Cadmium Influence on the Excretion of Sodium by Kidneys

The parenteral application of cadmium organic salt intensified sodium reabsorption in kidney's proximal tubuli of dogs[1,2]. In our experiments on rats we investigated this phenomenon in dependence on long term application of cadmium trace quantities administered in drinking water.

Material and methods. The total of 34 3-week-old female rats strain Wistar (substrain Konarovice) were placed in polyethylene laboratory cages and fed on Larsen's laboratory diet, its cadmium mean content being 0.004 ppm. Animals were given drinking water. A left-side nephrectomy was carried in one half of the animals. 30 days after the operation the animals began to drink a solution of 5 ppm cadmium as cadmium acetate dissolved in deionized water.

30 additional animals of the same age were used as a control group. The left side nephrectomy was carried out in one half of them at the same time as it was the case with cadmium-fed animals; 30 days after the operation all control animals began to drink a 1% solution of NaCl in deionized water, containing, in addition 50 ppm of Zn, 5 ppm of Cu, 10 ppm of Mn, 1 ppm of Co and Mo. They were fed on Larsen's diet too.

All experimental and control animals were kept under these conditions for 16 months, without interruption. Then, all animals were given intragastrically 3.33% (0.57 *M*) solution of NaCl in doses corresponding to 1.2% of the animals' body weight [3]. The rats were placed for 3 h in metabolic cages, their urine being collected in calibrated test tubes. Sodium concentration in urine was determined by flame spectrophotometer Zeiss. The student *t*-test was used for statistical evaluation. A difference with $P < 0.05$ was considered to be statistically significant.

Results and discussion. It is apparent from our results presented in the Table that cadmium-fed animals differed from the control animals, above all, in the values of excreted milliequivalents of sodium. This basic difference is of high statistical significance in the group of animals with nephrectomy ($P < 0.001$), and is also statistically significant in the group of animals with both kidneys. The salt load became evident in the diuresis of all groups of animals. The total volume of urine excreted by experimental female rats with nephrectomy during 3 h interval was the lowest of all groups of animals, the difference, however, being not statistically significant. The excreted urine volume, expressed as the percentage of the volume of NaCl solution applied, was lowest in both experimental groups, and especially so in the group of animals with nephrectomy. The exposure to cadmium, together with nephrectomy, had in this case a particularly clear effect on the reduced volume of excreted urine in dependence on the simultaneous salt load. The relation between the experimental group of animals likewise as the relation between the experimental and control animals with nephrectomy were, in this index, of statistical significance ($P < 0.05$ and $P < 0.001$).

The influence of chronic exposure to cadmium ions, therefore, manifests itself most clearly, in animals with nephrectomy, which excrete the salt load in a much reduced way. Coincident results in the group of experimental non-operated animals give evidence of this fact as not being provoked by nephrectomy, but being the direct consequence of the influence of accumulated cadmium in kidneys on excretion and reabsorption of renal sodium. Analysing cadmium content in kidneys of experimental rats with nephrectomy, the values 7.65 ± 0.76 µg Cd/g (wet wt.) and of the experimental animals with both kidneys 4.79 ± 1.33 µg Cd/g (wet wt.) were established. The kidneys of control animals with nephrectomy contained, under the same conditions 1.53 ± 0.11 µg Cd/g(wet wt.) and non-operated animals 1.29 ± 0.38 µg Cd/g (wet wt.)[4].

Urine excretion and sodium concentration in the urine of female rats drinking 5 ppm cadmium for 16 months 3 hours after the salt load (Mean ± standard error)

Group	administered mEq/l Na	Urine excretion (ml)	Urine excretion (% of administered volume of NaCl)	*Na* concentration (mEq/l)	*Na* excretion (% of administered dose)
Experimental animals with both kidneys $n = 15$	1740.01 ± 60.13	4.39 ± 1.83	137.47 ± 16.70	519.90 ± 58.51	32.88 ± 4.73
Control animals with both kidneys $n = 15$	1675.23 ± 50.30	3.95 ± 0.57	145.06 ± 26.18	758.36 ± 62.01	46.09 ± 6.20
Significance	n.s.	n.s.	n.s.	$P < 0.01$	n.s.
Experimental animals with nephrectomy $n = 19$	1756.72 ± 48.66	3.28 ± 0.84	99.53 ± 4.39	419.40 ± 89.50	28.31 ± 4.54
Control animals with nephrectomy $n = 15$	1694.22 ± 54.36	4.78 ± 0.42	165.47 ± 13.74	764.00 ± 30.43	45.12 ± 5.74
Significance	n.s.	n.s.	$P < 0.001$	$P < 0.001$	$P < 0.05$

The enhancement of the proximal tubular sodium reabsorption with the consequence of its reduced excretion from the body, leads to the accumulation of sodium ions in the body, this can constitute an important factor in developing some pathological states, and especially can play a role in developing cadmium hypertension; from this point of view it is, therefore, necessary to pay attention to this phenomenon.

Zusammenfassung. Spurenmengen von Cadmium, die Ratten während 16 Monaten im Trinkwasser verabreicht wurden, reduzieren bei einer einmaligen Kochsalzbelastung signifikant die Konzentration der Natriumionen im Harn.

J. LENER and J. MUSIL[5]

Institute of Hygiene and Epidemiology, Department of General and Occupational Hygiene, Švobárova 48, Praha 10 (Czechoslovakia), 26 February 1971.

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[5] New address: Department of Medical Basic Research Sandoz Ltd, CH-4002 Basel (Switzerland).