Cadmium, zinc, copper, and metallothionein levels in the kidney and liver of inhabitants of Upper Silesia (Poland)*

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Summary. The levels of Cd, Zn, Cu and metallothionein (MT) were determined in renal cortex and liver of 75 subjects deceased in the period 1986–1989 in the area of Upper Silesia (Katowice). The mean age of the population studied was 53.6 ± 14.6 years. The determined levels (mean ± SD) were: 43.1 ± 23.5 µg Cd/g; 52.5 ± 17.4 µg Zn/g; 2.2 ± 0.7 µg Cu/g; 0.80 ± 0.36 µmol Hg/g in renal cortex and 3.5 ± 2.5 µg Cd/g; 82.8 ± 34.3 µg Zn/g; 4.5 ± 2.6 µg Cu/g; 0.69 ± 0.44 µmol Hg/g in the liver. The level of Cd in renal cortex was 40% higher in smokers compared to nonsmokers and was independent of the gender. Whole-body retention of Cd was 34.1 ± 18.5 mg; smoking elevated the value from 27.1 to 38.2 mg. Compared with a similar study made in central Poland (Lódz), a significant difference was found only regarding the level of Zn and MT in the liver, pointing to the possibility that exposure to this element in the region of Upper Silesia may be higher.

Key words: Cadmium – Zinc – Kidney – Liver

Introduction

Cadmium is a well-known toxic metal occurring in the environment from natural and anthropogenic sources. It accumulates in the human organism during practically the whole life span, the kidneys and the liver being the main sites of accumulation. A 20-fold increase in the anthropogenic cadmium emission during the twentieth century has caused a five-fold increase in whole-body retention in humans [13].

Long-term low-level exposure to cadmium leads to tubular renal dysfunction [42]. The dysfunction is irreversible [18] and may be considered the first symptom of further renal, damage [45]; it is predictive of an exacerbation of the age-related decline in the glomerular filtration rate.

The best method for evaluation of cadmium exposure is biological monitoring, i.e., determining cadmium levels in the renal cortex (CdK), urine (CdU), and blood (CdB). However, critical values for the general population are still uncertain. The generally accepted critical concentrations of 200 µg/g and 10 µg/g creatinine for CdK and CdU, respectively, concern professional exposure [19]. International monitoring studies of cadmium levels in the blood, urine, and renal cortex of humans initiated in 1978 by UNEP/WHO have shown [55] great geographical differences in the levels of cadmium in humans (e.g., for renal cortex: Japan 61.1 mg/kg; Sweden 18.3 mg/kg) caused by the differences in daily intake. They have also proved the need for quality control along with monitoring studies.

Poland is considered one of the most polluted countries in Europe. Ecologically endangered regions constitute about 10% of the country’s surface area and are inhabited by one-third of the population [40]. Upper Silesia, the most industrialized region of the country, is also the most polluted. It constitutes 1% of the country’s surface area but is inhabited by 3 million people, almost 8% of the whole population [20]. It is in Upper Silesia that cadmium production was started in 1829, and in 1910 10% of the world’s cadmium still came from Upper Silesia [11]. The following information characterizes the recent situation in this region.

Katowice, with a population of 366 000, is the capital of Upper Silesia, and a centre for heavy industry in Poland. The mean monthly cadmium concentration in the air of the Katowice area (GOP) in 1982 was 17.0–110.0 ng Cd/m³ [16]. Jaworowski et al. [27] give a mean daily concentration in the centre of Chorzów (1981–1982) of 28.9 ng/m³. The fallout of cadmium was in the range of

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The maximum allowed concentration (yearly mean) according to Polish regulations [43] is 100 ng/m³, and the corresponding value recommended by the WHO [56] is 10–20 ng/m³.
Materials and methods

Subjects. The investigations were carried out on people who died in the years 1985–1989 as inhabitants of Katowice or its close surroundings. Autopsies were performed at the Department of Pathology, Silesian Medical University, Katowice, on average 32 ± 17.5 h after death. Renal cortex and liver samples were collected from persons not exposed to cadmium professionally who had no history of liver or kidney diseases and in whom no macroscopic lesions were found in these organs at autopsy. The information about the patient (taken from the case history) was included in a questionnaire concerning sex, age, body weight, kidney and liver weights, profession, place of residence, and smoking history. Unfortunately, full information was not always available.

The samples were obtained from 75 persons (42 women and 33 men) with a mean age of 53.6 years. Samples (ca. 20 g) of tissues were collected from the lower left pole of the kidney after separating the cortex from the medulla, and from the upper part of the left lobe of the liver. The samples were placed in acid-washed (10% HNO₃, 48 h) polyethylene containers, which were then closed tightly and stored until analysis at −20°C.

Measurements. Metals (cadmium, zinc, and copper) were measured by flame AAS (Pye-Unicam SP-192) with deuterium background correction after tissue digestion with a mixture of acids (HNO₃, H₂SO₄, HClO₄, Merck, Suprapure) as described elsewhere [4]. Three samples were always prepared from each tissue. The detection limits were: 0.02 μg Cd/ml, 0.01 μg Zn/ml, and 0.04 μg Cu/ml. The relative standard deviation for ten determinations at 0.2 μg/ml was 5%, 2%, and 5% for cadmium, zinc, and copper respectively.

Metallothionein was determined in full homogenates using the 203Hg method [58], giving a relative standard deviation of 5%. Levels of metallothionein were expressed in μmol Hg/g wet tissue to enable easy comparison with metallothionein levels determined by different methods.

Analytical quality assurance. Analyses of metals were performed under strictly defined conditions alongside analyses of internal laboratory control samples based on a CL-1 standard (lyophilized cabbage leaves) supplied by AGH (Poland) with certified metal levels. We also participated in the interlaboratory analytical quality control program conducted by the Institute of Veterinary Science (Poland). In three received samples our results were within 4% of the correct values. In addition, three samples of the renal cortex were analyzed by us (AAS method) and then using neutron activation analysis in the Institute of Chemistry and Nuclear Techniques in Warsaw; the maximum differences between the methods were 4%, 17%, and 20% for cadmium, zinc, and copper respectively.

Statistical analysis. All the determined values revealed log-normal distributions. In Table 1 all the parameters for each distribution are given along with the arithmetic mean and standard deviation for easier comparison with the results of other authors. Student’s t-test was used to evaluate statistical differences between groups. The whole-body retention of cadmium was calculated assuming that the kidney and liver contain half of the total cadmium content of the organism and that the ratio of cadmium concentration in the cortex to that in whole kidney is 1.25 [51].

Results and discussion

Cadmium

Cadmium levels in the renal cortex (CdK) and the liver (CdL) for the whole investigated population are shown in Tables 1–3. The dependence of CdK on age is shown in Fig. 1. We observed, in agreement with other data [48], a great range of concentrations (7.4–107.5 μg/g) with upper values in individual age groups increasing until about 50 years of age and then decreasing, in accordance with the well-known age dependence [19]. The upward trend with age is not evident, however, from the mean values (Table 3).

The CdK levels in Upper Silesia for nonprofessionally exposed persons (GM 36.1 μg/g) are the highest in Europe [22, 46, 53–55] (Fig. 2). Unexpectedly, however, these levels do not differ from the values found by us in Central Poland (GM 35.5 μg/g) [7]. A higher concentration can only be found in Japan [23, 55].

Smoking greatly increases the cadmium level in the organism [55]. In smokers CdK levels (47.7 μg/g) were ca. 40% higher than in nonsmokers (34.7 μg/g) (Tables 1, 2). The results are in agreement with literature data [22, 46, 54, 55]. The high mean CdK level (43.1 ± 23.5 μg/g), as well as the value for the 90th percentile (73.6 μg/g), is the result of smoking practised by the majority of the studied population. The tobacco produced in Poland contains high amounts of cadmium (1.3–3.2 μg Cd/ cigarette) [8, 57] that exceed the cadmium levels in imported cigarettes (0.5–1.0 μg/cigarette) [57]. No effect of sex on CdK has been found (Table 2).

The mean CdK level in Upper Silesia is lower than the generally accepted “critical concentration” of 200 μg/g [19]. It is, however, very close to the value 50 μg/g, at which “renal risk” was observed in 10% of general population in Belgium. Also Lindqvist et al. claim [32] that the critical value for the general population should be lower than the hitherto accepted value and they suggest 30 μg/g. One should also bear in mind the groups at particular risk, e.g., diabetes mellitus patients and people with calcium or iron deficiency, for whom the critical value may be even lower.

The levels of cadmium in the renal cortex should reflect the daily intake of cadmium with food. The daily cadmium intake with food averages 27.5 μg in Poland, and values as high as 77.8 μg have been reported [3]. Data for nonsmokers are of special value for the evaluation of the dependence between cadmium intake in food and its tissue levels. High CdK levels close to 30 μg/g in nonsmokers correspond to daily intakes of 15 μg (Belgium [9]) or nearly 30 μg (Poland [3]). The discrepancy

### Notes

1. The maximum allowed level in Poland is 3 mg Cd/kg soil [34].
2. The maximum allowed level in drinking water in Poland is 5 μg/dm³ [17].