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Lead Concentration in the Livers of Canadians

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Man's exposure to lead has been increasing in the industrialized world because the amount of lead mined and used in transportation, chemical and metal industries has been increasing from year to year. The normal sources of lead for man are diet, air and water; the diet providing 200-300 µg per day. Individuals living in large urban centres could inhale as much as 90 µg lead in a day and where lead pipes are used, water can also be a significant source of lead. Children may also ingest excessive amounts through contaminated dust, lead-based paint found in older homes, yellow, red and orange inks used on newsprint, tooth paste and lead in the paint coating of pencils (WHO, 1973; LIN-FU, 1973; BEATTIE et al., 1975).

The toxic effects of lead include anemia, neurological dysfunction, renal impairment, hypofertility in man, increased incidence of premature deliveries and mental retardation in children. Deficiences of nutrients such as iron, calcium and protein can enhance the toxic effects of lead (WHO, 1973; LANCRANJAN, 1975; FAHIM et al., 1976). In view of this, there is a need to monitor closely the trend in the body burden of lead in the industrialized world. A recent British study has concluded from the analysis of human tissues that present levels of lead in the environment are not hazardous to the health of the general population (BARRY, 1975). In Canada the lead content of rib samples obtained at autopsy from residents of Ontario and Québec was reported recently (CHERRY et al., 1976), but the concentration of lead in the soft tissues is not known. This report provides data on lead concentrations in the livers of Canadians taken at autopsies carried out in different parts of Canada.

MATERIALS AND METHODS

Liver specimens (10 g) were taken at 114 autopsies on accident victims and on 4 autopsies on stillborn infants, carried out in Fredricton, Hamilton,

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Ottawa, Saskatoon, Sudbury, Victoria and Windsor during 1969-1971 and shipped to Ottawa as described previously (SHAH and BELONJE, 1976). Approximately 2 g of the minced specimen was dissolved in 4 ml of 1:1 nitric acid in a 50 ml beaker by heating on a hot plate. After cooling, it was made to 20 ml by adding distilled demineralized water and stored in polyethylene vials. This solution was diluted ten times with distilled demineralized water and used for lead determination at 283.3 nM by flameless atomic absorption spectroscopy using a Varian Techtron AA5 with model 63 CRA according to the manufacturer's manual. The results were corrected for reagent blanks (Absorbance, 0.015 ± 0.012 S.D.-standard deviation-) and nonatomic absorption (0.005-0.020 absorbance). On the average 70% of the total signal was due to lead. The lead content of the liver samples was expressed as µg/g fresh weight.

The recovery of added lead to the sample matrix ranged from 98 to 104%. Five replicates of National Bureau of Standards (NBS) Reference Material 1577 bovine liver, treated in the same way, gave 0.33 ± 0.04 µg/g lead as compared to the certified value of 0.34 ± 0.08 (95% confidence interval) µg/g.

Statistical analysis: Since an examination of the data indicated that the distribution of lead concentrations was highly skewed, the data were transformed to logarithms before analysis. Observations of "not detected" were given a value of 0.1 µg/g before transformation.

Two different analyses were carried out on the data. For the first, the observations were subdivided into a group of persons under 19 years and the other group of those 19 years and older. The possibility of differences among cities was studied using a covariance analysis. A linear function of age and a factor for sex were used as the covariates.

In the second analysis, all the data except for those under one year of age were studied simultaneously. The data for the stillborn and infants were excluded because the values differed appreciably from those of the children. A multiple regression analysis was carried out using sex, city of death, age and the square of age as independent variables. The form of the function of age was allowed to differ for the two sexes.

RESULTS AND DISCUSSION

The lead content of the liver specimens from 4 stillborn infants, 83 male and 31 female accident victims are shown in Table 1. The frequency distribution

TABLE 1

Lead in the livers of accident victims (ug/g wet weight)

Age, yrs.	Male				Female			
	N	Mean	Median	Range	N	Mean	Median	Range
Stillborn	3	0.56	0.62	0.30-0.77	1	0.36		
0- 1	4	0.26	0.26	ND[1]-0.51	3	0.52	0.39	0.28-0.91
2-12	8	1.15	1.02	0.33-2.62	3	1.31	1.53	0.82-1.59
13-18	6	1.10	1.17	0.19-2.10				
19-45	36	2.09	1.73	0.58-6.47	15	0.74	0.66	0.29-1.97
46-65	22	1.30	1.18	0.31-2.93	6	0.63	0.63	0.28-1.04
>65	7	1.72	0.65	0.10-4.94	4	0.35	0.34	0.19-0.53

[1]Not detected

of liver lead levels in adult males and females is shown in Figure 1. It is evident from the results in Table 1 that at birth and during infancy the liver lead content was low in both sexes. BARRY (1975) also reported comparable results for eight infants under one year old. The mean and median liver lead levels in males from 2 to 19 years, were about 1 ug/g and for 3 females in the same age group the values ranged from 0.82 to 1.59 µg/g. These may be compared with a mean of 0.87 and 0.63 ug/g reported by BARRY (1975) for 2-9 and 13-16 year old British children.

The results in Table 1 show that adult females had a lower concentration of lead in the liver than male adults. A similar observation, was made by BARRY (1975) in men and women living in the industrialized north-west of England, although the differences between the sexes observed by us were much larger. The adult males had an average of 1.03 (Range 0.18-3.13) ug/g and the females had only 0.66 (Range 0.19-1.72) ug/g (BARRY, 1975). The corresponding values in the present investigation were 1.78 ug/g liver (Range 0.10-6.47) for males and 0.65 (Range 0.19-1.97) for females. In the U.S.A., white male residents of Cincinnati had a liver lead concentration of 0.97 ± 0.53 (Av. ± S.D.) ug/g (GROSS et al., 1975), whereas males and females in Baltimore were found to have an average of 2.5, median 2.2 and a range of 1.0-6.3 ug/g liver (POKLIS and FREIMUTH, 1976). TIPTON et al. (1965) reported the following values (median and range or S.D.) for liver specimens from different parts of the world: ug/g wet weight, U.S.A. 2.0, 1.4 (S.D.); Africa, 0.8, 0.1-9.3 (range); Near East 1.0, 0.3-4.1 (range); Far East 1.5, <0.1-1.73 (range); Switzerland 1.3, 0.4-4.9 (range).

The Baltimore samples were digested with nitric-perchloric acid before lead was chelated with ammonium pyrrolidine dithiocarbamate and extracted into methylisobutyl ketone. Lead was then determined by atomic absorption spectrophotometry (POKLIS and FREIMUTH, 1976). Recoveries of lead from spiked samples ranged from 98 to 107%. Our samples were dissolved in 1:1 nitric acid and lead was determined by flameless atomic absorption spectroscopy. The accuracy and the precision of this method was confirmed by analysing NBS reference material 1577, bovine liver. The Cincinnati samples however, were ashed in a muffle furnace overnight at 500°C and the results were lower than those obtained by the above two methods (GROSS et al., 1975). The British samples were digested with ammonium sulphate and nitric acid and ashed at 450°C in a muffle furance for a few minutes. This was

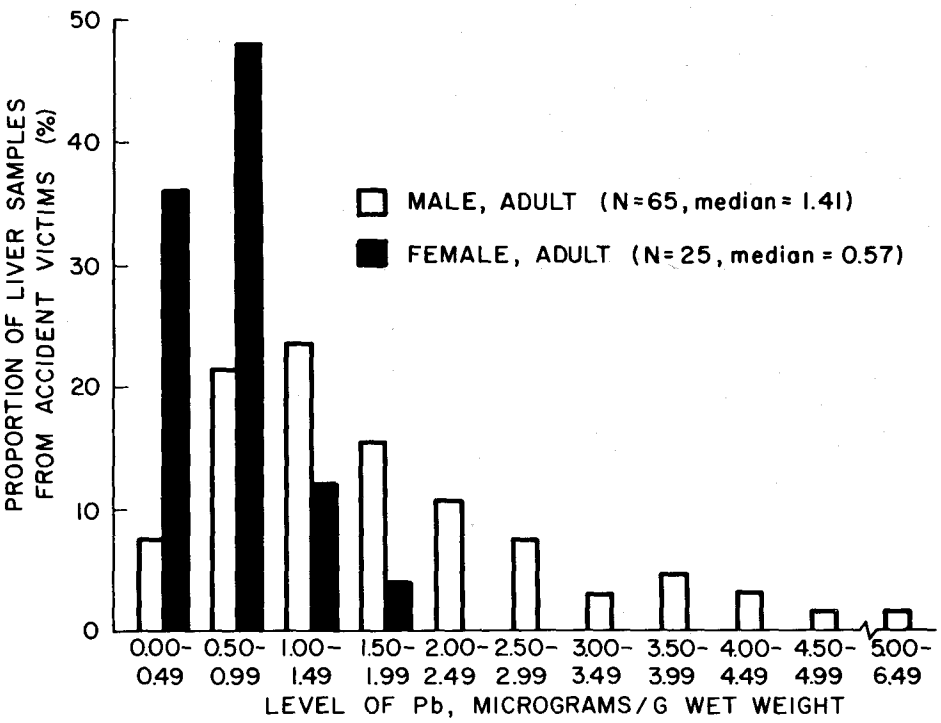

Figure 1. Frequency distribution of liver lead concentrations.

repeated until ashing was completed. The results for the British samples also tended to be lower than those obtained without the use of dry ashing in a muffle furnace. Indeed HAMILTON et al. (1972-73) who ashed the specimens at 100°C with nascent oxygen and determined the lead content by spark source mass spectrometry reported a relatively high value of 2.3 ± 0.6 (S.E.) ug/g liver for people living in the Surrey area of England. Specimens collected by TIPTON et al. (1965) from various parts of the world were dry ashed at 450°C in a muffle furnace and lead determination was carried out by emission spectrography (TIPTON et al., 1963). In view of the differences in methodology the absolute lead levels reported by different workers are probably not comparable although all mean or median values for adult males lie between 1.0 and 2.5 ug/g wet weight (GROSS et al., 1975). The differences in lead concentration due to age or sex, however, may be comparable.

The statistical analysis of our data indicated that in male adults the average lead concentration increased with age to a maximum at about the end of the fourth decade and then decreased. The number of samples from females, however, was not sufficient to draw any definite conclusion regarding the change in liver lead concentration with age. In British males and females the soft tissue lead was observed to peak in the 30 to 40-year-old group and then to decrease slightly, followed by a small rise after the seventh decade. Similar peaking of lead concentrations in liver and kidney during the fourth decade was also reported by SCHROEDER et al. (1961, 1968), although the concentrations of lead in the same organs of Japanese subjects were found to increase steadily throughout life (HORIUCHI et al., 1959). CHERRY et al. (1976) who analysed rib samples taken from residents of Ontario and Québec in Canada observed that in the males, lead concentration peaked at about 50 years and then decreased, although a similar trend was not seen in females. In agreement with our liver lead results they did note that the rib samples from females had lower lead concentrations than those from the males. The decrease in lead concentration in the males above 50 years was evident even when the results were expressed on ash weight basis indicating that it could not be attributed to loss of bone mineral. The decrease in the lead concentration in liver and rib bone may be interpreted to mean that the present level of general lead exposure in different parts of Canada is within the normal capacity of the body to handle lead.

The frequency distribution in Figure 1 brings out clearly the difference between the liver lead concentrations of adult males and females. The median value for the males was 1.41 and for the females was 0.57 ug/g, both being less than the corresponding mean values (1.78 and 0.65 ug/g). As the occupational history of the accident victims was not known, all the values were included in calculating the average and median values. There was an indication in Figure 1, however, that some of the males who had high liver lead levels might have experienced above normal exposure to lead.

The analysis of the data did not reveal any differences among cities. Although the autopsies were carried out in the seven cities mentioned, information on the place of residence of the victims was not available. It is likely however, that most of the individuals in this study lived in the industrial and nonindustrial areas represented by the cities. Also, no difference was noted by BARRY and MOSSMAN (1970) between tissue lead concentrations of urban or rural

residents. This may be attributed to the fact that for most individvals the major source of lead is the food and beverages consumed (UNDERWOOD, 1971). Indeed, food consumption patterns in Canada were not found to vary appreciably among different regions (HEALTH AND WELFARE CANADA, 1977).

SUMMARY

Liver specimens were collected at 114 (83 males and 31 females) autopsies on accident victims and on 4 stillborn infants in seven Canadian Cities. The age of the individuals ranged from newborn to 89 years with about 70% being 19-65 years. The level of lead in the samples was determined by atomic absorption spectroscopy using a carbon rod atomizer. The concentration of lead in liver was low (µg/g: Mean, 0.37, Median 0.28) at birth and during infancy. In the stillborn infants it ranged from 0.30 to 0.77 (Mean 0.51). Male adults had an average level of 1.78 (Median 1.41) whereas in adult females the corresponding values were 0.65 and 0.57. The liver lead concentration peaked at about the end of the fourth decade in adult males. There was no indication of any regional differences.

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