Lead Distribution in Soft Tissues of Baltimore Residents, 1973

by ALPHONSE POKLIS[1] and HENRY C. FREIMUTH[2]
Office of the Chief Medical Examiner
Baltimore, Md.

Lead is a ubiquitous component of the biosphere and as such is an inevitable component of the human body. Lead is present to some degree in all human tissues and fluids. This communication concerns the determination of the lead content of the liver,kidney, lung and pancreas of adult residents of Baltimore, Maryland sampled in 1973. The lead levels of these tissues are evaluated with reference to age and the relationship of lung lead to lead in other organs. The present data is compared to the results of similar studies on soft tissue lead.

Materials and Methods

Liver,kidney, lung and pancreas samples were collected from Baltimore City residents over seventeen years of age who had died suddenly and who had no apparent disease condition at the time of death. Causes of death for the subjects sampled were, typically, arterio-sclerotic cardio vascular disease and multiple injuries from auto accidents. Persons dying as a result of gunshot wounds or stabbing wounds were excluded from the study because of possible contamination of their organs by metal splinters containing lead. Each sample was placed in an individual polyethylene bag and labeled as to case number, age, sex, weight, race and apparent cause of death. The samples were quickly frozen and kept frozen until prepared for ashing.

The tissues were thawed, trimmed of fat and fascia, weighed and subjected to nitric perchloric acid wet digestion according to the procedure outlined by Freimuth (1960). The digests were neutralized and buffered to pH 8.5. The lead was chelated with ammonium pyrrolidine diethiocarbamate and extracted into methylisobutyl ketone according to the method of Yeager et al. (1971), and determined by atomic absorption spectrophotometry.

1. Office of the State Toxiologist, State University Station, Fargo, ND 58102
2. Department of Chemistry, Loyola College, Baltimore, MD 21210

Bulletin of Environmental Contamination & Toxicology,
Vol. 15, No. 3 © 1976 by Springer-Verlag New York Inc.

Recoveries of lead from spiked samples ranged from 98 to 107% of the added concentration. Six determinations on the same sample gave a standard error of seven percent.

Results and Discussion:

The results of the analysis of the liver, kidney, lung and pancreas of Baltimore residents are presented in Table 1.

TABLE 1.

LEAD CONTENT OF TISSUES (μg/g) FROM BALTIMORE RESIDENTS DYING DURING 1973

Tissue	No. of Samples	Mean	Median	Range
Liver	22	2.5	2.2	1.0-6.3
Kidney	21	1.8	2.0	0.40-3.8
Lung	22	0.85	0.80	0.21-1.8
Pancreas	21	0.98	0.92	0.49-1.8

A measurable concentration of lead was found in each sample. The highest concentrations were found in the liver and kidney. The relatively high levels of lead in these two organs is probably related to their excretory functions. The distribution of lead among the sampled tissues is in agreement with that observed by other investigators of "normal" or "natural" lead tissue levels (Barry & Mossman, 1970; Schroeder & Tipton, 1968; Kehoe et al, 1940). The mean concentration of lead for each organ in the present study exceeds the mean value of the older studies; however, it is difficult to construct a meaningful interpretation from this difference due to variations in sampling parameters and analytical methods in these studies.

Tipton and Shafer (1964) using rank correlation statistics, have demonstrated a significant correlation between the concentration of lead in the dry ash of lung and the concentration in the dry ash of other tissues. A high degree of correlation was noted between lung levels and brain, kidney, pancreas and liver lead levels. The lead concentrations in the present survey

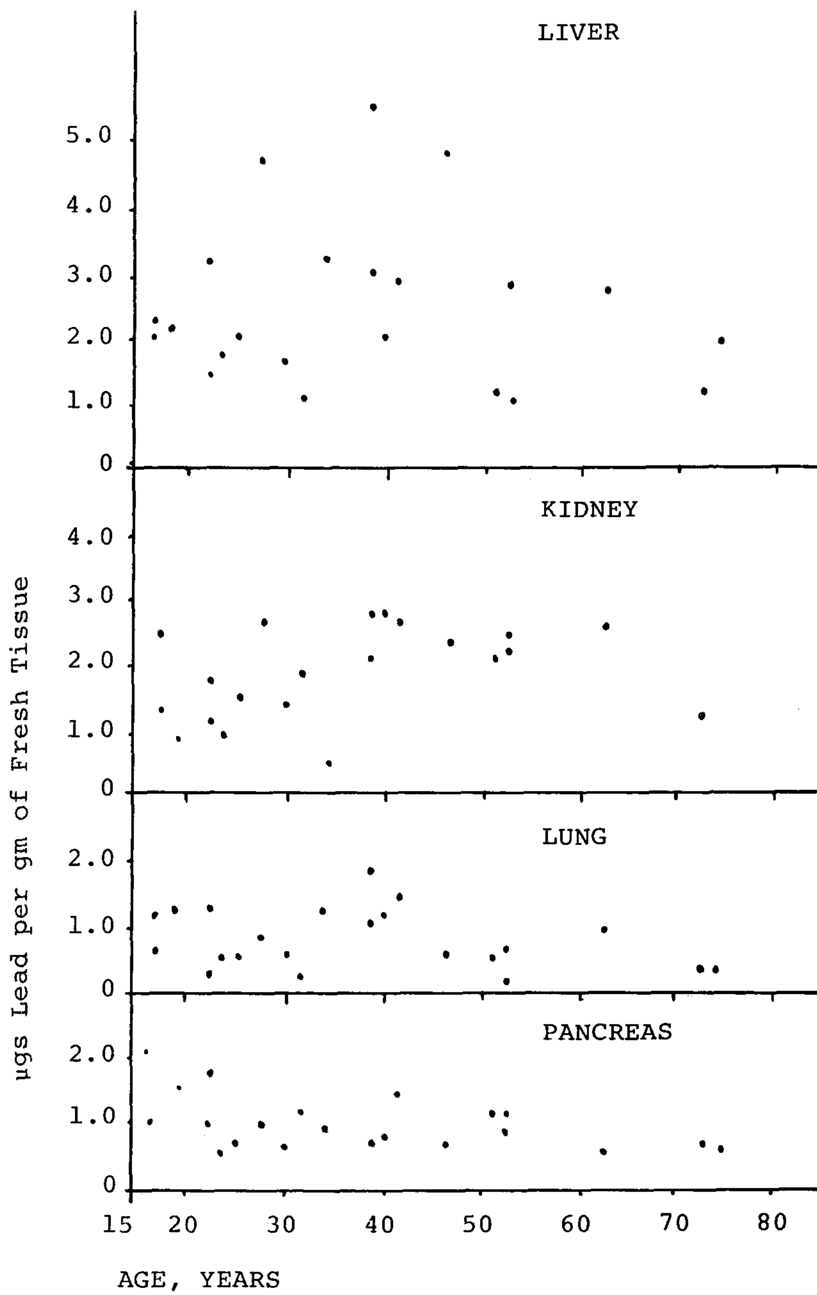

Figure 1. Relationship of age and tissue lead.

were calculated solely on a wet tissue weight basis and a significant lung lead-tissue lead correlation was observed only between lung and liver; rank correlation coefficient, r = 0.78 ($P< 0.01$). The wet weight of the lung varies with the mode of death and this may explain the lack of agreement with the previous dry ash study.

Schroeder and Tipton (1968) reported that lead accumulated with age in the liver, kidney, lung and pancreas of adults living in the United States, but no such age linked accumulation occurred in persons living outside the United States. The implication of this work is that in industrialized, urban areas persons are exposed to, and absorb lead in quantities that exceed their ability to excrete lead. The association between age and the lead content of the liver, kidney, lung and pancreas of Baltimore residents in the present study, are represented in figure 1. Paired rank correlation of the age and lead content of these tissues failed to reveal a significant relationship. A previous study of the lead content of soft tissues of residents in a densely populated industrialized area of England also demonstrated no significant increase in soft tissue lead with age (Barry and Mossman (1970).

The kinetics and metabolism of low levels of lead in the soft tissues of healthy adults are poorly understood. It has long been recognized there is a relationship between lead and calcium metabolism (Hammond,1969). The difference in the affinity for lead among the soft tissues and the variance in the lead content of a given tissue may be related to the factors regulating extraosseous calcium metabolism. Given the complexities of lead metabolism and the lack of correlation between age and soft tissue lead levels in both the present data and the data of Barry and Mossman(1970), it appears that age, per se, is not a significant factor in evaluating the lead content of the liver, kidney, lung or pancreas in healthy adults.

ACKNOWLEDGEMENT

This paper was supported in part by USPHS Grant 5T 01 GM 01218.

REFERENCES

BARRY, P. S. and D. B. MOSSMAN: Brit. J. Med. 27, 399 (1970)

FREIMUTH, H. C.: Toxicology: Mechanisms and Analytical Methods. (C.P. Stewart and A. Stolman, Ed.) Vol. 1, New York: Academic Press 1960.

HAMMOND, P. B.: Essays in Toxicology. (F.R. Blood, Ed.) Vol.1, New York Academic Press 1969.

KEHOE, R. A. et al: J. Nutr. 19, 579 (1940)

TIPTON, I. H. and J. J. Shafer: Arch. Environ. Health 8, 58 (1964)

SCHROEDER, H. A. and I. H. TIPTON: Arch. Environ. Health 17, 965 (1968)

YEAGER, D. W. et al: Environ Sci.Technol. 5, 1020 (1971)