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ASSESSMENT OF EXPOSURE TO LEAD AND CADMIUM THROUGH AIR AND FOOD IN INHABITANTS OF ZAGREB

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Exposure to lead and cadmium was monitored in a group of 17 non-smoking women in Zagreb. The monitoring included measurement of the intake of the two metals via air and the diet during a period of seven days. Duplicate daily diets and air filter samples were collected for analysis of lead and cadmium. To check the intake of lead and cadmium with the daily diet, faeces were collected and analysed. The mean gastrointestinal absorption was estimated to be 4.9 μg/day for lead and 0.75 μg/day for cadmium. These values were derived from faecal data which were assumed to be more reliable. The daily absorption through inhalation estimated on the basis of air analysis was 2.7 μg for lead and 0.03 μg for cadmium.

Key terms: blood analysis, daily diet, faeces analysis, gasoline, metals intake, monitoring, non-smokers, personal samplers, urban population.

To devise the best approach to assessing the exposure of urban populations to lead and cadmium, a pilot study on total exposure monitoring was undertaken in a small group of subjects living in Zagreb. The study was part of the UNEP/WHO project »Human Exposure Assessment Location« (HEAL) initiated on a global scale, with Zagreb as one of the four focal points.

Due to the high lead content of gasoline (0.6 g/L), as well as the fact that less than 10% of the gasoline used is lead free (mostly that used by foreign tourists), the Yugoslav urban population is exposed to much higher concentrations of lead in air than the city population in developed countries. From 30 to 50% of the total quantity of lead inhaled with particles of dust is absorbed from the lung (depending on respiratory volume, particle size and extent of physical activity) (1). Tobacco smoking is the primary source of non-occupational exposure to cadmium, which is absorbed to the same extent as lead after inhalation (2). Another major route of non-occupational

exposure to lead and cadmium is the diet. Their absorption from the diet is from 5 to 10%, depending on other dietary constituents. As the monitoring was designed to measure the intake from both routes of exposure, food and air, duplicate diet collection (including beverages) and estimation of exposure from inhaled air using personal monitors were performed for selected subjects during a seven-day period in the spring of 1988. In addition, faeces were collected from the same subjects in order to check the ingestion of these elements. To assess recent exposure to lead and cadmium, blood concentrations of these elements were also measured.

Such a detailed study of human exposure to lead and cadmium has not been performed earlier in this country. To enable a comparison between different focal points, the methodology of sampling blood, food, air and faeces was standardised in all the participating countries. However, as analytical methods were different, before the monitoring phase a stringent quality control (QC) was performed in each laboratory. This was organized and controlled by the WHO Technical Coordinating Centre (TCC) in Sweden (3). The results of both quality control and actual monitoring of human exposure to lead and cadmium in Zagreb are presented in this paper.

SUBJECTS AND METHODS

Quality Assurance

In the pre-monitoring (training) phase, QC of lead and cadmium determination was performed on samples of blood, air, food and faeces provided by the TCC. QC samples had the same matrix composition and approximately the same lead and cadmium concentration range as real samples from the preceding monitoring phase (3–6). Several QC runs were repeated until the results in the participating laboratories were within acceptable intervals. During the analysis of collected samples, two series of QC samples were analysed simultaneously. Acceptance criteria, i.e. a maximum allowable deviation (MAD) and an acceptance interval (AI) – for blood, air, food and faeces, were defined in advance by the TCC (7). The MAD interval was set to $\pm (5-10\%X + 2\delta)$ where X is the reference value and δ is an estimated error of the method, based on several QC runs.

Monitoring design

The study was carried out on 17 healthy women, non-smokers, selected from the Institute staff, aged 23–53 years. Lead and cadmium food intake (including beverages) was followed by collecting duplicate diets throughout the seven-day period. During the same period, 24-hour air collection was performed by using personal samplers. Total daily faeces were collected only during the first four days of monitoring i.e. between two carmine markers. The subjects received carmine (0.5 g in capsule) on the first day before the first meal and again on the fourth day. Faeces collection started with the first red-coloured faecal sample and ended with the second, which was discarded. At the end of the seven-day period, blood was collected for lead, cadmium and other analyses.

During the whole monitoring period the subjects kept a detailed record of their motion and activities, and of the food and beverages they consumed.

Blood sampling and analysis

Three millilitres of venous blood was sampled using Safety-Monovette blood collecting tubes (Sarstedt, Numbrecht) containing 1.5 mg K_2EDTA/ml blood, and stored at +4 °C. Blood lead (PbB) and cadmium (CdB) were determined by a method involving deproteinization of blood with 1 M HNO_3, followed by electrothermal atomic absorption spectrophotometry (ET-AAS) on a Perkin-Elmer 403, with deuterium background correction (DBC), HGA 72, AS-1 and recorder 056. The method applied (8) was a modification of those previously described by Stöppler (9, 10): the essential difference pertained to calibration which was performed by the addition of Pb + Cd-standards in human blood instead of in aqueous nitric acid. The detection limits of the method, when using »preconcentration« in the course of ET-AAS measurements (i.e. triple injection of 20 µl supernatant into graphite tube, the 2nd and 3rd following the pyrolysis step of previous aliquots), corresponded to PbB of 1.5 µg/L and CdB of 0.06 µg/L. In the same subjects 3 ml heparinized venous blood was simultaneously sampled for the determination of characteristic biological indicators of lead exposure: erythrocyte delta-aminolaevulinic acid dehydratase (ALAD) activity and erythrocyte protoporphyrin (EP) concentration. Blood haemoglobin (Hb) and haematocrit (Hct) were also determined because of the possible interference of anaemia, i.e. impaired iron metabolism, on the specificity of EP as a biological indicator of lead exposure. ALAD was measured within three hours after blood sampling (blood was stored at +4 °C) by the European standardized method (11), EP by the spectrofluorometric method (12), Hb by the standardized spectrophotometric method (13) calibrated by the ICSH-standard blood solution, and Hct by a standard haematological method. All the measurements were performed in duplicate, and the coefficients of variation (CV) were: 2% for ALAD, 4% for EP, 5% for Hb, and 6% for Hct.

Collection and analysis of airborne lead and cadmium

Air was sampled over 24 h at a flow rate of 2 – 2.5 l/min through membrane filters of 0.8 µm pore size (Millipore cellulose acetate/nitrate) by means of personal samplers (Casella Model T 13050). The device was carried by the subject throughout her stay outside home or workplace. A filter holder was attached to the subject's collar and was connected by a plastic tube to a pump, which was carried in the handbag. At home and at the workplace the sampler operated as a stationary device. Air filters were wet-digested with concentrated HNO_3 in glass beakers, heated on a hotplate at about 70 °C. The final solution was about 0.3 M HNO_3 and was adjusted to 10 ml. Standards were prepared by adding known amounts of Pb and Cd on blank filters and treated as described for the samples. Lead and cadmium were analysed by flame AAS (Pye Unicam SP9, with DBC and Slotted Tube Atom Trap system). The detection limits of this method were 21.3 µg/L for lead and 1.9 µg/L for cadmium.

Duplicate diet collection and analysis

Duplicate diets, i.e. everything consumed (meals and drinks) were collected in plastic containers, weighed and homogenized immediately after sampling and stored at −20 °C. Two replicates of about 10 g of each sample were dried at 105 °C and dry ashed at 450 °C (14) in quartz crucibles. The final solutions were prepared in 0.6 M HNO_3 and adjusted to 10 ml, and analysed for cadmium by flame AAS (Pye Unical SP9 with DBC). Lead was analysed by an ET-AAS method (Perkin-Elmer 4000 with DBC, HGA 400 and recorder 056). The detection limit for lead was 1.4 μg/L and that for cadmium 2.2 μg/L.

Faeces collection and analysis

Faeces were collected in plastic bags provided by the TCC and stored at −20 °C. Two replicates of about 5 g of each sample were weighed into porcelain crucibles. Drying, ashing and dissolving of samples was carried out in the same way as for duplicate diets. Lead and cadmium were analysed by flame AAS (Pye Unicam SP9 with DBC). The detection limits of the method were 43 μg/L for lead and 2.2 μg/L for cadmium.

RESULTS AND DISCUSSION

Quality assurance

The results of the lead and cadmium analyses performed on QC samples at the same time as the analyses of samples collected in the monitoring phase are presented in Figure 1 for blood, diet, faeces and air filters. All the laboratory values were well correlated to the reference values ($r > 0.90$, $P < 0.001$) and were found acceptable according to the criteria (MAD and AI lines). The QC of lead and cadmium in air filters gave acceptable values for a wide range of both elements. The range of lead in QC filters was 1–30 μg/filter and of cadmium 4–113 ng/filter. The lead values obtained for the monitoring samples were mostly below the lowest values in the QC samples. Cadmium air concentrations, i.e. the quantities collected in 24-hour filter samples, were mostly very low and in some of the samples below the detection limit of the method used (19 ng/filter).

Monitoring

Blood concentrations of both lead and cadmium are generally regarded as reflecting mainly recent exposure of an individual to these elements. In a previous WHO/UNEP monitoring study of non-occupational exposure to lead and cadmium in subjects living in Zagreb, the concentration of blood lead was found to be 81 ± 33 μg/L (mean ± standard deviation) in a group of 84 healthy adult women, non-smokers (7). In the present study the value obtained was lower, i.e. 50 ± 17 μg/L, which was confirmed by the finding of relatively higher ALAD and lower EP values. Considering the small number of subjects (Table 1) the prevalence of low Hb and Hct values in this study was

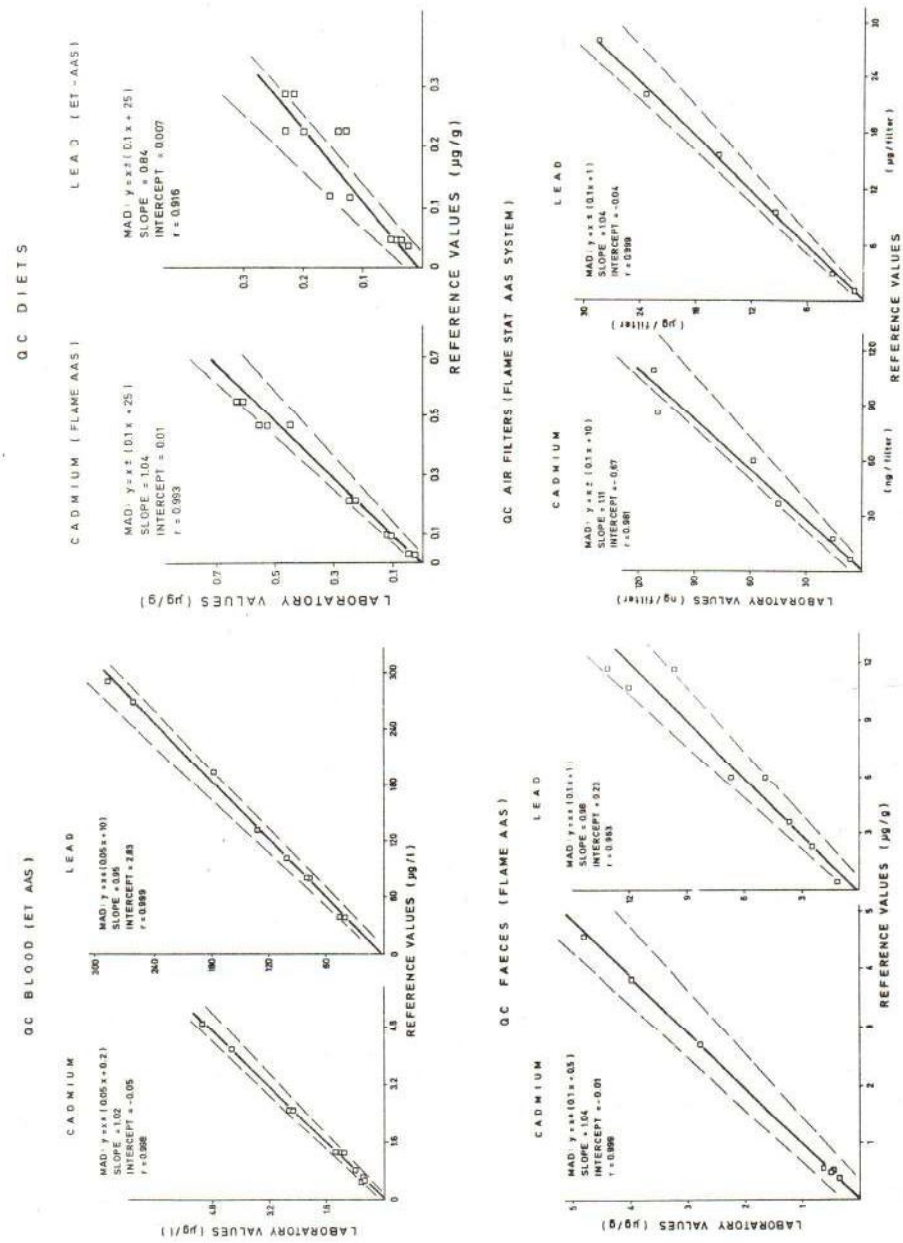

Fig. 1. *Cadmium and lead in QC samples of blood, diet, faeces and air filters related to reference values. Broken lines indicate acceptance interval (AI)*

Table 1.

Blood lead (PbB), blood cadmium (CdB), δ-aminolaevulinic acid dehydratase (ALAD), erythrocyte protoporphyrin (EP), haemoglobin (Hb) and haematocrit (Hct) in 17 adult women living in Zagreb

Subject No.	PbB (μg/L)	CdB (μg/L)	ALAD (European Units)	EP (μmol/L ercs)	Hb (g/L)	Hct (L ercs/L blood)
1	101	0.2	27.4	2.46	121.4	0.40
2	41	0.1	58.9	2.96	97.5	0.36
3	46	0.9	62.1	1.47	112.7	0.39
4	41	0.3	66.5	0.98	122.4	0.42
5	32	0.4	116.3	0.95	119.3	0.39
6	48	0.8	99.7	1.33	119.0	0.40
7	47	0.9	97.6	1.27	128.3	0.42
8	42	1.1	111.5	1.40	123.5	0.41
9	57	0.8	49.7	0.86	117.6	0.43
10	56	0.9	80.9	0.92	125.5	0.40
11	25	0.5	42.9	0.86	110.3	0.38
12	41	0.8	70.1	0.88	125.9	0.42
13	71	0.7	65.5	0.90	121.4	0.41
14	45	0.6	69.2	1.73	103.7	0.38
15	48	0.9	72.3	1.88	92.0	0.37
16	56	0.3	54.8	1.14	98.6	0.36
17	60	0.6	67.8	0.90	127.3	0.41
Mean ± SD	50 ± 17	0.7 ± 0.25	71.4 ± 23.7	1.35 ± 0.61	115.7 ± 11.3	0.39 ± 0.02
Range	25 – 101	0.2 – 1.1	27.4 – 116.3	0.86 – 2.96	92.0 – 128.3	0.36 – 0.43

rather great. However, low Hct values can decrease PbB values to some extent, and the correction of PbB results for Hct has occasionally been advocated (15). The blood cadmium concentrations found in the previous monitoring studies and those reported here are similar, i.e. 0.5 ± 0.35 and 0.7 ± 0.25 μg/L, respectively. During both monitoring periods, the air lead concentrations are assumed to have been comparable because of the high lead level in gasoline which is still in use. The lead and cadmium concentrations in the Zagreb air were found to be 412 ± 195 and 4.5 ± 2.4 ng/m^3 in the present study (Figure 2). They were 6 and 4 times higher than those in the Stockholm air (16), where low-lead gasoline (0.15 g/L) has been in use for many years and a considerable proportion of the gasoline now sold contains no added lead. However, relatively large intraindividual and interindividual differences were observed for lead and cadmium in inhaled air, as measured by personal samplers (Figure 2). This could also explain an apparent disagreement between the actual lead levels found in blood and in the air filter samples. The daily intake of both elements by inhalation was

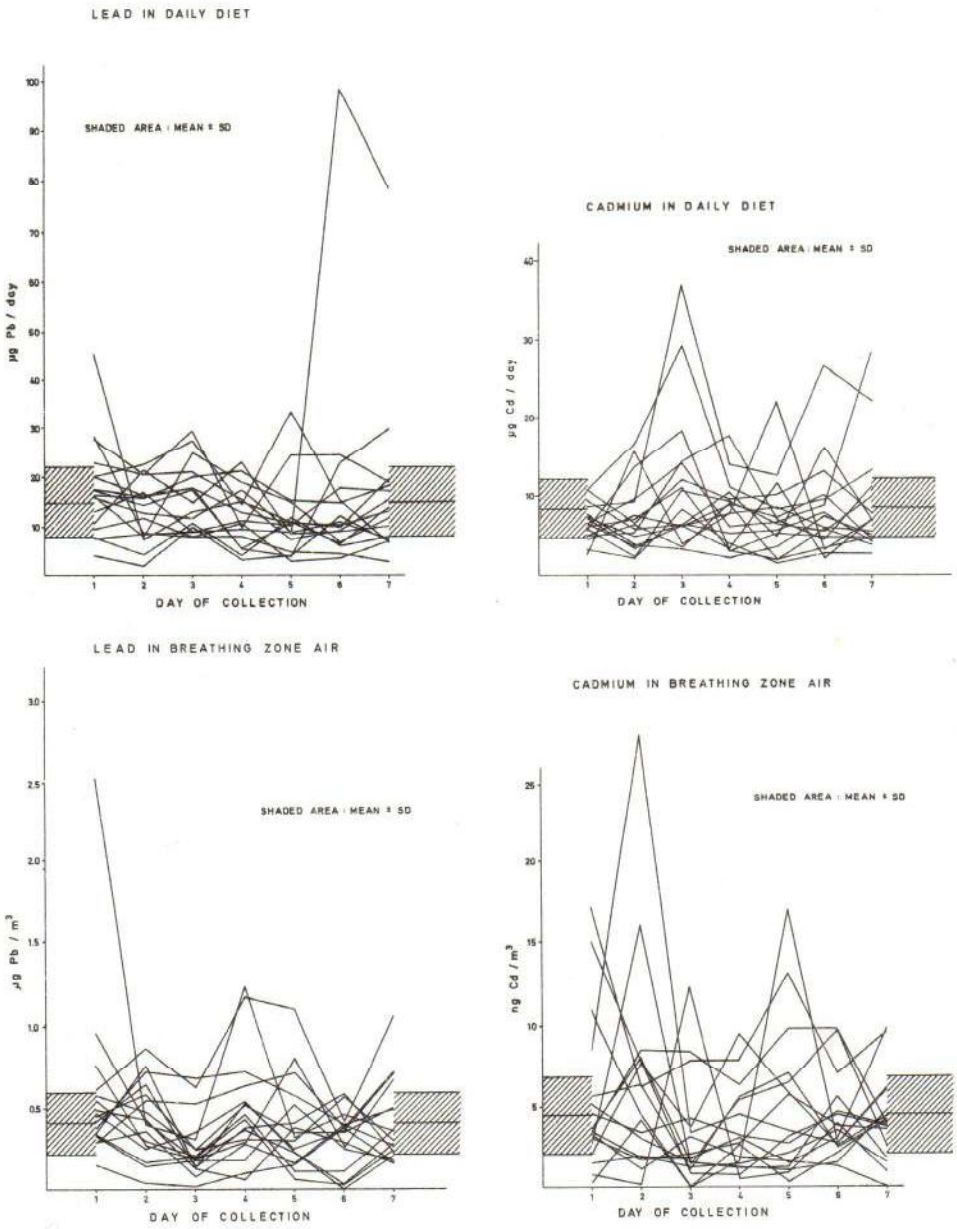

Fig. 2. *Lead and cadmium in collected duplicate diet samples (above) and in air filter samples (below) for each subject during 7 day-collection period. Shaded area presents mean ± SD of all subjects.*

Table 2.

Quantities of lead and cadmium inhaled, ingested (mean of 7 days) and eliminated by faeces (mean of 4 days) in 17 subjects

	Lead	Cadmium
Intake by inhalation (μg/day)	5.4 ± 2.5* (1.8 – 11.0) 4.8	0.06 ± 0.03 (0.02 – 0.13) 0.04
Intake by food (μg/day)	15 ± 7.2 (6.1 – 37) 15	8.5 ± 3.8 (3.5 – 18.6) 8.0
Faecal elimination (μg/day)	49 ± 23 (8.8 – 112) 50	15 ± 6.7 (5.0 – 25) 15

* Arithmetic mean ± standard deviation, range (in parentheses) and median

calculated assuming that about 13 m^3 of air was inhaled per person during each 24-hour period. It was also assumed that 50% of the airborne lead and cadmium was deposited in the alveoli and absorbed. For lead the average estimated absorbed quantity from the inhaled air amounted to 2.7 μg/day and for cadmium it was 0.03 μg/day (Table 2).

The individual data on the quantity of ingested food and the estimated lead and cadmium ingestion and excretion are shown in Figure 2 and Table 2. The mean quantity of lead ingested through the diet was estimated to be 15 μg/day, 10% of which is assumed to have been absorbed from the gastrointestinal tract. The ingestion of cadmium was estimated to be 8.5 μg/day of which only 5% is assumed to have been absorbable. Therefore the estimated mean absorbed quantities of lead and cadmium from food were 1.5 and 0.4 μg/day. The daily faecal excretions of lead and cadmium were 3.3 and 1.8 times higher than the amounts estimated to have been ingested with food. (Table 2). Since the analytical error can be regarded as being negligible, this difference remains to be explained by some other factors.

The quantities of food ingested by the subjects in the present study were considerably lower than those ingested by the subjects examined in Sweden, i.e. 1300 against 2300 g/day (17). Thus the estimated intake of lead and cadmium through food may not be representative for a longer period. This possibility should be taken into account when considering the apparent disagreement between the estimated amounts of ingested lead and cadmium and those excreted in the faeces, as well as the observed large individual differences in daily diet intake. The reason for this disagreement may have been additional oral intake for example from dirty hands, or difficulty to obtain faecal samples which corresponded exactly to the food ingested on a given day. Meanwhile, if it is assumed that the amount eliminated in faeces is the actual quantity of lead and cadmium which was ingested, then the absorbed amounts from the gastrointestinal tract would be 4.9 μg/day for lead and 0.75 μg/day for cadmium.

In conclusion, the present study in which exposure to lead and cadmium was monitored in a small segment of the Zagreb population indicated that a significant proportion of the total lead absorbed (over 36%) was due to lead-polluted urban air. The absorbed quantity of cadmium in this non-smoking population appeared to originate mostly from food and only a very small, insignificant amount came from inhaled air (about 4%). This exposure monitoring was only a preliminary study performed on a very small selected group of subjects. Future studies should include urban populations which are at highest risk, i.e. pregnant women and small children. The technique of exposure assessment could be slightly changed according to the experience gained during this preliminary study: filter collection could be prolonged several days to obtain higher values and facilitate measurements; faecal collection could be extended to a period of seven days to obtain more reliable results; detailed dietary record should be kept for all subjects to evaluate why the values of the duplicate diet methods were underestimated.

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Sažetak

ODREĐIVANJE IZLOŽENOSTI OLOVU I KADMIJU PUTEM ZRAKA I HRANE U STANOVNIKA ZAGREBA

Izloženost olovu i kadmiju mjerena je na skupini od 17 žena nepušačica u Zagrebu. Praćenje je uključivalo određivanje unosa ovih elemenata putem zraka i dnevne prehrane. Primijenjena je metoda »dvostrukih obroka« i osobni sakupljači čestica zraka, uzastopce tijekom sedam dana. Kao kontrola unosa ovih elemenata putem hrane poslužilo je sakupljanje i analiza fekalija. Dobiveni rezultati upućuju na to da su vrijednosti dobivene iz fekalija pouzdanije te su one uzete za procjenu apsorpcije. Gastrointestinalna apsorpcija olova procijenjena je na vrijednost od 4,9 μg/dan, a kadmija na 0,75 μg/dan. Dnevno apsorbirana doza određena iz koncentracije elemenata u zraku daje vrijednost od 2,7 μg/dan za olovo, a 0,03 μg/dan za kadmij.

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Ključne riječi: analiza krvi, dnevna prehrana, analiza fekalija, benzin, unos metala, praćenje, nepušači, osobni sakupljači, gradsko stanovništvo.