less accurate correction can be obtained by using a non-absorbing lead line that is near the lead analytical line. Information on use of these correction techniques can be obtained from instrument manufacturers manuals.

If instrumental correction is not feasible, the interference can be eliminated by use of the ammonium pyrrolidinecarbodithioate-methylisobutyl ketone, chelation-solvent extraction technique of sample preparation.[8]

4. Precision and Bias

4.1 The high-volume sampling procedure used to collect ambient air particulate matter has a between laboratory relative standard deviation of 3.7% over the range 80 to 125 $\mu g/m^3$.[9] The following equations give the precision of lead measurements made on 3/4" x 8" strips cut from exposed glass fiber filters using the hot extraction procedure.[15]

$$x = 1.73 + 0.01c$$

$$y = 4.82 + 0.03c$$

where

x = within laboratory standard deviation, µg Pb/strip

y = between laboratory standard deviation, µg Pb/strip

c = measured lead concentration, µg Pb/strip

Similar information is being obtained for the ultrasonic extraction procedure.

4.2 Single laboratory experiments indicate that there is no significant difference in lead recovery between the hot and ultrasonic extraction procedures.[16]

5. Apparatus

5.1 Sampling.

5.1.1 High volume sampler. Use and calibrate the sampler as described in reference 10.

5.2 Analysis.

5.2.1 Atomic Absorption Spectrophotometer. Equipped with lead hollow cathode or electrodeless discharge lamp.

5.2.1.1 Acetylene. The grade recommended by the instrument manufacturer should be used. Change cylinder when pressure drops below 50-100 psig.

5.2.1.2 Air. Filtered to remove particulate, oil and water.

5.2.2 Glassware. Class A borosilicate glassware should be used throughout the analysis.

5.2.2.1 Beakers. 30 and 150 ml. graduated, Pyrex.

5.2.2.2 Volumetric flasks. 100-ml.

5.2.2.3 Pipettes. To deliver 50, 30, 15, 8, 4, 2, 1 ml.

5.2.2.4 Cleaning. All glassware should be scrupulously cleaned. The following procedure is suggested. Wash with laboratory detergent, rinse, soak for 4 hours in 20% (w/w) HNO_3, rinse 3 times with distilled-deionized water, and dry in a dust free manner.

5.2.3 Hot plate.

5.2.4 Ultrasonication water bath, unheated. Commercially available laboratory ultrasonic cleaning baths of 450 watts or higher "cleaning power", i.e., actual ultrasonic power output to the bath have been found satisfactory.

5.2.5 Template. To aid in sectioning the glass-fiber filter. See Figure 1 for dimensions.

5.2.6 Pizza cutter. Thin wheel. Thickness <1 mm.

5.2.7 Watch glass.

5.2.8 Polyethylene bottles. For storage of samples. Linear polyethylene gives better storage stability than other polyethylenes and is preferred.

5.2.9 Parafilm "M".* American Can Company, Marathon Products, Nennah, Wisconsin, or equivalent.

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*Mention of commercial products does not imply endorsement by the Environmental Protection Agency.

6. Reagents

6.1 Sampling.

6.1.1 Glass fiber filters. The specifications given below are intended to aid the user in obtaining high quality filters with reproducible properties. These specifications have been met by EPA contractors.

6.1.1.1 Lead content. The absolute lead content of filters is not critical, but low values are, of course, desirable. EPA typically obtains filters with a lead content of <75 µg/filter.

It is important that the variation in lead content from filter to filter, within a given batch, be small.

6.1.1.2 Testing.

6.1.1.2.1 For large batches of filters (≥ 500 filters) select at random 20 to 30 filters from a given batch. For small batches (< 500 filters) a lesser number of filters may be taken. Cut one 3/4" x 8" strip from each filter anywhere in the filter. Analyze all strips, separately, according to the directions in Sections 7 and 8.

6.1.1.2.2 Calculate the total lead in each filter as

$$F_b = \mu g\ Pb/ml \times \frac{100\ ml}{strip} \times \frac{12\ strips}{filter}$$

where:

F_b = Amount of lead per 72 square inches of filter, µg.

6.1.1.2.3 Calculate the mean, $\overline{F}_b$, of the values and the relative standard deviation (standard deviation/mean x 100). If the relative standard deviation is high enough so that, in the analysts opinion, subtraction of $\overline{F}_b$, (Section 10.3) may result in a significant error in the µg Pb/m^3, the batch should be rejected.

6.1.1.2.4 For acceptable batches, use the value of $\overline{F}_b$ to correct all lead analyses (Section 10.3) of particulate matter collected using that batch of filters If the analyses are below the LDL (Section 2.3) no correction is necessary.

6.2 Analysis.

6.2.1 Concentrated (15.6 M) HNO_3. ACS reagent grade HNO_3 and commercially available redistilled HNO_3 has been found to have sufficiently low lead concentrations.

6.2.2 Distilled-deionized water. (D.I. water).

6.2.3 3 M HNO_3. Add 192 ml of concentrated HNO_3 to D.I. water in a 1 ℓ volumetric flask. Shake well, cool, and dilute to volume with D.I. water. CAUTION: Nitric Acid Fumes Are Toxic. Prepare in a well ventilated fume hood.

6.2.4 0.45 M HNO_3. Add 29 ml of concentrated HNO_3 to D.I. water in a 1 ℓ volumetric flask. Shake well, cool, and dilute to volume with D.I. water.

6.2.5 Lead Nitrate, $Pb(NO_3)_2$. ACS reagent grade, purity 99.0%. Heat for 4 hours at 120°C and cool in a desiccator.

6.3 Calibration standard.

6.3.1 Master standard, 1000 µg Pb/ml. Dissolve 1.598 g of $Pb(NO_3)_2$ in 0.45 M HNO_3 contained in a 1 ℓ volumetric flask and dilute to volume with 0.45 M HNO_3. Store in a polyethylene bottle. Commercially available certified lead standard solutions may also be used.

7. Procedure.

7.1 Sampling. Collect samples for 24 hours using the procedure described in reference 10 with glass-fiber filters meeting the specifications in 6.1.1. Transport collected samples to the laboratory taking care to minimize contamination and loss of sample.

7.2 Sample Preparation.

7.2.1 Hot Extraction Procedure.

7.2.1.1 Cut a 3/4" x 8" strip from the exposed filter using a template and a pizza cutter as described in Figures 1 and 2. Other cutting procedures may be used.

Lead in ambient particulate matter collected on glass fiber filters has been shown to be uniformly distributed across the filter [1,3,11] suggesting that the position of the strip is unimportant. However, other studies [12,17] have shown that when sampling near a road-way lead is not uniformly distributed across the filter. Therefore, when sampling near a road way, additional strips at different positions within the filter should be analyzed.

7.2.1.2 Fold the strip in half twice and place in a 150-ml beaker. Add 15 ml of 3 M HNO_3 to cover the sample. The acid should completely cover the sample. Cover the beaker with a watch glass.

7.2.1.3 Place beaker on the hot-plate, contained in a fume hood, and boil gently for 30 min. Do not let the sample evaporate to dryness. CAUTION: Nitric Acid Fumes Are Toxic.

7.2.1.4 Remove beaker from hot plate and cool to near room temperature.

7.2.1.5 Quantitatively transfer the sample as follows:

7.2.1.5.1 Rinse watch glass and sides of beaker with D.I. water.

7.2.1.5.2 Decant extract and rinsings into a 100-ml volumetric flask.

7.2.1.5.3 Add D.I. water to 40 ml mark on beaker, cover with watch glass, and set aside for a minimum of 30 minutes. This is a critical step and cannot be omitted since it allows the HNO_3 trapped in the filter to diffuse into the rinse water.

7.2.1.5.4 Decant the water from the filter into the volumetric flask.

7.2.1.5.5 Rinse filter and beaker twice with D.I. water and add rinsings to volumetric flask until total volume is 80 to 85 ml.

7.2.1.5.6 Stopper flask and shake vigorously. Set aside for approximately 5 minutes or until foam has dissipated.

7.2.1.5.7 Bring solution to volume with D.I. water. Mix thoroughly.

7.2.1.5.8 Allow solution to settle for one hour before proceeding with analysis.

7.2.1.5.9 If sample is to be stored for subsequent analysis, transfer to a linear polyethylene bottle.

7.2.2 Ultrasonic Extraction Procedure.

7.2.2.1 Cut a 3/4" x 8" strip, fold and place in a beaker as described in Sections 7.2.1.1 and 7.2.1.2 except that a 30-ml beaker covered with Parafilm is used instead of a 150-ml beaker covered with a watch glass. The Parafilm should be placed over the beaker such that none of the Paraflim is in contact with water in the ultrasonic bath. Otherwise, rinsing of the Parafilm (Section 7.2.2.3.1) may contaminate the sample.

7.2.2.2 Place the beaker in the ultrasonication bath and operate for 30 minutes.

7.2.2.3 Quantitatively transfer the sample as follows:

7.2.2.3.1 Rinse Parafilm and sides of beaker with D.I. water.

7.2.2.3.2 Decant extract and rinsings into a 100-ml volumetric flask.

7.2.2.3.3 Add 20 ml D.I. water to cover the filter strip, cover with parafilm, and set aside for a minimum of 30 minutes. This is a critical step and cannot be omitted. The sample is then processed as in Sections 7.2.1.5.4 through 7.2.1.5.9.

NOTE: Samples prepared by either procedure are now in 0.45 M HNO_3.

8. Analysis.

8.1 Set the wavelength of the monochromator at 283.3 or 217.0 nm. Set or align other instrumental operating conditions as recommended by the manufacturer.

8.2 The sample can be analyzed directly from the volumetric flask, or an appropriate amount of sample decanted into a sample analysis tube. In either case, care should be taken not to disturb the settled solids.

8.3 Aspirate samples, calibration standards and blanks (Section 9.2) into the flame and record the equilibrium absorbance.

8.4 Determine the lead concentration in µg Pb/ml, from the calibration curve, Section 9.3.

8.5 Samples that exceed the linear calibration range should be diluted with HNO_3 of the same concentration as the calibration standards and reanalyzed.

9. Calibration.

9.1 Working standard, 20 µg Pb/ml. Prepare by diluting 2.0 ml of Master standard (6.3.1) to 100 ml with 0.45 M HNO_3. Prepare daily.

9.2 Calibration standards. Prepare daily by diluting the working standard with 0.45 M HNO_3 as indicated below. Other concentrations may be used.

Volume of 20 µg/ml Working Standard, ml	Final Volume, ml	Concentration µg Pb/ml
0	100	0.0
1.0	200	0.1
2.0	200	0.2
2.0	100	0.4
4.0	100	0.8
8.0	100	1.6
15.0	100	3.0
30.0	100	6.0
50.0	100	10.0
100	100	20.0

9.3 Preparation of calibration curve. Since the working range of analysis will vary depending on which lead line is used and the type of instrument, no one

set of instructions for preparation of a calibration curve can be given. Select at least six standards (plus the reagent blank) to cover the linear absorption range indicated by the instrument manufacturer. Measure the absorbance of the blank and standards as in Section 8.0. Repeat until good agreement is obtained between replicates. Plot absorbance (y-axis) versus concentration in μg Pb/ml (x-axis). Draw (or compute) a straight line through the linear portion of the curve. Do not force the calibration curve through zero.

To determine stability of the calibration curve, remeasure - alternately - one of the following calibration standards for every 10th sample analyzed: concentration $\leq$ 1 μg Pb/ml; concentration $\leq$ 10 μg Pb/ml. If either standard deviates by more than 5% from the value predicted by the calibration curve, recalibrate and repeat the previous 10 analyses.

10. Calculation.

10.1 Measured air volume. Calculate the measured air volume as

$$V_m = \frac{Q_i + Q_f}{2} \times T$$

where:

V_m = Air volume sampled (uncorrected), m^3

Q_i = Initial air flow rate, m^3/min.

Q_f = Final air flow rate, m^3/min.

T = Sampling Time, min.

The flow rates Q_i and Q_f should be corrected to the temperature and pressure conditions existing at the time of orifice calibration as directed in addendum B of reference 10, before calculation of V_m.

10.2 Air volume at STP. The measured air volume is corrected to reference conditions of 760 mm Hg and 25°C as follows. The units are standard cubic meters, sm^3.

$$V_{STP} = V_m \times \frac{P_2 \times T_1}{P_1 \times T_2}$$

V_{STP} = Sample volume, sm^3, at 760 mm Hg and 298° K

V_m = Measured volume from 10.1

P_2 = Atmospheric pressure at time of orifice calibration, mm Hg

P_1 = 760 mm Hg

T_2 = Atmospheric temperature at time of orifice calibration, °K

T_1 = 298°K

10.3 Lead Concentration. Calculate lead concentration in the air sample.

$$C = \frac{(\mu g\ Pb/ml \times 100\ ml/strip \times 12\ strips/filter) - \overline{F}_b}{V_{STP}}$$

where:

C = Concentration, $\mu g\ Pb/sm^3$

$\mu g\ Pb/ml$ = Lead concentration determined from Section 8

100 ml/strip = Total sample volume

12 strips/filter = $\frac{\text{Useable filter area, 7" x 9"}}{\text{Exposed area of one strip, 3/4" x 7"}}$

$\overline{F}_b$ = Lead concentration of blank filter, µg, from Section 6.1.1.2.3

V_{STP} = Air volume from 10. 2

1. Quality Control.

3/4" x 8" glass fiber filter strips containing 80 to 2000 µg Pb/strip as lead salts) and blank strips with zero Pb content should be used to letermine if the method - as being used - has any bias. Quality control charts should ›e established to monitor differences between measured and true values. The frequency ›f such checks will depend on the local quality control program.

To minimize the possibility of generating unreliable data, the user should ollow practices established for assuring the quality of air pollution data,[13] nd take part in EPA's semi-annual audit program for lead analyses.

2. Trouble Shooting.

1. During extraction of lead by the hot extraction procedure, it is mportant to keep the sample covered so that corrosion products - formed on ume hood surfaces which may contain lead - are not deposited in the extract.

2. The sample acid concentration of 0.45 M should minimize corrosion f the nebulizer. However, different nebulizers may require lower acid oncentrations. Lower concentrations can be used provided samples and tandards have the same acid concentration.

3. Ashing of particulate samples has been found, by EPA and contractor aboratories, to be unnecessary in lead analyses by Atomic Absorption. herefore, this step was omitted from the method.

4. Filtration of extracted samples, to remove particulate matter, was pecifically excluded from sample preparation, because some analysts have observed osses of lead due to filtration.

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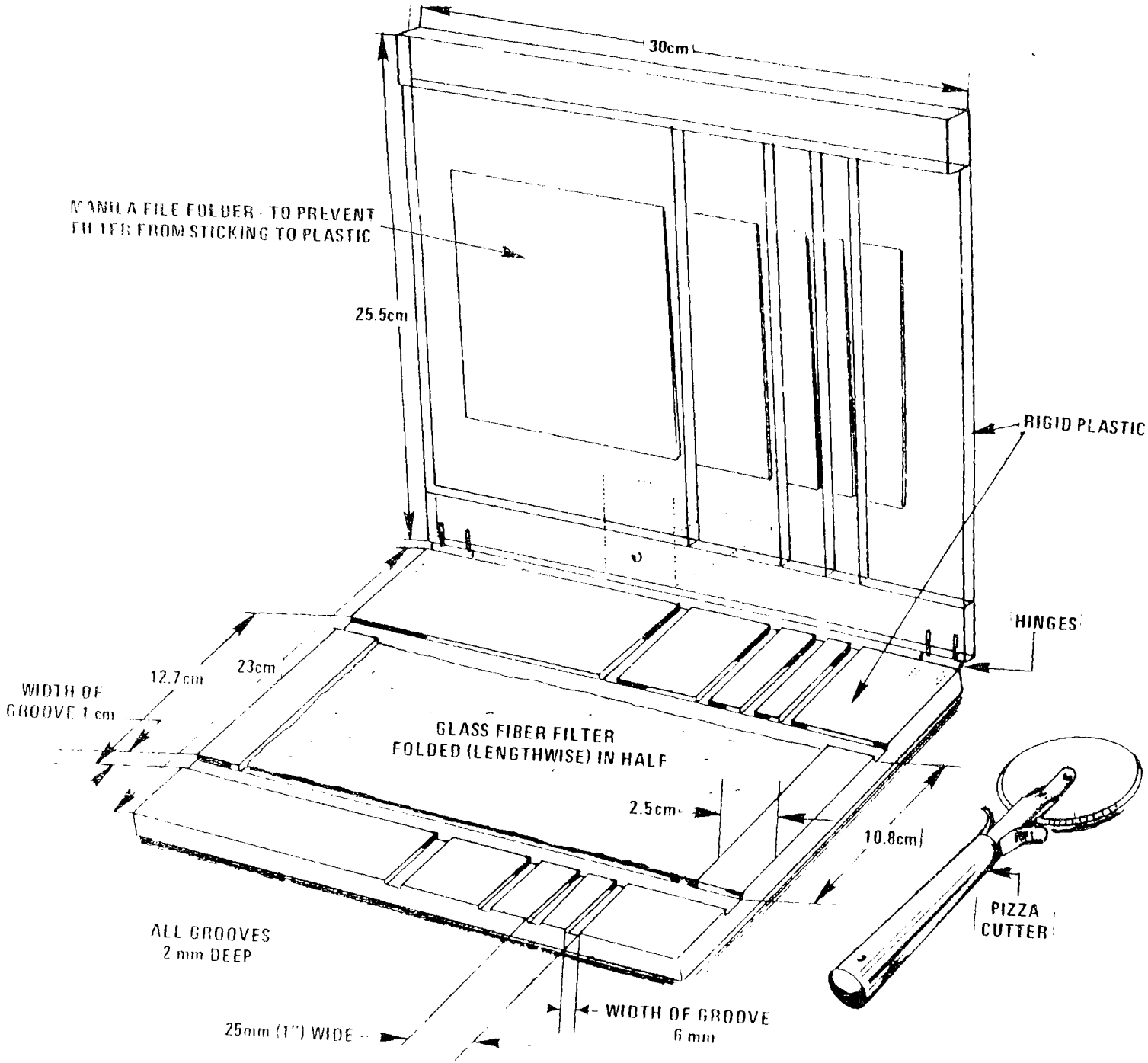
30cm
MANILA FILE FOLDER - TO PREVENT
FILTER FROM STICKING TO PLASTIC
25.5cm
RIGID PLASTIC
HINGES
12.7cm
23cm
WIDTH OF
GROOVE 1 cm
GLASS FIBER FILTER
FOLDED (LENGTHWISE) IN HALF
2.5cm
10.8cm
PIZZA
CUTTER
ALL GROOVES
2 mm DEEP
25mm (1") WIDE
WIDTH OF GROOVE
6 mm

Figure 1

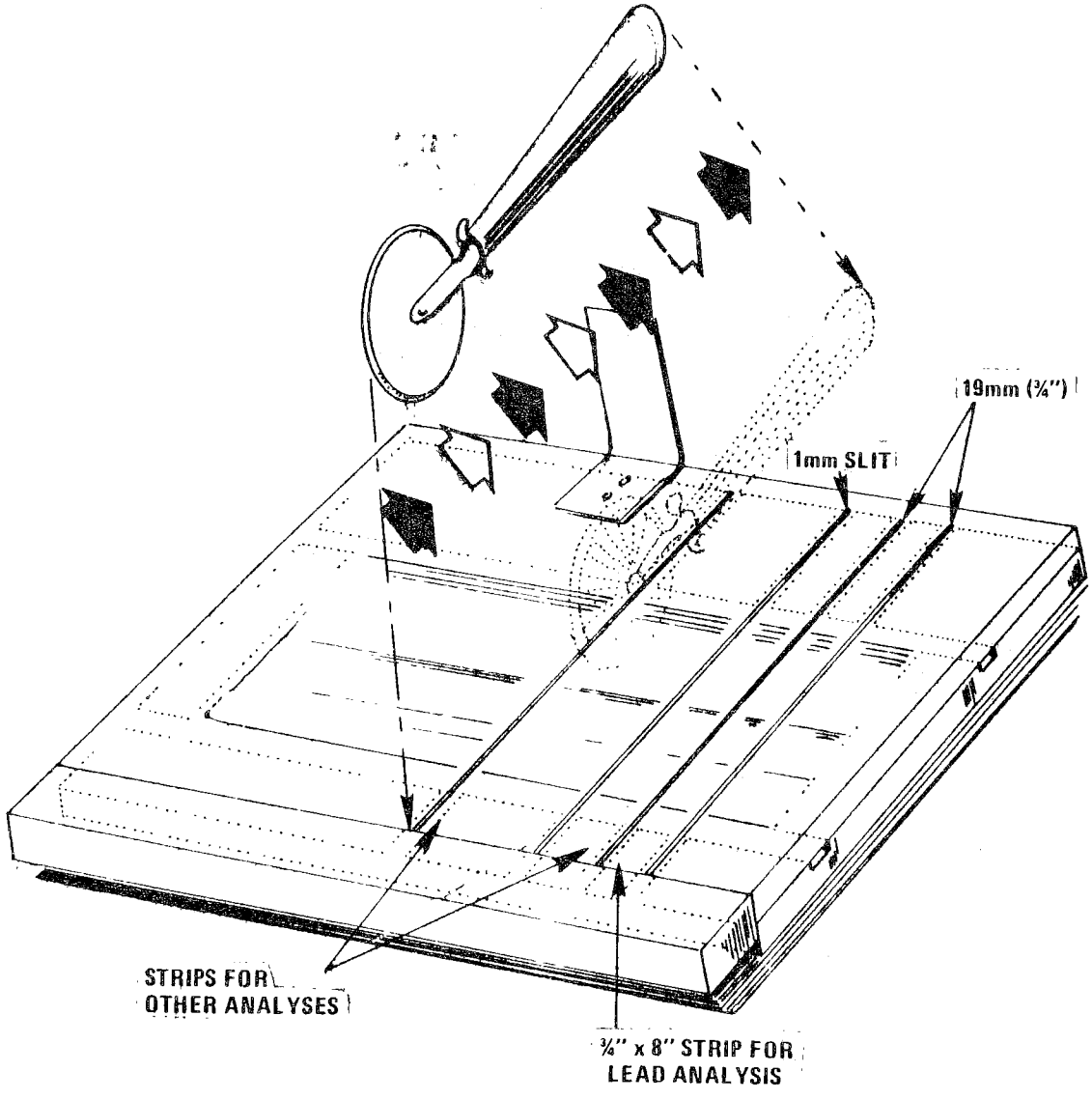
19mm (¾")
1mm SLIT
STRIPS FOR
OTHER ANALYSES
¾" x 8" STRIP FOR
LEAD ANALYSIS

Figure 2