

Lead Intake from Food and from the Atmosphere

Goldsmith and Hexter (1) cite data (2) which appeared to fit a curve purported to indicate the human response to respiratory exposure to lead in the ambient atmosphere. They report a direct relationship between slight differences in the concentration of lead in the atmosphere at various sites and the concentration of lead in the blood of groups of persons whose daily activities have been carried out at more than one of these sites.

Our experiment was designed to portray the intake of lead by human subjects, from the air and from food and beverages, as balanced against the output through the respiratory, alimentary, and urinary tracts, during a series of periods (16 to 20 weeks each) of exposure to air containing a known concentration of highly dispersed lead. The duration of the experimental exposure per week was increased in a regular progression. The response to increases in either the alimentary or the respiratory intake of lead compounds is reflected as increases in the rate of the excretion of lead in the urine and in the concentration of lead in the blood. These responses vary directly with the size of the dose of lead and with the duration of exposure.

In the experimental approach to the significance of lead in the ambient air of the community, the investigative effort has consisted in measurements of the lead content of the atmosphere at various sites, and in measurements of the concentration of lead in the blood (in some instances, in the urine as well) of groups of persons in the population. This approach has two weaknesses. First, it provides no information as to the variability of rate of absorption of lead from the alimentary tract, even though this may vary from less than 0.1 mg to more than 2.0 mg per day (the mean intake of individuals over long periods of time ranges from 0.12 to 0.38 per day). Second, it yields only the crudest of estimates of the average respiratory exposure of individuals in terms of the time spent at specific sites, indoors or out-of-doors, or at work, at rest, or asleep. The assumptions that must be made in order to employ the analytical findings on the blood of individuals or groups in the general population as indicators of

their *respiratory* exposure to lead are much too gross and uncertain to permit valid conclusions. This can be done only when the respiratory exposure is great enough to exceed the alimentary exposure by a significant margin.

The available evidence yields the strong implication that the quantity of lead absorbed from the alimentary tract of individuals in the general population is greater than that absorbed from the respiratory tract. It is unfortunate, therefore, that Goldsmith and Hexter have elected to ignore the clear evidence of the irregular but progressive increase in the alimentary intake of lead by our subjects.

ROBERT A. KEHOE

*Department of Environmental Health,
University of Cincinnati,
Cincinnati, Ohio 45219*

References

1. J. R. Goldsmith and A. C. Hexter, *Science* **158**, 132 (1967).
2. R. A. Kehoe, *Proc. XV Congr. Intern. Med. Travail, Vienna* **3**, 83 (1966).

7 December 1967

Kehoe's comment is welcome, since much of the data we used in our report was from the work he did.

Kehoe criticizes our report on four grounds: (i) we ignored individual variations in absorption of lead from the alimentary tract; (ii) the estimates of our respiratory exposure are too crude; (iii) "valid conclusions" can be drawn only when the respiratory exposure exceeds alimentary exposure by a significant margin; and (iv) we ignored a progressive increase in the alimentary intake of lead by his subjects. To reply in sequence:

1) Evaluation of community exposure by epidemiologic methods has weaknesses, but not those cited by Kehoe. While no information on individual variations in alimentary absorption was used, with averaged data based on an adequate sample of persons individual variability ceases to be such an important factor.

2) The estimates of respiratory exposure would be crude if applied to an individual, but again when one deals with a great many observations individual variability is less of a factor.

3) The argument that valid conclusions can be drawn only when the respiratory exposure significantly exceeds alimentary exposure is like saying we should be less influenced by the words

we read than the words we hear, because there are more of the latter! The argument ignores modern methods of data analysis (such as regression analysis) capable of detecting and accurately estimating small effects in the presence of uncontrolled or random variations which are much greater. Furthermore, the absorption ratio from respiratory ingestion is three to ten times that from oral ingestion, so that the amounts actually absorbed by the two routes are of similar magnitude.

4) In rereading his article we are unable to find "clear evidence" for a progressive increase in alimentary intake by his subjects. Indeed, data are not shown for all subjects. However, alimentary intake is given for the two subjects with the highest respiratory exposure; these show an irregular but progressive *decrease* during the last third of the exposure period, when both respiratory exposure and blood lead were *increasing*.

Finally, we have a further support for the validity of the regression relationship. Thomas *et al.* recently measured blood lead levels for persons living near freeways and for persons near the coast in Los Angeles; average blood lead levels for males of 22.7 and 16.0 $\mu\text{g}/100\text{ g}$ were observed (1). The appropriate estimate of average atmospheric lead concentrations, 2.5 $\mu\text{g}/\text{m}^3$ (2) for Los Angeles composite, is from the Three City Study. An estimate for coastal atmospheric exposure can be based on recent data from the Los Angeles Air Pollution Control District. The average of samples taken at the Long Beach Marina, 23 March to 14 April 1967, is 0.34 $\mu\text{g}/\text{m}^3$. These points are consistent with the values expected from the regression we reported. We believe this supports our previous conclusion that further increases in atmospheric lead will result in higher blood lead levels in populations in a predictable relationship.

JOHN R. GOLDSMITH

ALFRED C. HEXTER

*California State Department of
Public Health, Berkeley 94704*

References

1. H. V. Thomas *et al.*, *Arch. Environ. Health* **15**, 695 (1967). The two groups were household samples selected in the Los Angeles area, from areas with similar socioeconomic and demographic characteristics.
2. U.S. Public Health Service, *Survey of Lead in the Atmosphere of Three Urban Communities*, Publ. No. 999-AP-12 (1965), p. 32.

22 January 1968