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*Letter to Editors*

# Effect of Long-Term Cadmium Intoxication on Selected Biochemical Parameters in Experimental Animals

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## Abstract

Cadmium (Cd) is a dangerous occupational and environmental toxin. Exceeding its permissible weekly uptake in meals is a disturbing phenomenon. The aim of this study is to assess the effect of long-term uptake of cadmium chloride on selected biochemical parameters and oxidative stress biomarkers in animal models. Long-term intoxication with cadmium chloride elevated blood serum concentration of urea, creatinine, glucose, AspAT and A1AT activity as well as TBARS and protein carbonyl group concentrations; TBARS concentration in erythrocytes was also elevated as well as 8-hydroxy-2'-deoxyguanosine excretion with urine.

**Keywords:** cadmium, serum biochemical parameters, oxidative stress biomarkers.

## Introduction

As a result of human economic activity, the natural circulation of elements (including harmful ones) is subject to various distortions. Harmful heavy metals numbered among impurities which penetrate the human organism cannot be avoided. They are found in drinking water, atmospheric air and even in food [1]. Recently, the effect of long-term exposure to cadmium compounds has been widely investigated [2, 3, 4, 5, 6], and perhaps most disturbing is the possibility of exceeding permissible temporary uptake of cadmium in meals [1]. Vegetable products are the main carrier of cadmium compounds in food rations as they are exposed to the most pollution in the natural environment.

The aim of this study is to assess the effect of long-term uptake of cadmium chloride on selected biochemical parameters and oxidative stress biomarkers in experimental animals.

## Material and Methods

The study comprised 20 male Wistar rats divided into two groups of ten each. The control group received drinking water for 3 months, while the investigated group received drinking water with 50 mg/1 of cadmium chloride also for 3 months. The animals were kept in animal quarters at constant temperature and humidity with free access to "Murigran" chow for small laboratory animals. After 3 months the animals were placed in metabolic cages where urine was collected and then in general narcosis they were terminated and blood collected for determination.

Blood serum, urea, creatinine and glucose levels and AspAT and A1AT activity were determined with Bio-Merieux apparatus and reagents. Peroxidase - antioxidative balance was estimated based on:

a) content of compounds reacting with thiobarbituric acid (TBARS) in blood serum and erythrocytes hemolysate according to Rice-Evans [7],

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b) content of protein carbonyl groups ace. to Levin et al., [8] and Reznik et al. [9],

c) concentration of 8-hydroxy-2'-deoxyguanosine in urine with HPLC method.

The obtained results were statistically analysed with Mann-Whitney nonparametric test with Statistica program No SP7105488009G51.

The Bioethics Committee of the Military Medical University gave consent No 117/00 to conduct the investigations.

## Results

Administration of 50 mg/1 cadmium chloride to rats in drinking water for 3 months resulted in elevation of the analysed biochemical parameter concentrations in relation to the control group (Table 1).

Cadmium chloride increased statistically significant serum TBARS concentration in erythrocyte hemolysate, plasma protein carbonyl groups content and 8-hy-droxy-2'-deoxyguanosine concentration in urine in relation to the control group (Table 2).

## Discussion

Cadmium is one of the most dangerous occupational and environmental toxins. It accumulates in the human organism mainly in liver and kidneys, where it causes functional changes and then interstitial fibrosis [10, 11]. As a result of long-term exposure to cadmium there may come to grade I urethras impairment and then to glomerular filtration impairment [12, 13], which might justify the elevation of urea and creatinine concentration in own studies. Rikans et al. [14], observed Kupffer's cell activation and liver cell infiltration with proinflammatory cytokins after cadmium intoxication; whereas Shaikh et al. [5], observed oxidative stress intensification after cadmium administration defined as intensification of liver peroxidation of fats and liver glutathione exhaustion. It is possible that the described processes taking place in liver are responsible for the increase of AspAT and A1AT activity in our investigations. Increase of glycemia as the result of long-term uptake of cadmium chloride is probably connected with the decrease of insulin excretion by pancreas, which was reported by other authors [15]. Cadmium ions, when bound with various proteins disturb different metabolic cycles. They evoke, among others, oxidative phosphorylation disorder, inhibit tissue respiration and activity of enzymes responsible for sodic and potassium ion transport [6, 16]. Cadmium decreases the activity of antioxidative system elements (superoxide dismutase, catalase) as well as glutathione content and leads to the production of oxygen reactive forms [6, 17]. The increase of the concentration of compounds reacting with thiobarbiturate acid (TBARS) in blood serum, erythrocyte hemolysate and 8-hydrexy-2'-deoxyguanosine in urine in the carried out own investigations may be the confirmation of these reports. A significant increase of 8-hydroxy-2'--deoxyguanosine in urine is disturbing in the group of animals on cadmium chloride, as it speaks for a significant damage of DNA thread which was reported earlier by other authors [18, 19]. In 1993 cadmium was recognised as a carcinogen mainly related to pulmonary carcinoma [20, 21, 22]. The search for compounds being able to limit harmful consequences of long-term environmental or occupational exposure to cadmium should thus be widely carried out.

Table 1. Effect of cadmium chloride on rat blood serum selected biochemical parameters.

| | Urea mg % | Creatinine mg % | AspAT U/l | AlAT U/l | Glucose mg % |
|---|---|---|---|---|---|
| Controls | 33.0 ± 4.31 | 0.43 ± 0.05 | 110.4 ± 15.5 | 42 ± 5.49 | 140 ± 15.85 |
| Cadmium | 50.2 ± 3.12 | 0.7 ± 0.03 | 142 ± 33.15 | 77 ± 20.76 | 170 ± 6.99 |
| Statistical significance | $p < 0.01$ | $p < 0.01$ | | $p < 0.05$ | $p < 0.05$ |

Table 2. Effect of long-term administration of cadmium chloride on selected oxidative stress biomarkers.

| | TBARS in serum µM/l | TBARS in erythrocyte hemolysates nM/g Hb | Protein carbonyl groups nM/mg of protein | 8-hydroxy-2'-deoxyguanosine in urine nM |
|---|---|---|---|---|
| Controls | 3.02 ± 0.4 | 16.4 ± 4.35 | 4.02 ± 0.94 | 0.45 ± 0.17 |
| Cadmium | 3.87 ± 0.37 | 36.5 ± 7.3 | 11.4 ± 2.1 | 84.14 ± 39.6 |
| Statistical significance | | $p < 0.01$ | $p < 0.001$ | $p < 0.001$ |

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