

ETHYL CORPORATION

MEDICAL DEPARTMENT
Laboratory of Environmental Medicine
1700 Perdido St.
NEW ORLEANS, LOUISIANA 70112

TELEPHONE: AREA 504
522-2906
522-4745

April 25, 1969

Robert A. Kehoe, M.D.
Professor Emeritus of Occupational Medicine
Kettering Laboratory
Department of Environmental Health
College of Medicine
University of Cincinnati
Cincinnati, Ohio 45219

Dear Doctor Kehoe:

I was sorry I didn't have a chance to talk to you in Houston; I was there only because of a commitment I had for the seminar on TLVs. I left right after the Kettering luncheon because Elsie is bed-fast with an acute disc and I didn't want to be gone any longer than necessary.

I read your paper on lead with real interest and pleasure as I found you taking after the specters which have been raised about lead. You made so many good statements that had to be made.

I spent some time thinking about what I could say constructively and I have five general points I would like to review for you:

1. Though you have stated in your paper that there is "no -- effect" on man at present at ambient levels of lead -- I would like to hear it said several times.
 - a. On Page 35 you do state that the evidence in public literature would say that there are no effects.
 - b. On Page 11 you talk of industrial experience which has amply demonstrated the syndrome of clinical lead poisoning. Though there may be biochemical effects which are a part of this syndrome which have not yet been unraveled, it is indeed unlikely that there are clinical findings of any consequence that will not be found with clinical lead poisoning. In other words, any effect in the dose-response curve should be exaggerated at the dose level of clinical lead poisoning.

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- c. On Page 17 you discuss the knowledge, information and judgment of today, and at this time, you can add that your own experience tells us what happens with lead poisoning. And again, the dose response concept would say that it is extremely unlikely that there will be untoward effects found at low levels which will not be found with clinical lead.
2. The problem of accumulation of lead that results solely from exposure to ambient lead is difficult to define because of the variability of a lifetime of ingested lead. The Tipton data certainly does not answer the question. If you plot the bone lead of Tipton (I wonder why Tipton did not plot bone lead as she did every other tissue) would suggest that there is no accumulation after the age of 40. I personally found Tipton's data quite consistent with your previous statements on this subject.
 3. I would like to have you include Dr. Goldsmith in your references -- I would hope that you would include his long article published by the State of California in 1967, as well as his Science article. I would also like you to discuss, in this paper, his log-log curve, plotting blood lead vs. ambient lead. And finally, I would like to have you discuss ALA vs. lead in blood; I have been told that Goldsmith will discuss this last subject in his APCA paper.
 4. I would also think it worthwhile to discuss the uncertainties of lead analysis. I am referring specifically to the AIHA survey on lead analysis which was completed a year or two ago. I think these uncertainties are most pertinent when we discuss the minor differences which have been attributed to exposure to ambient lead. This topic could easily be included at the end of the first paragraph on Page 33.
 5. On Page 19 you discuss the large amounts of lead that have been spread across the surface of the earth as the result of mining. I would like to see a reduction in the scope and magnitude of this discussion. My questions specifically would be whether you could not leave out the sentence that begins "The uses have necessitated mining, crushing, grinding, etc." down to the bottom of the page that ends with "would be incomplete at best and probably misleading, quantitatively". Though your statement is certainly true, the result

Of this dispersing of lead over the earth is difficult to quantitate and your statement could be read as saying that the contamination has been considerable. I think that the significance of this contamination has not been established or shown to be significant in any manner. I do not believe that the omitting of this section will effect your total paper since the dispersal of lead in the environment is not directly relevant to the lead in man.

There are in addition, there are a few specific details I would like you to consider:

1. Though I personally do not differentiate between the use of the words, "criteria" and "standard", others have defined the two words to have separate meanings.
2. When you discuss the source of the excess 20 micrograms which a subject must absorb on Page 26, should not the sentence read as follows "in these experiments, the output of lead exceeded the measured intake by about 0.02 mg. (20 micrograms) per day"?

It is difficult for me to rationalize the source of this 20 micrograms which is excreted and not necessarily absorbed.

If the man excretes 20 micrograms per day from respiratory intake:

$$\begin{array}{lll} ? \text{ ug/m}^3 \times 20 \text{ m}^3/\text{da.} \times 50\% \text{ retention} & = & 20 \text{ ug excreted/da.} \\ ? \text{ ug/m}^3 \times 20 & \times 1/2 & = 20 \\ ? \text{ ug/m}^3 & & = 20 \times 2/20 = 2 \text{ ug/m}^3 \end{array}$$

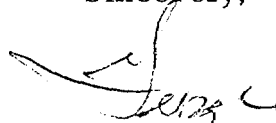
Therefore, if retention in the lung is 50%, then these subjects must be exposed to 2 micrograms/m³; if, retention is only 25%, then the subjects were exposed to 4 micrograms per cubic meter.

3. On Page 29 you compare blood leads: .02 mg. vs. .03 mg. -- more than a third higher. Should not this be more than 50% higher?
4. On Page 45 and 46 you discuss the paper by Nozaki and I wonder whether a graph of his data would not be helpful in presenting your subject.
5. On Page 51, you discuss the relationship of alkyl lead compounds on urine and blood. I wonder whether this parenthetical statement is necessary to the paper.

6. On Pages 13, 54 and 55, you have used the phrase, delta-amino-levulinic-acid. It is my impression that only delta-amino should be hyphenated, or the phrase should be delta-aminolevulinic acid.

I trust that my comments have been constructive and helpful. Please call me collect if I have been vague in my comments.

Sincerely,



George Roush, Jr., M. D.
Medical Director

GR/n
Enclosure (1)

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