BEFORE THE
ENVIRONMENTAL PROTECTION AGENCY

TESTIMONY OF JEROME F. COLE, Sc.D., DIRECTOR,
ENVIRONMENTAL HEALTH, LEAD INDUSTRIES ASSOCIATION, INC.

ON THE PROPOSED NATIONAL AMBIENT AIR
QUALITY STANDARD FOR LEAD
DOCKET OAQPS 77-1

February 16, 1978

My name is Jerome F. Cole. I am Director of Environmental Health for the Lead Industries Association, Inc. (LIA), a non-profit trade association whose 80 member companies include virtually all producers and commercial consumers of lead, including primary smelters and refiners, secondary smelters and refiners, battery makers and pigment, chemical and solder manufacturers. I am also Vice President of the International Lead Zinc Research Organization, the research arm of the international primary lead, zinc and cadmium industries. As indicated by the attached statement of my credentials, my responsibilities over the past nine years have included lead health issues. As an outgrowth of my work in this area, I am quite familiar with medical studies and other scientific literature bearing on the potential adverse health effects of lead.

I am here today on LIA's behalf to present its comments in opposition to the Environmental Protection Agency's proposal to adopt an ambient air quality standard

for lead of 1.5 ug/m^3, monthly average, and in support of LIA's position that a standard no lower than 5 ug/m^3, 90-day average would more than adequately protect human health and should be adopted instead.

The Medical Issues

LIA objects to EPA's proposed standard because it is designed to protect children from a biological effect of lead, erythrocytic protoporphyrin (EP) elevation, without a shred of scientific evidence that this effect is in any way injurious to health -- and, indeed, in the face of a wealth of evidence that this effect is not injurious. Moreover, there is no evidence to support EPA's choice of 15 ug/dl as the threshold at which the effect with which EPA is concerned occurs, and the evidence points to a higher threshold.

While there is no evidence to support EPA's conclusions as to the health effects of EP or its blood lead threshold, there is a wide body of medical evidence showing that children with blood lead levels lower than 40 ug/dl suffer no adverse health effects associated with lead. To underscore that point, and provide further evidence on the subject, LIA has asked three medical experts to appear at these hearings: Drs. Chisolm, McNeil and McCabe, all of whom appeared today as witnesses. In addition, a statement by Dr. Ronald K. Panke,

who served on the Technical Advisory Committee of the Shoshone Lead Project, is appended to my testimony.

In light of the testimony of these experts and the wealth of other evidence on the subject, LIA urges that, if we accept the proposition that children are especially sensitive to lead, the proper function of an ambient air quality standard for lead should be to ensure that blood lead levels in children are kept below 40 ug/dl.

As I will show later in my statement, an ambient air standard for lead of at least 5 ug/m^3 90-day average will accomplish that goal. However, as my statement will also show, even if EPA were to pursue its approach of basing the standard on the threshold of EP elevation, but were to recognize, as the evidence demonstrates, that the threshold is 20-25 ug/dl, an ambient air quality standard for lead of 5 ug/m^3 or higher would be dictated.

The Economic Issues

Mr. Kenneth Wise of Charles River Associates has stated that the work his firm has done thus far indicates that adoption of the proposed standard would result in widespread closures throughout the lead industry because the proposed standard is economically and technologically infeasible.

The one point I wish to make on this subject is that, whether or not one agrees with EPA's bald proposition

that economics are not relevant to standard-setting, there is no basis for assuming that Congress intended that EPA would adopt standards causing the widespread economic and social damage that would result if EPA's proposal were adopted solely in order to protect against biological effects which have no adverse impact on human health.

With that point in mind, let me turn to a fuller discussion of the medical issues.

EP Elevation Is Not An Adverse Health Effect

The heme synthetic pathway is not 100 percent efficient. Heme pockets occupied by protoporphyrin without iron are normally present in erythrocytes, but their presence is not considered to indicate dysfunction of the hematopoietic system. At some blood lead level, however, EP begins to rise in response to lead. At this point, the activity of lead on the hematopoietic system may be accurately described as decreasing the efficiency of iron insertion into protoporphyrin. EPA has concluded that, at the threshold for this response, there is an adverse health effect because heme synthesis is impaired. EPA's conclusion has no scientific basis, and it is inaccurate to describe the phenomenon involved as a medically significant impairment of heme synthesis. A medically significant impairment of heme synthesis and hence an adverse health effect only occurs when the production of heme and

hemoglobin are reduced. If an elevation of EP indicates impairment of the production of heme in any meaningful way then, at the threshold for elevation, hemoglobin levels would decline. This plainly is not the case as hemoglobin levels are not reduced until blood lead levels exceed 40 ug/dl.

We believe that EPA has confused a metabolic indicator of lead interference with an adverse health effect. And, as I have pointed out earlier, there is no medical evidence that EP elevation is or indicates an adverse effect on human health.

There are several points in the process of heme production where lead interferes. When it does, there are also compensating feedback mechanisms to adjust various reactions in order to continue to meet the body's heme requirements. For example, lead interferes with heme synthesis by inhibiting the enzyme aminolevulinic acid dehydratase. In EPA's own view, however, at low blood levels this should not be regarded as physiological impairment. It is similarly inaccurate to view lead interference with iron insertion into protoporphyrin with resulting erythrocytic accumulation of protoporphyrin -- that is, elevated EP -- as, by itself, an adverse health effect.

In short, only if it could be demonstrated that there are deleterious effects associated with EP elevation at levels below that at which heme production

is actually reduced would it be correct to consider EP an adverse health effect. There is no medical evidence of any such effects. Hence, the mere existence of the phenomenon of EP elevation does not imply adverse health impact.

Moreover, even accepting EPA's determination that children should be protected, LIA contends that EPA erred in two important respects in developing an ambient air quality standard for lead.

First, EPA improperly concluded that the threshold for EP elevation occurs at 15 ug/dl. The evidence reviewed in the Criteria Document suggests that EP elevation takes place at a higher level than 15 ug/dl. H. Roels, et al., have reported from their studies that a threshold value for EP response to blood lead is clearly observed at approximately 20 ug/dl, not 15-20 as EPA reports. Moreover, testimony already given at these hearings showed that by applying more accurate statistical techniques to the Piomelli study, EP is first observed to rise at 20 ug/dl rather than 15.5 as reported.

Dr. Chisolm has noted the necessity of correcting any EP data for iron status. As pointed out by Dr. Chisolm, inspection of the Stockman report, which takes iron status into account, would suggest that the blood lead threshold for the EP response is in the 21-30 ug/dl blood lead range and probably is about 25 ug/dl. While the EPA Criteria Document states that Piomelli considered iron status in

his study no such evidence appears in the cited abstract. Further, while on October 18, 1977, we requested that Dr. Piomelli supply any data he had on iron status, he has not to date supplied any such data to us.

Second, EPA has selected a ratio of 1:2 for children's blood lead response to air lead, based on studies cited in the Criteria Document. The Criteria Document only reveals two studies dealing with air lead/ blood lead ratio in children, one of which, that by Yankel and von Lindern, calculated the ratio as 1 to 1.2-1.4. As is pointed out in the Criteria Document, the Goldsmith study on which EPA has apparently based its judgment has methodological faults. It has been subject to criticism because air and blood lead samples were not taken at the same time and blood lead determinations were based on unfrozen samples which in many instances were analyzed 8-9 months after drawing. Therefore, the only relevant data without obvious methodological faults leads to the conclusion that a ratio of 1:1.4 is more accurate than the 1:2 ratio selected by EPA.

The results of employing EPA's arithmetical approach to developing a standard -- but using a 20-25 ug/dl threshold for EP elevation and a 1:1.4 air lead/ blood lead ratio, both of which factors are quite conservative -- are as follows:

EP Elevation Threshold	20 ug Pb/dl	25 ug Pb/dl
Non-Air Contribution	12 ug Pb/dl	12 ug Pb/dl
Allowable Contribution From Air Sources 20 ug Pb/dl - 12 ug Pb/dl	8 ug Pb/dl	13 ug Pb/dl
Ambient Air Standard 8 ug Pb/dl x $\frac{1 \text{ ug/m}^3 \text{ air}}{1.4 \text{ ug/dl blood}}$ =	5.7 ug Pb/m^3	9.3 ug Pb/m^3

The Ambient Air Standard For Lead Should Be Designed To Protect Against Hemoglobin Decrease

In the explanatory statement accompanying the proposed standard, EPA notes that the standard "is based on a goal for total lead exposure lower than previously advocated by other Federal agencies," and raises the question whether the standard should be based on some lead-related effect more severe than EP elevation. The answer to that question, in LIA's view, is yes for the reasons I have given.

LIA urges that the ambient air quality standard for lead should be based on the earliest adverse health effect of lead: a decrease in hemoglobin which can be detected well before clinical anemia results. In children, the earliest statistically measurable decrease in hemoglobin has been reported at blood lead levels of 40 ug/dl. A standard based on a geometric mean blood lead level which will ensure that 99.5 percent of the critical population's blood lead falls below this level will provide a high degree of

protection. LIA calculates that at a geometric mean blood lead of 20.3 ug/dl (geometric standard deviation of 1.3), 99.5 percent of the population's blood lead level would fall below 40 ug/dl. Therefore, LIA proposes the use of a geometric mean blood lead level of 20.3 ug/dl as the target blood lead level.

EPA's methodology for calculating the standard requires that the geometric mean be converted to an arithmetic mean for comparison with the 12 ug/dl non-air source contribution to blood lead. A geometric mean blood lead level of 20.3 ug/dl converts to an arithmetic mean of 21.0 ug/dl.

	1:1.4 Air Lead/Blood Lead Ratio
Arithmetic Mean Target Blood Lead	21 ug Pb/dl
Non-Air Source Contribution to Blood Lead	12 ug Pb/dl
Allowable Air Contribution to Blood Lead	9 ug Pb/dl
Resulting Standard	6.4 ug Pb/m^3

Hence, adoption of a 5 ug/m^3 lead standard would provide a comfortable safety factor.

Averaging Time

EPA has concluded that the averaging period for the proposed lead standard should be a calendar month based

on the average of 24-hour measurements. The net result, statistically, is the imposition of a lead standard much lower than 1.5 ug/m^3. It is possible to achieve each monthly average with reasonable certainty only by installing equipment capable of consistent operation at levels far below the lead standard.

LIA proposes use of a 90-day averaging period. Increasing the length of the averaging period will not adversely affect blood lead levels but will permit the use of equipment capable of operation at levels closer to the standard while still meeting it.

In conclusion, I want to stress the fact that LIA believes that while a lead standard higher than 5 ug/m^3 is supportable, a 5 ug/m^3 standard (90-day averaging time) would provide a more than adequate margin of safety and, we believe, would in most cases be technologically and economically feasible.

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Born: Cincinnati, Ohio, August 8, 1940
Married: Virginia E. Vaughn, July 6, 1963
Children: Two -- Cheryl, Born May 1, 1965
Robert, Born August 10, 1970

Present Address: 156 Rolling Ridge Road
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Academic Experience

B.S. in Pharmacy, University of Cincinnati, 1962.

M.S., University of Cincinnati, 1966
Dissertation Title -- "A Critical Review of the Literature Pertaining to the Insecticide Endrin"

Sc.D. in Environmental Health, University of Cincinnati, 1968
Dissertation Title -- "Endrin and Dieldrin: A Comparison of Hepatic Excretion in the Rat"

Employment

3/63-9/64 -- Fidelity Prescriptions, Inc., 201 S. Main Street, Dayton, Ohio - Pharmacist

1/68-1/69 -- Procter and Gamble Co., Ivorydale Technical Center, Cincinnati, Ohio - Corporate Industrial Hygienist

1/69-Present -- International Lead Zinc Research Organization, Inc., 292 Madison Avenue, New York, New York -
1/69-1/74 -- Manager, Environmental Health Research
1/73-1/76 -- Deputy Director
1/76-present -- Vice President
And (Since 1/71) -- Lead Industries Association, Inc., 292 Madison Avenue, New York, New York -- Director, Environmental Health

Military

6/62-6/65 -- Ohio National Guard (Active Duty with U.S. Army from 9/62-3/63)

6/65-7/67 -- U.S. Army Reserve

7/67-7/68 -- U.S. Air Force Reserve

Military obligations completed. Honorable discharge.

Miscellaneous Organizations and Activities

Member -- American Industrial Hygiene Association
Chairman - Committee on Proposed Limits for Occupational Exposure

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Member -- Air Pollution Control Association

Continued . . .

Certification -- Registered Pharmacist, Ohio

Member -- Rho Chi Honorary Pharmaceutical Fraternity

Member -- Society of Toxicology

Member -- Society of Geochemistry and Health

Member -- New York Academy of Sciences

Member -- National Academy of Sciences Medical and Biological Effects of Environmental Pollutants Committee Panel on Zinc

Member -- Industrial Hygiene Roundtable