Lead Levels in Human Lungs

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Determination of the blood lead concentration has been accepted as the most valid and reliable indicator of excessive lead absorption in man. Recently, it was documented that determination of blood lead concentration alone is an inadequate measure of occupational lead exposure (VITALE et al. 1975). In an extensive study of the distribution of trace elements in human tissues TIPTON and SHAFER (1964) found very high correlation between lung and almost all other soft tissues on examination of lead concentrations. These correlations indicate that a large part of the lead deposited in the lungs is absorbed into the rest of the body, and that the lung lead concentration reflects the total body burden.

It has been stated (LEE et al. 1968) that lead in urban and suburban atmospheres is present predominantly in particles able to penetrate and be retained in both the tracheobronchial and alveolar regions of the respiratory tract. Thereby, lung tissue reflects the lead absorbed by the body and redeposited in the lung tissue in addition to any lead that has been deposited in the respiratory tract but not yet absorbed (STRINGER et al. 1974). Although the dietary intake of lead is greater than that taken in by inhalation, the absorption in the respiratory tract is so much more effective than the gastrointestinal absorption that the contribution to the total body lead absorption may be of similar magnitude, at least in city atmospheres (GOLDSMITH and HEXTER, 1967). If uniformity in dietary habits in urban and rural residents is assumed, differences in body tissue lead levels should therefore reflect differences in ambient air lead levels. Thus, the determination of lung lead levels is well suited for comparative analyses of lead exposure of different populations.

MATERIALS AND METHODS

Samples of lung tissue were obtained from autopsies on 250 individuals who died in Sør-Trøndelag county, Norway,

Bulletin of Environmental Contamination & Toxicology,
Vol. 18, No. 6 © 1977 by Springer-Verlag New York, Inc.

in the period 1974-76. During the first part of this investigation all samples brought to our laboratory were analysed. In the latter part of the investigation only samples from subjects under fifty years of age were analysed. This selection was carried out in order to achieve a satisfactory age distribution in the population analysed. The majority, 139, of the individuals came from the city of Trondheim (population 120,000), whereas the remaining 111 were from mainly rural areas.

The analyses were carried out by the flameless atomic absorption technique described by STEGAVIK et al. (1976), which allows analyses of small lung tissue samples. From each sample, fixed and stored in formalin, ten replicas, each weighing approximately 2 mg, were analysed in the carbon rod atomic absorption spectrophotometer. All samples were taken from the apical segment of the right lower lobe. The concentration of lead is expressed as parts per million (ppm) dry weight. For each subject the following parameters were collected: age, sex, place of residence and occupation. In addition, the pertinent facts were obtained from the subject's medical histories. Unfortunately, it was not possible to obtain information concerning the smoking habits of each subject.

RESULTS

In Table 1 the results from the lung tissue analyses for lead are presented. From the total of 250 individuals investigated 6 distinguished themselves as occupationally exposed. All 6 had a lead concentration in the lungs of over 1.30 ppm dry weight, which is well above the lung lead level in any other individual analysed (Table 1). Of the 6 occupationally exposed individuals, all of whom were men, 3 were vehicle drivers and 3 were employed in industry (factory and machine shop). It is of course quite possible that several of the others, without our knowledge, may have been occupationally exposed to a greater or lesser degree. However, it is only these 6 individuals, with an evident connection between lung lead levels and occupation, who are excluded from further treatment of the analytical results.

Table 1 reveals that the mean lead concentration in lung tissue is greater in the urban population group than in the rural one (0.50 ppm as opposed to 0.37). The difference between the means is highly significant ($p < 0.01$).

In the urban group the mean lung lead concentration in males and females is more or less alike (0.50 and 0.49 ppm), whereas males in the rural population have a

somewhat higher lead content in the lung tissue than the female group (0,39 ppm as opposed to 0.33). However, none of these differences is statistically significant.

TABLE 1

Mean concentration of lead (ppm dry weight) in human lung tissue in an urban and a rural population in Norway. Influence of sex and occupation is indicated.

Group	No. of individuals	Mean lung lead	Range	SD
Occupationally exposed (urban and rural)	6	1,81	1,32-2,45	0,42
Urban	136	0,50	0,15-1.24	0.21
Male	76	0.50		
Female	60	0.49		
Rural	108	0.37	0,10-1.08	0.20
Male	70	0,39		
Female	38	0.33		

Fig. 1. illustrates the influence of age on the lung lead concentration within the two population groups. The mean lead value for the urban group exceeds that of the rural one for all age levels except for children in their first year of life, where the rural group has the higher value. The latter group, however, consists of only four samples, and the difference may not be meaningful. There seems to be a tendency towards lead accumulation in the lungs in both population groups with increasing age, this tendency being most prominent in the urban group. In the ninth and tenth decade of life there is a decrease in pulmonary lead concentrations in both groups.

TABLE 2

Influence of days of hospitalization on lung lead levels. Stillborns not included.

Days in hospital	No. of indv.	Mean lung lead	SD
0	78	0.42	0.19
1 or more	158	0.45	0.22

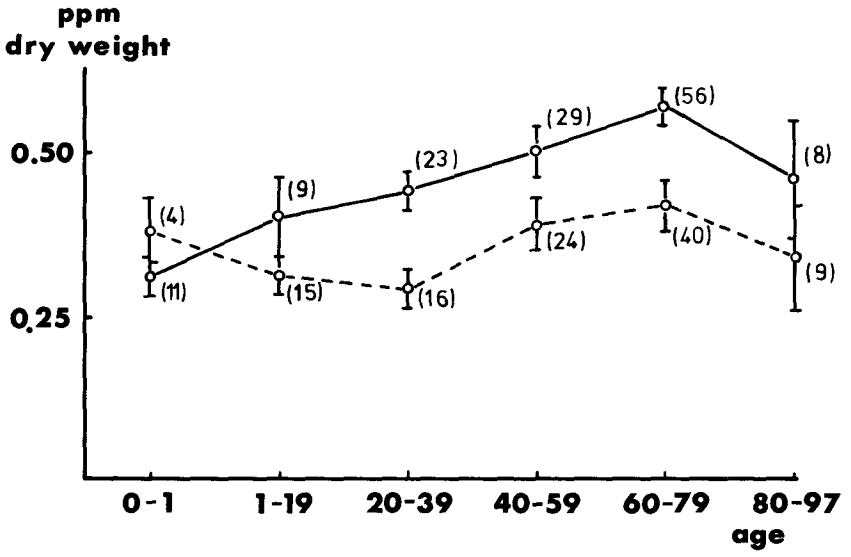
In Table 2 the difference in lung lead levels between those individuals having a record of hospitalization prior to death, and those having none is recorded. Table 3 displays the mean lung lead levels for the individuals when grouped according to cause of death.

TABLE 3

Influence of cause of death on lung lead levels.

Diagnosis at death	No. of indv.	Mean lung lead	SD
Malformations and trauma at birth	12	0.31	0.12
Accidents	53	0.43	0.19
Pneumonia	18	0.44	0.22
Malignant tumours, lung metastases	12	0.47	0.20
Brain haemorrhage and thrombosis	18	0.38	0.19
Acute haemorrhages	10	0.56	0.31
Thromboses	16	0.40	0.19
Major infections	14	0.38	0.17
Infarct of the heart	34	0.45	0.19
Heart failure	13	0.57	0.22
Malignant tumours	21	0.45	0.23
Uremia	14	0.54	0.26
Miscellaneous	9	0.38	0.30

Fig. 1. Influence of age on lung lead concentration in an urban (solid line) and a rural (broken line) population in Norway. Figures in parantheses refer to the number of subjects analysed. Standard error is indicated.



DISCUSSION

A recent article (MYLIUS and OPHUS, 1976) stressed the importance of selecting specimens from the same and preferably the lower part of the lung when comparative studies of pulmonary lead concentrations in different human populations are made. The pulmonary distribution of lead, like other nonessential trace-elements (MOLOKHIA and SMITH, 1967), seems to be quite heterogenous, due to differences in exposure and physiological response.

Estimates of the normal range of lung lead values in human populations have varied, as might be expected, when different analytical techniques are empoloyed and samples are taken from groups with presumed differences in lead exposure. Nevertheless, the range of pulmonary lead concentrations found in this investigation, 0.10 to 2.45 ppm, is within the range found by STRINGER et al. (1974) in their AAS investigation of lung lead levels in 66 Americans (0.10 to 3.04 ppm).

The relatively high content of lead in the lungs of the 6 occupationally exposed individuals (Table 1) indicate that occupation is the most crucial parameter correlated with lung lead concentration. It seems that individuals working in areas of high atmospheric lead concentrations may be placed in high risk categories. However, a larger number of occupationally exposed individuals is necessary to assess the influence of occupation on lung lead concentration.

Increased blood and tissue lead levels have been reported in children and adults living in areas of excessive exposure to lead (THOMAS et al. 1967), although the significance of the rural-urban blood lead gradient has been debated (GOLDWATER and HOOVER, 1967). The proximity of the subject's place of residence to heavy automobile traffic is particularly important for the total lead burden imposed (THOMAS et al. 1967; WALDRON, 1976; CAPRIO et al. 1974). In our investigation there is a significant difference between the mean lung lead levels in the urban and the rural groups. This is due, undoubtedly, to the use of lead alkyls in petrol, which constitutes the major lead source in city atmospheres (CHOW and EARL, 1970).

Differences in mean blood lead levels between males and females have been noted by several investigators (HOFREUTHER et al. 1961; STUIK, 1974). With regard to pulmonary lead levels similar differences have been found (STRINGER et al. 1974; TIPTON and SHAFER, 1964), males always showing significantly higher lead concentrations

than females. In these investigations occupationally exposed individuals were not excluded. When the values for the six individuals (all men) with an evident occupational exposure are excluded in our study, no significant differences between males and females can be found. This may indicate that the observed differences mainly might be due to differences in the degree of occupational exposure to lead.

In a comprehensive study of the lead concentration in the lungs of 209 subjects from the US and 167 from abroad SCHROEDER and TIPTON (1968) found that with age there was an increase in the lung lead levels of Americans, whereas people from other parts of the world had an almost constant lead level in all soft tissues (except aorta). The concentration of lung lead in the Americans appeared to increase with each decade of life up to the seventh, whereas the mean value was lower in the eighth decade of life. In our investigation there is a tendency towards pulmonary accumulation of lead with age in both the urban and the rural groups although most evident in the former (Fig. 1). This may, in fact, imply that people in this area have an excessive exposure to lead, causing imbalance between absorption and excretion of this potentially harmful metal. The decrease in pulmonary lead concentrations in the ninth and tenth decade of life is evident in both groups and is in accordance with the findings of SCHROEDER and TIPTON (1968). In their study of 66 American city dwellers STRINGER et al. (1974) also found an increase in pulmonary lead concentration with respect to age except for men in their ninth decade of life who had significantly lower pulmonary lead concentrations than those in their seventh and eighth decades. They speculated that this decrease resulted from a reduced bulk food intake together with a reduced tidal volume in elderly people.

Relatively high lead levels are found in the lung tissue of stillborns and infants (Fig. 1). The placental permeability of lead (primarily inorganic lead) and the presence of lead in newborns has been established by several investigators (GERSHANIK et al. 1974; SCANLON, 1971; CARPENTER, 1974). The importance of the innate lead is difficult to assess, but the possibility of permanent neurological damage in children with excessive lead absorption cannot be excluded (BRYCE-SMITH, 1974; LIN-FU, 1972).

LIN-FU (1972) stated that to assess a given blood lead value the interval from cessation of excessive lead absorption should be considered. In view of the relatively short biological half-life of most lead compounds in human lung tissue (HURSH and MERCER, 1970; BOOKER et al. 1969), this seems quite relevant. On examining the

influence duration of hospitalization has on lead levels, we find (Table 2) no correlation with the pulmonary lead concentration at time of death. Furthermore, when comparing lead levels in individuals who were accident victims and in hospitalized patients who had died as a result of illness, no correlation to the pulmonary lead concentration can be found. These results indicate that lead inhaled but not yet absorbed, contributes less to the lung levels measured than lead redeposited in the lungs from the bloodstream.

It is known that various diseases, for instance cancer, may cause erratic distributions of metals in human tissues (TIETZ et al. 1957; MULAY et al. 1971). When we examine a possible connection between cause of death and lung lead levels we find (Table 3) that the difference between the mean of each subgroup and the mean of the nonoccupationally exposed population is not statistically significant, except for infants who had died of malformations and trauma at birth. However, a slight increase in lung levels can be observed in patients who had died from illnesses involving circulatory disturbances. This may indicate impairment of normal lead excretion.

SUMMARY

This study shows that the concentration of lead in the lungs of an urban population is significantly higher than in a rural group. The pulmonary lead levels seem to be age- and occupation-related.

Aknowledgement

We wish to thank amanuensis Kåre Stegavik who kindly put the facilities of the Department of Physics, NLHT, University of Trondheim, at our disposal and Med. lab. tech. Aileen Murdoch Larsen for valuable assistance during preparation of the manuscript.

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