Pak. j. sci. ind. res., vol. 37, nos. 6-7, June-July 1994

HUMAN EXPOSURE TO POLLUTANTS

Part-I. Blood Lead and Cadmium Levels in a Sample of Population of Karachi

A.H.K. Yousufzai, Qamar Khalid and Liaquat Sultana
PCSIR Laboratories Complex, Karachi-75280, Pakistan

(Received February 25, 1993; revised December 30, 1993)

A study was carried out to see the blood lead and cadmium levels in fifty employees working at PCSIR Laboratories Complex, Karachi. These employees belonged to various socio-economic groups and had their residences in different areas of Karachi. Sixty two percent staff had blood lead level between 100-200 µg/L. The highest blood lead level (>400 µg/L) was found in volunteers working as garage staff. No significant difference was found between the blood lead levels of volunteers belonging to different socio-economic and age groups. Only 8% of the staff had blood lead levels below 100 µg/L. Lead in the dust collected from the residences of the volunteers was also estimated for lead and correlated with blood lead levels. Blood cadmium levels were also estimated. These were found to be higher in smokers and tobacco chewers. A definite correlation was observed between blood cadmium levels and smoking habits.

Key words: Blood lead, Blood cadmium, Household dust.

Introduction

The use of lead ware in kitchen and houses, sweet additives containing lead salts, lead pipes and pans was the cause of sapping the vigour of Roman elite. In present days, use of lead additives as antiknock agents in gasoline is posing a similar problem in the metropolitan cities of the world. There are more than six hundred and fifty thousand registered vehicles plying in Karachi emitting exhaust laiden with lead and carbon. A mean concentration of 2989 ppm lead was found in the street dust of the city. It has also been calculated that about 28,447 kg of lead is being spread into the environment of Karachi every year [1]. An average of 8 $\mu g/m^3$ of lead at or below four feet level from the ground is generally present near the moving traffic [2]. The rate of deposition of inhaled lead from fresh car exhaust in lungs has been found to be about 60% whereas, from other urban air it is about 50%. This difference may be due to the formation of large aggregates of lead particles with other material present in the atmosphere [3]. Children in Karachi often play in close proximity to moving traffic because of non availability of proper play grounds and are, therefore, more exposed to automative exhaust.

Investigations have also shown that lead levels in blood are generally higher in city dwellers than in the people living in the country side [3]. The difference can be linked to a relatively larger number of vehicles plying in the urban areas. It has been found that lead can be absorbed both from the respiratory and the alimentary tracts, especially, when a significant portion of the inhaled lead particles is greater than 1µm in diameter [9]. In USA, 36.7% reduction in blood lead levels was observed between the years 1976 and 1980 after the introduction of unleaded petrol showing clearly that leaded gasoline is one of the significant sources of lead pollution in the urban environment [5]. The lead found in human body may originate from working environment, ambient air, drinking water, food, or non-food items such as paints etc. Approximately, 90% of the total body burden of lead is present in the bones and teeth as stable fraction [4]. The half life of lead in blood is only about 18 days [5].

Cadmium is also a cumulative toxic agent and its half life in blood is estimated to be 2-3 months [6]. The best way to estimate total exposure to lead and cadmium is through biological monitoring because sufficient information is now available on their transport and participation in different metabolisms [4,7,9,13,14,15,17]. The level of lead and cadmium in blood is the best indicator of current exposure and reflects a dynamic equilibrium between exposure, adsorption, distribution and elimination [10]. The main object of the present studies was to investigate the extent of human exposure to lead present in the environment of Karachi and cadmium absorbed through smoking tobacco to correlate the level of exposure with the concentrations of lead and cadmium present in the blood of volunteers.

Experimental

Forty eight male and two female workers of the Pakistan Council of Scientific and Industrial Research Laboratories Complex, Karachi belonging to different income groups and living in different localities of Karachi, served as the study group. A detailed questionnaire was also prepared for each participant in which general information about identity, personal history, consumption of food and beverages, health status, recreation activities, housing conditions, mode of transport to and from Laboratories and type of condiments used, was collected. Sampling was carried out during the months of July and August 1991. Sample of blood was taken from the antecubital vein of each of the 50 volunteers and processed according to the method described by Bruaux and Svartengren [7]. The supernatant was analysed for lead and cadmium, using Hitachi Z-8, 000 Atomic Absorption Spectrophotometer, equipped with Zeeman background corrector and graphite furnace. To avoid sample contamination, all persons involved in the sampling and analysis were given precise instructions in order to minimise the risk for contamination. All material used for sampling were checked for heavy metal contamination before use and, if necessary, washed with dilute nitric acid and deionized water. Calibration curve was prepared by standard addition method (Fig. 1). The correlation coefficient was 0.9986 with 96-98% recovery.

Results were statistically computed and analysed by student's "t" test and recorded in Tables 1-5.

Results and Discussion

Table 1 shows percentage distribution of volunteers with respect to their blood lead levels. It can be observed here that 8% of volunteers had blood lead levels less than 100 μg/L., 62% between 100-200 μg/L, 20% between 200-300 μg/L, 6% between 300-400 μg/L and 4% above 400 μg/L. Approximately 10% of the volunteers who had their blood lead levels above 300 μg/L belonged to the group of volunteers professionally exposed to lead. They included drivers, persons working on liquid toner photocopying machines (6%) and garage staff (4%). The highest lead level was found to be 485 μg/L in the blood of a motor mechanic. It has been shown that lead levels as low as 70 μg/L can exert irreversible neurotoxic effects, whereas, 80 μg/L shortened the red blood cell life and 250 μg/L caused irreversible chronic nephropathy and an irreversible loss of IQ in children [6]. Thus there is need to exercise caution in the safety limits for blood lead levels, because lead is a cumulative poison and, therefore, even a subclinical lead exposure can cause a variety of mental and behavioural disorders.

Blood lead levels of volunteers of different age groups, socioeconomic status and using different type of condiments are given in Table 2. No significant difference was observed between blood lead levels and age, blood lead levels and socioeconomic status and blood lead levels and type of condiments used, though slightly higher but statistically non-significant blood lead levels were observed in middle income group. The non-significance shown here among different groups may primarily be due to the fact that the volunteers are exposed to automotive exhaust during their journey to and from the Laboratories whereas they spend 42 hrs a week in PCSIR and are exposed to an environment low in lead level (0.005 mg Pb/m^3). It has been reported that exposure to 0.01 mg Pb/m^3 does not cause any significant increase in lead concentration in blood or urine [9]. Moreover, intake of food, water and beverages is purely a personal matter. Consumption of contaminated food and beverages constitutes, a variable part of total blood lead level. In addition cleaning, washing and cooking may change the lead concentration in food and water [15].

Tetraethyl lead and tetramethyl lead are still extensively used as fuel additives in many countries. Both are volatile and poorly soluble in water. In Pakistan, 0.84 g/L tetraethyl lead is being used in 'Super Petrol' [19]. Tetraethyl lead breaks down during combustion forming trialkyl lead compounds which are readily soluble in water and are, therefore, quickly ad-

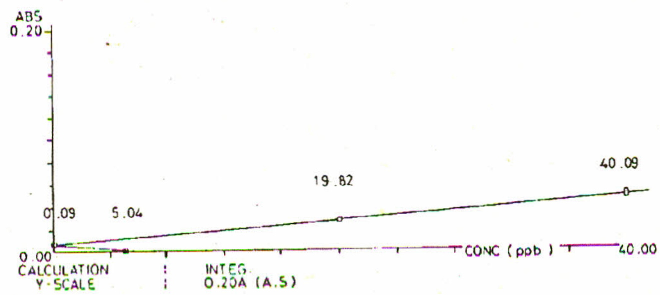

Fig. 1. Pb calibration curve by standard addition method.

TABLE 1. PERCENTAGE DISTRIBUTION OF VOLUNTEERS WITH RESPECT TO THEIR BLOOD LEAD LEVELS.

Blood lead level μg/L		Percent distribution
Below	100	8
Between	100 - 200	62
	200 - 300	20
	300 - 400	6
Above	400	4

TABLE 2. LEAD LEVELS (μg/L) IN THE BLOOD OF VOLUNTEERS ACCORDING TO AGE, SOCIOECONOMIC STATUS AND THE TYPE OF CONDIMENTS USED.

Age		Socioeconomic group Grade/Basic pay scale			Condiments used	
Below 40 years	Above 40 years	1 - 5	6 - 15	16 - 20	Packed	Unpacked
184±17.3 (28)	185±12.2 (22)	161±15.8 (18)	213±28.8 (18)	161±13.3 (14)	163±13.3 (10)	192±14.8 (25)

All values were statistically non-significant when compared with the respective controls.

sorbed in the body by inhalation. It has also been estimated that 30-50% of lead in inspired air may ultimately be retained by the body [8]. Thus lead in air, introduced primarily from the combustion of leaded gasoline is considered as a significant source of lead pollution. Base-line data presented earlier [1], also confirmed that main contributor of lead in the urban environment of Karachi is the automative exhaust. It is, therefore, understood that most of the lead in the blood of these employees is due to exposure during travel to and from the Laboratories.

There are three possible routes of entry of lead in the body *viz.*, inhalation, oral ingestion and percutaneous absorption. The latter route is considerably less significant than respiratory and gastro-intestinal routes for uptake of inorganic lead. To evaluate, as possible source of lead exposure, samples of household dust were also collected from the houses of the volunteers and analysed for lead content. The results showing lead levels in the blood of volunteers and in the dust collected from their residences are recorded in Table 3. No correlation was found between the two parameters possibly due to the fact that ingested lead is less efficiently absorbed than the inhaled lead [9]. Table 4 describes the level of lead in the blood and household dust of the professionally exposed and other volunteers. No correlation was found in the levels of blood lead and house hold dust in either group. In fact, comparatively low lead level in the household dust of professionally exposed volunteers substantiate the contention that ingestion of dust was not a significant source of lead exposure here.

Table 5 shows the level of cadmium in the blood of the smokers and betel chewers. A significant relationship was found between blood cadmium levels and smoking habits. Cadmium levels were found to be higher in regular smokers as compared to the level obtained in non-smokers (P<0.01). Highest values were reached in betel/tobacco chewers (P<0.001). There was also an upward but statistically non-significant trend between the blood cadmium values reached in occasional smokers as compared with the non-smokers. The blood cadmium levels have usually been regarded as indicator of recent exposure and 60% of the blood cadmium level in non-smoker adults could be attributed to the influence of body burden i.e., long term integrated exposure to cadmium, but 5-6 fold increase in "Normal" blood cadmium level of adults, non-occupationally exposed to cadmium, may occur through smoking habits [10]. It is difficult to explain why smoking cigarettes should give rise to such a marked increase in blood cadmium levels. However, Telismna [11] by using smoking machine have found that mainstream smoke of a cigarette contains 0.2-0.3 µg Cd/cigarette [11] and it has been estimated that smoking 20 cigarettes per day would result in an inhalation of about 2-4 µg Cd/day. Now assuming that 25-50% of the cadmium is absorbed through pulmonary route, 20 cigarettes/day would result in a daily reflection of 1-2 µg Cd in their blood [12]. A value of below 5 µg Cd/L in blood has been quoted by WHO as "NORMAL" in the adult population, non-occupationally exposed to cadmium. In our volunteers, these values have been estimated to be far

TABLE 3. LEAD LEVELS IN THE BLOOD OF VOLUNTEERS AND IN THE DUST COLLECTED FROM THEIR RESIDENCES.

Specimen	PCSIR and Muhammad Khan Goth	F.B. Area and Liaquatabad	Korangi, Landhi and Rifah-e-Aam Society	Gulshan-e-Iqbal and Azeem Goth	New Karachi North Karachi North Nazimabad Nazimabad
Blood (µg/L)	172±11.3	128±33	198±25.7	157±25.7	149±16.7
Household dust (mg/L)	4.18±0.88	213±30.5	145±31.6	165±19.4	106±10.0

All values were statistically non-significant when compared with the respective controls.

TABLE 4. LEAD LEVELS IN THE BLOOD AND HOUSEHOLD DUST OF PROFESSIONALLY EXPOSED AND OTHER VOLUNTEERS.

	Blood (µg/L)	Dust (mg//L)
Professionally exposed volunteers	291± 54 (6)	62 ± 18.7 (6)
Other volunteers	172 ± 8.7 (43)	136 ± 13.8 (43)

TABLE 5. LEVEL OF CADMIUM (µg/L) IN THE BLOOD OF VOLUNTEERS.

Non-smoker	Occasional smoker	Regular smoker	Betel/Tobacco chewers
0.4 ± 0.10 (22)	0.6 ± 0.28 (4)	1.3 ± 0.37* (11)	1.9 ± 0.26** (11)

The values were significantly different when compared with the values obtained in non-smokers. * P < 0.01, ** P < 0.001.

below the normal values indicated by WHO. Thus, in non-smokers the mean cadmium level has been found to be 0.4 µg/L, in occasional smokers 0.6 µg/L, in regular smokers 1.3 µg/L and in betel tobacco chewers 1.9 µg/L. It may be mentioned here that in our target group, tobacco chewers used an average of upto 3.5 g of tobacco per day, whereas, the smokers used 19.3 g of tobacco per day in cigarettes. It seems, therefore, that the absorption of cadmium is more via ingestion of tobacco than through pulmonary absorption. The data also indicate that use of tobacco and the smoking habits are directly responsible and related to the raised cadmium levels reached in the blood.

References

1. A.H.K. Yousufzai, Pak. j. sci. ind. res., **34**, 167 (1991).
2. Martha L. Lepow, Env. Res., **10**, 415 (1975).
3. H.A. Waldron, Nature, **253**, 345 (1975).
4. Lars Friberg and Marie Vahter, Env. Res., **30**, 95 (1983).
5. W.T. William Manser, Lead, JPMA, **2**, 296 (1989).
6. William W.T. Manser, Plumbum: Karachi Quo Vadis, JPMA, **227-228** (1988).
7. P. Bruaux, L. Friberg, Assessment of Human Exposure to Lead: Comparison between Belgium, Malta, Mexico and Sweden, Karolinska Institute, Stockholm (1985).
8. Howard W. Mielke, Bruce Banke, Sarah Burroughs and Nancy Hassinger, Env. Res., **34**, 64 (1984).
9. Robert A. Kehoe, Food. & Chemical Toxicology, **25**, 425 (1987).
10. Recommended Health Based Permissible Levels in Occupational Exposure to Heavy Metals, Reports of Study Group, Tech. Report No. 647, WHO (1980 b).
11. S. Telismna, J. Azaric and D. Pripc Majic Bull. Environ. Contam. Toxicol., **36**, 491 (1986).
12. Carl Gustaf Elinder, Lars Friberg, Biger Lind and Muhammad Jawaid, Env. Res., **30**, 233 (1983).
13. P.B. Hammond, E.J.O. Flaherty and P.S. Gartside, J. Cosmet. Toxicol., **19**, 6310638 (1981).
14. Lead, Environmental Aspects, Environmental Health Criteria 85, WHO (1989).
15. Guidelines on Studies in Environmental Epidemiology, Env. Criteria 27, WHO (1983).
16. An Introductory Guide to Human Exposure: Field Studies, Survey Methods and Statistical Sampling, PEP: HEAL: 91.26.
17. S.R.V. Raghavan, B. Dwight Culver and Harvey C. Gonick, Eng. Res., **22**, 264 (1980).
18. Hakan Bjorki-Lund, Lars Olson, Ake Seiger and Barry Heffer, Env. Res., **22**, 224 (1980).
19. Specification for Petrol (Motor Gasoline), Pakistan Standard 1430, Ammendment 1985 (UDC:: 665.733.5).