File Needham

Lead Industries Association, Inc.
295 Madison Avenue • New York, N.Y. 10017 • Tel. (212) 578-4750 • Fax (212) 684-7714

October 4, 1991

TO: Distribution

FROM: Jeff Miller

RE: LEAD HEALTH EFFECTS WHITE PAPER

We have commissioned a science writer, Martha King, to draft a white paper which will provide a balanced prospectus on the blood lead level of U.S. children and a review of the health effects reported by scientific studies. This paper will be used for distribution to the media as well as by our members and interested organizations for distribution to employees, customers, concerned community groups and political leaders.

Attached is a draft outline of that white paper. We ask you to review it and return any comments to me by Wednesday, October 9. Thanks for your assistance in this matter.

JTM/kb

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Blood Lead Levels and the Health Effects of Lead Exposure

PART ONE: MEASURING LEAD EXPOSURE IN THE UNITED STATES

I. Exposure to lead is dropping in the U.S.

American children of the 1990's have been exposed to less lead than U.S. children in 1935. The mean level has dropped by more than half in the past ten years.

How we know: Cite NHANES II survey, 1980. [Define methods, who was tested, where, and over what time period.] Compare to EPA review staff report, 1989, concluding that U.S. preschool children now have mean blood lead levels of 4 - 5 μg/dl.

II. Supporting evidence for dropping lead levels in the United States.

EPA's estimates are supported by TK. [We need stronger support here. On what studies are the EPA's estimates based?] Three recent studies of blood lead levels in children living near two former and one still active lead smelters in Utah, Montana, and New York, provide additional data. [Get additional information on Midvale, Utah; Butte, Montana; and Wallkill, New York.]

The historical record shows that the U.S. decline began as long as 50 years ago. [Figure 1 "U.S. Blood-Lead Values, 1935-1990" should be redrawn. Those symbols are strange. Charts relating to those data points don't use the symbols, thus the whole thing looks odd.]

Declines are now appearing in UK, New Zealand, Germany, Denmark, Sweden, Belgium and Canada. [Cite studies, by first author's last name and year of publication. Briefly note what was found.]

PART TWO: TEN POSSIBLE HEALTH EFFECTS OF LOW-LEVEL LEAD EXPOSURE

1. Red Blood Cell Formation

Anemia is a classic symptom of lead poisoning. The effect of lead on the blood is one of the most intensely studied topics in all of toxicology, but no sign has yet been found that lead levels lower than 30 μg/dl can impair red blood cell production.

Recent studies on this question by Moore (1985) and Paglia & Valentine (1985) showed no reduction in hemoglobin (the part of the red blood cell that carries oxygen) or any other effect on the process to build new red blood cells when blood lead levels were lower than 30 μg/dl.

The Moore study found signs of decreases in hemoglobin in some children at or above 40 μg/dl, and in adults at 50 μg/dl. This was termed pre-clinical anemia...an early warning of serious hazard. Moore also noted the decreased activity of enzyme (ALA-D) [which does what?] Paglia and Valentine noted decreases in enzyme Py-5-N [which does what?]

Neither group of researchers could find any evidence that lower levels of these enzymes affected either the function or the lifetime of red blood cells. However, both enzymes are so sensitive to the presence of lead that the investigators could not find a dose limit -- a level too small to provoke these effects.

The same is true for protein-bound lead complexes in the nucleus of the kidney's tubule cells. They too are found in the presence of extremely low doses of lead. Many scientists think these "inclusion bodies" are part of a natural detoxification mechanism.

Circulating lead is ultimately excreted or permanently incorporated into bones or teeth. As lead clears from the blood, the number of inclusion bodies drops and other changes in the tubule cells reverse. There is no evidence that inclusion bodies constitute a health risk when lead levels are low. Instead, they are seen as a biological effect of the presence of lead,

which Moore pointed out is not necessarily detrimental to health. [Use exact quote.]

II. Blood Pressure (Hypertension)

The U.S. Environmental Protection Agency cited eight recent studies to be reviewed to answer questions about whether low blood lead levels cause elevated blood pressure -- perhaps through subtle kidney damage. High blood lead is known to damage the kidneys, and lead poisoning may promote total kidney failure. No study has ever shown that low to moderate blood lead levels harm kidney function.

Two of the studies (Parkinson and Fanning) focused on lead industry workers and found no relationship between blood lead levels and high blood pressure.

Three others (Elwood, Granjean, and Pocock) were inconclusive.

Three (Neri, Sharp, and Schwartz) presented facts that suggest a connection, although both Neri and Sharp called the connection "weak".

All but the Schwartz studies were complete in time to be reported on at the 1987 International Symposium on Lead and Blood Pressure [sponsored by whom? held where?]

The International Symposium debated the ways these scientists had analyzed their information and could not agree that any association between low lead levels and high blood pressure exists. Members did agree that the relationship is very weak if it does exist. According to symposium chairman, Dr. [first name] Kannel of Boston University School of Medicine, "The relationship does not fulfill the usual criterion for causality." [Explain what this means.]

Schwartz study reanalyzed NHANES II data.

Coate & Fowles (1989) and Prikle (1985) raised questions about using NHANES II data for this purpose. [Provide details.]

It is important to note that there is no good clinical evidence -- information on patients who are being treated for high blood pressure -- to support the suspicions extracted from population studies.

III. Neurological Effects on Children

A. Attention Span in Children

In 1979, child psychiatrist Dr. Herbert L. Needleman first suggested that low lead exposure affected attention span and, consequently, school performance in children. [Briefly describe who, how, what and where of his first study.]

He followed 132 of the original group and published a study in 1990 asserting that a child's lead burden could be used to predict poor school performance and that lead levels he termed high were also associated with poorer eye-hand coordination, longer reaction time, poor vocabulary, and lower scores on tests of grammatical-reasoning. (Please see the Section on Learning Ability, below.)

Finally, using a special technique called meta-analysis, Needleman examined 12 other studies and reported in 1991 that lead exposure causes deficits in intelligence test scores (I.Q.).

Only one year after Needleman's first paper, Bornschein [Say who he is] and his colleagues analyzed his methods. This group questioned the way the lead levels were measured, the way the study children were selected, the way teachers' ratings were interpreted, and even how much difference there really was in the behavior of "low" and "high" lead children. [Do you want details here?]

Mention high concern of public on this issue - both here and in Europe.

In the U.S.: The Expert Committee on Pediatric Neurobehavioral Evaluation, created by EPA to sort through criticisms and new studies, did not support Needleman's findings. [Reference to their document. Quote their conclusion.]

In Europe: Winneke (1990) WHO/CEC collaborative study in eight European medical centers did not agree with Needleman's meta-analysis. [Quote.]

In Britain: Pocock (1987), surveying results of [How many] studies in England, found "no evidence that low lead levels currently experienced by British children has any relevance to their intellectual development."

B. Learning Ability in Children

Five recent studies have questioned the effect of lead on intelligence and psychometer development by following groups of children exposed to lead before and after birth. They are McMichael (1986, 1988), Ernhart (1988), Cooney (1989), Bellinger (1990), and Dietrich (1990) -- in Port Pirie [Where?]; Cleveland; Sydney, Australia; Boston; and Cincinnati, respectively.

Scientists expected that the adverse results of low lead levels would be readily apparent in these children. But the results of the studies are not clear or consistent.

In one study, the problems related to prenatal lead exposure disappeared spontaneously by the time the children were age two. In another study, they disappeared by age four and a half, with the help of good pre-school experiences. [Cite studies, add some details.] Two other studies showed no effect of prenatal lead levels or infant development up to the age of two.

On the other hand, post-natal levels [At what level?] were associated with moderate reductions in I.Q. scores at age 4 in one of the Australian groups, and the Cleveland group found that continued lead exposure was linked to delays in some aspects of language development.

A second Australian study found no I.Q. problems in children with exposures lower than 30 μg/dl. The principal investigator of this series concluded that there are no detectable neurological or behavioral effects of lead exposure when the levels are lower than 25 μg/dl. [Add names and additional detail.]

If the predictions based on [The OECD document] were accurate, the harmful effects of low lead levels on infants and children in these five studies would have been unmistakably clear.

C. Hearing Ability in Children

In 1987, Schwartz and Otto examined the NHANES II data and concluded that lead reduced hearing in children with blood lead levels as low as 4 to 6 μg/dl. These scientists found no lower limit to lead levels associated with hearing damage.

Question: Is this the only study that has been done since the 1986 criteria document cautioned against forming conclusions on this subject in the absence of more data?

The NHANES II study was not designed to examine hearing. No attempt was made to identify or remove any of the large number of variables that affect hearing development and auditory nerve conduction in young children.

[Check who published Schwartz & Otto...Any usable editorial comments?]

IV. Reproduction and Lead Exposure

Lead poisoning (blood lead levels of 50 μg/dl or higher) has been clearly associated with decreased fertility, spontaneous abortions, and still births. Moore's 1982 study [Of small group, where?] found that mothers with blood lead levels of 21 μg/dl and newborns with umbilical cord blood lead level of 17 μg/dl are also associated with higher rates of premature birth. Another study (McMichael, 1986) [Of who, where] found the significant blood lead values to be even lower - 11.2 for the mothers and 10.1 for the infants.

Two other studies - Bornschein (1988) and Bellinger (1988) could find no connection between the mother's blood lead level and premature delivery. [Details on who, where.]

None of these studies involved a large number of mothers. These blood lead scores qualify as low-level, although they are in the high end of the range for the general population -- which is from 4.4 to 13 μg/dl. (In pre-school children, the 1990 range is 4 to 5 μg/dl.) Why the results of these four studies are inconsistent is not clear.

Data from studies of lead exposure on male reproduction are also inconsistent and hard to compare because of the different study methods the various investigators used. In several small studies, high blood lead levels (over 50 μg/dl) are associated with lowered sperm count, more sperm with poor swimming ability, and larger numbers of abnormal sperm. There have also been some findings of abnormal seminal fluids. There are no data about low lead exposures [Check this]. Coste (1991) reviewed [How many?] studies that reported semen abnormalities [Check this) and determined that the men who had these abnormalities were able to cause normal rates of pregnancy in their partners.

V. Low Birth Weight

There have been 13 recent studies on the possible relationship between low lead levels and low birth weight. Eleven found no supporting evidence. The two exceptions were studies that were not well controlled for the effect of tobacco or drug use by the pregnant mothers. Both these factors are well known causes of low birth weights -- and both should be determined through laboratory tests, because questionnaires on such sensitive topics often have wide margins of error.

The popular press has occasionally referred to birth defects associated with low lead levels. While low birth weight and prematurity clearly increase the risk of developmental problems as well as serious illness during the infant's first year, no studies have ever linked the mother's lead level to physical deformities in the newborn.

VI. Physical Developmental in Infants

Silbergeld's analysis of the NHANES II data resulted in the suggestion that childhood lead exposure is associated with small stature

and slow physical growth. None of the five studies on learning ability (cited above) uncovered this trend even though the infants were periodically measured.

In addition, a ten year study of 104 children who had been lead poisoned [Cite author and blood lead levels of study children] found their growth matched that of their unpoisoned siblings. [Add a few more details.]

VII. Metabolism of Vitamin D

Vitamin D metabolism is mediated through the kidney -- and lead poisoning is known to damage the kidneys. Researchers have therefore raised questions about low lead levels and Vitamin D in children. [Details on Koo study.] Children with adequate nutrition and blood lead levels as high as 23.6 μg/dl showed no apparent sign of inadequate vitamin D. (Note: The U.S. Centers for Disease Control terms blood lead of 25 μg/dl an "undue exposure level.")

VIII. Release of Lead Stored in Bones

Laboratory studies have established that 90 percent of the human body's lead burden is eventually stored in the bones. As time passes, a given dose of lead is buried more and more deeply in the bone matrix, where it is out of circulation and incapable of interfering with body chemistry.

Can stress or hormonal changes, such as those that women experience at menopause, cause the bone to release stored lead? An analysis of the NHANES II data reported a significant increase in blood lead levels following menopause in 2,981 women (Silbergeld, 1988).

No high blood lead levels were found in Ewers' 1990 study of 3,098 older women, not even in women whose bones were actively demineralizing due to osteoporosis. This study was conducted [In what medical centers over what period of time] rather than through reworking data from a study that was not designed to examine this question.

IX. The Eleventh Issue: Does Lead Cause Cancer?

In 19__, the U.S. Environmental Protection Agency did not include carcinogenicity in the list of health effects to be reviewed before developing new federal rules on lead safety. [Cite EPA, advanced notice of proposed rulemaking, document #, etc.]

Laboratory rats and mice have developed benign and malignant kidney tumors after high doses of lead. Investigations designed to provoke other cancers in laboratory animals exposed to lead have not been conclusive. Data from studies of people exposed to lead in the workplace, which might show that lead exposure increases cancer risk, are inadequate or confusing. Many other contributing factors were involved in these studies.

Therefore, the EPA's Science Advisory Board classified lead as a B2 carcinogen but stressed that understanding of the mechanism is limited, many gaps in the data exist, and noted that extremely high levels of lead exposure were used in the laboratory studies before any kidney tumors developed. The board concluded: "The B2 classification is not considered to provide a sufficient basis for [requiring a] quantitative risk assessment."