Cadmium determination in urine by atomic absorption spectrometry as a screening test in industrial medicine

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Lehnert, G., Klavis, G., Schaller, K. H., and Haas, T. (1969). *Brit. J. industr. Med.*, **26**, 156-158. **Cadmium determination in urine by atomic absorption spectrometry as a screening test in industrial medicine.** Cadmium was determined by atomic absorption spectrometry in the urine and serum of 18 workers exposed to cadmium dust and fume in a zinc smelting plant. In all cases the concentrations of cadmium in the serum were within the normal range, but those in the urine were elevated. The elevated urine concentrations showed that the exposed group had taken up more cadmium than normal, although they showed no clinical symptoms of cadmium poisoning. Analysis of urine for cadmium is therefore of value in preventive medicine, and should be used for periodic check-ups on those occupationally exposed.

Today cadmium is mainly used as a component of low melting alloys, in pigment manufacture, in electroplating, and in the making of cadmium accumulators. The significance of cadmium for industrial medicine lies in the fact that closely above its melting point its vapour pressure is so high that toxic vapour concentrations may result. The risk to health is mainly associated with the production of alloys containing cadmium and cadmium-coated metals, especially with smelting, casting, welding, and cutting.

The signs and clinical characteristics of the toxic effects of cadmium have been reported in several publications and were summarized by Baader (1960), Browning (1961), Hunter (1962), and Koelsch (1962, 1963, 1966). Whereas the diagnosis of acute intoxication is relatively easy, signs of chronic poisoning could be overlooked. As with other toxic metals (Lehnert, 1968), estimations of cadmium in biological specimens from people occupationally exposed may be used to diagnose incipient poisoning and to monitor the working environment. Atomic absorption spectrometry both simplifies and increases the precision of cadmium estimations (Lehnert,

Schaller, and Haas, 1968). Our first results using this method on urine and serum from exposed workers are reported here.

The exposed group

The group consisted of 18 workers in a zinc smelting plant. Regular medical examinations since the beginning of 1967 had failed to reveal any clinical symptoms attributable to cadmium. There was, however, undoubtedly exposure to cadmium, as the dusts in the roasting plant contained 0.05 to 3% cadmium as well as zinc, lead or mercury. Cadmium was present usually as the sulphide, but in the zinc plant the cadmium fumes leaving the distillation furnace were burnt to cadmium oxide.

Materials and methods

Cadmium determinations in serum and urine were performed as described by Lehnert and his colleagues (1968). The urine specimens were collected in polyethylene bottles, which had previously been rinsed with nitric acid (1:3). The blood was drawn with plastic syringes and needles which were used only once. All

glass material was washed with nitric acid and distilled water. Ten millilitres of serum or 50 ml. of urine were wet ashed with 10 ml. 65% HNO_3 , 5 ml. 96% H_2SO_4 , and 5 ml. 70% HClO_4 . The white residues were dissolved in dilute hydrochloric acid and adjusted to pH 2.5 using a pH meter. Two millilitres of 5% aqueous ammonium-pyrrolidine dithiocarbamate solution were added forming a cadmium chelate, which was extracted into 2 ml. methyl isobutylketone (MIBK).

For the final measurement in the organic phase a Beckman atomic absorption spectrometer was used in combination with a Beckman spectrophotometer DU (lamp current, 5 mA; turbulent flow burner assembly with three sprayer burners; triple-pass; air pressure, 11 psi; hydrogen pressure, 2 psi; slit, 0.06 mm.; wavelength 2288 Å (228.8 nm.); 10-in. recorder). The instrument was calibrated with standard solutions treated in the same way. With the given instrument settings a detection limit of 0.012 µg. cadmium/100 ml. urine or 0.06 µg. cadmium/100 ml. serum could be obtained. Sensitivity could be slightly improved by reducing the volume of organic solvent using the improved hollow cathode lamp and by expanding the scale. The precision of the method was $\pm 10\%$, and mean recoveries were 96%.

Cadmium levels in the urine were corrected to creatinine content and are expressed as µg. cadmium/g. creatinine.

Results and discussion

Lehnert and his colleagues (1968) found that, in specimens taken from adults who had not been exposed to cadmium in the course of their work, the

cadmium concentrations were 0.33 ± 0.24 µg./100 ml. serum (mean $\pm$ S.D.) and in urine 0.63 ± 0.34 µg./g. creatinine (24-hour excretion in urine was 0.98 ± 0.36 µg.).

In specimens from exposed workers (Table) all the serum concentrations were within 3 standard deviations of the normal mean and could not, therefore, be regarded as abnormal; but urines from 14 of the 18 persons examined exceeded the normal average urine concentration by more than 3 standard deviations. There was no correlation between the duration of exposure to cadmium and cadmium concentrations in either the serum (correlation coefficient = -0.124) or the urine (correlation coefficient = 0.060) (see also the Figure). This is in agreement with our experience with other heavy metals (Lehnert, 1968).

The discrepancy between the (normal) serum cadmium concentrations and the (pathological) rates of cadmium elimination is remarkable. There was no correlation between these variables ($r = -0.377$), since theoretically a dynamic steady state should exist between the cadmium-influx, -interflux, and -efflux. The incongruity might be explained if, as Friberg (1952) states, cadmium is mainly bound to the erythrocytes. We will examine this question in further studies to be reported later.

As there were no clinical signs which could be attributed to cadmium the increased urinary cadmium did not indicate cadmium poisoning. The

TABLE
DETERMINATION OF Cd (II) IN SERUM AND URINE OF 18 CADMIUM-EXPOSED WORKERS

Name	Age (years)	Work-place	Exposure (years)	Analyses	
				Serum (µg. Cd (II)/100 ml.)	Urine (µg. Cd (II)/g. creat.)
B.F.	36	Roasting plant	2	0.08	2.55
D.W.	28	Roasting plant	10	0.04	2.05
D.H.	35	Mercury plant	12	0.23	1.48
F.G.	38	Fine zinc plant	2.5	0.90	0.80
G.A.	49	Mercury plant	4	0.17	3.60
H.W.	38	Mercury plant	11	0.25	1.40
K.D.	37	Roasting plant	18	0.36	3.91
K.A.	46	Roasting plant	4	—	5.62
M.H.	31	Fine zinc plant	11	0.0	2.71
P.K.	59	Fine zinc plant	11	0.15	5.31
P.J.	34	Mercury plant	0.055	0.07	5.60
P.H.	41	Fine zinc plant	10.5	0.24	3.40
R.H.	34	Mercury plant	9	0.05	2.62
R.H.	31	Electroplating process	8	0.14	2.72
S.A.	56	Roasting plant	5	0.32	2.56
S.E.	30	Roasting plant	1	0.11	1.15
T.G.	35	Roasting plant	20	0.11	3.43
T.H.	51	Roasting plant	10	0.0	6.17
				0.0 — 0.90	1.15 — 6.17 ¹

¹ Mean differs from mean of normals ($P < 0.001$)

FIGURE Urinary cadmium plotted against years of exposure for the 18 workers from a zinc smelting plant: correlation coefficient (r) = 0.06; $\mu\text{g. Cd/g. creatinine} = 3.05 + 0.18 \times \text{years}$.

results, however, indicate an increased uptake of cadmium. The analysis of urine from workers

believed to be exposed can be used to monitor the working conditions, and periodical estimations should make it possible to introduce protective measures in time to prevent the development of organic damage.

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