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CADMIUM AND SELENIUM IN ANIMAL TISSUES AND THEIR INTERACTIONS AFTER AN EXPERIMENTAL ADMINISTRATION TO RATS

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ABSTRACT

The aim of this work was to find the cadmium and selenium accumulation in the experimental rat tissues and their interactions in relation to their accumulation in the animal tissues. Male rats were dosed with Cd (2 mg.kg^{-1} b.w., i.p. and 30 mg.l^{-1}, p.o. in drinking water) and Se (2 mg.kg^{-1} b.w., i.p. and 5 mg.l^{-1}, p.o. in drinking water). Thirty six hours after an i.p. and after 90 days of p.o. administration of Cd and Se, the sample of liver, kidney, adipose tissue and muscle tissue (*m. quadriceps femoris*) were collected and contents of Cd and Se were analyzed using ETAAS (Cd) and HG-AAS (Se) methods. After an i.p. administration of Cd, a significant increase in Cd ($P<0.0001$) and Se ($P<0.01$) in liver, Cd ($P<0.05$) in kidney and adipose tissue and Cd ($P<0.0001$) and Se ($P<0.01$) contents in muscle tissue were found. After the peroral Cd administration, an increase in Cd ($P<0.0001$) in liver, Cd and Se ($P<0.0001$) in kidney, Se in adipose tissue ($P<0.05$) and in muscle tissue ($P<0.0001$) contents were observed. After an intraperitoneal administration of Se, the increase in Se in liver and kidney ($P<0.0001$), in muscle ($P<0.05$) and the decrease in kidney Cd contents ($P<0.0001$) were found. After the peroral administration of Se, the significant increase in Se in liver, kidney, adipose tissue ($P<0.0001$) and in muscle ($P<0.01$) contents were recorded. The results suggest that the separate administration of Cd causes the increase in Se content in tissues and also Se administration results in decrease in Cd kidney content.

Key words: cadmium; selenium; accumulation; animal tissues, interactions

INTRODUCTION

Cadmium (Cd) is an extremely toxic metal commonly found in industrial workplaces, particularly where any ore is being processed or smelted. Exposure to cadmium occurs as a result of atmospheric emission during Cd production and processing, from combustion of fossil energy sources, waste and sludge, phosphate fertilizers, and deposition of waste and slag at disposal sites. Meat, fish, and fruits generally contain up to 50 µg Cd.kg^{-1} on fresh weight basis, whereas vegetables, potatoes, and grain products may contain up to 150 µg Cd.kg^{-1} fresh weight. Higher concentrations are found in the kidneys of animals slaughtered for food, in wild mushrooms, and in seafood such as mussels and oysters (Fried and Rozman, 2008).

Cadmium has a strong preferential affinity for the liver and the kidney over a wide range of exposure levels. In general, about 50 % of the total body burden is found in these two organs. It is widely distributed elsewhere in the body at low concentrations relative to the liver and kidney (Hammond and Beliles, 1980). The kidney and liver are considered to be the major organs which accumulate cadmium and so are probably the most susceptible organs to cadmium effects (Yamano et al., 1999; Yiin et al., 1999). Cadmium causes tissue damage

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in humans and animals and many toxicological studies have found the functional and structural changes in the kidneys, liver, lungs, bones, ovaries and fetal effects (Chang et al., 1981; Friberg et al., 1986; Kukner et al., 2007; Massányi et al., 2007). Cd has been suggested to practice some of its toxic effects by disturbing metabolism of essential metals, such as Se. Zinc and selenium are believed to be the antagonists of cadmium toxic effects.

Selenium is known due to its antioxidant role in living systems and therefore it is considered to be an essential element for humans and animals. However, routine selenium supplementation is not recommended if the Se intake is equal to 80 µg per day or greater (Burk, 2002). The best way of adequate selenium intake is via food and increase of its content in plants by soil or foliar application has been under investigated during the last decade (Ducsay and Ložek, 2006; Zhao and McGrath, 2009). The treatment with Se during Cd exposure has been demonstrated to have beneficial effects on Cd-induced toxicity (Chen et al., 1975; Sugawara and Sugawara, 1984; Jamba et al., 2000; Kimáková et al., 2006; El-Sharaky et al., 2007). However, the co-effect of the trace element on the toxicity caused by Cd is not yet well studied. Xiao et al. (2002) assessed the protective effect of Se and Zn on Cd induced oxidative stress, only in the kidney of the rat, besides, Cd was administrated using the i.p. route. Alterations in selenium consumption and metabolism are reflected by changes in the activity of a selenoenzyme, glutathione peroxidase. Moreover, dietary selenium partially protected against lung injury caused by cadmium administered intratracheally (Reddy et al., 1978).

The aim of the work was to determine the effect of cadmium and selenium administered separately on the status of both metals in selected rat tissues. The present study was, therefore, undertaken to investigate if there exists the mobilization of the metal in organism after the antagonists administration, it means, an effect of cadmium on cadmium and selenium tissue concentrations and effect of selenium on cadmium and selenium concentrations on selected tissues.

MATERIAL AND METHODS

Fifty male Wistar rats were divided to five groups, cadmium-treated groups (G and J), selenium-treated groups (D and I) and control, untreated group (K), each containing 10 males. The males were housed individually in plastic cages in an environment maintained at 20-24°C, 55 ± 10% humidity, with access to water and food (feed mixture M3, Machal, Czech Republic) *ad libitum*. Sexually mature male rats of group G were administered a single intraperitoneal dose (2 $mg.kg^{-1}$ b.w.) of cadmium ($CdCl_2$, Reachem, Slovak Republic) and young 4 weeks old rats of group J were dosed with a daily Cd intake of 30 $mg.l^{-1}$ in drinking water for 90 days. Adult male rats of group D were administered a single intraperitoneal dose (2 $mg.kg^{-1}$ b.w.) of selenium (Na_2SeO_3, Sigma, USA) and young 4 weeks old males of group I were dosed with a daily Se intake of 5 mg/l in drinking water for 90 days. The liver, kidney, adipose tissue and muscle tissue (*m. quadriceps femoris*) were sampled 36 hours after the cadmium and selenium administration (groups G and D) and 90 days after the daily Cd and Se intake (groups J and I). The samples were weighed and stored at -20°C and then analyzed. Cadmium was analyzed using the electrothermal atomic absorption spectrometry (ETAAS) and selenium was determined using the hydride generation atomic absorption spectrometry (HG-AAS) (EL, s.r.o. Spišská Nová Ves, Slovak Republic).

RESULTS AND DISCUSSION

Rats exposed to cadmium showed significant accumulation of cadmium in liver, kidney and muscle tissue when dosed intraperitoneally and in liver and kidney after a peroral administration (Table 1). The kidney and liver are recognized as the main cadmium storage tissues (Jihen et al., 2008; Kolesarova et al., 2008). Selenium appears to antagonize cadmium, especially in acute exposures. In a mouse study, after acute cadmium exposure, a significant decrease in cadmium levels was observed in the kidneys and liver following an eight-week daily selenium supplementation (Jamba et al., 1997). It was interesting to follow the selenium content in the selected tissues after the cadmium administration. In our experiment, selenium content significantly increased in liver and muscle tissue after the i.p. exposure to 2 mg $Cd.kg^{-1}$ body weight and also significantly increased in kidney, adipose and muscle tissues after the daily peroral intake of 30 mg $Cd.l^{-1}$ in the drinking water during 90 days (Table 1). It suggests that increase in intake of cadmium mobilizes the selenium storage in the tissues which can be manifested by its increase in the main storage organs, but also in the muscle tissue. Similarly, Ongjanovic et al. (2008) reported that with increased Cd concentration in the liver and kidneys, Se concentration also rises, although it was not administered additionally.

Selenium was found to have a protective effect by decreasing Cd content in the liver and kidneys (Chen et al., 1975). However, it has also been observed that co-administration of cadmium and selenium (200 ppm + 0.1 ppm, respectively) in drinking water for five weeks did not decrease Cd concentration in the liver nor in the kidney, and only diminished the toxic effects of Cd in these organs (Jihen et al., 2008). In our study, selenium administration significantly ($P<0.0001$) decreased the cadmium concentration in the kidney in the

Table 1: Cd and Se concentrations in selected rat tissues after experimental cadmium and selenium administration (mg.kg^{-1})

		Liver $\bar{x}$ ± SD	Kidney $\bar{x}$ ± SD	Adipose tissue $\bar{x}$ ± SD	Muscle tissue $\bar{x}$ ± SD
Control group (K)	Cd	0.0059±0.001	0.0125±0.002	0.0050±0.001	0.0056±0.001
	Se	1.085±0.262	1.527±0.239	0.137±0.048	0.250±0.001
Group G	Cd	20.27±9.30++++	11.30±12.9+	1.17±1.47	0.088±0.051++++
	Se	2.775±1.378++	1.781±1.207	0.179±0.165	0.32±0.077+
Group J	Cd	0.0652±0.025++++	0.284±0.153++++	0.0108±0.016	0.013±0.025
	Se	1.292±0.22	2.46±0.538++++	0.398±0.331+	0.607±0.113++++
Group D	Cd	0.068±0.003	0.0061±0.0019++++	0.005±0.001	0.005±0.001
	Se	3.105±1.09++++	7.23±4.00++++	0.122±0.062	0.304±0.061+
Group I	Cd	0.010±0.009	0.012±0.007	0.005±0.001	0.006±0.002
	Se	3.266±1.271++++	4.256±0.977++++	0.424±0.112++++	0.423±0.149++

+P<0.05; ++P<0.01; ++++P<0.0001; SD – standard deviation

intraperitoneally exposed group. The selenium content significantly increased in liver, kidney and muscle tissue after the i.p. Se administration and in all observed tissues in the peroral selenium group (Table 1).

The mechanism of protective effects of selenium on cadmium toxicity and increased Se content in organs like liver and kidney after the cadmium intake can be explained by antagonism to Cd-induced DNA damage and by the fact that selenium is an essential constituent of a number of enzymes, some of which have antioxidant functions. Deficiency of the element in animals makes them susceptible to injury by certain types of oxidative stress (Burk, 2002). This protection includes the capability of Se to alter the distribution of Cd in tissues and induces binding of the Cd-Se complexes to proteins, which are similar to metallothioneins (Jamba et al., 1997; Combs and Gray, 1998; Ognjanovic et al., 2008). The lipid peroxidation, one of the main manifestations of the oxidative damage plays an important role in the toxicity of many xenobiotics. Intoxication with cadmium causes a significant increase of lipid peroxidation in liver and kidneys of rats (Ognjanovic et al., 2008) which are also the main organs accumulating the cadmium. Therefore, increase in selenium content in these organs can be connected with the protective role of selenium in oxidative stress induced by cadmium.

CONCLUSION

The major aim of this work was to evaluate the Cd and Se contents in the selected organs after the separate administration of both metals. The distribution of cadmium and selenium in the organism is in correlation. Increased intake of cadmium caused redistribution of selenium mainly in the main storage organs, liver and kidney and in adipose and muscle tissues. On the other hand, selenium intake decreased Cd content in the kidney. Increase in selenium content in these organs alters the distribution and accumulation of cadmium in tissues.

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