Role of Airborne Lead in Increased Body Burden of Lead in Hartford Children*

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The ingestion of airborne lead fallout is the mechanism responsible for increased lead body burdens found in 10 urban Connecticut children. The mean indoor lead levels found in housedust was 11,000 μg/g; highest concentrations occurred on windowills and in floor dust. The mean lead content of Hartford street dirt was 1,200 μg/g; levels were highest near the street and next to the buildings. The mean lead concentration of hand samples taken from the subject children was 2,400 μg/g; the mean weight of hand samples was 11 mg. The concentration of lead in dirt and household dust was high enough to theoretically result in excessive lead accumulation in young children who are putting their dusty, dirty hands in their mouths during play. While we believe that lead emitted from automobiles contributes significantly to air, dirt and dust lead levels the environmental impact of reducing or eliminating lead from gasoline is not yet completely understood.

In 1971, nearly 25% of urban 1–5 year olds from low income families attending the University of Connecticut clinics had blood lead levels >40 μg-% and 2–3% had levels >60 μg-%. Rural children of comparable age attending well child clinics in Dutchess County, New York, and Litchfield County, Connecticut, had only a 9% prevalence of blood lead levels >40 μg-%. Mean blood lead levels in the two groups were 32 and 21 μg-%, respectively. Although ingestion of old leaded paint was probably a significant factor among the children with lead levels >60 μg-% in the urban group and >40 μg-% in the rural group, many of the urban children with blood lead levels of 40–60 μg-% had no history of pica for paint, nor was leaded paint (defined as >1% lead) found in the dwellings. There was no rural counterpart for this group. (1) These data have lead to a reconsideration of the potential sources of environmental lead other than leaded paint among those urban children whose blood

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lead levels are in the 40–60 μg-% range (borderline elevated).

During the summer of 1973, the role of atmospheric lead and lead fallout into dust and dirt was investigated among 10 children whose average age was 4 1/3 years and who had chronically elevated lead levels (40–120 μg-%) for 6–24 months. Half of this group had levels > 80 μg-% more than 3 months previous to initiation of the present study and required chelation therapy. All had blood lead levels of >40 μg-% when the present study was initiated. A history of pica in the past was obtained for all but one child. Where a source of leaded paint had been found previously in the environment, corrective measures had been taken. Thus, continuing exposure to leaded paint was believed to have been eliminated and yet blood lead levels remained significantly elevated.

The types of data obtained in the present investigation included the following: (1) obtaining surface dirt and dust samples (10 of each type) where children played on preweighed self-adhesive labels by pressing the labels on the surface (single, localized application and analyzing for quantity of lead in the particles which adhered (2)); (2) obtaining a sample of dirt from hands of the 10 study children and 12 siblings and playmates in the same manner as above; and (3) observations of 3–6 hr duration of normal play, with emphasis on documentation of frequency of children putting hands or nonfoods in their mouths.

Laboratory Analyses

All environmental samples were tested for lead content at the Connecticut State Department of Health Laboratory by the atomic absorption spectroscopy technique.

Results

The mean lead level for 94 samples of outdoor dirt near 13 dwellings was 1200 μg/g. The levels were highest near the street and next to the buildings. The mean level of lead in household dust was 11,000 μg/g. The highest concentrations occurred on windowsills and in floor dust. The lowest concentrations of lead in dirt and dust were in and around a city housing project where there was little traffic, while the highest concentrations in dirt and dust were found in and around the residence of one child whose house was on a heavily trafficked thoroughfare. The mean lead concentration of hand samples from the 22 children was 2400 μg/g; the mean weight of hand samples was 11 mg.

Nine of the 10 study children exhibited some degree of excessive "mouthing" behavior. Three children were observed to have their hands or nonfood items in the mouth at least 50% of the time.

Comments

It is believed that fallout of lead from the air is a significant contributor to the lead present in dirt and dust found in urban streets, parks, and homes. The mean lead content of the indoor dust samples was greater than 10 times the level found in outdoor dirt. Dust from pulverized leaded paint chips, small paint chips themselves, in addition to different mechanisms and rates of removal (e.g., wind and rain outdoors, housekeeping procedures indoors) may have caused the higher lead levels found in indoor dust; however, high lead concentrations were found in dust on unpainted indoor surfaces suggesting contamination from outside. Results in the present investigation for lead in urban dirt are within the range of those reported by Hunt (3), while the findings in the house dust sample parallel those of Sayre et al. (4).

The findings of the present study have clear health implications. It has been estimated that the maximum permissible daily intake (MPDI) of lead in preschool age children before accumulation occurs is between 70 to 500 μg (Table 1). If a small child playing in dirt having a lead concentration of 1200 μg/g puts his fingers or hands in his mouth 10 times a day each time ingesting about 10 mg of dirt, he would
| MPDI, µg | Adult | Child (2 yr) | Comment | Reference |
|---------|-------|--------------|---------|-----------|
| —       | 300   |              | Total intake, includes air intake of 6 µg | (5)       |
| 600     |       |              | Ingested | (6)       |
| —       | 138   |              | Ingested | (7)       |
| 430 (7 µg Pb/kg body weight) | 90 * | Ingested foodstuffs; 10% absorption and retention rate; any increase in lead intake from drinking water or air would reduce this allowable level | (8)       |
| —       | 125 * (10 µg Pb/kg body weight) | Total intake (ingested and inhaled) | (9)       |
| 280 (4 µg Pb/kg body weight) | 50 * | Total absorbed intake, if one assumes a 10% absorption and retention rate, this corresponds to a total lead intake for 2-yr old child of 500 µg | (10)      |

*Assumed body weight of 2-yr-old child is 12.5 kg.

theoretically ingest 120 µg of lead. Substituting household dust with 11,000 µg/g of lead would result in 1100 µg ingested, more than twice the highest MPDI.

Consequently, it is quite possible for a young child who is frequently putting his hands in his mouth during play in a heavily leaded environment to ingest excessive quantities of lead and accumulate an increased body burden of lead.

### Summary and Conclusions

An attempt was made to evaluate the role of airborne lead and its subsequent fallout in the production of increased lead burden in 10 urban children. The concentration of lead in dirt and household dust was high enough to theoretically result in excessive lead accumulation in young children who are putting dusty, dirty hands in their mouths during play. While we believe that lead emitted from automobiles contributes significantly to air, dirt, and dust lead levels the environmental impact of reducing or eliminating lead from gasoline and replacing lead with nonleaded additives is not yet completely understood; certainly one alternative solution to removing lead from gasoline is to institute a mass transit system that significantly reduces inner city traffic density.

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### REFERENCES

1. Cohen, C., Bowers, G., and Lepow, M. Epidemiology of increased lead burden: a comparison of urban and rural children. JAMA, in press.
2. Darrow, D. K., and Schroeder, H. A., Childhood exposure to environmental lead. Paper presented at the Symposium on Protein—Metal Interactions, American Chemical Society, Chicago, Illinois, August 29, 1973.
3. Hunt, W. F., Jr., et al. A study in trace element pollution in 77 midwestern cities. In: Trace Substances in Environmental Health. IV. D. D. Hemphill, Ed., University of Missouri Press, Columbia, Missouri, 1971.
4. Sayre, J. W., et al. House and hand dust as a potential source of childhood lead exposure. Am. J. Dis. Child., in press.

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5. King, B. G. Maximal daily intake of lead without excessive body burden in children. Am. J. Dis. Child., 122: 337 (1971).
6. Kehoe, R. A. The metabolism of lead in man, in health and disease. The Harben Lectures, 1960. J. Roy. Inst. Publ. Health, 24: 1, 101, 129, 177 (1961).
7. Barltrop, D. Sources and significance of environmental lead for children. In: Proceedings of the International Symposium on the Environmental Health Aspects of Lead. Commission of the European Communities, Luxembourg, 1973.
8. Evaluation of certain food additives and the contaminants mercury, lead and cadmium. Sixteenth Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series No. 505, FAO Nutrition Meeting Report Series No. 51, Geneva, Switzerland, 1972.
9. Alexander, F. W., Delves, H. T., and Clayton, B. E. The uptake and excretion by children of lead and other contaminants. In: Proceedings of the International Symposium on the Environmental Health Aspects of Lead. Commission of the European Communities, Luxembourg, 1973.
10. Edwards, H. W. Ed. Impact on Man of Environmental Contamination Caused by Lead. Colorado State University, Fort Collins, Colorado, 1972.