Statement of

Edward J. Baier, Deputy Director
National Institute for Occupational Safety and Health
Center for Disease Control
Department of Health, Education, and Welfare

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I welcome this opportunity to appear here today to discuss the effects of occupational lead exposure upon human health including the results of recent NIOSH studies of lead workers. With me today are the following: Dr. Kenneth Bridbord, Office of Extramural Coordination and Special Projects, Dr. Edward Baker, Bureau of Epidemiology, Dr. Theodore Thoburn, Division of Surveillance, Hazard Evaluation and Field Studies, Mr. John Bryant, Division of Physical Sciences and Engineering, and Mr. Robert Schutz, Testing and Certification Branch.

As early as the first century A.D., lead was recognized as an occupational hazard, but it was not until the 1800's, however, that systematic studies were conducted on health effects of lead exposure. Symptoms associated with excessive lead absorption have been documented in painters, printers, potters and glass workers. Exposure to excessive quantities of lead is known to have significant effects on the kidney, the peripheral and central nervous systems, and the hematopoietic system.

NIOSH estimates that more than 1 million American workers are occupationally exposed to lead. Some of the highest exposures are likely to occur in industrial processes such as primary and secondary lead smelters and lead battery operations. Lead exposure occurs in more than 100 other occupations. Relatively high exposures can be encountered in occupations such as welding, metal burning, painting and printing, and lower exposures occur among workers exposed to the
handling or combustion of gasoline containing lead additives. These occupational exposures increase the underlying body burden of lead which is derived from ambient air, food, and water. Not included in our estimates are workers exposed to lead in mining, crushing, and milling operations, for which the Department of the Interior has regulatory responsibility.

Occupational exposure to inorganic lead was high on the NIOSH list of priorities and was the subject of one of the first criteria documents we issued. The criteria document, dated in 1972 and transmitted to the Occupational Safety and Health Administration (OSHA) in January, 1973, recommended reducing the existing Federal standard for maximum lead concentration from 200 to 150 micrograms per cubic meter of air as a time weighed average (TWA) exposure for an 8-hour workday, 40-hour workweek. In August, 1975 NIOSH recommended this level be reduced below 150 ug/m$^3$. NIOSH felt at the time that available data were not sufficient to define precisely how much below 150 ug/m$^3$ the air lead concentration should be set, although a level below 50 ug/m$^3$ was not felt to be necessary. In October, 1975, OSHA proposed a standard of 100 ug/m$^3$.

There is no existing OSHA standard for maximum permissible blood lead levels. For that matter, there is no existing biological standard for any substance in the workplace. The 1972 criteria document recommended a biological standard of 80 ug per 100 grams of whole blood in adults. In 1975, NIOSH recommended reducing the maximum
permissible level to 60 ug/100 g and that level was proposed by OSHA in October 1975.

In testimony last March before the Subcommittee on Manpower, Compensation, and Health and Safety, House Committee on Education and Labor, the Director of NIOSH reviewed the occupational lead problem and also sharply criticized the routine, prophylactic use of chelating agents in workers continually exposed to lead. Since that testimony, the Food and Drug Administration circulated warnings to practicing physicians throughout the country against prophylactic chelation, and OSHA also took action against this practice. Today, NIOSH wants to reiterate its concerns about prophylactic chelation. While NIOSH believes that the use of chelating agents may be justified in acute lead intoxication, the evidence does not exist to justify their use on a prophylactic basis. A further evaluation of the chelating agent problem is being submitted for the record with this testimony.

The toxicology and metabolism of lead have been extensively studied. Inorganic lead is absorbed from the lungs and the digestive tract. Lead is transported in the blood throughout the body. Lead is stored in the bones and affects the bone marrow, causing an interference in the production of hemoglobin which can lead to anemia. The interference with the synthesis of heme causes an accumulation of heme precursors in the blood, which are useful for measuring the degree of lead intoxication, as well as the quantity of lead absorbed into the body. It is apparent from the analysis of heme precursors
that lead interferes in at least two sites in the hemoglobin synthesis pathway. At one site lead interferes with the enzyme aminolevulic acid dehydrase (ALAD) leading to an increase of aminolevulinic acid (ALA) which can be measured in the urine. Lead interferes with the incorporation of iron into the protoporphyrin ring, resulting in an accumulation of protoporphyrins in the blood. The protoporphyrins may be measured as free erythrocyte protoporphyrin (FEP) by extraction from the red blood cells, or are measured directly in the blood in the form of zinc protoporphyrin, or ZPP. FEP and ZPP thus measure the same biological phenomenon.

In addition to the bone, lead is also stored in other vital organs and tissues including the kidneys and the nervous system. Lead can be mobilized from various storage sites and excreted from the intestinal tract and the kidneys.

Repeated exposure to lead can result in accumulation in the amount of lead stored in the body. Continued chronic exposures to high levels of lead, even intermittent, can cause death or permanent damage to the nervous system, serious damage to the kidneys and impairment of red blood cell production. Once the kidneys are damaged by lead, the ability of the body to excrete lead through the kidneys is impaired, thus making lead in urine a poor screening test for lead absorption.
Workers exposed to excessive levels of lead may feel weak, tired, and irritable. They may experience trembling, severe colic and digestive disturbances, and convulsions. A characteristic sign of severe lead poisoning is "wrist drop", caused by damage to the nerves controlling the extensor muscles of the forearm, wrist and fingers.

Of considerable concern are the effects resulting from long-term lead exposure. There is evidence that prolonged exposure can increase the risk of nephritis, mental deficiency, premature aging, and high blood pressure.

Another category of adverse effects includes the so-called "subclinical" changes produced by lower exposures to lead. These changes are generally measured only by laboratory tests and would not necessarily be evident by routine physical examination. These workers may have early damage to the nervous system, muscular weakness, behavioral disturbances, and interference with red blood cell production. The use of the term "subclinical", however, does not mean that these changes are without significance from a health point of view.

One of the most frequently measured of these "subclinical" changes is the excretion of ALA in the urine. Increased excretion of ALA in the urine is observed in workers as blood lead levels rise above 40 to 50 ug/100 g. A report by a National Academy of Science committee studying the effects of airborne lead concluded that this
increased excretion is significant. Another National Academy of
Science committee, this time studying the effects of lead poisoning in
children, concluded that . . . "environmental limits set to prevent
reversible effects in the hematopoietic system should serve to prevent
potentially irreversible effects in the nervous system."

Perhaps the most frequently employed measure of lead absorption
into the body is the quantity of lead in the blood. Most clinical
measures of lead toxicity have been related to blood lead
measurements. One of the greatest difficulties with the measurement
of blood leads is the high level of skill required in analytical
techniques and the great care demanded to avoid the risk of sample
contamination by lead or of lead loss. The proficiency record of
laboratories in blood lead determinations has at times been less than
adequate as shown by a recently completed Center for Disease Control
(CDC) study of commercial clinical laboratories. It was disturbing to
find that only one-third of all commercial laboratories in this study
performed acceptably, but it is encouraging that in another CDC
proficiency testing survey about two-thirds of State and public
laboratories did perform well. A copy of the commercial laboratory
proficiency testing study is being submitted for the hearing record.
The poor record of commercial laboratories on blood lead testing is
but one of the reasons why NIOSH opposes setting an occupational lead
standard based solely upon blood lead levels.
In contrast to blood lead, the ZPP test offers certain advantages. For example, ZPP is a relatively stable indicator reflecting lead absorption over a several month period; ZPP does not suffer from problems of lead contamination or lead loss; and ZPP provides an index not only of lead absorption, but also of lead effect. A major advantage of the ZPP test is that rapid, reliable and economic instruments are available which allow instantaneous readout of ZPP tests following finger stick blood specimens. One of the major problems with ZPP is that this is a very recently developed test and only limited data are available on blood lead-ZPP correlations. Further, ZPP may present calibration problems, and careful attention must also be given to quality control procedures. Under these circumstances, it would seem wise to develop a biologic screening approach which incorporates ZPP or an equivalent screening test with blood lead determinations. Provisions for biologic monitoring must, however, be accompanied by specific quality control requirements.

Another reason why NIOSH opposes setting an occupational standard based solely on biologic monitoring is that this would discourage the development and implementation of adequate engineering controls and would place undue reliance upon personal protective equipment such as respirators. NIOSH believes it is necessary to reiterate its basic recommendation that employee exposure should be reduced to the lowest possible level by use of engineering controls. Respirators may be used to protect employees while engineering controls are being installed or to temporarily supplement such controls. NIOSH does not
recommend continual respirator use as the primary method of controlling any employee's exposure. Respirators must be regarded as temporary solutions to problems or as devices available for emergency respiratory protection, not as permanent answers to employee exposure. A list of respirator requirements for the lead standard is being submitted for the record.

NIOSH endorses the concept of an occupational standard for lead which includes provisions for both environmental and biological monitoring. For example, air lead determinations can help to prevent excess lead absorption. The principal advantage of an air lead determination is to prevent excessive exposure. Since lead is absorbed by routes in addition to inhalation, a biological measure of lead absorption is necessary.

NIOSH's concern about the lead problem is evidenced by our continuing research and health hazard evaluations and other activities in lead operations. NIOSH is currently updating the lead criteria document. NIOSH and the Bureau of Epidemiology, Center for Disease Control (CDC), have conducted comprehensive occupational surveys of lead exposure at primary and secondary lead smelters. The largest of these studies, undertaken in response to requests by union representatives and state health officials, was conducted at a primary lead smelter at Kellogg, Idaho, and involved about 500 workers. Within approximately the last year NIOSH–CDC has examined nearly 1,000 workers occupationally exposed to lead, including those at Kellogg.
An interim report of our results from certain aspects of the Kellogg study is being submitted for the record as are results from other recently completed NIOSH-CDC lead studies. These studies included investigations at a secondary lead smelter in Memphis, Tennessee; a lead chemicals plant in Joplin, Missouri; a secondary lead smelter in Salt Lake City, Utah; a secondary lead smelter in Eagen, Minnesota, and a secondary lead smelter in Atlanta, Georgia. In addition, NIOSH has also supported other lead studies such as the investigation of a secondary lead smelter at Vernon, California, to be presented later at these hearings by scientists at the Mount Sinal School of Medicine. This study is being submitted for the record by NIOSH.

Among the major findings of our studies are the following:

First, epidemiologic studies of workers at 5 different lead plants across the U.S. have shown unacceptably high blood lead levels and symptoms of lead poisoning in every plant studied. Hematologic, neurologic, and renal damage due to lead were also encountered. Inappropriate medical practices were noted including the misuse of oral chelating drugs and the allowing of lead-poisoned workers to continue to be exposed to lead during chelation therapy.

Second, the usefulness of monitoring blood lead and/or protoporphyrin levels as indicators of lead toxicity was clearly shown by the high degree of correlation between these measurements and the
signs and symptoms of lead toxicity. The newly developed portable method of zinc protoporphyrin (ZPP) determination has been shown to be a promising method of screening lead-exposed workers.

Third, in several locations, home contamination with lead dust from lead plants resulted in increased lead absorption among workers' children; and in one location several cases of lead poisoning were observed. These studies illustrate the importance of good work practices such as not bringing contaminated work clothes and shoes home and showering before going home, and other practices minimizing exposure to the worker and to his or her family.

Fourth, chronic neurologic and renal effects of lead exposure were demonstrated in our studies. Neuropathy was noted at a relatively low blood lead level (81ug/100 ml) and after only 2 months of exposure. Abnormal glomerular function (elevated blood urea nitrogen and creatinine levels and decreased glomerular filtration rates) and abnormal tubular function (impaired urinary concentrating ability and reduced lead clearance rates) were noted in lead workers. These studies clearly demonstrated an increase in symptoms compatible with lead intoxication and a decrease in hemoglobin concentrations as blood lead levels rise above 60 ug/100 ml.

These findings emphasize the need for improved industrial hygiene measures in the lead industry and the importance of biological monitoring in preventing lead toxicity. Based on the data contained
in these studies, a blood lead standard no higher than 60 ug/100 ml is recommended. These data clearly establish the need to reduce the maximum blood lead levels for an individual worker from 80 ug/100 ml to 60 ug/100 ml. A blood lead level of 60 ug/100 ml is equivalent to a blood lead level of 57 ug/100 g, but this difference is well within the analytic variability for blood lead determinations. The decreased urinary lead excretion observed in workers with evidence of kidney damage argues against the retention of urinary lead as a screening test.

The results from these studies demonstrate that workers in this country are being exposed to high concentrations of lead in the air and are absorbing dangerous amounts of lead into their bodies. This is occurring despite the fact that hazards of lead exposure are well recognized and technology to reduce these hazards is, in our opinion, available.

This situation clearly demonstrates the inadequacy of current OSHA compliance programs aimed at reducing lead exposure. Data on lead compliance activities supplied to us from OSHA further confirm the inadequate enforcement of the existing lead standard. The fact that these compliance programs are inadequate reflects a lack of vigorous enforcement of the existing lead standard, not a lack of knowledge of how to control the lead hazard.
Data supplied to us from General Motors involving a lead battery plant at Muncie, Indiana, clearly demonstrate what can be done to control lead exposure at an older battery plant. The majority of departments tested at this plant had average air lead exposures during 1976 below 100 ug/m$^3$ based upon personal monitors. Blood lead levels in over 90% of the workers were 60 ug/100 ml or lower. Average exposures at this plant using personal monitors were below 100 ug/m$^3$ in the assembly areas and below 150 ug/m$^3$ in the oxide pasting and grid casting areas.

If blood lead levels are used as criteria for technical feasibility, the data reported recently from Finland are especially encouraging. Assuming that keeping 90% or more of individual blood lead levels at 60 ug/100 g or lower is a good indication of technical feasibility to meet a 100 ug/m$^3$ standard and, assuming that respirators were not generally used in these industries, then, the following industries or operations in Finland have clearly demonstrated such feasibility: crystal glass manufacturing, car radiator repair, lead glazing, cable manufacturing, scrap metal shop work, car repair, machine shop work, aluminum manufacturing, sheet metal work, paint manufacturing, shipbuilding, iron and steel founding, painting, service station work, plumbing, manufacturing electric lamps, telephone repair and installation, manufacturing radio and telephone equipment, and traffic police work.
The Finnish experience also confirms what is generally known in
the United States, i.e., that lead scrap smelting and storage battery
manufacturing present significant problems with respect to lead
absorption, but not problems which are insurmountable.

One of the important issues to be considered at this hearing
involves the relationship between exposure to lead in air and
absorption of lead into the body, measured in terms of blood lead
level. The OSHA proposal concluded that in order to keep blood lead
levels in individual workers below 60 ug/100 g, air lead levels would
have to be reduced below 100 ug/m$^3$, calculated as an 8-hour time
weighed average, 40-hour workweek. The data available to NIOSH
generally support this conclusion.

The basis for our agreement with this earlier assessment is
spelled out in two attachments to this testimony. One attachment
reviews the published studies relating blood lead levels to air lead
exposure covering primarily the equivalent occupational exposure range
up to 50 ug/m$^3$. This assessment, based on about 10 studies, concluded
that to keep blood lead levels in male workers below 40 ug/100 g, air
lead exposures have to be kept under 50 ug/m$^3$. An additional
observation from the general population is that men have higher blood
lead levels than women with comparable environmental exposures. The
reason for this is not clearly understood. The second assessment
involves an extensive analysis of air lead and blood lead data at the
General Motors battery plant at Muncie. These important data indicate
that a good correlation exists between air lead exposure (especially when based on personal samplers) and blood lead over a range of air leads up to about 200 ug/m$^3$. These data further indicate that if yearly average personal sampler air lead exposure in a department is kept below 100 ug/m$^3$, yearly average blood leads in over 90% of workers will be under 60 ug/100 ml. Similarly, if yearly average personal sampler air leads in a department are kept under 50 ug/m$^3$, yearly average blood leads will be 40 ug/100 ml or lower over half of the workers. One of the greatest impacts of reducing lead exposure in air from 200 to 100 ug/m$^3$ is a great increase in the number of workers with blood lead levels 40 ug/100 ml or lower.

In reviewing the relationship between air lead exposure and blood lead level, it is also apparent that a linear relationship between air lead and blood lead does not occur over the whole range of exposures. Incremental changes in air lead exposure in the range up to 100 ug/m$^3$ produce greater increases in blood lead than do similar increases in the range from 100–200 ug/m$^3$.

A problem of special significance in setting a lead standard, in part because of our national commitment to equal employment opportunity, is the concern for female employees of childbearing age. Lead absorbed in the bloodstream of pregnant women can cross the placenta and enter the blood of the fetus where it may cause neurologic damage to the child. Lead is also found in mothers' milk. Other special concerns are susceptible workers known to have certain
clinical conditions such as kidney problems, neurologic disorders, and anemias. Enzyme disorders and inherited hemoglobin abnormalities may also pose a similar problem.

Adequate data do not currently exist to estimate what percentage of the workforce is made up of individuals with increased susceptibility to lead intoxication. Blood lead levels required to adequately protect susceptible individuals are not well established. The fact that certain individuals may be more susceptible to lead effects is an argument for setting a blood lead standard to protect against early biochemical changes induced by lead. Protecting against these earlier changes will hopefully assure protection against the more serious and potentially irreversible lead effects, even in susceptible individuals. Women show early abnormalities in heme synthesis at lower blood leads than men, and show greater abnormalities in heme synthesis at the same blood lead level as men. Conversely, women in the general population tend to have lower blood lead levels than men exposed to similar environmental levels. NIOSH does not believe that adequate data exist to fully evaluate whether women per se are more susceptible than men to lead effects.

The real issue of susceptibility to lead and women involves effects of lead upon the fetus. NIOSH does not recommend that women of childbearing age be excluded from work involving exposure to lead, but NIOSH does recommend that present and future lead standards be
vigorously enforced as one way to provide additional protection to the fetus.

The issues of lead reproductive effects on men, as well as women, chromosomal damage, and lead carcinogenicity were discussed in a recent Department of Health, Education and Welfare report dealing with human health consequences due to lead exposure from automobile emissions. A copy of this report is being submitted for the record. The data on chromosomal damage among workers occupationally exposed to lead provide contradictory results, and no clearcut conclusions can be drawn. The data on carcinogenicity are also not substantial enough to consider lead a human carcinogen, although lead remains a suspect carcinogen based upon limited animal data. Further studies could, of course, change this conclusion.

Historic data involving undoubtedly very high exposures document that lead compounds can be used as abortifacients and that women occupationally exposed to lead have increased miscarriage rates. Lead is known to cross the placenta and lead concentrations in maternal blood and fetal/newborn blood correlate with each other, with newborn blood lead levels being somewhat lower. Recent studies have shown that blood lead levels between 50 and 80 ug/100 ml are associated with diminished fertility of male workers based upon analysis of sperm. What is not clear, is precisely the level of blood lead in the mother or father which would protect against lead effects in the newborn, including those involving the nervous system. Limited data in
experimental animals suggest that blood lead levels in the 35 to 45 ug/100 g range at birth may be associated with subtle neurologic damage. Data from young children suggest that the risk of neurologic damage increases as blood lead levels rise above 40 ug/100 g. Among the factors which make extrapolation of these data to precise standards difficult are the relative hemoconcentration of the newborn, the relative hemodilution of the pregnant woman and the possibility, not proven, of mobilization of lead from the skeleton during pregnancy. The fact that a nursing infant can be exposed to lead via mother's milk is an additional complicating factor.

The existence of potentially susceptible groups to lead, the possibility of damage to the fetus at blood lead levels in the 30 to 40 ug/100 g range and the possibility of damage to male germ cells at blood lead levels of about 50 ug/100 ml present difficult questions in recommending biologic standards for lead absorption.

Studies suggesting the need for establishing a blood lead standard at less than 60 ug/100 g would place considerable emphasis upon a limited number of observations which have not been confirmed by multiple investigators. To do this would place the recommendation of a blood lead level in the workplace on less firm ground than if NIOSH continued to endorse a blood lead maximum of 60 ug/100 g. The opinion of the Institute is to adhere to its earlier recommendation, i.e., 60 ug/100 g particularly since recent studies clearly support the need to reduce blood lead levels from 80 to 60 ug/100 g.
In adhering to the 60 ug/100 g figure, NIOSH has not relinquished its concern for possible effects that may occur below 60 ug/100 g. Adherence to this 60 ug/100 g figure should not be interpreted as firm NIOSH opposition to establishing a lower blood lead standard. In fact, NIOSH endorses a lower blood lead standard as a future goal to provide greater assurances of safety. The OSHA proposal would establish an action level of 50 ug/m$^3$ for lead in air. As noted above, exposures of 50 ug/m$^3$ or less would keep blood lead levels in virtually all workers at about 40 ug/100 g or lower. This should protect against "subclinical" effects of lead including the hematopoietic system and against other potential effects of lead as noted above. For workers exposed above the action level but below the maximum air lead level of 100 ug/m$^3$, NIOSH endorses a vigorous medical surveillance program involving, for example, ZPP testing or other screening tests to identify workers with blood lead levels of 40 ug/100 g or higher. This 40 ug/100 g figure corresponds to a biological "action level". Workers with blood lead levels of 40 ug/100 g or higher should have periodic medical exams to identify, as early as possible, any adverse effects which may occur. Such a program of medical surveillance will also help to identify workers who may be highly susceptible to lead effects. NIOSH estimates that even at the proposed air standard of 100 ug/m$^3$, less than half of the workers will have blood lead levels above 40/100 g.

Before closing our formal presentation, I should like to list the additional information which NIOSH is now submitting for the record.
Letter from Director, NIOSH to Acting Deputy Assistant Secretary, OSHA, August 4, 1975.

A report of recent medical studies of five U.S. lead plants.

Initial report of an investigation of employees of a secondary lead smelter in Vernon, California.

Interim report on analysis of blood and air lead data from General Motors, Delco Battery Plant, Muncie, Indiana.

A report on the relationship between air lead exposure and blood lead levels in occupational situations.

Summary report on proficiency testing of blood lead, 1976.

A review of the recent literature concerning the relationship of free erythrocyte protoporphyrin, zinc protoporphyrin and blood lead level.

A review of prophylactic chelation therapy in occupational lead poisoning.

Recommendations for respirator selection.

Follow-up of a health hazard evaluation, involving lead exposure and kidney disease.

Supplemental information on sampling and analytic procedures and engineering control technology.

Interim report on Bunker Hill Study.

Report on human health consequences due to lead exposure from automobile emissions.

Recommendations for the prevention of lead poisoning in children.*

We are now ready to respond to questions concerning our prepared remarks or the backup material submitted for the record.

*A copy of these above listed attachments to the testimony may be obtained from the National Technical Information Service, Springfield, Virginia 22161, PB281735/AS.