The Lead Content of the Trachea in Baltimore Residents

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Lead is an ubiquitous environmental contaminate and, as a result, one of the most abundant nonessential trace metals in man. The lead content of human tissues has been determined by various researchers; however, only Tipton and Cook (1963) have reported the lead content of the trachea. Part of this study involved the determination of trachea lead in residents of Baltimore, Maryland sampled prior to 1957. This present communication presents the lead content of the trachea of seventeen Baltimore residents sampled in 1972. The lead content of these tissues are evaluated with particular reference to the lead "body burden" of Baltimore residents.

Materials and Methods

Tracheas were collected at autopsy from seventeen residents of Baltimore who had died suddenly and who had no apparent disease conditions at the time of death. The samples were carefully handled to prevent contamination and were placed in individual polyethylene bags and quickly frozen. Each sample was labeled as to case number, age, sex and manner of death of the subject.

The tracheas were thawed, trimmed of excess connective tissue, weighed and wet digested with nitric and perchloric acid. The digest was extracted and analyzed by Atomic Absorption Spectrophotometry by the method of Yeager et al (1971). Recoveries of lead spiked samples were 98-102%. The standard error of the method was less than five percent.

Results and Discussion

The results of the analysis of the samples collected in the present study are presented in Table I. The concentration of lead in the seventeen tracheas ranged from 0.6 - 8.8 micrograms per gram of fresh tissue. The distribution of lead in the samples exhibited a Poisson distribution; therefore, the square root of the mean was taken as the measure of deviation in the data. Paired rank correlation was used to evaluate the increase of lead in the trachea with age. Contrary to the observation of Schroeder and Tipton 1968), no significant correlation of trachea lead and age was demonstrated by the present data.

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Table I

Age, cause of death, and concentration of lead in the trachea of 1972 residents of Baltimore, Maryland.

	Age	Cause of Death	Micrograms Lead/Gram of Fresh Trachea
1	17	Intravenous Narcotism	5.9
2	17	Multiple Injuries	4.1
3	23	Intravenous Narcotism	8.8
4	23	Bronchopneumonia	3.3
5	24	Multiple Injuries	0.7
6	28	Multiple Injuries	3.1
7	30	Bronchopneumonia	1.5
8	31	Multiple Injuries	0.6
9	34	Multiple Injuries	3.4
10	38	A.S.C.V.D.[1]	6.2
11	41	Drowning	3.0
12	46	Multiple Injuries	1.4
13	51	A.S.C.V.D.	2.1
14	52	Diabetes Mellitus	2.3
15	53	A.S.C.V.D.	1.3
16	62	A.S.C.V.D.	2.3
17	74	Multiple Injuries	1.5
		Mean	3.0
		S.D.	1.7

1. Arterial Sclerotic Coronary Vascular Disease

The lead content of human tissues is influenced by a variety of biological and environmental factors. When attempting to evaluate the tissue content of lead, geographic locale is an important consideration. Hall (1972) noted the difference in blood lead levels between suburban and downtown residents of Philadelphia and Kubota et al (1968) reported the variance in blood lead levels between subjects in different U.S. cities. Schroeder and Tipton (1968) have observed a difference in tissue lead between urban and rural dwellers.

Table II

Comparison of the Mean Trachea Lead of Baltimore Residents in Present Study, 1972, with Study of Tipton et al (1957) [1]

	Number of Samples Analyzed	Lead Concentrations (ug/g) Range	Mean	S.D.
1957	13	0.4 - 4.4	1.5	1.2
1972	17	0.6 - 8.8	3.0	1.7

1. Students "t" value, 2.58 ($P < 0.02$)

Table II presents a comparison of the mean trachea lead concentration of the samples collected in 1972 with the mean trachea lead concentration of Baltimore residents reported by Tipton et al (1957). Within 99% confidence limits, there has been a significant increase in mean trachea lead in Baltimore residents over the fifteen years between sampling. Although lead kinetic and metabolic balance of specific tissues and organs is poorly understood, trachea, like bone, is generally considered a storage sink of body lead. From the increase in trachea lead it may be concluded that from 1957 - 1972 there has been an increase in the lead "body burden" of Baltimore residents. The high lead levels found in the younger subjects, Table I, also lend support to this conclusion. The increase in body lead may not be sufficient to affect the public health of Baltimore residents; however, it does reflect an increase in lead exposure in the Baltimore environment.

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