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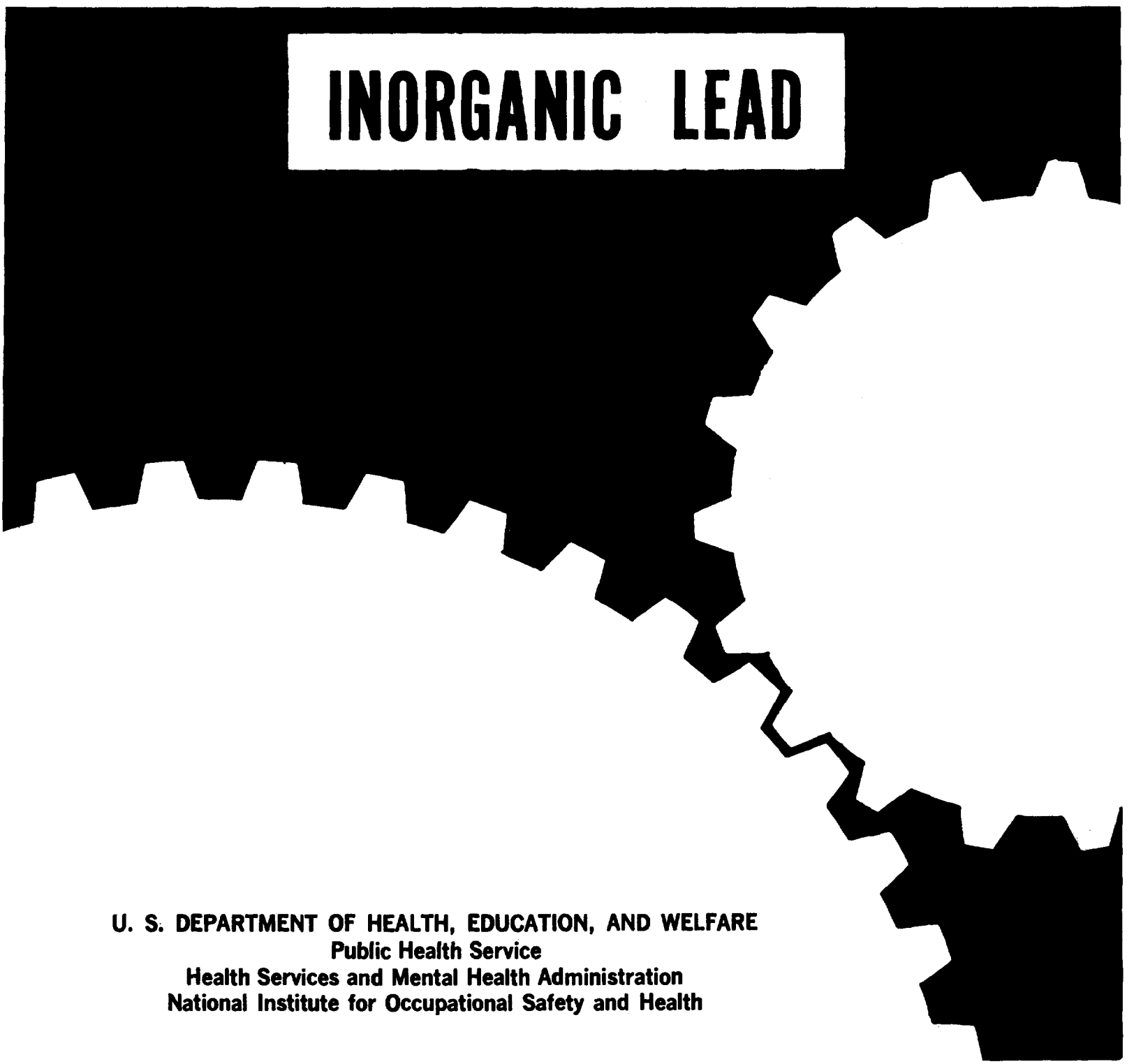
OCCUPATIONAL EXPOSURE TO INORGANIC LEAD



U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
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1972

PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace. To provide relevant data from which valid criteria and effective standards can be deduced, the National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices.

It is intended to present successive reports as research and epidemiologic studies are completed and sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on inorganic lead by my staff and the valuable constructive comments by the Review Consultants on Inorganic Lead, by the ad hoc committee of the American Academy of Industrial Hygiene, by Robert B. O'Connor, M. D., NIOSH consultant in occupational medicine, and Edwin C. Hyatt on respiratory protection. The NIOSH recommendations for standards are not necessarily a consensus of all of the consultants and professional societies that reviewed this criteria document on inorganic lead. A list of the NIOSH Review Committee members and of the Review Consultants appears on pages iii and iv.

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The Office of Research and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for inorganic lead. Keith H. Jacobson, Ph.D., had program responsibility and Robert E. Seiter served as criteria manager.

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CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR INORGANIC LEAD

Table of Contents

I. RECOMMENDATION FOR AN INORGANIC LEAD STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that employee exposure to inorganic lead in the workplace be controlled by adherence to the following sections. The standard is designed to protect the health and safety of workers for an 8-hour day, 40-hour week over a working lifetime; compliance with the standard should therefore prevent adverse effects of lead on the health and safety of workers. The standard is measurable by techniques that are valid, reproducible, and available to industry and government agencies. Sufficient technology exists to permit compliance with the recommended standard. The criteria and standard will be subject to review and revision as necessary.

"Inorganic lead" means lead oxides, metallic lead, and lead salts (including organic salts such as lead soaps but excluding lead arsenate). "Exposure to inorganic lead" is defined as exposure above half the recommended workroom environmental standard. Exposures at lower environmental concentrations will not require adherence to the following sections, except for Section 7(a).

Section 1 - Environmental (workplace air)

(a) Concentration

Occupational exposure to inorganic lead shall be controlled so that workers shall not be exposed to inorganic lead at a concentration greater that 0.15 mg Pb/m^3 determined as a time-weighted average (TWA) exposure for an 8-hour workday.

(b) Sampling, Collection, and Analysis

Procedures for collection of environmental samples shall be as provided in Appendix I, or by an equivalent method. Analysis of samples shall be as provided in Appendix II, or by any method shown to be equivalent in precision and accuracy to the method specified in Appendix II.

Section 2 - Medical

Medical monitoring (biologic monitoring and medical examinations) shall be made available to workers as outlined below.

(a) Biologic monitoring

Biologic monitoring shall be made available to all workers subject to "exposure to inorganic lead." It consists of sampling and analysis of whole blood, or alternatively, of urine for lead content. Such monitoring shall be performed to ensure that no worker absorbs an unacceptable amount of lead. Unacceptable absorption of lead posing a risk of lead poisoning is demonstrated at levels of 0.080 mg Pb/100 g of whole blood or greater, or at levels of 0.20 mg Pb/liter of urine (with urine specific gravity corrected to 1.024) or greater.

Procedures for sampling and analysis of blood or urine for lead shall be as described in Appendix II, or by any method shown to be equivalent in precision and accuracy. In the case of urine, "spot" urine specimens of about 100 ml shall be collected during a workday, and urine specimens with a specific gravity less than 1.010 shall be discarded and another sample obtained.

Half of all workers subject to "exposure to inorganic lead" shall be offered biologic monitoring every 6 months, so that each worker

shall have blood sampling and analysis made available to him yearly. If urine sampling and analysis are chosen instead of blood lead sampling and analysis to satisfy the biologic monitoring requirement, every worker shall have urine sampling and analysis made available to him at 6 month intervals. The schedule of biologic monitoring, above, may be altered if indicated by a professional industrial hygiene survey. If environmental sampling and analysis show that environmental levels are at or greater than the environmental limit, the interval of biologic monitoring shall be halved, i.e. blood analysis shall be conducted quarterly, with each worker sampled semi-annually, or urinalysis shall be conducted quarterly on every worker. This increased frequency shall be continued for at least 6 months after the high environmental level has been shown.

If a worker's urine lead level is found to be 0.20 mg/liter or greater, calculated to a specific gravity of 1.024, a blood sample shall be obtained and analyzed within two weeks. If a blood lead level of 0.080 mg Pb/100 g or greater is found, and confirmed by a second sample to be taken within two weeks, steps to reduce his absorption of lead shall be taken as soon as the high levels are confirmed. Steps to be considered should include improvement of environmental controls, of personal protection or personal hygiene, and use of administrative controls. A medical examination for possible lead poisoning shall be made available, and the OSHA area industrial hygienist shall be informed of the results of the biologic

sampling of those workers with confirmed, high biologic levels of lead.

Biologic monitoring shall also be made available where the OSHA area industrial hygienist has reason to believe operations produce unusual exposure excursions or that environmental samples do not adequately describe worker exposure.

(b) Medical examination

Medical examinations shall be available when a variance has been granted permitting administrative controls or use of respiratory protection, for workers with unacceptable absorption of lead as judged by biologic monitoring, or when environmental levels are at or above the environmental standard.

These examinations should be made available prior to employee placement and annually thereafter. They should include a physical examination, complete blood counts, blood lead determinations, routine urinalysis (specific gravity, sugar and protein determinations, and microscopic examination), and should record any signs or symptoms of plumbism, if present. Where urine is selected instead of blood for biologic monitoring, the preplacement examination should also include a urinary lead determination. Each employee who absorbs unacceptable amounts of lead as indicated by biologic monitoring shall be examined as soon as practicable after such absorption is demonstrated and confirmed, and at least every 3 months thereafter until his blood or urine lead levels have returned to normal, i.e. below 0.080 mg/100 g of blood or 0.20 mg/liter of urine. If clinical evidence of plumbism

is developed from these medical examinations, the worker shall be kept under a physician's care, in accordance with applicable Workman's Compensation provisions, until the worker has completely recovered or maximal improvement has occurred.

Medical records shall include information on all biologic determinations and on all required medical examinations. These records shall be available to the medical representatives of the employer, of the Secretary of Labor, of the Secretary of Health, Education, and Welfare, and, at the employee's request, to the employee's physician. These records shall be kept for at least five years after the last occupational exposure to inorganic lead.

Section 3 - Labeling (Posting)

Areas where exposure to lead at levels over one-half the workroom air standard is likely to occur shall be posted with a sign reading:

LEAD (Pb)

DANGER!

High concentrations of fume or dust
may be hazardous to health.
Provide adequate ventilation.

If environmental levels are at or greater than the environmental limit, or if a variance permitting use of respiratory controls has been granted, add information to the label or placard describing the location of the respirators.

Section 4 - Personal Protective Equipment and Work Clothing

Subsection (a) shall apply whenever a variance from the standard recommended in Section 1 is granted under provisions of the Occupational Safety and Health Act, or in the interim period during the application for a variance. When the limits of exposure to lead prescribed in paragraph (a) of Section 1 cannot be met by limiting the concentration of lead in the work environment, an employer must utilize, as provided in subsection (a) of this Section, a program of respiratory protection to effect the required protection of every worker exposed.

(a) Respiratory Protection

Engineering controls shall be used wherever feasible to maintain lead dust and fume concentrations below the prescribed limits. Appropriate respirators shall be provided and used when a variance has been granted to allow respirators as a means of control of exposure to routine operations and while the application is pending. Administrative controls should also be used to reduce exposure. Pespirators shall also be provided and used for nonroutine operations (occasional brief exposures above the TWA of 0.15 mg/m^3 and for emergencies); however, for these instances a variance is not required but the requirements set forth below continue to apply. Appropriate

respirators as described in Table I-1 shall only be used pursuant to the following requirements:

(1) For the purpose of determining the class of respirator to be used, the employer shall measure the atmospheric concentration of inorganic lead in the workplace when the initial application for variance is made and thereafter whenever process, worksite, climate or control changes occur which are likely to affect the lead concentration. The employer shall test for respirator fit and/or make lead measurements within the respiratory inlet covering to ensure that no worker is being exposed to inorganic lead in excess of the standard either because of improper respirator selection or fit.

(2) Employees experiencing breathing difficulty while using respirators shall be referred to a physician for evaluation. This evaluation should investigate if the employee has adequate ventilatory capacity and any evidence of obstructive lung disease. Employees with inadequate ventilatory capacity or evidence of obstructive lung disease shall not wear types A, B, and E respirators.

(3) A respiratory protective program meeting the general requirements outlined in section 3.5 of American National Standard for Respiratory Protection Z88.2-1969 shall be established and enforced by the employer.

(4) The employer shall provide respirators in accordance with the Table below and shall assure that the employee uses the respirator provided.

(5) If both fume and dust are present, the recommended usage is that for fume.

(6) Respiratory protective devices described in the following Table I-1 shall be either those approved under the following listed regulation or those approved under 30 CFR 11, published March 25, 1972. The termination date of currently approved respirators described in 30 CFR 11 shall apply.

(i) Reusable or replaceable filter-type air-purifying respirator - - - 30 CFR 14 (Bureau of Mines Schedule 21 B)

(ii) Powered air-purifying positive-pressure respirator - - - 30 CFR 14 (Bureau of Mines Schedule 21 B)

(iii) Type C positive-pressure supplied air respirator - - 30 CFR 12 (Bureau of Mines Schedule 19 B)

(7) Usage of a respirator specified for use in higher concentrations of lead is permitted in atmospheres of lower concentrations.

(8) Employees shall be given instruction on the use of respirators assigned to them, cleaning of the respirators, and how to test for leakage.

TABLE I-1

Requirements for Respirator Usage

at Concentrations Above the Standard

Exposure	8 Hr TWA mg/m^3	*Respirator type
Inorganic lead dust	less than 1.5	A,B
	less than 15.0	C
	greater than 15.0	D
Inorganic lead fume	less than 1.5	E
	less than 15.0	F
	greater than 15.0	D

* A - Reusable or replaceable filter-type air-purifying dust respirator

B - Single-use dust respirator

C - Powered air-purifying positive-pressure dust respirator

D - Type C positive-pressure supplied air respirator

E - Replaceable filter-type air-purifying fume respirator

F - Powered air-purifying positive-pressure fume respirator

(b) Work Clothing

(1) Each employee subject to exposure above the environmental standard of Section 1 should wear coveralls or similar full body work clothing and hat, which should be worn during the working hours in areas where there is exposure to lead. Workers subject to "exposure to inorganic lead" at or below the recommended standard should change into work clothing before starting work, and should remove work clothing before leaving work. This work clothing need not afford full body coverage.

(2) Work clothing should be vacuumed before removal. Clothes shall not be cleaned by blowing or shaking.

(3) Work clothing should be changed at least twice a week and more frequent changes, especially in high exposure areas, are suggested.

(4) Adequate shower facilities should be available and used.

(5) When in the judgment of the OSHA area industrial hygienist contamination of clothing or exposed body surfaces can produce significant secondary exposures, items (1), (2), (3), and (4) above shall be mandatory.

Section 5 - Apprisal of Employees of Hazards from Lead

(a) Each employee exposed to lead shall be apprised at the beginning of his employment or assignment to a lead area of all hazards, relevant symptoms, appropriate emergency procedures, and proper conditions and precautions for safe use or exposure and shall be instructed as to availability of such information which shall be kept on file including that prescribed in (b) below and shall be accessible to the worker at each place of employment where lead is involved in unit processes and operations.

(b) Information as specified in Appendix III shall be recorded on U. S. Department of Labor Form OSHA-20, "Material Safety Data Sheet", (see page IX-4 and IX-5), or on a similar form approved by the Occupational Safety and Health Administration, U. S. Department of Labor.

Section 6 - Work Practices

(a) Emergency Procedures

(1) Procedures including fire fighting procedures shall be established and implemented to meet foreseeable emergency events.

(2) Respirators shall be available for wearing during evacuation procedures if long distances need to be traversed; supplied air respirators shall be available for employee use where equipment or operations cannot be abandoned.

(b) Exhaust Systems

Where a local exhaust ventilation and collection system is used, it shall be designed and maintained to prevent the accumulation of lead dust and fume.

(1) Hazardous types of exposure should not be scattered throughout a plant but, rather, concentrated in a single area where special control procedures can be utilized.

(2) Air from the exhaust ventilation systems shall not be recirculated into the workroom, and should not be discharged outside the plant so as to create an air pollution problem.

(c) General Housekeeping

(1) Vacuuming shall be used wherever practicable and no dry sweeping or blowing shall be performed.

(2) Emphasis shall be placed upon cleanup of spills, periodic repair of equipment and leaks, proper storage of materials, and collection of lead-containing dust.

Section 7 - Sanitation Practices

(a) Food Facilities

Food preparation, dispensing (including vending machines), and eating shall be prohibited in lead work areas.

(b) Locker Facilities

Work and street clothing should not be stored in the same locker.

Section 8 - Monitoring, Recordkeeping, and Reporting Requirements

Workroom areas where it has been determined, on the basis of an industrial hygiene survey or the judgment of a compliance officer, that environmental levels do not exceed half the environmental standard shall not be considered to have inorganic lead exposure. Records of these surveys, including the basis for concluding that air levels are below half the environmental standard, shall be kept.

Requirements set forth below apply to inorganic lead exposures.

(a) Employers shall monitor environmental levels of lead at least every 6 months, except as otherwise indicated by a professional industrial hygiene survey. If environmental levels are at or above the standard, environmental levels shall be monitored every 3 months. This increased frequency of monitoring shall be continued at least 6 months (i.e. two more quarterly monitoring periods) after the last sampling that demonstrated levels at or above the environmental limit.

Periodic environmental sampling shall be performed to coincide with periodic biologic sampling, i.e. shall be performed within 2 weeks of biologic sampling.

Breathing zone samples shall be collected to permit construction of a time-weighted average exposure for every operation. The following number of samples shall be collected and analyzed, as a minimum, based on the number of workers exposed in any given work area:

Number of Employees Exposed	Number of Samples
1-20	50% of the number of workers
20-100	10 samples plus 25% of the excess over 20 workers
over 100	25 samples plus 5% of the excess over 100 workers

(b) When any time-weighted average exposure is at or above the environmental standard, the OSHA area industrial hygienist shall be notified.

(c) Records shall be maintained for all sampling schedules to include the sampling methods, analytical methods, type of respiratory protection in use (if applicable), and the concentrations of lead in each work area. Records shall be maintained so that they can be classified by employee. Each employee shall be able to obtain information on his own environmental exposure.

(d) Medical records shall include information on all biologic determinations and of all required medical examinations. These records shall be kept for at least five years following the last occupational exposure to inorganic lead.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to inorganic lead. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to ". . . develop criteria dealing with toxic materials and harmful physical agents and substances which will describe . . . exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that any recommended criteria for a standard should enable management and labor to develop better engineering controls resulting in more healthful work practices and should not be used as a final goal.

These criteria for a standard for inorganic lead are in a continuing series of criteria developed by NIOSH. The proposed standard applies only to the processing, manufacture, and use of lead products as applicable under the Occupational Safety and Health Act of 1970.

The occupational safety and health aspects of the mining and milling of lead ores are covered by provisions of the Federal Metal and Non-metallic Mine Safety Act (30 U.S.C. 725 et seq.) under which provisions the Bureau of Mines has responsibility.

These criteria were developed to assure that the standard based thereon would (1) protect against development of acute and chronic plumbism, (2) be measureable by techniques that are valid, reproducible, and available to industry and governmental agencies, and (3) be attainable with existing technology.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

In excess of a million tons of lead are processed yearly. The total usage of lead has remained relatively stable during recent years, but the consumption by various industries has changed. For example, there has been a decrease of lead usage in the manufacture of house paints and a simultaneous increase in the manufacture of lead storage batteries. The particular properties of lead (Table X-1[1]) have made it useful for many applications.

Scrutiny of Table X-2 (from U. S. Bureau of Mines[2]) gives an idea as to the relative proportion of lead usage for various industries. Metal products and miscellaneous categories account for the bulk of lead consumption. The refining and processing necessary to form these products include heating, grinding, and volatilization and therefore produce potentially hazardous industrial atmospheres. The impression should not be left that all workers in these industries are jeopardized, but rather that such uses of lead places them at risk of lead absorption.

Table X-3 (Gafafer[3]) lists specific occupations and trades where lead exposure occurs. The diversity of occupations displayed in these tables shows why a precise measure of the extent of lead exposure is non-existent. The National Academy of Sciences' recently published document on lead[4] agrees, stating, "A reliable definition of the extent of risk of occupational lead exposure is unavailable." Because of the changing usage of lead in industry and the widely varied trades where exposure occurs, the United States has no reporting system whereby the prevalence of occupational lead poisoning can be analyzed.

Consider Table X-4[5] which gives examples of general exposure from industrial operations utilizing lead. Simultaneous examination of these tables should give at least a general overview of the extent of occupational exposure to lead. Specific levels for operations within lead-using industries are presented in Part IV, Environmental Data.

Historical

Lead has been used for thousands of years because of its availability and desirable properties. Its low melting point (327 C), ductility, malleability, and weathering resistance enabled its use without the need for the more complex equipment that, in modern times, has enabled the use of other metals such as steel that have more desirable properties for many applications.

In the 1800's, there was an increasing recognition of hazards to health associated with lead. It was found that lead could be absorbed by inhalation and ingestion, and that lead absorption was responsible for loss of movement in printers' fingers exposed to heated lead type and for "dry grippes" in pottery and glass workers. In 1839, Tanquerel des Planches[6] published a treatise on lead diseases, to which Dana later added notes on the effects of using lead pipes. Progress in recognizing signs of lead absorption was made during the 19th Century also. Burton,[7] described in 1840 the "Burtonian Line", a blue line on the gums, as a sign of lead absorption, and chemical methods for detection of lead in blood or urine were developed.

The prevalence of lead poisoning in ancient times is speculated upon, and it has been suggested that Rome fell because of the prevalence of lead

poisoning (plumbism) in its citizens. It seems likely that, with the ignorance that existed on the hazards of lead and on methods of limiting exposure, there was a significant incidence of plumbism until its recognition in recent times generated preventive procedures.

Effects on Humans

A description of effects of lead absorption can be graphic if based on effects seen in industries earlier in this century. Thus, Mayers[8] can describe effects of lead poisoning, from studies of many years ago, such as loss of appetite, metallic taste in the mouth, constipation and obstipation, anemia, pallor, malaise, weakness, insomnia, headache, nervous irritability, muscle and joint pains, fine tremors, encephalopathy, and colic. In lead colic, there may be severe abdominal pain, such that abdominal surgery has occasionally been performed. In workers, as pointed out by Mayers,[8] who have had repeated attacks of lead colic over many years, there is a tendency towards the occurrence of weakness of extensor muscle groups. This weakness may progress to palsy, often observed as a characteristic "wrist drop" or "foot drop."

The important routes of absorption of lead by man and animals are ingestion and inhalation. Eating of lead-bearing paint by children and drinking of lead-contaminated, illicitly distilled whiskey are important sources of non-industrial poisoning. Other sources include exposure to buring battery casings, drinking of liquids from improperly fired, lead-glazed containers, and high levels of airborne lead. But man absorbs lead in small amounts not normally leading to poisoning from his food and water, and from the air. These sources lead to the "normal" body burden

of lead. Thus, the lead absorbed in the course of occupational exposure is superimposed on lead abosrbed from other means.

Descriptions of lead poisoning appear in many texts and reviews, for example Airborne Lead in Perspective, a report of the National Academy of Sciences,[4] and The Diseases of Occupations by Hunter.[9] The rest of this section pertains to the occupational aspects of lead poisoning, with a few notes on effects seen only in children.

Lead can interfere with the synthesis of heme, thereby altering the urinary or blood concentration of enzymes and intermediates in heme synthesis or their derivatives. Thus, lead poisoning can lead to accumulation of non-heme iron and protoporphyrin-9 in red blood cells, an increase in delta-aminolevulinic acid (ALA) in blood and urine, an increase in urinary coproporphyrin, uroporphyrin, and porphobilinogen, inhibition of blood ALA-dehydratase (ALA-D), and an increased proportion of immature red cells in the blood (reticulocytes and basophilic stippled cells).

Anemia from lead poisoning is associated with a reduced red cell life span and with reticulocytosis and basophilic stippled cells in peripheral blood. Symptoms of this anemia include irritability, fatigue, pallor, and sallow complexion. Bone marrow preparations show increased numbers of sideroblasts, and this is useful in differential diagnosis of lead poisoning from iron deficiency anemia.

Gastrointestinal sequelae of lead poisoning include intestinal colic, nausea often without vomiting, and constipation (or, occasionally, diarrhea). Headache usually occurs before or about the time of onset of colic.

Peripheral and central nervous system effects occur in severe poisoning. Peripheral neuropathy of lead poisoning involves considerable loss of motor function but little loss of sensory function. Extensor muscles of the hand and feet are often involved; extensor weakness normally precedes wrist drop or palsy.

Encephalopathy may be either acute or chronic. Acute encephalopathy may follow ingestion or inhalation of large amounts of lead, and may develop quickly to seizures, coma, and death from cardiorespiratory arrest. Chronic encephalopathy usually occurs in children after excessive ingestion of lead, and leads to loss of motor skills and of speech, and to development of behavioral disorders. Lead encephalopathy, often involving psychosis, also occurs from absorption of alkyl lead compounds.

Nephropathy is another effect of lead poisoning. There may be a progressive and irreversible loss of kidney function, with progressive azotemia, and occasionally hyperuricemia with or without gout. Children have developed renal dwarfism, hypertension, marked retention of urea, and low urinary concentration; some children with acute encephalopathy have developed a form of Fanconi syndrome, a kidney disease indicative of severe injury of the promixal renal tubules. Nephritis in adults is not common, but ischemic nephritis may occur after prolonged absorption of lead.

Epidemiologic Studies

Lane[10] examined the causes of death of storage battery workers, including retired workers, and compared data from this group with data from all English and Welsh males of similar ages during the same period of time. Among the retirees who had been exposed to lead, there were found to be greater numbers of deaths than would have been expected, for their ages, from data on the population as a whole. Most of this excess in expected mortality was accounted for by vascular lesions in the central nervous system. Lead workers who died during employment also showed an excess of deaths from this cause.

Another study of electric storage battery workers was conducted by the Public Health Service over 30 years ago.[11] In this study, the incidence of various disease states was studied in relation to lead exposure of 766 workers, most of whom (75%) had worked in storage battery plants for more than five years and some of whom (12%) had worked there for twenty years or more. The incidence of disease (other than plumbism) in men exposed at levels of 0.15 mg/m^3 and higher (high exposure group) was compared to the incidence in men exposed below 0.15 mg/m^3 (low exposure group). Special attention was given to cardiovascular disease because of the common belief that chronic plumbism results in arteriosclerosis; however, the data developed by the PHS team did not show that more severe exposure to lead is associated with a significantly higher incidence of vascular disease. The incidence of arteriosclerotic-hypertensive disease was not significantly different in the high and low exposure groups. The responses to a standard

exercise, in terms of return to pre-exercise pulse rates and to systolic and diastolic blood pressure, were also compared, and again the two groups were found not to be significantly different from each other. These lead workers were also found not to be significantly different from other, non-lead, workers in terms of blood pressure. From this, it was concluded that exposure to lead in the storage battery industry does not cause cardiovascular effects.

A contrary conclusion was reached by Dingwall-Fordyce and Lane[12] in a study of British battery workers. A significant excess of deaths from cerebrovascular accidents was found in pensioners who had had exposure to lead of sufficient degree to have caused mean urinary lead levels of 0.25 mg/liter during many years of lead work. They compared three groups of workers--those with no occupational lead exposure, those with negligible exposure, and those occupationally exposed to lead*--with the general population of English and Welsh males of similar ages. They found a significant excess of death, over that predictable from the population at large, among retirees in the highest exposure group, and this was largely attributable to cerebrovascular accidents. They also examined records of deaths due to cancer in lead workers, both employed and retired, and concluded that there was no association between malignant disease and lead absorption. While they found an excess of deaths from cancer in the negligible exposure group (in the last decade of the 35-year figures only), there was

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*Urinary lead levels in this group averaged between 0.10 and 0.25 mg/liter for a 20-year period.

a slight decrease in deaths, from that expected from statistics on the whole population, among workers absorbing more than negligible amounts of lead, hence their conclusion that malignant disease is not related to lead absorption. As improved working conditions decreased lead exposure, the excess of cerebrovascular deaths diminished.

Malcolm[13] recently conducted similar investigations of past and present employees exposed to lead. Since 1927, airborne lead to which these men had been exposed had been limited to 0.15 mg/m^3, according to Malcolm. He divided the workers into three groups: (A) no exposure, (B) mild exposure, and (C) severe exposure. Average blood lead* in group (C) workers, since 1961, has been 0.065 mg/100 g, from which it may be inferred that the 0.15 mg/m^3 air concentration was sometimes exceeded. Urinary leads in subgroups averaged 0.09 to 0.180 mg/liter, and averaged 0.119 mg/liter for the entire group of workers.

Based on comparison of blood pressures of the two exposed groups (B and C) with the control group (A), it was concluded that there was no occupationally induced hypertension (although there might have been lead-induced hypertension before improved hygienic measures were instituted). There was a non-significant increase in chest disease among older retired workers, attributed to other causes, since most of these pensioners lived in an urban area with a higher rate of death from chest disease than that in the country as a whole.

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*Concentrations of lead in blood are expressed as weight units (such as mg) per 100 ml or 100 g of whole blood. European workers more commonly express blood lead as weight units per 100 ml of blood, while American workers more commonly express blood lead as weight units per 100 g of blood. This document will follow the American custom except in referring to studies reporting blood lead in weight units per 100 ml. The difference between the two expressions is small, about 5% or less. Thus, a blood lead concentration of 0.080 mg/100 g would be equivalent to about 0.084 mg/100 ml.

Unlike the findings of earlier investigators (Dingwall-Fordyce and Lane,[12] and Lane[10]) Malcolm found no evidence of increased frequency of cerebrovascular death in his study, which included deaths occurring between 1963 and 1967, while data from the two earlier reports included deaths from 1926 to 1960. Thus, if all three reports are correct in their conclusions, it would seem that improving hygiene has diminished lead-induced cerebrovascular disease.

For years, chronic nephritis was thought to be a consequence of plumbism, and an analysis of death rates in the U.K. in 1921[10] and in 1931[13] shows a considerable excess in plumbers and painters due to nephritis and to cerebrovascular disease. The question of nephropathy from lead has also been raised by Henderson and Inglis,[14] who showed a relationship between chronic nephritis and excessive lead absorption as indicated by elevated lead levels in bone.

Lane[15] described 9 deaths from renal failure in lead workers, men who had been exposed for long periods at lead concentrations around 0.5 mg/m^3. Terminally, they all had evidence of chronic azotemic nephritis. These men, all of whom worked in storage battery industries for over 20 years, died between the ages of 42 and 52 (average age at death was 48.4). Other than two episodes of colic, there had been no previous history of lead intoxication.

In the United States, there have been few reports of renal disease in lead workers,[13] though the PHS survey of storage battery workers discovered an increased incidence of albuminuria in affected workers.

Animal Toxicity

Unlike toxicologic studies of many industrial substances, experimental animal studies of either inorganic or organic lead have contributed far less to an understanding of the toxicology of lead and its compounds than studies on man, and hence have directly contributed very little to the criteria for the standard for lead. The reason is that until recently, much of the investigative effort was directed to the effects of lead on the red blood cell, its urinary intermediates and lead content of blood and urine, all readily investigated in man. Moreover, many of the studies in man or animals relate to detecting changes in biologic constituents of the blood and urine, and hence are relevant more to criteria for biologic standards than to air standards. Thus, the experimental studies discussed herein will be confined to those that confirm or extend the findings in man in these areas and which are related, even if only indirectly, to the criteria for the air standard.

In recent years, research investigations have broadened to include biologic systems other than the erythropoietic, and in this way may ultimately provide new criteria for standards. Lead intoxication has been studied for its effects on the rat thyroid, comparative changes in kidneys of rat and man, and the effect of certain trace metal deficiencies on the toxicity of lead. But only a beginning has been made in our understanding of the action of lead on the nervous system; behavioral effects have been studied in rats following exposure to tetraethyl lead after the finding of marked metabolic changes in the brain from its administration.

a. Experimental Animal Toxicology. The USPHS-sponsored conference on environmental lead[16] in 1965, although oriented towards the community

environment, marked a turning point in experimental animal investigations on lead. Up to this time, animal studies relating to standards criteria used hematologic disturbances for the most part as a focal point of investigations because of their practical usefulness as criteria for judging harmful exposures to lead.

b. Biosynthesis of Heme. Following the first evidence by Rimington[17,18] that lead interfered with the incorporation of iron into the protoporphyrin molecule, and the subsequent demonstration by Eriksen[19] and others that lead also interfered with an early step in heme synthesis catalyzed by delta-amino-levulinic acid dehydratase (ALA-D), Kreimer-Birnbaum and Grinstein[20] confirmed in rabbits poisoned by lead the earlier findings of Eriksen and others. As determination of ALA-D in the red blood cell became recognized as the most sensitive criterion of response to lead exposure yet discovered, it was applied to the control of lead exposures among industrial workers. It was soon suspected, however, when red cell ALA-D was markedly inhibited in the absence of subjective symptoms of lead poisoning and at blood levels within currently accepted normal limits[21-23] that, as a criterion for overexposure of lead workers, ALA-D was of less value than had been anticipated. Studies in dogs[23] confirmed this suspicion; dogs that had been given lead acetate for a period (46 weeks) until their red cell ALA-D was nearly or completely inhibited and were bled to a reduction of from 30 to 40% in hemoglobin, red cell count and hematocrit ratio, recovered to normal hematologic values as well as did controls not treated with lead. Thus, animal studies resolved the important issue of the relative usefulness of the measure, reduction in red cell ALA-D, as an indicator of response to lead exposure, and hence as a criterion for a lead standard, albeit a criterion only indirectly related to an air standard; measurement of changes in ALA-D is too sensitive

to be usefully applied to workers exposed to lead at this stage of knowledge.*

c. Other Animal Studies on Hematologic Effects of Lead. In addition to the inhibitory effects of lead on the biosynthesis of heme, animal studies have included 1) the stimulation of erythropoietic activity[24]; 2) increased rate of basophilic stippling[15]; 3) reticulocytosis[25]; 4) concentration of coproporphyrins in urine and certain tissues[26]; and 5) the effect of lead on iron metabolism in hemoglobin formation.[27]

d. Serum Protein Changes. Changes in the patterns of the proteins in human blood serum, consisting of a decrease in albumin-globulin ratio with marked increases in the alpha- and beta-globulins, have been confirmed in animals.[28]

Similar confirmation has been made in animals of the findings in man of reduced quantities of mucoid and sialic acid, prosthetic groups of conjugated proteins,[29] reductions of which were used as a warning of impending lead poisoning in industry. Unfortunately, other common conditions such as inflammation also cause changes in the amounts of these blood constituents.

A distinct relationship has been found between lead poisoning and the metabolism of nicotinic acid[30]; animals poisoned by lead showed a marked decrease in the nicotinic acid content of blood (and urine), indicating an increased utilization of this constituent by lead, and suggesting that lead exerts serious effects on the pyridine nucleotides, either by blocking

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*This is not to detract from the major recommendation of the PHS conference on lead[16] to search for ever-more sensitive indicators of response, because much of value on the mechanism of lead in the biosynthesis of heme has resulted, but it does clearly point out 1) that ultra-sensitive methods may not always have practical utility in estimating and controlling workers exposure, and 2) that, inasmuch as highly sensitive methods are used as the criteria for many, if not most, of the air standards in the U.S.S.R., these standards must be carefully reexamined in the light of their appropriateness and suitability.

their synthesis or by accelerating the degradation of nicotinic acid. These changes have been suggested as a means of assessing the severity of lead poisoning.

In line with the general opinion that toxic substances adversely affect the body's resistance to disease by interfering with natural immunologic processes, Fonzi et al.[31] showed that lead-treated and actively immunized animals developed lesser amounts of gamma globulin than did immunized controls. Similarly, lysozyme, another part of the defense mechanisms of the body, was progressively reduced in the blood serum of dogs administered lead salts for a prolonged period.[32]

Although shifts in the body's inorganic elements (copper, calcium phosphorus, sodium and potassium[33]) from lead poisoning have been reported,[34] their significance in over-all body metabolism is yet to be clearly demonstrated.

e. Endocrine Changes. The effects of lead exposure on some aspects of endocrine function have been studied in animals, as well as in man. The excretion of steroids was studied in the urine under different conditions of lead exposure in the hope of finding some evidence of their relation to lead absorption. Adrenal steroids were reported at first to decrease, then to increase considerably during advanced stages of lead intoxication.[35] Vitamin C content of the adrenal gland was decreased in the guinea pig following exposure to lead.[36]

Relatively little use has been made of animals in the study of other endocrine functions, these functions being readily studied in man. Sandstead[37] has, however, reported that lead, like other heavy metals, impairs the uptake of iodine by the thyroid, and that the conversion of iodine to protein-bound iodine is retarded; females were more affected than males.

f. Renal Changes. Goyer[38] has recently reviewed the current state of knowledge of the effects of lead on the kidney; his review is based in large part on his investigations and those of his associates. Prominent among their findings of acute lead poisoning in animals were 1) formation of intranuclear inclusion bodies, 2) mitochondrial swelling with impairment of oxidative and phosphorylative processes, and 3) aminoaciduria (apart from the long-recognized delta-aminolevulinic aciduria); the intranuclear inclusion bodies were a lead-protein complex that may have adaptive function in excessive lead exposure. The acute renal changes progress to a diffuse nephropathy with tubular atrophy and dilation. Rats developed hyperuricemia and in chronic lead poisoning, renal adenocarcinoma. In all but the last, the findings made in rats paralleled those seen in man.

g. Trace Metal Interactions. In recognition that lead poisoning is often associated with an iron-deficiency anemia, the interaction of lead on iron deficiency was studied in the rat.[39] An enhancement of lead retention and toxicity was found in the iron-deficient animals as measured by elevated ALA excretion.

h. Effects on the Nervous System. Despite the fact that the nervous system can be affected by lead, comparatively little experimental attention has been directed to gaining an understanding of the manner in which lead acts on this system. Behavioral response studies in animals, predominantly by Soviet scientists, comprise most of the research effort, although of late, Xintaras and associates have initiated investigations in behavioral toxicology.

Using a range of atmospheric concentrations of lead oxide dust, Gusev[40] found that at a level of 11 $\mu g/m^3$ disturbed reflexes began to occur at 1.5

to 2 months of exposure, whereas no impairment of reflexes was seen at levels averaging about 1 $\mu g/m^3$; base-line conditioned reflex activity returned 10 to 23 days after cessation of exposure. Although no changes in the formed elements of the blood were seen, despite a lead content in rat bone of 10-fold higher than that of animals on the lower dose, histopathologic changes in the central nervous system were seen in both rats and rabbits at the 11 $\mu g/m^3$ level.

Shalamberidze[41] reported disturbed conditioned reflexes in rats exposed to lead sulfide ore dust at a level averaging 48 μg Pb/m^3, 6 hours daily for 6 months. However, because of the insolubility of the sulfide supported by health experience with lead sulfide, responses to lead sulfide at that level are unlikely.

Xintaras studied the applicability of evoked response in the rat's cortex in air pollution toxicology[42,43]; in rats intoxicated with lead acetate he found electroencephalographic changes similar to changes in man.[43] From studies of alterations in rapid eye movements during sleep, he concluded that lead may cause impaired neural control in rats.[43]

i. Developmental Effects. Although mice nursing on dams fed diets containing high levels (1% or 4%) of lead carbonate showed evidence of faulty growth and various neurologic changes,[44] recent evidence reveals a low degree of teratogenic effects in rats and mice.[45]

Correlation of Exposure and Effect

Tsuchiya and Harashima[46] studied storage battery workers and compared airborne lead with urinary lead, urinary coproporphyrin, basophilic stippling of erythrocytes, and specific gravity of blood as indications of anemia.

To control urinary coproporphyrin to normal levels (below 50 μg/liter), they recommended a TLV of about 0.12 mg/m^3 for daily 8- to 10-hour exposures. However, the workers studied by these investigators worked 48 to 60 hours a week. With the increased lead absorption from these working hours, a lower standard than that suitable for a 40-hour week would be indicated. If other criteria were chosen on which to base an air limit, other limits would have been selected; 0.10 mg/m^3 would have been recommended to keep urinary lead levels below 0.15 mg/liter, 0.14 mg/m^3 to keep basophilic stippling at 0.3 per thousand, and 0.14 to 0.15 mg/m^3 to prevent anemia. They did not use blood lead as a criterion of effect.

The study of Williams, King, and Walford[47] was based on observations of storage battery workers who worked a 40-hour week, and were stable in their exposure. They had worked without job change for a year, there was no recent absence for sickness or vacation, and no change in overtime or productivity for 6 months.

Workers in the plastics department were exposed to airborne lead levels of about 0.01 mg/m^3, while workers in lead handling departments were exposed to higher levels, up to about 0.3 mg/m^3. Specific gravities of urine samples averaged 1.020 in the morning and 1.022 at lunch time. They concluded that air levels of 0.20 or 0.15 would result in the blood and urinary lead levels given in Table X-5 (urinary lead levels were corrected for a specific gravity of 1.024; it should be noted that a urinary level of 0.20 mg/liter corrected to a specific gravity of 1.024 would be 0.133 mg/liter corrected to 1.016).

These investigators also showed a very low correlation ($r = 0.09$) between airborne lead and blood hemoglobin levels.

Selander and Cramer[48] compared blood lead, urinary lead, and urinary ALA in lead workers. They found several workers with high urinary lead and ALA values in relation to blood lead and attributed this to a metabolic influence of lead; ALA excretion in these workers had seldom fallen to normal values. They recommended that workers removed from lead overexposure not be allowed to return until ALA excretion was normal.

A statement by a group of experts (R.E. Lane, D. Hunter, D. Malcolm, M.K. Williams, T.G.F. Hudson, R.C. Browne, R.I. McCallum, A.R. Thompson, A.J. deKretser, R.L. Zielhuis, K. Cramer, P.S.I. Barry, A. Goldberg, T. Beritic, E.C. Vigliani, R. Truhaut, R.A. Kehoe, and E. King)[49] on diagnosis of inorganic lead poisoning suggests ranges of indices of lead absorption for occupationally acceptable exposures with the following upper limits:

Blood lead: 0.08 mg/100 ml
Urinary lead: 0.15 mg/liter
Urinary coproporphyrin: 0.50 mg/liter
Urinary ALA: 20 mg/liter

They point out that these values may not be applicable when there are low hemoglobin levels or where chelating agents have been used.

Stankovic[50] reported on blood and urine lead concentrations, urinary coproporphyrin, and urinary ALA in workmen exposed to lead at various concentrations of lead in air. In workmen exposed to 0.15 mg/m^3 and below, the highest individual blood lead found was 0.06 mg/100 g, the highest urine lead 0.12 mg/liter, the highest urinary coproporphyrin 0.186 mg/liter, and the highest urinary ALA 11.85 mg/liter. There were 48 workers exposed to air lead levels of 0.025 to 0.15 mg/m^3, whose mean blood lead level was 0.05 mg/100 g (range of 0.03 to 0.06). However, the number of workers exposed to or near 0.15 mg/m was not stated.

Zielhuis[51] has reviewed and analyzed the data of several other investigators of human absorption of lead, in terms of the relationships between blood lead, ALA, and coproporphyrin. He concluded from analysis of these data that a combination of blood lead greater than 0.08 mg/100 g with values of urinary lead greater than 0.15 mg/liter or urinary ALA greater than 20 mg/liter or urinary coproporphyrin greater than 0.80 mg/liter is evidence of an unacceptable degree of occupational exposure to lead. He did not review the relationships between airborne lead and the several indices of biological effect of absorbed lead.

The selection of 0.08 mg Pb/100 g of whole blood has been described by Kehoe[52] as the critical concentration of lead in blood below which no case of even mild poisoning has been induced by lead. The higher the concentration of blood lead above 0.08, the greater the likelihood of lead poisoning, though higher concentrations did not mean lead poisoning in all individuals. The scientific consensus supports the view of Kehoe as it applies to adults.

However, even in the hands of the best analyst, there may be a 10% error in a specific lead determination. Thus, an analysis showing a blood level of 0.08 mg/100 g may have a true value of almost 0.09. This may account for the recommendation of some authorities[48] that blood lead levels be kept below 0.07 mg/100 g.

IV. ENVIRONMENTAL DATA

Information presented in this section was selected to satisfy two purposes: (1) link measured environmental and biological levels to specific lead using industries, and (2) to link exposure levels to clinical lead intoxication. Table X-4 (from Elkins[5]) gives an overview of in-plant lead levels from various industries. Specific data for industries and a discussion of the exposures therein follow. The principal plant types covered are printing, storage battery manufacturing, and welding operations. Note that the general concentrations of lead in in-plant air range from negligible to those indicative of imminent danger. Scrutiny of specific plant operations is necessary to determine where the hazards exist and how priorities for control should be developed.

(a) Printing

The necessary characteristics of type metal prescribe the use of lead alloys. Examinations of Table X-6 (from Brandt[53]) shows that many areas could presently comply with a 0.15 mg/m^3 standard. Others such as the remelt room and stereotype room will require additional control measures.

Table X-7 (from Ruf[54]) associates exposure levels to significant functions performed by workers in the printing industry. They are obviously not 8-hour TWA levels but are nevertheless indicative of conditions. Most of the higher exposures occur while either some mechanical action is applied either to the metal (such as drossing and filing) or near the melting pots. In the former, large amounts of dust are generated, and in the latter the lead fumes present the problem.

Table X-8 shows data of Belknap [55] on calculated exposures in printing industries. The calculations were based on time spent by printers at various tasks and used data of Ruf [54] summarized in Table X-7. Calculated air exposures and urinary lead levels are shown for various operations. These air concentrations (or urinary levels) may be erroneous, because much less urinary lead would be expected at the listed air concentrations.

(b) Storage Battery Manufacture

Tables X-9 and X-10 furnish data on levels found in plants where storage batteries are produced. The percentages of workers exposed to air-lead levels greater than 0.15 mg/m^3 is important. Table X-9 directs attention to the operations where the serious hazards occur. The levels shown are serious in that they are above the recommended concentration, but also appear to be in a range that are responsive to conventional industrial hygiene control techniques.

(c) Welding and Cutting of Steel

Welding or cutting of lead bearing steels results in the generation of lead fume in significant concentrations. This is also the case when these operations are performed on steels which are either galvanized, zinc-silicate coated, or painted with lead pigmented paints. Elkins [5] observed that at 507 C the vapor pressure of lead (VP = 0.000016 mm Hg) is high enough to produce a concentration after oxidation of 0.18 mg/m^3 of lead fume. During welding or cutting temperatures may reach 1000 to 3000 C.

Table X-11 contrasts lead fume exposures when welding galvanized steel and zinc-silicate coated steel. The worst exposures occurred when welding the zinc-silicate coated steel. Electric arc welding produced an average concentration of 5.63 mg/m^3 and oxy-acetylene produced 1.96 mg/m^3 of lead. The information presented in Table X-11 developed by Pegues.[56] Samples are well identified, providing a clear picture of lead exposure in these welding operations. Note that with good ventilation breathing zone samples can be controlled to within the recommended standard. Note also that room air samples downwind from the welder can rise to levels which jeopardize the health of other workers. In Table X-12 (from Tabershaw[57]), limited data are presented to illustrate the exposures of those workers who perform cutting operations on painted structural steels. The urinary lead data indicate that sufficient protection from lead fume is not given through the use of the indicated respirators, and further controls are needed.

(d) Workers Whose Occupational Exposure is Out-of-Doors

Policemen, firemen, taxi drivers, vehicle tunnel attendants, garage mechanics, and service station attendants are examples of occupational groups who work out-of-doors, but are nonetheless exposed to lead. The primary source of this exposure is the lead salts emitted from internal combustion engines which burn leaded gasoline. Tables X-13 and X-14 were taken from a U.S. Public Health Service survey[58] of lead in the atmosphere and describe lead levels in blood and urine. This same survey shows that these workers are placed in atmospheres containing various amounts of lead for their 8-hour workday. Few of the samples indicate levels which even approach the biologic standard; however, the distribution of the samples does demonstrate the need for monitoring these individuals for lead exposure. There are many levels shown in these tables which are in excess

of normal (not occupationally exposed) levels, and this fact shows that there is absorption of lead on the job.

(e) Miscellaneous

Limited data for lead exposures in many other industries prevent a detailed analysis here. Nonferrous foundries often utilize lead alloys. Berg and Zenz[59] reported on one such foundry and stated that atmospheric lead concentrations have risen in the past twenty years. They stated that from 108 samples collected between 1943 and 1947, there were average concentrations as follows: 0.14 mg/m^3 in the melting room and 0.18 mg/m^3 in the pouring floor area. The results from 40 samples of 1953-1954 produced the following increases: 0.28 mg/m^3 in the melting room and 0.29 mg/m^3 in the pouring floor area. Extensive modification and increased ventilation reduced the concentration from 0.28 mg/m^3 to 0.03 mg/m^3. Attention to the processes and analysis of what operations produced the high concentrations facilitated the control of the lead hazard.

Leaded steel production sometimes generates hazardous occupational exposures to lead. Ruhf[60] reported that the highest atmospheric lead concentrations prevailed during the steel pouring operation in which the lead is added. Other elevated exposures were measured in processes such as the rolling mills. However, because of the intermittent nature of the operations the time weighted average exposure was below the <u>then</u> current limit of 0.20 mg/m^3. Ruhf further described control measures and manufacturing techniques whereby lead exposure can be minimized.

V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

The American Conference of Governmental Industrial Hygienists (ACGIH)[61] has reviewed previous standards for lead in the work environment, and has commented that there are few meaningful data relating to the threshold limit value, probably because most authorities rely primarily on other tests for estimating lead hazards, such as urinary and blood leads, urinary coproporphyrin and ALA, as well as examination of the blood for stippled cells.

Nevertheless, attempts were made to control occupational lead poisoning by establishing acceptable air levels to guide engineering control measures. Although the point is not documented, it seems that at one time an air limit value of 0.5 mg/m^3 was used. In the 30's and 40's, a value of 0.15 mg/m^3 was a common, but often unachieved, goal based on a recommendation of a 1928 PHS survey of storage battery workers published in 1933.[62]

This value continued to be the one most often accepted until 1957, when the ACGIH increased the TLV to 0.20 mg/m^3, based in part on data of Elkins[5] showing that exposure at 0.20 mg/m^3 would result in urinary excretion at 0.20 mg/liter.

In 1971, the Conference recommended lowering of this value back to 0.15 mg/m^3. This appears to have been based in part on the recommendations of the International Subcommittee for Occupational Health, Permanent Commission and International Association of Occupational Health[63] at a 1968 meeting in Amsterdam, and on the results of the study by Williams, King, and Walford.[47]

The International Subcommittee recommended a time-weighted average concentration for a 40-hour week of 0.15 mg/m^3, on the basis that it corresponded to an acceptable blood concentration of 0.07 mg/100 ml.

The current workroom air standard established under the Occupational Safety and Health Act of 1970 (published in Part 1910.93 of the Federal Register, Volume 36, Number 157, pages 15101-15107, dated August 13, 1971) is 0.2 mg/m^3; this is a time weighted average, and is based on American National Standards Institute Z37.11-1969.[64] This ANSI standard provided no basis for its recommendation.

Basis for Recommended Environmental Standard and Biologic Monitoring

Earlier in this century, efforts to reduce occupational lead poisoning were based on adherence to hygienic workroom air guides. As more knowledge developed, increasing attention was given to blood and urinary lead levels as guides to reduction of occupational poisoning. Concomitantly, there was increasing attention to better lead analyses. There was also an increasing knowledge of the relationship between levels and rates of absorption and excretion, blood lead levels, and health status.

The PHS study by Dreessen et al.[11] was undertaken during the period that the workroom air guide of 0.15 mg/m^3 was accepted, but failure to achieve control of airborne lead to this level was common, so findings of slight effects among workers in lead-using industries by Dreessen and co-workers did not invalidate the guide. Though not documented, it appears that many industries have rotated their workers to various jobs to keep blood lead levels below 0.08 mg/100 g; thus, exposure to unsafe workplace air levels did not result in adverse effects on health.

Consequently, there is a little definitive information from experience in the United States and other countries on the suitability of 0.15 or 0.20 mg/m^3 as an air-lead level to which workers can be safely exposed over a working lifetime.

However, much experience has accrued to show that absorption of lead in amounts resulting in blood lead concentrations of 0.08 mg/100 g or less will not lead to adverse effects on health, and there is information from studies in other countries relating airborne lead levels to blood lead.

It was previously concluded (III. Biologic Effects of Exposure; Correlation of Exposure and Effect) that a blood lead level of 0.08 mg/100 g is useful for delineating acceptable from nonacceptable lead absorption. While levels below 0.08 mg/100 g are indicative of acceptable occupational lead absorption and, if also representative of past absorption of lead by an individual person, also indicative of insignificant risk of lead poisoning, it should not be concluded that lead poisoning will occur if blood lead levels exceed 0.08 mg/100 g. However, there is an increasing risk of poisoning as levels increase above 0.08 mg/100 g, so absorption of lead should be held to amounts that will result in blood lead levels less than 0.08 mg/100 g. As Kehoe[65] has stated, "...lead poisoning occurs in man only when certain well-defined conditions have been fulfilled" and that this is quantitatively applied by "...the relationship between the current rate and the extent of the absorption of the inorganic compounds of lead, and the concentration of lead in an accessible tissue of the living body, namely, the blood." Thus, a biologic standard of 0.08 mg of lead per 100 g of whole blood is recommended; it provides a margin of safety in adults,

but probably not in children. The extent of this margin of safety is not known, but it seems likely that there will be few, if any, cases of lead poisoning below 0.09 mg/100 g.

Kehoe[65] also pointed out the usefulness of urinary lead as an index of current absorption of lead, but added that it was a quantitatively less certain index than blood lead. It may be consistent with this view that Williams, King, and Walford[47] found that the best correlation between airborne lead and biochemical index of effect was with blood lead ($r = 0.90$) and less correlation with urinary lead ($r = 0.82$). The study of Williams and co-workers[47] indicates that blood levels of 0.08 mg/100 ml is associated with a urinary lead level of 0.20 mg/liter. It has been commonly accepted that 0.20 mg/liter is a safe level in urine, based in part on the findings of Elkins.[5] However, it is important to note that Elkins' studies involved calculation of specific gravity of urine to a value of 1.024. The studies of Williams et al.[47] also calculated urinary specific gravity to 1.024. (Urinary lead levels of 0.20 mg/liter, adjusted to a specific gravity of 1.024, would be 0.133 mg/liter if the specific gravity were calculated to 1.016.) Thus, the conclusion of Zielhuis[51] that urinary lead greater than 0.15 mg/liter, uncorrected for specific gravity, represents unacceptable absorption of lead is consistent with the selection of a biologic standard for urinary lead of 0.20 mg/liter, so long as the specific gravity correction is used.

ALA and coproporphyrin assays, and blood examinations for hemoglobin, reticulocytes, and stippled cells are useful in the assessment of worker health, but are less useful than blood lead as a single criterion for

interpreting the acceptability of lead absorption, since no one of these measurements is a specific index of lead absorption, as is urinary or blood lead.

It should be emphasized that blood lead and urinary lead are good indices of current absorption of lead (in the absence of anemia or absorption of chelating agents), but are not necessarily indications of body burden of lead or of the state of health of the individual. Bone lead is probably more indicative of total body burden than is blood lead, but it is not feasible to sample bone for routine lead assay. As to state of health, overabsorption of lead by an individual in the past may have led to a high body burden of lead and may also have contributed to a state of current ill-health in the individual, all without causing currently high blood or urinary levels of lead.

Since the studies of relationship between health and airborne lead levels are inadequate, it is concluded that an air standard should be recommended from data on the relationship between airborne lead and biochemical indices of effect, most importantly, blood lead. There are several studies that point to 0.15 mg/m^3 as the level of airborne lead that will result in biochemical indices showing acceptable absorption of lead, in other words, showing that occupational exposure at 0.15 mg/m^3 will not result in adverse effects on the health of workers.

Tsuchiya and Harashima[46] studied storage battery workers in Japan and compared airborne lead with urinary lead, urinary coproporphyrin, basophilic stippling of erythrocytes, and, as an index of anemia, specific gravity of blood. They recommended airborne lead levels on the basis of acceptable

levels of these biochemical indices. On the basis of acceptable urinary lead levels of 0.15 mg/liter, corrected to a specific gravity of 1.024, they recommended a threshold limit value of 0.10 mg/m^3. If a higher urinary lead level is accepted, as recommended in the preceding discussion of the relationship between acceptable lead absorption and urinary lead excretion, a higher air standard would result. It should be noted that the workers studied by Tsuchiya and Harashima worked 8 to 10 hours, 6 days a week, and they observed that a higher air level would have been recommended for a 40-hour week.

The study most directly relevant to the development of a recommended workplace air standard is the study of Williams, King, and Walford.[47] Their data (Table X-5), from studies of storage battery workers stable in their employment (40-hour work week, no job change in the past year, no recent absence or sickness, no change in overtime or productivity), showed that exposure at 0.15 mg/m^3 resulted in a mean blood lead of 0.060 mg/100 ml. Were mean blood lead the criterion of effect, an air standard much higher than 0.15 mg/m^3 could be recommended, but in order to keep most or all workers' blood lead below 0.084 mg/100 ml (0.080 mg/100 g), it is believed that a mean of about 0.060 mg/100 ml should be achieved. The data of Williams and associates[47] does not provide a basis for interpreting the percentage of workers exposed at 0.15 mg/m^3 that will have blood levels above 0.084 mg/100 ml. However, it is believed that a small percentage will have blood lead levels at or above 0.084 mg/100 ml or 0.080 mg/100 g, so it is recommended that workers be monitored biologically, by periodic assays of blood lead, or of blood and urinary lead.

Stankovic[50] also compared airborne lead with blood and urinary lead, and in workmen exposed to lead at 0.15 mg/m^3 and below, the highest individual blood lead found was 0.06 mg/100 g, and the highest urinary lead 0.12 mg/liter. However, the number of workers exposed at or near 0.15 mg/m^3 was not stated, so his finding of 0.06 mg/100 g as the highest individual blood lead is not believed to contradict the previously stated inference that some workers exposed at 0.15 mg/m^3 will have blood lead levels at or above 0.08 mg/100 g (especially those workers absorbing abnormal amounts of lead from nonoccupational sources).

It is of interest that conclusions of experts[63] support the recommended standard, but since data and arguments supporting their conclusions were not presented, their recommendations have not been given weight in deriving the recommended occupational air standard.

The rationale for the recommended work practices and sanitation practices was principally derived from Kehoe.[66] They are normal industrial hygiene procedures used to control occupational exposures to various dusts and fumes.

If worker exposures exceed 40 hours a week, the same TWA of 0.15 mg/m^3 should be used unless exposures so greatly exceed 40 hours a week that nonworking (excretion) time is significantly reduced; exposures up to 50 hours a week should not significantly affect the time for excretion of absorbed lead. However, maintenance of the same TWA means a proportionate reduction in average concentration as exposures exceed 40 hours a week. To achieve a TWA of 0.15 mg/m^3, the average concentration should be 0.15 mg/m^3 for a 40-hour week and 0.12 mg/m^3 for a 50-hour week.

Basis for Environmental Sampling and Analytical Method

Various methods of sampling air and of analysis of these samples have been considered, and recommended methods are presented in Appendixes I and II.

The recommended method of sampling air involves collection of 100 liters of air or more, use of breathing zone samplers with sampling at a rate of 2 liters/min., and collection on 0.45μ cellulose membrane filters. Other sampling rates and other collection media (filter paper, nitric acid impinger, electrostatic precipitation) are capable of giving equivalent results. The recommended procedure is described in Appendix I.

For analysis of lead in blood, atomic absorption spectrophotometry[67-71] and dithizone colorimetry[72,73] were considered. Appreciable consonance can be demonstrated between results obtained with atomic absorption and dithizone methods. Both methods have been used for analysis of air samples, and both are concluded to be capable of giving accurate results. After a review of the several procedures involving atomic absorption spectrophotometry, it was concluded that no one of these procedures has been sufficiently standardized. Individual laboratories get excellent results with a specific procedure, but these procedures have not been compared in a number of laboratories. Dithizone colorimetry, on the other hand, has been used for a long time and has been thoroughly studied. The procedures, interferences, sensitivity, and replicability have been studied and are described by Keenan, Byers, Saltzman, and Hyslop.[72] The recommended procedure is described in Appendix II.

Dithizone colorimetry is a wet chemical method requiring equipment found in most chemical laboratories, but requires meticulous attention to detail and to the prevention of loss and the exclusion of contamination.

Results of lead analysis by this method obtained by well trained technicians are often superior to results obtained by other methods of analysis.

Basis for Biologic Analytical Method

Blood lead was selected as the best method, and urinary lead as an acceptable method, for judging lead absorption, for reasons discussed in earlier sections (see "Basis for Recommended Environmental Standard and Biologic Monitoring").

Specific details for collection of biologic specimens for lead analysis have been described in a booklet Methods for determining lead in air and in biological materials, published by the American Public Health Association. Keppler et al.[75] described the initiation of interlaboratory evaluations of lead in an attempt to improve accuracy and reproducibility of laboratory analyses through a system of accreditation. Subsequent reports[76,77] have described some of the results, from which it is apparent that lead analysis is subject to significant error unless a very high degree of care is used.

Methods for the collection of blood and urine are described by Keenan et al.[72] (Appendix II). While lead-free Vacutainers are convenient, any lead-free tube can be used for collection and shipment or storage of blood prior to analysis. No aliquots can be taken unless blood-clotting has been prevented, either by taking aliquots before clotting or by prevention of clotting, e.g., by heparinization. Single use needles ("throw-aways") are acceptable, but must be lead-free, thus must not be lead-soldered.

Methods for the determination of lead in biological materials include dithizone colorimetry,[72,73,78] spectrography,[79] polarography,[80] and atomic absorption spectrophotometry.[69,71,81] In addition, many biochemical tests, reviewed by Chisolm,[82] have been developed; these depend on the lead-induced upset in heme synthesis. Among these biochemical tests are determination of coproporphyrin excretion,[83] urinary ALA[26,84] and ALA-D in blood.[85,86] Additional methods, such as cell stippling, porphobilinogen determinations, and examination of intranuclear inclusion bodies have received less acceptance. These biochemical indices are not recommended at this time. They can be sensitive, perhaps too sensitive, but they are not specific for lead, and are judged to be less useful than blood and urinary lead determinations for estimating the absorption of lead. However, future developments may resolve some of the present objections to the routine use of these indices of alterations of heme synthesis in the assessment of lead absorption.

The dithizone procedure is recommended for analysis of lead in blood and urine. As discussed in the previous section (Basis for Environmental Sampling Method), the method is capable of good results if meticulous attention is given to details, including sources of contamination and loss. Cholak[87] has stated that with careful control the procedure can detect as little as 0.5 μg, with a precision of + 0.5 μg, and that, with modifications, as little as 0.2 + 0.1 μg can be determined. The recommended method as described by Keenan et al.[72] is given in Appendix II. Bismuth is a possible, but uncommon, interfering substance, which can be removed by extraction.

VI. REFERENCES

1. Weast RC (ed): Handbook of Chemistry and Physics--A Ready Reference Book of Chemical and Physical Data, ed 52. Cleveland, Chemical Rubber Publishing Co, 1971-1972

2. Minerals Yearbook 1969: Metals, Minerals and Fuels. US Bureau of Mines, US Government Printing Office, 1969, vol I-II, p 638

3. Gafafer WM (ed): Occupational Diseases--A Guide to Their Recognition, PHS publication 1097. US Dept Health, Education, and Welfare, Public Health Service, 1966, pp 170-72

4. Committee on Biologic Effects of Atmospheric Pollutants: Airborne Lead in Perspective. National Academy of Sciences, National Research Council, Division of Medical Sciences, 1971

5. Elkins HB: The Chemistry of Industrial Toxicology, ed 2. New York, John Wiley & Sons Inc, 1959, pp 49-57

6. Tanquerel des Planches L: Lead Diseases: A Treatise (from Traite des maladies de plomb, ou saturnines, Paris, 1839) as cited in Hunter D: The Diseases of Occupations, ed 4. Boston, Little, Brown & Co, 1969, p 330

7. Burton H: Med-Chir Tr, London, 1840, XXIII, 63-79, as cited in Garrison FH: An Introduction to the History of Medicine--With Medical Chronology Suggestions for Study and Bibliographic Data, ed 4. Philadelphia, WB Saunders Co, 1929, p 444

8. Mayers MR: Occupational Health--Hazards of the Work Environment. Baltimore, Williams & Wilkins Co, 1969, pp 69-70

9. Hunter D: The Diseases of Occupations, ed 4. Boston, Little, Brown & Co, 1969, pp 234-89

10. Lane RE: Health control in inorganic lead industries--A follow-up of exposed workers. Arch Environ Health 8:243-50, 1964

11. Dreessen WC, Edwards TI, Reinhart WH, Page RT, Webster SH, Armstrong DW, Sayers RR: The Control of the Lead Hazard in the Storage Battery Industry, PH bulletin 262. Federal Security Agency, US Public Health Service, US Government Printing Office, 1941

12. Dingwall-Fordyce I, Lane RE: A follow-up study of lead workers. Br J Ind Med 20:313-15, 1963

13. Malcolm D: Prevention of long-term sequelae following the absorption of lead. Arch Environ Health 23:292-98, 1971

14. Henderson DA, Inglis JA: The lead content of bone in chronic Bright's disease. Australasian Ann Med 6:145-54, 1957

15. Lane RE: The care of the lead worker. Br J Ind Med 6:125-63, 1949

16. Symposium on Environmental Lead Contamination, Dec 1965, PHS publication No 1440, 0-210-345. US Dept Health, Education, and Welfare, Public Health Service, 1966

17. Rimington C: Haems and porphyrins in health and disease. I. Acta Med Scand 143(fasc III):161-96, 1952

18. Rimington C: The biosynthesis of haemoglobin. Br Med J 2:189-93, 1956

19. Eriksen L: Lead intoxication--I. The effect of lead on the in vitro biosynthesis of heme and free erythrocyte porphyrins. Scand J Clin Lab Invest 7:80-85, 1955

20. Kreimer-Birnbaum M, Grinstein M: Porphyrin biosynthesis--III. Porphyrin metabolism in experimental lead poisoning. Biochem Biophys Acta 111:110-23, 1965

21. Hernberg S, Nikkanen J, Mellin G, Lilius H: δ-Aminolevulinic acid dehydrase as a measure of lead exposure. Arch Environ Health 21:140-45, 1970

22. Millar JA, Cumming RLC, Battistini V, Carswell F, Goldberg A: Lead and δ-aminolaevulinic acid dehydratase levels in mentally retarded children and in lead-poisoned suckling rats. Lancet 2:695-98, 1970

23. Maxfield ME, Stopps GJ, Barnes JR, Snee RD, Azar A: Effect of lead on blood regeneration following acute hemorrhage in dogs. (Accepted for publication by Am Ind Hyg Assoc J)

24. Goldblatt MW, Goldblatt J: Some Aspects of Industrial Toxicology, in Merewether ERA: Industrial Medicine and Hygiene. London, Butterworth Co Publishers Ltd, vol 3, p 459, 1956

25. Baikie AG, Valtis DJ: The oxygen consumption of the blood in experimental lead poisoning. Br J Exp Pathol 35:434-38, 1954

26. Haeger-Aronsen B: Studies on urinary excretion of δ-aminolaevulic acid and other haem precursors in lead workers and

lead-intoxicated rabbits. Scand J Clin Lab Invest 12(suppl. 47):1-128, 1960

27. Vannotti A, Imholz A: Die Beziehungen des Reticulumendothels zum Umsatz des Nichthamoglobineisens. Z Gesamte Exp Med 106:597-611, 1939

28. Afonova VN: The metabolism of proteins in the blood serum in experimental lead poisoning. Sb Nauch Tr Ryazan Med Inst 12:129-32, 1960

29. Camerada P, Congiu M, Leo P: Comportamento del seromucoide e dell'acido sialico (sangue e tessuto nervoso) nell'intossicazione sperimentale da piombo nell ratto. Rass Med Sarda 66:253-63, 1964

30. Caccuri S: Primi risultate sull'azione dell'acido nicotinico sulla porfirinuria nell'saturnismo sperimentale. Folio Med 26:345, 1940

31. Fonzi S, Pengue L, Raddi R: Processi immunitari nella intossicazione sperimentale da piombo--Nota VI. Variazioni degli anticorpi antitifici (completi ed incompleti) in corso di immunizzazione attiva specifica. Lav Umano 19:324-28, 1967

32. Ambrosio L, Mazza V: Comportamento del lisozima nel siero di sangue e negli organi nelle intossicazioni sperimentali (piombo, benzolo, manganese). Riv Ist Sieroter Ital 29:252-62, 1954

33. Rubino GE, Pagliardi E, Prato V, Giangrandi E: Erythrocyte copper and porphyrins in lead poisoning. Br J Haemat 4:103-07, 1958

34. Kosmider S, Crabski J, Stradowski J: Plasma sodium, calcium and potassium levels in rabbits during experimental acute lead poisoning. Arch Immunol Ther Exp 11:303-06, 1963

35. Kanner NL: [Influence of lead on the functional state of the adrenal cortex.] Probl Endokr Gormonoter 11:83-88, 1965 (Rus)

36. Gontea I, Sutescu P, Stanciu V, Lungu D: [Vitamin C in lead poisoning in the guinea pig.] Igiena 13:501-09, 1964 (Rum)

37. Sandstead HH: Effect of chronic lead intoxication on vivo I^{131} uptake by the rat thyroid. Proc Soc Exp Biol Med 124:18-20, 1967

38. Goyer RA: Lead and the kidney, in Current Topics in Pathology. New York, Springer-Verlag, 1971, vol 55, pp 147-76

39. Six KM, Goyer RA: The influence of iron deficiency on tissue content and toxicity of ingested lead in the rat. J Lab Clin Med 79:128-36, 1972

40. Gusev MI: [Limits of allowable lead concentrations in the air of inhabited localities], in Ryazanov VA (ed): [Limits of Allowable Concentrations of Atmospheric Pollutants], Bk 4, pp 5-31, 1960, translation, US Dept Commerce, Off Tech Serv, 1961 (Rus)

41. Shalamberidze OP: [Limit of allowable concentration of lead sulfide in atmospheric air], in Ryazanov VA (ed): [Limits of Allowable Concentrations of Atmospheric Pollutants], Bk 5, pp 29-38, translation, US Public Health Service, 1962 (Rus)

42. Xintaras C, Johnson BL, Ulrich CE, Terrill RE, Sobecki MF: Application of the evoked response technique in air pollution toxicology. Toxicol Appl Pharmacol 8:77-87, 1966

43. Xintaras C, Sobecki MF, Ulrich CE: Sleep: Changes in rapid-eye-movement phase in chronic lead absorption. Toxicol Appl Pharmacol 10:384, 1967; abst

44. Rosenblum WI, Johnson MG: Neuropathologic changes produced in suckling mice by adding lead to the maternal diet. Arch Pathol 85:640-48, 1968

45. McClain RM, Becker BA: Placental transport and teratogenicity of lead in rats and mice. Fed Proc 29:347, 1970; abst

46. Tsuchiya K, Harashima S: Lead exposure and the derivation of maximum allowable concentrations and threshold limit values. Br J Ind Med 22:181-86, 1965

47. Williams MK, King E, Walford J: An investigation of lead absorption in an electric accumulator factory with the use of personal samplers. Br J Ind Med 26:202-16, 1969

48. Selander S, Cramer K: Interrelationships between lead in blood, lead in urine, and ALA in urine during lead work. Br J Ind Med 27:28-39, 1970

49. Lane RE, Hunter D, Malcolm D, Williams MK, Hudson TGF, Browne RC, McCallum RI, Thompson AR, deKretser AJ, Zielhuis RL, Cramer K, Barry PSI, Goldberg A, Beritic T, Vigliani EC, Truhaut R, Kehoe RA, King E: Diagnosis of inorganic lead poisoning: A statement. Br Med J 4:501, 1968

50. Stankovic MK: Biochemical tests for the appraisal of exposure to lead. Arch Environ Health 23:265-69, 1971

51. Zielhuis RL: Interrelationship of biochemical responses to the absorption of inorganic lead. Arch Environ Health 23:299-311, 1971

52. Kehoe RA: The Harben Lectures 1960. The metabolism of lead in man in health and disease--Present hygienic problems relating to the absorption of lead, lecture 3. J R Inst Public Health 24:177-203, 1961

53. Brandt AD, Reichenbach GS: Lead exposures at the government printing office. J Ind Hyg Toxicol 25:445-50, 1943

54. Ruf HW: Studies on the lead hazards in certain phases of printing. I. Actual lead exposures as measured by the amount of lead in printing atmospheres. J Ind Hyg Toxicol 22:445-63, 1940

55. Belknap EL: Studies on the lead hazards in certain phases of printing--II. Actual lead absorption as measured by physical examinations, blood and urine studies. J Ind Hyg Toxicol 22:463-71, 1940

56. Pegues WL: Lead fume from welding on galvanized and zinc-silicate coated steels. Am Ind Hyg Assoc J 21:252-55, 1960

57. Tabershaw I, Ruotolo BPW, Gleason RP: Plumbism resulting from oxyacetylene cutting of painted structural steel. J Ind Hyg Toxicol 25:189-91, 1943

58. Survey of Lead in the Atmosphere of Three Urban Communities, PHS publication 999-AP-12. US Dept Health, Education, and Welfare, Public Health Service, 1965

59. Berg BA, Zenz C: Environmental and clinical control of lead exposure in a non-ferrous foundry. Am Ind Hyg Assoc J 28:175-78, 1967

60. Ruhf RC: Lead exposure control in the production of leaded steel. Am Ind Hyg Assoc J 24:63-67, 1963

61. American Conference of Governmental Industrial Hygienists: Transactions of the thirty-third annual meeting of the American Conference of Governmental Industrial Hygienists, Toronto, Canada, May 24-28, 1971. Lead, pp 129-132

62. Russell AE, Jones RR, Bloomfield JJ, Britten RH, Thompson LR: Lead poisoning in a storage battery plant, PHS bulletin 205. US Treasury Dept, Public Health Service, 1933

63. Permanent Commission and International Association of Occupational Health: Report on the Meeting of International

Subcommittee for Occupational Health in International Conference on Inorganic Lead - 28-29 Nov, 1968, Amsterdam. Ind Med Surg 38:10-11, 1969

64. USA Standard Acceptable Concentrations of Lead and its Inorganic Compounds, USAS Z37.11-1969 (revision of Z37.11-1943). New York, United States of America Standards Institute Inc (now American National Standards Institute Inc), 1969

65. Kehoe RA: Standards with respect to atmospheric lead. Arch Environ Health 8:348-54, 1964

66. Kehoe RA: Industrial Lead Poisoning, in Patty FA: Industrial Hygiene and Toxicology, revised ed 2. New York, John Wiley & Sons Inc, vol II, pp 941-80, 1963

67. Analytical Methods for Atomic Absorption Spectroscopy. The Perkin Elmer Corp, 1971

68. Slavin W: Atomic Absorption Spectroscopy. New York, Interscience Publishers, pp 118-22, 1968

69. Farrelly RO, Pybus J: Measurement of lead in blood and urine by atomic absorption spectrophotometry. Clin Chem 15:566-74, 1969

70. Hwang JY, Ullucci PA, Smith SB Jr, Malenfant AL: Atomic Absorption Spectrometric Determination of Lead in Blood by the Solid Phase Sampler Technique. Application Report, Lexington, Mass, Instrumentation Laboratory Inc, 1971

71. Pierce JO II, Cholak J: Lead, chromium, and molybdenum by atomic absorption. Arch Environ Health 13:208-12, 1966

72. Keenan RG, Byers DH, Saltzman BE, Hyslop FL: The "USPHS" method for determining lead in air and in biological materials. Am Ind Hyg Assoc J 24:481-91, 1963

73. Cholak J, Hubbard DM, Burkey RE: Microdetermination of lead in biological material with dithizone extraction at high pH. Anal Chem 20:671-72, 1948

74. Subcommittee on Chemical Methods, Committee on Ventilation and Atmospheric Pollution, Industrial Hygiene Section: Methods for Determining Lead in Air and in Biological Materials. New York, American Public Health Association, 1944

75. Keppler JF, Maxfield ME, Moss WD, Tietjen G, Linch AL: Interlaboratory evaluation of the reliability of blood lead analyses. Am Ind Hyg Assoc J 31:412-23, 1970

76. Weil CS: Critique of interlaboratory evaluation of the reliability of blood-lead analyses. Am Ind Hyg Assoc J 32:304-12, 1971

77. Donovan DT, Vought VM, Rakow AB: Laboratories which conduct lead analysis on biologic specimens. Arch Environ Health 23:111-13, 1971

78. Vinter P: The estimation of lead in blood. J Med Lab Technol 21:281-86, 1964

79. Cholak J, Story RV: Spectrographic and analysis of biological material--III. Lead, tin, aluminum, copper, and silver. Ind Eng Chem Anal Ed 10:619-22, 1938

80. Cholak J, Bambach K: Determination of lead in biological material: A polarographic method. Ind Eng Chem Anal Ed 13:583-86, 1941

81. Berman E: The determination of lead in blood and urine by atomic absorption spectrophotometry. Atomic Absorp Newsletter 3:111-14, 1964

82. Chisolm JJ Jr: Disturbances in the biosynthesis of heme in lead intoxication. J Pediatr 64:174-87, 1964

83. Askevold R: Routine analysis of porphyrines in urine. Scand J Clin Lab Invest 3:318-19, 1951

84. Mauzerall D, Granick S: The occurrence and determination of δ-aminolevulinic acid and porphobilinogen in urine. J Biol Chem 219:435-46, 1956

85. Granick S, Mauzerall D: Porphyrin biosynthesis in erythrocytes: II. Enzymes converting δ-aminolevulinic acid to coproporphyrinogen. J Biol Chem 232:1119-40, 1958

86. Bonsignore D: [The erythrocyte aminolevulinic (ALA) dehydratase activity as a diagnostic test in occupational lead poisoning] Med Lav 57:647-54, 1966 (It)

87. Cholak J: Analytical methods for determination of lead. Arch Environ Health 8:222-31, 1964

VII. APPENDIX I

METHOD FOR SAMPLING OF LEAD IN AIR

Lead dust or fume is collected on 0.45 μ cellulose membrane filters mounted in either 2- or 3-piece filter cassettes. Air is drawn through the filter by means of a pump at a rate of 2 liters a minute (not less than 1 nor more than 4 liters per minute). A minimum sample of 100 liters shall be collected. Larger sample volumes are encouraged provided the filters do not become loaded with dust to the point that loose material would fall off or the filter would become clogged.

With each group of samples, one filter, labeled as a blank, shall be submitted; no air shall be drawn through this filter.

The sample cassettes, if shipped, must be packed in a suitable container to prevent damage in transit. Loss of sample shall be prevented; loss of loose deposits on the filter can be prevented by mounting a clean filter in the cassette on top of the sample filter.

Ash the filter and analyze for lead as described in Appendix II.

Other collection methods shown to be equivalent may be used.

VIII. APPENDIX II

DITHIZONE METHOD OF ANALYSIS OF LEAD IN AIR AND BIOLOGIC SAMPLES

The following directions for analysis of lead are taken from the first part of the report, "The 'USPHS' Method for Determining Lead in Air and in Biological Materials" by Keenan, Byers, Saltzman, and Hyslop.[72] Additional information on the reproducibility and accuracy of the method is given in other portions of the report.

REAGENTS

Analytical grade reagents are used. Purification is essential when analyzing biological tissues and fluids because of the very low levels of lead in these materials; purification of reagents may not be required for air samples containing quantities of lead sufficiently greater than that present in the reagent blank. A reagent blank sample is carried through the entire procedure with each set of unknown samples (air, biological, or other type) and its analyzed lead content is subtracted from each analytical result to calculate the net quantity of lead in each unknown sample.

A boiling rod is used to prevent bumping in the flasks when distilling reagents. This is prepared by cutting 3 or 4 mm O.D. glass tubing to a length which is one cm greater than the height of the flask. The tubing is sealed at a spot about one cm above the bottom end which is fire-polished but left open. Before each use, the liquid is shaken out of

the bottom section and the rod inserted in the flask. As the flask is heated a steady stream of air and vapor bubbles issues from the open space, thus providing nuclei for smooth boiling.

Double-distilled Water - To distilled water in an all borosilicate-glass still add a crystal each of potassium permanganate and barium hydroxide and redistill. Use for reagent and biological sample solutions unless tests indicate that single-distilled water is satisfactory; single-distilled water is usually adequate for determinations on air samples.

Nitric Acid, Concentrated - Redistill in an all borosilicate-glass still the ACS reagent grade acid, 69.0% minimum, specific gravity 1.42. Use an electric heating jacket on the boiling flask to minimize danger of its breakage, and a boiling rod to prevent bumping, which otherwise would be severe. Discard the first 50 ml of distillate; this may be combined with the acid allowed to remain in the flask at the end of the distillation and used for washing glassware. The reagent is conveniently dispensed from a small automatic burette. No grease should be used on the stopcock.

Nitric Acid, 1:99 - Dilute 10 ml of the redistilled, concentrated acid to one liter with doubled-distilled water.

Ammonium Hydroxide, Concentrated - Distill in an all borosilicate-glass still 3 liters of the ACS reagent grade, 28.0% minimum specific gravity 0.8957 at 60 F, into 1.5 liters of double-distilled water,

contained in a 2-liter reagent bottle which is chilled in an ice bath. Continue the distillation until the bottle is filled up to the previously marked 2-liter level. Submerge the condenser tube deeply in the water in the receiver, but withdraw it before discontinuing the heat to avoid siphoning back of distillate. This reagent may be prepared more conveniently from tank ammonia, using a small wash bottle to scrub the gas and a sintered glass delivery tube which extends to the bottom of the reagent bottle. The ammonia gas is absorbed in double-distilled water until the solution reaches the desired specific gravity.

Chloroform - Use a brand with a statement on the label that the chloroform passes the American Chemical Society test for suitability for use in dithizone procedures. In addition, each batch of chloroform should be purchased in glass containers only and should be tested as follows in the laboratory to make sure that it is satisfactory for preparing the dithizone solutions: add a minute quantity of dithizone to a portion of the chloroform in a test tube, shake gently, then stopper with a cork. The faint green color should be stable for one day. Our experience has indicated that the procedures for reclaiming used chloroform are tedious, time-consuming, sometimes unsuccessful, and no longer warranted in view of the commercial availability of acceptable reagent grades.

Extraction Dithizone - Dissolve 16 mg of diphenylthiocarbazone (dithizone), Eastman Kodak Co. No. 3092, or equivalent, in one liter of chloroform. Store in a brown bottle in the refrigerator.

Standard Dithizone - Dissolve 8 mg of diphenythiocarbazone in one liter of chloroform. Store in a brown bottle in the refrigerator but allow to warm to room temperature before using. Age for at least one day, then standardize as described in the procedure. Restandardize every few months.

Sodium Citrate - Dissolve 125 g of the 2 $Na_3C_6H_5O_7 \cdot 11\ H_2O$ salt in sufficient distilled water to provide a solution nearly 500 ml in volume. Adjust the pH to 9-10, using a very small quantity of phenol red indicator solution (strong red color) and fresh, pHydrion test paper to check the pH. Extract in a large separatory funnel with a 100 mg per liter solution of dithizone and finally with the extraction dithizone reagent until a green extract is obtained with the latter reagent. Add a small volume of lead-free citric acid until an orange color (pH 7) appears. Extract the excess dithizone repeatedly with chloroform until a colorless extract is obtained. Remove the last traces of chloroform.

Hydroxylamine Hydrochloride - Dissolve 20 g of the salt in distilled water to provide a volume of 65 ml. Add a few drops of m-cresol purple indicator, then add ammonia until the indicator turns yellow (pH 3). Add a sufficient quantity of a 4% solution of sodium diethyldithiocarbamate to combine with metallic impurities, then mix. After a few minutes extract repeatedly with chloroform until the excess carbamate reagent has been removed, as indicated by the absence of a yellow color in the final chloroform extract tested with a dilute copper solution.

To the aqueous solution of the hydroxylamine hydrochloride add redistilled, 6N hydrochloric acid until the indicator turns pink, and adjust the volume to 100 ml with double-distilled water.

Potassium Cyanide - (Danger! Highly poisonous!!) To 50 g of potassium cyanide in a beaker, add sufficient distilled water to make a sludge. Transfer the sludge to a separatory funnel previously marked to show 100-ml volume. Add a small amount of distilled water to the beaker and warm. (Potassium cyanide cools the solution as it dissolves, thus retarding the solution process.) Add this warm water to the separatory funnel but do not permit contents to exceed the 100-ml mark. Shake, then let stand until the contents come to room temperature. A practically saturated solution results.

Extract the lead by shaking repeatedly with portions of the extraction dithizone solution until the lead has been removed. Part of the dithizone dissolves in the aqueous phase but enough remains in the chloroform to color it. A green extract indicates that all the lead has been completely extracted. Most of the dithizone in the aqueous phase is then removed by repeated extractions with pure chloroform. Dilute the concentrated solution of potassium cyanide with double-distilled water to 500 ml. It should not be necessary to filter the solution, if the directions are followed precisely. Extraction is carried out before dilution because the higher pH of the dilute solution is less favorable.

(NOTE: A colorless solution usually results if above directions are followed. Occasionally aging results in a brown color or precipitate due to polymerization of hydrogen cyanide. This does not interfere with use of the reagent if it is carefully decanted. Old potassium cyanide reagent may lose enough strength to cause insufficient complexing of large amounts of zinc.)

Ammonia-cyanide Mixture - Mix 200 ml of the purified 10% potassium cyanide reagent with 150 ml of distilled ammonium hydroxide (specific gravity 0.9, corresponding to 28.4% NH_3) and dilute to one liter with double-distilled water. If the measured specific gravity of the ammonia is not 0.9, use the equivalent volume as calculated from a table of specific gravity vs. percentage ammonia.

Standard Lead Solution - Dissolve 1.5984 g of pure lead nitrate in one liter of 1:99 nitric acid to provide a strong stock solution containing one mg Pb per ml. Pipet exactly 20 ml into a 500-ml volumetric flask and make to mark with 1:99 nitric acid to give a dilute stock solution containing 40 µg Pb per ml. (A standard lead solution, 10 µg Pb/ml, was stable in 1:99 nitric acid for three years.) Prepare a working solution, containing 2 µg Pb per ml, just before it is needed by pipetting 5 ml of the dilute stock solution into a 100-ml volumetric flask and making to mark with 1:99 nitric acid.

Phenol Red - 0.1% aqueous solution.

Ashing Aid Acid - Dissolve 25 g potassium sulfate in sufficient redistilled concentrated nitric acid to make 100 ml.

White Petrolatum - Supplied in a glass jar, for greasing stopcocks. To check on the purity, put a pinch of this petrolatum in a beaker, add a few milliliters of the standard dithizone and swirl. If the dithizone is no longer green after a few minutes, the material is unsatisfactory for greasing stopcocks.

APPARATUS

A Beckman Model DU Spectrophotometer has been used in this laboratory since this instrument became available in the 1940s. However, the Beckman Model B and the Bausch and Lomb Spectronic 20 have been shown to give comparable results for blood lead determinations, provided that appropriate standardizations are conducted with each instrument. Other laboratories, whose results are reported in this paper, have presumably used a diversity of available photometers and spectrophotometers. In our laboratory, 22 x 175 mm matched test tubes are used in most spectrophotometric procedures employing the Model DU, which is fitted with a tube holder which does not interfere with the use of the instrument with regular cells. These same tubes are used in the Model B fitted with a test tube adaptor. A 3/4-inch tube, supplied by the manufacturer, is used in the Bausch and Lomb Spectronic 20.

Borosilicate glassware is used throughout the procedures (except for vacutainers used for blood sampling). Ashing is performed in 125- or 250-ml Phillips beakers. Automatic burettes are used for the addition of most reagents. The extractions are conducted in the Squibb-type, 125-ml separatory funnels supported in electrically operated shakers provided with timer switches. The stopcocks of the separatory funnels

are greased with white petrolatum (purchased in a glass jar rather than in a metal can or tube) unless Teflon stopcocks, which require no grease, are used. All glassware should be reserved for trace analysis only to avoid possible gross contamination.

Soak all ashing beakers in a detergent solution (Alconox or Duponol is suitable) immediately after each usage to prevent any material from drying on the surfaces. Rinse 8-10 times with hot water and store in a dust-proof drawer or cabinet until needed. Use the following acid cleaning, lead-freeing techniques immediately before the next use of the glassware: Rinse the ashing beakers with a saturated solution of sodium dichromate in concentrated sulfuric acid. Leave a 1-2 ml portion in the beaker or flask (proportionally less in a small volumetric flask!). Add about 5-10 ml of warm tap water and allow the hot solution to flow over all inner surfaces to remove the last traces of grease. Rinse with three or four portions of cold tap water. Rinse with one portion of either concentrated or 1:1 nitric acid, as preferred. (This wash nitric acid may be used repeatedly until it loses its strength.) Then rinse successively with three or four portions each of tap water, distilled and double-distilled water. Set the beakers upright on the bench and cover with a clean dust-case or a large piece of filter paper (or otherwise protect from dust). Under no circumstances is glassware turned upside down to drain on a towel or cheesecloth placed on a laboratory bench. Use an oven operating at 105 C if dry glassware is required.

Separatory funnels are rinsed with tap water immediately after use. If a high lead sample was present or if a visible precipitate remains on the inside, it is rinsed with a small portion of 1:1 wash nitric acid (which is discarded), followed by tap water. The stoppered funnels are stored in double-deck racks. Immediately before use, stopcocks are regreased if necessary. Then the funnels are rinsed with wash acid, four times with tap water, and four times with distilled water. Each rinse is accomplished by shaking with the stopper, then draining through the stopcock with two or three turns.

Spectrophotometer tubes are rinsed four times each with tap and distilled water immediately after use. They are placed upright in a large beaker and dried in an oven at 105 C, then stored under a dust-cover. Occasionally they are cleaned with dichromate-sulfuric acid and nitric acid as described above.

(NOTE: With this method of cleaning glassware we have never encountered cross-contamination from chromium, lead, or from any other trace element being determined routinely in this laboratory.)

ANALYTICAL PROCEDURE

1. Warm the sample ash (prepared as described in the following sections) with 2 ml of concentrated nitric acid for a few minutes, then add 25 ml of distilled water, heating on the hotplate until a clear solution is obtained.

2. Cool to room temperature. Add to the solution in the beaker one ml of hydroxylamine hydrochloride, 4 ml of sodium citrate (10 ml is required for a urine sample), one drop of phenol red indicator, and titrate to a strong red color with concentrated ammonia reagent. Add a few drops excess of ammonia to make sure that the pH is between 9 and 10, using fresh pHydrion test paper to check the pH.

(NOTE: Phenol red has a weak orange-red color in strong acid, yellow in weak acid, and a red color in alkaline solution. Do not mistake the

first color for that produced in alkaline medium!)

3. Transfer the sample quantitatively with double-distilled water rinsings to a 125-ml Squibb separatory funnel containing 5 ml of the potassium cyanide reagent.

4. Add 5 ml of the extraction dithizone and shake two minutes, after releasing the initial pressure by momentarily opening the stopcock of the inverted separatory funnel. Allow the chloroform layer to settle.

5. Draw off most of the extraction dithizone into a second funnel containing exactly 30 ml of 1:99 nitric acid.

6. Add a second 5-ml portion of extraction dithizone to the first funnel and shake as before. Allow the layers to separate and combine the extracts in the second funnel. Continue this process with fresh portions of extraction dithizone until the reagent remains green. A rough estimate of the lead present in the sample may be made on the basis of 20 μg for each cherry-red 5-ml extract portion.

7. Shake the second funnel for two minutes to transfer the lead to the 1:99 nitric acid layer. Allow the layers to separate. Discard the chloroform layer.

8. Shake the nitric acid solution with approximately 5 ml of reagent chloroform and let settle. Drain the settled chloroform through the stopcock bore as completely as possible without loss of the aqueous layer. Evaporate the last drop of chloroform clinging to the upper surface of the liquid.

(NOTE 1: Start a zero lead standard at the beginning of this step by placing 30 ml of 1:99 nitric acid in a separatory funnel. This zero lead standard will be used to set the spectrophotometer at zero absorbance for

each series of samples being analyzed.)

(NOTE 2: If the quantity of lead estimated for any sample exceeds the 25 µg range of the colorimetric determination, pipet an appropriate aliquot of the nitric acid solution at the end of step 7 into a clean separatory funnel containing 5 ml 1:99 nitric acid to minimize errors caused by possible leakage of the stopcock, add sufficient additional 1:99 nitric acid to make 30 ml total volume, and continue with step 8.)

(NOTE 3: Start lead standards at this point if required. Add 5-ml portions of 1:99 nitric acid to each of four separatory funnels, then 2.5, 5.0, 7.5, and 12.5 ml of dilute standard lead solution (2 µg Pb/ml) from a burette, respectively to the separatory funnels, finally add the proper quantity of 1:99 nitric acid to make total volume 30 ml in each. Continue with step 8.)

9. Add 6.0 ml of the ammonia-cyanide mixture, exactly 15.0 ml of the standard dithizone, and shake two minutes. Allow the layers to separate. Drain the chloroform layer containing the lead dithizonate into a clean, dry test tube, and cork the tube immediately.

10. Decant this solution carefully into a dry photometer tube leaving the water behind. If any water spots are visible in the optical light path, transfer again to another photometer tube.

11. Set the spectrophotometer at a wavelength of 510 nm.

12. Set the instrument at zero absorbance using the zero lead standard solution.

13. Read the absorbances of the samples and of the reagent blank.

14. Calculate the lead content of each by multiplying its absorbance by the standardization factor (which is the slope of the standardization plot in micrograms of lead per unit of absorbance.) Subtract the blank value from the gross lead content of each sample to obtain the net amount of lead expressed in micrograms.

SPECIAL MATERIALS FOR BLOOD SAMPLING

1. Vacutainers, Becton-Dickinson, No. 3208, 20-ml or 10-ml, complete with stoppers are used for blood sampling. The vacutainers are used repeatedly and are lead-freed by the technique described previously. Blood is removed from the vacutainers and the stoppers, after each use, by soaking in cold tap water. When no further visible trace of blood remains on these items, they are soaked overnight in the detergent solution. They are then rinsed repeatedly with hot tap water to remove alkaline materials. The vacutainers are then subjected to the chromic and nitric acid cleaning procedures. The stoppers are soaked for 20- to 30-minute periods, three times, with single distilled water and finally three times with double distilled water. The lead-freed vacutainers are dried at 105 C, fitted with clean stoppers, and stored in a drawer reserved for them. Layers of cheesecloth are placed between the separate layers of vacutainers and the drawer is sealed with masking tape to prevent the admittance of any dust. They are evacuated just before shipment to the field. A vacuum tester is used both in the laboratory and field to test for loss of vacuum, which usually will not occur until stoppers have been used several times.

2. Vacuum Tester, High Frequency, Fisher Cat. No. 1-179, or equivalent.

3. Needles, Becton-Dickinson, Gauge 20, one and one-half inches in length, stainless steel, B-D No. 3200 N. As these needles are used repeatedly, check the tips for burrs by drawing them across the thumb nail. When burrs develop either discard the needles or file off the burrs. After filing, they must be recleaned. Vacutainer needles are soaked in a dilute detergent solution. A Becton-Dickinson Needle Cleaner, No. 3200 C, is used to force detergent solution and subsequent rinse water through the needles. Needles are subjected to thorough rinsing with distilled water. They are then placed in steritubes and either autoclaved or heated for two hours in a drying oven operating at 180 C. The steritubes are then fitted with rubber caps.

4. Steritubes, Becton-Dickinson, No. 3200 D, with rubber caps.

5. Stillets for No. 3200 N needles, 20 Gauge, two and seven-eighths inches long.

(These BD items are available from the Becton-Dickinson Company, Rutherford, New Jersey.)

COLLECTING AND ASHING BLOOD SAMPLES

Collect a 10-ml sample of whole blood using a lead-free vacutainer and a sterilized, stainless steel needle. In the laboratory, transfer the sample to a weighed, lead-free, 125-ml borosilicate Phillips beaker. No aliquoting of the blood is permissible, as most of the lead is present in the clot. Determine the weight of the blood sample to the nearest 0.01 gram, weighing rapidly to minimize evaporation. Add 2 ml of ashing aid acid reagent. Add 7 ml of concentrated nitric acid. (This ashing system

permits the analyst to handle a large number of samples at a time as the blood clot breaks up readily and smoothly without bumping and without requiring the constant attention of the analyst.) Place the samples on a hotplate operating about 130 C and evaporate just to dryness. After the water is driven off in the initial evaporation to dryness, keep the beaker covered with a lead-free watchglass to increase the reflux action of the concentrated acid. This serves to wash solids down from the sides to the hotter zone at the bottom, and also reduces the amount of acid needed. Cool the beaker briefly and then add successive portions of the nitric acid ranging from 2 ml down to 0.5 ml as the ashing proceeds. Do not remove the watchglass at any time but merely slide it back sufficiently to facilitate each new addition of the acid. Each time, as soon as the residue becomes light colored, heat on a 400 C hotplate just long enough to blacken the residue, then remove and cool the sample. Throughout the remainder of the ashing procedure, alternately heat the sample with a few drops of nitric acid on the 130 C hotplate and bake the residue for the few minutes required to darken it on the 400 C hotplate. Finally, the residue will remain pale yellow or light brown (due to iron content) after heating for 5-10 minutes at the high temperature. Avoid excess baking at this stage as the ash will become decomposed to a difficultly soluble form. It is now ready for solution and analysis. Report results as milligrams of lead per 100 grams of whole blood.

COLLECTING AND ASHING URINE SAMPLES

Use lead-free, narrow-mouthed, reagent-type, borosilicate, 250-ml bottles provided with standard taper glass stoppers to collect grab samples of

urine. Add 2.0 ml of a 37% formalin solution as a preservative, shaking the bottle 10-12 times after the contribution of the urine to mix the specimen with the formalin thoroughly.

Alternatively, urine specimens may be collected in 125-ml polyethylene bottles containing as a preservative 100-200 mg of EDTA (acid form) per bottle. This is convenient and economical for shipping samples considerable distances.

If the urine sample is clear and only one or two days old, measure a 50 ml portion into a graduated cylinder. However, if the sample is older, much of the lead may be in a sediment or on the walls of the bottle and must be dissolved before aliquoting. Transfer the entire specimen to a glass-stoppered graduated cylinder, record the volume, rinse the sample bottle with three small portions of concentrated nitric acid and add these rinsings to the cylinder. Mix thoroughly (Caution! Old samples may foam over.) Note the total volume and remove an aliquot equivalent to 50 ml of urine for analysis. Transfer the aliquot portion to a lead-free, 250-ml borosilicate Phillips beaker and add 5 ml of redistilled concentrated nitric acid. Evaporate just to dryness on a hotplate operating at about 130 C. Cool, add sufficient nitric acid to moisten the residue and cover the beaker with a lead-free watchglass. Heat on the 130 C hotplate and then alternately bake for a few minutes and digest with minimal amounts of nitric acid (as described in the ashing method for blood) until a white residue remains after the final heating for 5-10 minutes at the high temperature. The sample is now ready for solution and analysis. Report results as milligrams of lead per liter of urine.

PROCEDURE FOR AIR SAMPLES

It is convenient to wash out samples in electrostatic precipitator tubes with redistilled ethanol, using a special policeman made with a rubber disc cut to fit the tube like a piston, and transferring the sample through a short stem funnel into a 250-ml Phillips beaker; gently evaporate just to dryness. (Ethanol is helpful in removing greasy deposits on the walls of the precipitator tube. Some chemists may prefer hot 1 to 5% nitric acid to transfer the sample.) Transfer impinger samples or membrane filter samples to Phillips beakers. If little ash is expected (usually for impinger or membrane filter samples), add 2 ml of ashing aid acid reagent. (The presence of this salt will prevent loss of lead by glazing onto the surface of the beaker during ashing.) Otherwise add 1-2 ml nitric acid. Evaporate to dryness. Continue ashing with nitric acid at a moderate heat until organics are destroyed.

Dissolve the ash in 2 ml of concentrated nitric acid and distilled water and then transfer quantitatively to a 100-ml volumetric flask and make to mark. Pipet a suitable aliquot into a separatory funnel, containing about 5 ml of double-distilled water, add sufficient additional double-distilled water to make the total volume about 25 ml, and apply the Analytical Procedure, starting with step 2. In step 3, as the sample is already in a separatory funnel, merely add the cyanide. The amount of lead present in the aliquot may be estimated as described in step 6. If it is less than a few micrograms, an additional aliquot may be added to the same funnel, and the pH readjusted with ammonia. The extraction is then continued, and extracts combined with those collected previously in the second funnel. If the estimated amount

of lead exceeds the range of the method (25 micrograms), take an aliquot as described in Note 2, step 8.

When calculating the results, make allowance for the total number of aliquots. If convenient, aliquot the reagent blank in the same manner so that the correction represents the same amounts of ashing and extraction reagents as are present in the sample. However, the blank correction is usually small for air samples. Report results as milligrams of lead per cubic meter of air.

IX. _ APPENDIX III

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material containing lead shall be provided in the appropriate section of the Material Safety Data Sheet or other approved form. If a specific item of information is inapplicable (i.e. flash point) initials "n.a." (not applicable) should be inserted.

(i) The product designation in the upper left hand corner of both front and back to facilitate filing and retrieval. Print in upper case letters in as large print as possible.

(ii) Section I. Name and Source

(A) The name, address, and telephone number of the manufacturer or supplier of the product.

(B) The trade name and synonyms for a mixture of chemicals, a basic structural material, or for a process material; and the trade name and synonyms, chemical name and synonyms, chemical family, and formula for a single chemical.

(iii) Section II. Hazardous Ingredients

(A) Chemical or widely recognized common name of all hazardous ingredients.

(B) The approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range of maximum amount, i.e., 10-20% V; 10% max. W.

(C) Basis for toxicity for each hazardous material such as established OSHA standard in appropriate units and/or LD_{50}, showing amount and mode of exposure and species or LC_{50} showing concentration and species.

(iv) Section III. Physical Data

(A) Physical properties of the total product including boiling point and melting point in degrees Fahrenheit; vapor pressure, in millimeters of mercury, vapor density of gas or vapor (air = 1), solubility in water, in parts per hundred parts of water by weight; specific gravity (water = 1); volatility, indicate if by weight or volume, at 70° Fahrenheit; evaporation rate for liquids (indicate whether butyl acetate or ether = 1); and appearance and odor.

(v) Section IV. Fire and Explosion Hazard Data

(A) Fire and explosion hazard data about a single chemical or a mixture of chemicals, including flash point, in degrees Fahrenheit; flammable limits, in percent by volume in air; suitable extinguishing media or agents; special fire fighting procedures; and unusual fire and explosion hazard information.

(vi) Section V. Health Hazard Data

(A) Toxic level for total compound or mixture, relevant symptoms of exposure, skin and eye irritation properties, principal routes of absorption, effects of chronic (long-term) exposure, and emergency and first aid procedures.

(vii) Section VI. Reactivity Data

(A) Chemical stability, incompatibility, hazardous decomposition products, and hazardous polymerization.

(viii) Section VII. Spill or Leak Procedures

(A) Detailed procedures to be followed with emphasis on precautions to be taken in cleaning up and safe disposal of materials leaked or spilled. This includes proper labeling and disposal of containers containing residues,

contaminated absorbants, etc.

(ix) Section VIII. Special Protection Information.

(A) Requirements for personal protective equipment, such as respirators, eye protection and protective clothing, and ventilation such as local exhaust (at site of product use or application), general, or other special types.

(x) Section IX. Special Precautions.

(A) Any other general precautionary information such as personal protective equipment for exposure to the thermal decomposition products listed in Section VI, and to particulates formed by abrading a dry coating, such as by a power sanding disc.

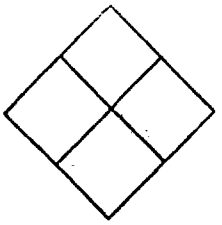
(xi) The signature of the responsible person filling out the data sheet, his address, and the date on which it is filled out.

(xii) The NFPA 704M numerical hazard ratings as defined in section (c) (5) following. The entry shall be made immediately to the right of the heading "Material Safety Data Sheet" at the top of the page and within a diamond symbol preprinted on the forms.

PRODUCT DESIGNATION

MATERIAL SAFETY DATA SHEET

Form Approved
Budget Bureau No.
Approval Expires
Form No. OSHA



SECTION I SOURCE AND NOMENCLATURE

MANUFACTURER'S NAME	EMERGENCY TELEPHONE NO.
ADDRESS (Number, Street, City, State, ZIP Code)	
TRADE NAME AND SYNONYMS	CHEMICAL FAMILY
CHEMICAL NAME AND SYNONYMS	FORMULA

SECTION II HAZARDOUS INGREDIENTS

BASIC MATERIAL	APPROXIMATE OR MAXIMUM % WT. OR VOL.	ESTABLISHED OSHA STANDARD	LD_{50}		LC_{50}	
			ORAL	PERCUT.	SPECIES	CONC.

SECTION III PHYSICAL DATA

BOILING POINT °F.	VAPOR PRESSURE mm Hg.
MELTING POINT °F.	VAPOR DENSITY (Air=1)
SPECIFIC GRAVITY (H_2O=1)	EVAPORATION RATE (_____=1)
SOLUBILITY IN WATER Pts/100 pts H_2O	VOLATILE % Vol. % Wt.
APPEARANCE AND ODOR	

SECTION IV FIRE AND EXPLOSION HAZARD DATA

FLASH POINT	FLAMMABLE (EXPLOSIVE) LIMITS	UPPER
METHOD USED		LOWER
EXTINGUISHING MEDIA		
SPECIAL FIRE FIGHTING PROCEDURES		
UNUSUAL FIRE AND EXPLOSION HAZARDS		

PRODUCT DESIGNATION

SECTION V HEALTH HAZARD DATA	
TOXIC LEVEL	CARCINOGENIC
PRINCIPAL ROUTES OF ABSORPTION	SKIN AND EYE IRRITATION
RELEVANT SYMPTOMS OF EXPOSURE	
EFFECTS OF CHRONIC EXPOSURE	
EMERGENCY AND FIRST AID PROCEDURES	

SECTION VI REACTIVITY DATA
CONDITIONS CONTRIBUTING TO INSTABILITY
CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION
INCOMPATIBILITY (Materials to Avoid)
HAZARDOUS DECOMPOSITION PRODUCTS

SECTION VII SPILL OR LEAK PROCEDURES
STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED
WASTE DISPOSAL METHOD

SECTION VIII SPECIAL PROTECTION INFORMATION	
VENTILATION REQUIREMENTS LOCAL EXHAUST	PROTECTIVE EQUIPMENT (Specify Types) EYE
MECHANICAL (General)	GLOVES
SPECIAL	RESPIRATOR
OTHER PROTECTIVE EQUIPMENT	

SECTION IX SPECIAL PRECAUTIONS
PRECAUTIONS TO BE TAKEN IN HANDLING AND STORAGE
OTHER PRECAUTIONS

Signature _____ Address

Date

TABLE X-1

Physical Properties of Lead

Property	Value	
Atomic Number	82	
Atomic Weight	207.19	
Thermal Conductivity	0.346 watts/cm C	(25 C)
Density	11.344 g/ml	(16 C)
Melting Point	327.5 C	
Boiling Point	1744 C	
Electrical Resistivity	20.6 μohm-cm	(20 C)

Adapted from reference[1]

TABLE X-2

Lead consumption in the United States, by products 1969

(Short tons)

Product	1969
Metal products:	
Ammunition	79,233
Bearing metals	17,406
Brass and bronze	21,512
Cable covering	54,203
Calking lead	44,857
Casting metals	9,918
Collapsible tubes	12,484
Foil	5,881
Pipes, traps, and bends	19,407
Sheet lead	25,818
Solder	72,626
Storage batteries:	
Battery grids, posts, etc.	280,386
Battery oxides	302,160
Terne metal	1,583
Type metal	25,660
Total	973,134
Pigments:	
White lead	6,617
Red lead and litharge	79,898
Pigment colors	14,670
Other	1,201
Total	102,386
Chemicals:	
Gasoline antiknock additives	271,128
Miscellaneous chemicals	602
Total	271,730
Miscellaneous uses:	
Annealing	4,252
Galvanizing	1,797
Lead plating	406
Weights and ballast	17,366
Total	23,821
Other, unclassified	18,287
Grand total	389,358

Adapted from Reference 2

TABLE X-3

Potential Occupational Exposures to Inorganic Lead

Babbitters
Battery makers
Bookbinders
Bottle cap makers
Brass founders
Brass polishers
Braziers
Brick burners
Brick makers
Bronzers
Brush makers
Cable makers
Cable splicers
Canners
Cartridge makers
Ceramic makers
Chemical equipment makers
Chippers
Cutlery makers
Demolition workers
Dental technicians
Diamond polishers
Dye makers
Electronic device makers
Electroplaters
Electrotypers
Emery wheel makers
Enamel burners
Enamelers
Enamel makers
Farmers
File cutters
Filers
Flower makers, artificial
Foundry molders
Galvanizers
Glass makers
Glass polishers
Gold refiners
Gun barrel browners
Incandescent lamp makers
Insecticide makers
Insecticide users
Japan makers
Japanners
Jewelers
Junk metal refiners
Lacquer makers
Lead burners
Lead counterweight makers
Lead flooring makers
Lead foil makers
Lead mill workers
Lead miners
Lead pipe makers
Lead salt makers
Lead shield makers
Lead smelters
Lead stearate makers
Lead workers
Linoleum makers
Linotypers
Lithographers
Match makers
Metal burners
Metal cutters
Metal grinders
Metal miners
Metal polishers
Metal refiners
Mirror silverers
Motor fuel blenders
Musical instrument makers
Painters
Paint makers
Paint pigment makers
Patent leather makers
Pearl makers, imitation
Pipe fitters
Plastic workers
Plumbers
Pottery glaze mixers
Pottery workers
Putty makers
Riveters
Roofers
Rubber buffers
Rubber makers
Scrap metal workers
Sheet metal workers
Shellac makers
Ship dismantlers
Shoe stainers
Shot makers
Solderers
Solder makers
Steel engravers
Stereotypers
Tannery workers
Temperers
Tetraethyl lead makers
Tetramethyl lead makers
Textile makers
Tile makers
Tin foil makers
Tinners
Type founders
Typesetters
Varnish makers
Wallpaper printers
Welders
Zinc mill workers
Zinc smelter chargers

From reference 3

Operation	General Exposure Incidence of Plumbism
Metalizing	High
Paint spraying: red lead	High
Brush painting: red lead	Some
Paint sanding, scraping	High
Leaded iron pouring	High
Bearing bronze pouring	Some
Bearing bronze grinding	Low
Storage-battery manufacturing:	
Mixing	Some
Pasting	Some
Grouping	Some
Separating	Low
Casting	Low
Lead smelting, refining	Some
Lead burning	Some
Homogenizing	Some
Painted-steel burning	Some
Lead powder mixing	Some
Lead sanding, grinding	Some
Paint mixing	Low
Painting, N.O.C.	Low
Paint spraying: chrome yellow	Low
Wire patenting	Low
Steel tempering	Low
Bronze pouring	Low
Bronze grinding	Low
Lead casting	Low
Printing:	
Stereotyping	Low
Linotyping	None
Soldering, tinning	Low
Lead sawing	Low
Lead glass working	Low
Gasoline-tank cleaning	Low

TABLE X-4

from Operations Utilizing Lead

Average Lead Concentrations Found			
Air (mg/m^3)		Urine (mg/l)	
Avg	Max	Avg	Max
1.8	3.5		
		0.26	0.35
0.32		0.30	0.48
19.5			
1.86	3.4	0.54	0.82
0.84		0.33	
0.73	3.8	0.70	1.00
0.75	2.1	0.26	0.48
0.50	4.0	0.22	0.68
0.15	0.41	0.15	0.27
0.26	0.65	0.19	0.31
0.35	1.45	0.35	0.88
0.57	1.5	0.26	0.37
3.0			
		0.41	0.50
2.2	10.2	0.22	0.32
4.2	7.4	0.26	
1.75	5.8	0.17	0.29
		0.09	0.16
3.9		0.10	
0.29	0.60	0.12	0.21
0.13	0.22	0.10	0.21
0.34	1.56	0.20	0.34
0.47	1.24	0.17	0.34
0.12	0.35	0.14	0.37
0.26	0.51	0.15	0.22
0.07	0.24	0.08	0.14
0.25	0.62	0.15	0.23
0.25			
0.01	0.02	0.05	0.10
		0.07	0.14

TABLE X-5

Biochemical Values at Two Airborne Levels of Lead

Air Pb conc. mg/m	Biochemical Test, mean (and 95% confidence limits)			
	Blood Pb mg/100 ml	Urine Pb mg/liter	Urine Coproporphyrin (Donath)	Urine ALA* mg/100 ml
0.20	0.070 (0.048-0.092)	0.143 (0.056-0.230)	4.2 (2.4-6.0)	1.8 (0.3-3.3)
0.15	0.060 (0.038-0.082)	0.118 (0.031-0.205)	3.6 (1.8-5.4)	1.4 (0.1-2.9)

* ALA values were determined by a method which probably gives higher values than do other methods, thus a high "normal" value.

From Williams, King and Walford [47]

TABLE X-6

Representative Lead Exposures in the Printing Industry

Location	Nature of Operations or Exposure	Lead Concentration in mg/m^3			Remarks
		Max.	Min.	Ave.	
Linotype Room	Lead concentration about 12" above lead pot of one of centrally located machines	0.027	0.007	0.014	Pot temperature ranged from 515° to 550° F.
	Exposure of machine operators	0.020	0.006	0.012	
Monotype Room	Lead concentration about 12" above lead pot of one of centrally located machines	0.570	0.056	0.163	Pot temperature ranged from 660° to 835° F.
	Exposure of machine operators	0.096	0.027	0.056	
Remelt Room	Average room concentration	0.158	0.004	0.041	Melt kettles enclosed are exhaust ventilated
	Workers' exposure while filling molds	0.132	0.035	0.073	Worker's face about 18 to 24[11] above molds while being poured. Lead temperature 600° to 700° F.
	Room concentration while drossing kettles and while removing copper plates from electrotype	0.257	0.149	0.196	Several kettles drossed during sample but only one kettle door open at a time
Composing Room	Average room concentration	0.118	0.016	0.062	
Stereotype Room	Concentration at or near the breathing level of workers operating lead pots, pouring molds, etc.	0.026	0.003	0.008	Pot temperature ranged from 550° to 600° F.
	Exposure of operators of trimming and finishing machines such as saws, bevelers, planers and routers	0.442	0.002	0.104	

Adapted from reference [53]

TABLE X-7

REPRESENTATIVE LEAD EXPOSURE IN PRINTING OPERATIONS

Description of Exposure	No. Of Samples	Range mg/m^3	Mean mg/m^3
Lead Concentrations over Linotype Melting Pots	9	< 0.01 - 0.054	0.029
Concentrations While Cleaning Linotype Plungers	6	0.06 - 2.8	0.783
Concentrations Around Metal Pots While Removing Dross	9	1.4 - 160.0	29.30
Atmospheric Lead at Breathing Zone of Linotype Operators	17	< 0.01 - 0.049	0.021
Atmospheric Lead in Hand Composing Areas Adjacent to Linotypes	7	< 0.01 - 0.045	0.017
Lead in General Atmosphere of Monotype Rooms	12	< 0.01 - 0.060	0.028
Lead Concentration 6 inches Above Monotype Metal Pots	22	< 0.01 - 10.0	1.070
Lead Concentrations 19 inches Above Monotype Metal Pots	8	< 0.01 - 0.38	0.148
Atmospheric Lead in Vicinity of Unexhausted Remelt Furnace During Various Phases of Operation			
1. Loading & Heating	8	< 0.01 - 0.16	0.052
2. Cleaning & Drossing	7	5.10 - 50.0	15.26
3. Pouring	7	0.094 - 0.78	0.313
Atmospheric Lead in Vicinity of Exhausted Remelt Furnace During Various Phases of Operation			
1. Loading & Heating	2	0.881 - 0.15	0.116
2. Cleaning & Drossing	2	1.8 - 5.3	3.55
3. Pouring	2	0.053 - 0.15	0.102

Sampling - Electrostatic Precipitator
Analysis - Dithizone
Adapted from reference [54]

TABLE X-8

Representative Lead Exposure in the Printing Industry

	Years in Printing	Calculated Exposure mg/m^3	Urine Lead mg/liter
Linotype Operators			
	9	0.03	-
	16	0.03	0.11
	15	0.10	0.04
	6	0.02	-
	20	0.02	0.17
	15	0.02	0.11
	19	0.02	0.17
	38	0.02	-
	12	0.02	-
	22	0.02	-
	11	0.02	-
	40	0.09	0.16
	18	0.02	0.11
	3	0.02	0.32
	8	0.04	0.21
	6	0.02	0.19
	4	**0.02**	**0.24**
	15	0.10	0.28
	20	0.10	0.26
Monotype Operators			
	3	0.04	0.03
	10	0.09	0.28
	19	0.06	0.17
	7	0.04	0.10
	17	0.06	0.18
Remelt Men			
	2	0.38	0.17
	7	0.15	0.13
	1	0.04	0.28
	10	0.09	0.06
	3	0.50	-
	5	0.03	-
	9	0.13	0.19

TABLE X-8 Cont

	Years in Printing
Stereotypers	
	1
	10
	4
	1
Others	
	26
	1
	2
	6
	10

Sampling - Electrostatic Precipitator
Analysis - Dithizone

Adapted from Reference 55

Calculated Exposure mg/m^3	Urine Lead mg/liter
0.09	0.27
0.10	0.17
0.08	0.29
0.10	0.26
0.02	0.23
0.03	0.36
0.07	0.23
0.02	-
0.02	-

TABLE X-9

Representative Mean Lead Exposures and Biologic Lead Levels for Workers in the Storage Battery Industry

Job	Number Workers	Air Lead Concentration, mg/m^3		Blood Lead Concentration, µg/100g Blood		Urine Lead Concentration, µg/Liter	
		Mean	SE	Mean	SE	Mean	SE
Machine pasting	6	0.218	0.025	74.2	4.7	163.8	21.2
Hand pasting	8	0.150	0.029	63.2	9.2	111.3	14.1
Forming	9	0.134	0.013	63.0	2.7	114.0	7.2
Casting	6	0.052	0.003	-	-	87.9	6.8
Plastics department A	5	0.012	0.0008	27.2	1.4	34.5	3.2
Plastics department B	5	0.009	0.0008	29.1	1.6	34.8	2.0

Adapted from reference [47]

TABLE X-10

Average and Median Blood Lead Content in mg/100 g of Blood in Storage-Battery Workers, by Exposure and Duration of Employment.

Duration of Lead Exposure, Years	Air Lead Content, mg/m^3				
	0-0.074	0.075-0.14	0.15-0.29	≥0.3	% >0.15
0-4					
Number	17	16	32	20	
Average	0.0187	0.0316	0.0378	0.0463	59
Median	0.021	0.030	0.038	0.050	
5-9					
Number	10	13	40	24	
Average	0.0278	0.0405	0.0501	0.0505	74
Median	0.033	0.040	0.043	0.050	
10-14					
Number	23	24	30	32	
Average	0.0198	0.0375	0.0502	0.0481	57
Median	0.018	0.038	0.046	0.048	
15+					
Number	44	30	59	45	
Average	0.0293	0.0407	0.0457	0.0493	58
Median	0.023	0.036	0.045	0.045	

Analysis - Dithizone

Adapted from references 4 and 11

TABLE X-11

REPRESENTATIVE LEAD EXPOSURES WHILE PERFORMING

WELDING OPERATIONS UNDER VARIOUS CONDITIONS

Coating	Type weld	Location of sampling probe	Lead	Avg.
POOR VENTILATION†		EXPERIMENTAL AREA	mg/m^3	
Zinc-silicate	Elect. arc	2' directly above welding	15.2	
" "	" "	3' above and 2-1/2 back of welding*	0.86	
" "	" "	3' above and 2' back of welding*	3.27	5.63
" "	" "	3' above and 2' back of welding*	3.65	
" "	" "	Attached to welder's shoulder*	5.16	
Zinc-silicate	Oxy-acetylene	1' above and 1' back of welding*	3.53	
" "	" "	3' above and 2-1/2' back of welding*	1.24	
" "	" "	3' above and 2-1/2 back of welding*	1.56	
" "	" "	3' above and 2' back of welding*	1.80	1.96
" "	" "	3' above and 2' back of welding*	1.80	
" "	" "	3' above and 2' back of welding*	1.76	
" "	" "	3' above and 2' back of welding*	2.00	
Galvanized steel	Elect. arc	2' above and 1' back of welding*	0.40	
" "	" "	2' above welder's face	0.69	
" "	" "	6' above floor, 5' in front of welder	0.35	0.52
" "	" "	Attached to welder's shoulders*	0.64	
Galvanized steel	Oxy-acetylene	2'above and 2' back of welding*	0.66	
" "	" "	3' above and 2-1/2' back of welding*	0.24	
" "	" "	2' above and 1' back of welding*	0.41	0.43
" "	" "	6' above and 5' back of welder	0.30	
" "	" "	3' above and 1' back of welding	0.55	

TABLE X-11 (CONTINUED)

Coating	Type weld	Location of sampling probe	Lead	Avg.
Clean steel	Elect. arc	2' above and 1' back of welding. (Control sample)	0	
" "	Oxy-acetylene	20' from welding enclosure (Room air. Control sample)	0	
" "	Elect. arc	20' from welding enclosure (Room air. Control sample)	0	
GOOD VENTILATION		(BREATHING ZONE SAMPLES)		
Zinc-silicate	Oxy-acetylene cutting	Attached near welder's nose**	0.18	
Zinc-silicate	Electric arc beading	Inserted in welder's hood**	0.08	
Zinc-silicate	Electric arc welding	Inserted in welder's hood**	0.14	
Galvanized steel	Oxy-acetylene cutting	Attached near nose**	0.01	
Galvanized steel	Electric arc welding	Inserted in welder's hood**	0.01	
ROOM AIR SAMPLES		(DOWNWIND FROM WELDER)		
Zinc-silicate	Elect. arc	3' downwind from the welder. 3' from floor	0.81	
" "	" "	3' downwind from the welder. 3' from floor	0.76	0.78
" "	" "	20' downwind from the welder. 3' from floor	0.26	
" "	" "	20' downwind from the welder. 3' from floor	0.24	0.25
" "	" "	20' downwind from the welder. 6' from floor	0.27	
" "	" "	20' downwind from the welder. 6' from floor	0.53	0.40

TABLE X-11 (CONTINUED)

Coating	Type weld	Location of sampling probe	Lead	Avg
OUTDOOR SAMPLES		(10 MPH WIND)		
Zinc-silicate	Elect. arc	Welder sat upwind. Probe inserted in hood.	0.06	
Galvanized steel	Elect. arc	Welder sat upwind. Probe inserted in hood.	0.01	
Galvanized steel	Oxy-acetylene (cutting)	Welder sat upwind. Probe was held 3" from nose.	0.00	

† Samples were not collected inside welder's hoods.
* Sample probe located near welder's face.
** Welder located upwind from welding.

Analysis - Dithizone
Adapted from Reference 56

TABLE X-12

Lead Exposures and Urinary Lead Levels from the Cutting of Painted Structural Steel

Exposures (Breathing Zone)	No.	Exposure mg/m^3
	1	0.18
	2	0.50
	3	2.40
	4	1.70
	Avg.	1.20

Urine-Lead	Respirator	Sp. Gr.	Mg. Lead/Liter Urine	
	Mech. Filter	1.014	0.06	
	Mech. Filter	1.025	0.34	
	Mech. Filter	1.026	0.30	
	Mech. Filter	1.030	0.53	
	Mech. Filter	1.016	0.36	
	Mech. Filter	1.020	0.58	Avg. 0.39
	Mech. Filter	1.034	0.28	
	Mech. Filter	1.025	0.70	
	Mech. Filter	1.031	0.50	
	Mech. Filter	1.020	0.49	
	Mech. Filter	1.030	0.33	
	Mech. Filter	1.020	0.26	
	Canister-Type	1.020	0.26	Avg. 0.25
	Canister-Type	1.030	0.24	

Adapted from Reference 57

TABLE X-13

DISTRIBUTION OF PERSONS IN VARIOUS OCCUPATIONAL GROUPS ACCORDING TO CONCENTRATIONS OF LEAD IN BLOOD-CINCINNATI

Lead in blood, mg/100g	Service station attend-ants 1956	Refinery handlers of gasoline 1956	Park-ing attend-ants 1956	Garage Me-chanics 1956	Drivers of cars		Police			Fire-men 1963	Post-Office Emp. 1963	City Health Dept. Emp. 1963
							Traffic officers		All police*			
					1956	1963	1956	1963	1963			
0-0.009												
0.010-0.019	1	2				1		3	12	18	22	10
0.020-0.029	42	30	1	8	17	4	7	23	78	123	90	24
0.030-0.039	71	46	26	43	19	9	9	9	27	44	24	2
0.040-0.049	14	8	20	72	9		1	4	5	6	2	
0.050-0.059	2			25							1	
0.060-0.069			1	4				1	1		1	
Totals	130	86	48	152	45	14	17	40	123	191	140	36
Mean	0.028	0.027	0.034	0.038	0.033	0.031	0.031	0.030	0.025	0.025	0.023	0.021
Std. Dev.	0.007	0.006	0.006	0.009	0.006	0.006	0.006	0.009	0.007	0.006	0.007	0.005

*Includes traffic officers for 1963.

From reference 58

TABLE X-14

DISTRIBUTION OF PERSONS IN VARIOUS OCCUPATIONAL GROUPS ACCORDING TO CONCENTRATIONS OF LEAD IN URINE-CINCINNATI

Lead in urine, mg/100g	Service station attend-ants 1956	Refinery handlers of gasoline 1956	Park-ing attend-ants 1956	Garage Me-chanics 1956	Drivers of cars		Police			Fire-men 1963	Post-Office Emp. 1963	City Health Dept. Emp. 1963
							Traffic officers		All police*			
					1956	1963	1956	1963	1963			
0-0.009	1	1	1	4	1			2	2			
0.010-0.019		1	4	2	28		9		6	47	49	12
0.020-0.029	74	49	21	39	11	5	5	13	29	71	52	18
0.030-0.039	33	22	12	33	2	4		7	21	36	19	6
0.040-0.049	13	9	7	30	2	4	3	8	30	19	9	1
0.050-0.059	5		2	21		1		2	12	9	1	
0.060-0.069	3	4	1	16				1	7	2		
0.070-0.079				4	1			1	3	1		
0.08-0.12	1			3				3	6			
Totals	130	86	48	152	45	14	17	37	116	185	130	37
Mean	0.027	0.028	0.028	0.040	0.020	0.036	0.023	0.039	0.038	0.027	0.022	0.022
Std. Dev.	0.010	0.013	0.011	0.020	0.011	0.010	0.011	0.020	0.018	0.011	0.009	0.007

*Includes traffic officers for 1963.

From reference 58

* U.S. GOVERNMENT PRINTING OFFICE: 1973—759-574/1384