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CADMIUM-INDUCED CHANGES IN HAEMATO-BIOCHEMICAL PARAMETERS, LIPID PEROXIDATION AND GLUTATHIONE CONTENT IN BLOOD OF RATS

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Cadmium (Cd^{+2}) is an ubiquitous toxic metal that may induce oxidative damage by disturbing the prooxidant-antioxidant balance in the blood. Wistar albino rats (3 months old), were injected with a single dose of $CdCl_2$ (0.4 mg Cd/kg i.p. and sacrificed after 24^h). The hematological parameters: red blood cells count (RBCs), hematocrite (Ht) value and hemoglobin (Hb) concentration were significantly decreased in the blood of Cd treated rats. The activities of enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST) in plasma, as well as concentration of blood glucose were significantly increased in animals treated with cadmium in comparison to control values. The treatment with Cd increased lipid peroxidation (LP) and reduced glutathione (GSH)

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contents in the blood, suggesting that the Cd induced oxidative stress. These results of our study suggested that Cd induced alterations in hemato-biochemical parameters and LP and GSH content.

Key words: cadmium, hemato-biochemical parameters, lipid peroxidation, glutathione, blood, rat

INTRODUCTION

Cadmium (Cd) is a very toxic heary metal, an important pollutant of environment (present in soil, water, air, food and in cigarette smoke) which causes poisoning in different organisms (STOHS and BAGCHI, 1995). After the intake and resorption, cadmium enters the blood were it binds to erythrocytes and proteins of low molecular mass forming metalothioneins (MT) (THORNALLEY and VAŠAK, 1985). Cadmium is then transported into most of tissues and organs in which it also induces the forming of metalothioneins (WAISBERG *et. al.*, 2003). From totally accumulated cadmium in organism, about 75% is deposited in liver and kidneys (OGNJANOVIĆ *et al.*, 1995; ŠTAJN *et al.*, 1997). However, cadmium is accumulated in most of other tissues and organs, such as pancreas, salivary glands, testes, heart, brain or brown adipose tissue (KOSTIĆ *et al.*, 1993a; ŽIKIĆ *et al.*, 1998).

Binding of cadmium to erythrocytes causes their destruction and increased hemolysis, haematological values alterates (decrease of haematocrite values, haemoglobin concentration and total red blood cells count), absorption of intestinal iron is decreased and anemia appears (FOX and FRY, 1970; PRIGGE *et al.*, 1977; KOSTIĆ *et al.*, 1993b). Above mentioned parameters can be taken as the sensitive indicators of cadmium toxicity.

Investigations on different organisms have shown that cadmium causes significant metabolic and histological alterations, disturbs biological systems and decreases body mass growth and mass growth of certain organs (OGNJANOVIĆ *et al.*, 1995). Cadmium has negative effects in energy metabolism (KOSTIĆ *et al.*, 1993b), membrane transport (GRABOWSKA and GUMINSKA, 1987) and protein synthesis (OGNJANOVIĆ *et al.*, 1995).

FARISS (1991) has shown that the scavengers of free radicals and antioxidants may be used in the protection against cadmium toxicity. Some antioxidants, such as vitamin E (Vit E), ascorbic acid (AsA), glutathion (GSH) and selenium (Se) exert protective effects against oxidative damages in different tissueas (SHUKLA and CHANDRA, 1989).

These results of our study suggested that Cd induced alterations in hemato-biochemical parameters and LP and GSH content.

MATERIALS AND METHODS

Wistar albino male, 3 months old rats (weighing 280±30g) were used in experiment. The animals were kept at 21±2°C and exposed to 12 h light/dark cycle. All rats were housed in individual cages and given a standard diet and tap water ad

libitum. The rats of the experimental group were injected i.p. with a single dose of 0.4 mg Cd/kg body weight, as CdCl2, in 0.1 ml saline and killed 24 h after injection. The control animals received the equivalent volume of saline (0.1 ml/kg body weight). Every group consisted of 7 animals.

After the last treatment the animals were sacrificed by decapitation between 8^h and 10^h and fresh blood was immediately collected in to heparinized tubes. Number of red blood cells (RBC) and haematocrit (Hct) value were determined by standard haematological techniques (CHANARIN, 1989). The haemoglobin (Hb) concentration was determined by the cyanmethaemoglobin method (DRABKIN and AUSTIN, 1935). The blood glucose concentration was measured by the ortho-toluidine colorimetric method (HULTMANN, 1959). The concentration of LP was assayed as thiobarbituric acid-reactive substances (TBARS) in the blood according to OHKAWA *et al.* (1979). Concentration of GSH in whole blood was measured by standard method of BEUTLER (1975).

Aliquots of blood for the determination of transaminase were centrifuged to separate plasma and red blood cells. Plasma specimens were used for determination of activities of ALT and AST by spectrophotometricy method (WOOTON *et al.*, 1964).

All values were computed as means ± S.D. The statistical analysis of the data was performed using the Student's t-test. Differences from controls were considered significant at $p < 0.05$.

RESULTS

Results presented in Table 1 clearly show that intraperitoneal administration of Cd results in significant decreases of RBCs count, Ht value and Hb concentration ($p<0.05$) when compared to control animals.

Table 1. - Haematological parameters (haematocrit values - Htc, haemoglobin concentration Hb and red blood cells count - RBC) in the blood of rats after exposure to cadmium (Cd) compared with the controls

	Hct (L/L)	Hb (mmol/L)	RBC (10^{12}/L)
Controls	0.45 ± 0.06	8.24 ± 0.11	7.91 ± 0.21
Cd	0.41 ± 0.03 *	7.56 ± 0.10 *	5.11 ± 0.11 *

Means ± SEM from 7 animals in each group.
Significantly different from controls: * $p<0.05$

The concentration of glucose was increased in blood of rats after i.p. administration of cadmium ($p<0.05$), as well as significant increase of the activity of transaminases (ALT and AST) in the plasma ($p<0.05$) of cadmium exposed rats was alls shown in our experiments (Table 2).

Table 2. - Biochemical parameters values (concentration glucose in the blood, and the activities of alanin aminotransaminase - ALT and aspartat aminotransaminase - AST in the plasma) of rats after exposure to cadmium (Cd) compared with the controls

	Glucose (mmol/L)	ALT (U/mL)	AST (U/mL)
Controls	4.91 ± 0.13	14.84 ± 0.34	66.31 ± 1.86
Cd	6.41 ± 0.17 *	19.91 ± 0.27 *	94.93 ± 1.78 *

The results of our experiments show that the LP and GSH concentrations were significantly increased in the blood of rats after acute administration of Cd ($p<0.05$) (Table 3).

Table 3. - Concentration of lipid peroxides (LP) and glutathione (GSH) from whole blood of rats after exposure to cadmium (Cd) compared with the controls

	LP (nmol/ml)	GSH (nmol/g Hg)
Controls	1.21 ± 0.04	65.82 ± 6.59
Cd	2.35 ± 0.09 *	88.09 ± 7.52 *

Means ± SEM from 7 animals in each group.
Significantly different from controls: * $p<0.05$

DISCUSSION

Cadmium (Cd^{+2}), a potent toxic metal, is very harmful to the environment and to humans because of in vivo accumulation in liver, kidney and other tissues, causing metabolic, histological and pathological changes. Some evidence has suggested that cadmium induces a pro-oxidant state in tissues and cells exposed to it (WARREN *et al.*, 2000).

Our previous investigations showed that chronic treatment with Cd induced oxidative damage in erythrocytes of rats and goldfish, causing destruction of cell membrane and increase lipid peroxidation, as well as alteration of the AOS, energy metabolism and the appearance of anemia (KOSTIĆ *et al.* 1993, ŽIKIĆ *et al.* 1997, OGNJANOVIĆ *et al.* 2000, PAVLOVIĆ *et al.* 2001, ŽIKIĆ *et al.* 2001).

The results obtained in our study show that treatment with Cd induces anemia (decrease of RBCs count, Ht value and Hb concentration) in rats (Table 1). It is well known that the presence of Cd in organism decreases the level of iron in blood (KOSTIĆ *et al.* 1993) and causes the decrease of Hb concentration. The decrease of Ht value in hemolysed plasma of rats exposed to Cd indicates the increased destruction of erythrocytes (SHUKLA *et al.* 1996, HAMADA *et al.* 1998).

The results obtained in this work (Table 2) show that the concentration of blood glucose was significantly increased after acute administration of cadmium. The increased activity of transaminase enzymes (ALT, AST) in plasma

was also observed. These results are in accordance with results obtained in previous investigations and point to the damage of liver and disturbed carbohydrate and protein metabolism (CHAPATWALA *et al.*, 1980; RAJANNA *et al.*, 1984). Similar results were obtained in our previous investigations in rats after chronic treatment with cadmium (ŠTAJN *et al.*, 1993), where it was shown that cadmium caused hyperglycemia and proteinemia. In previous investigations it was also shown that cadmium increased the activity of transaminase enzymes (ALT, AST) in serum of rabbits (PISCATOR and AXELSSON, 1970) and in plasma of rats (RAJANNA *et al.*, 1984; ŠTAJN *et al.*, 1993). These enzymes have an important role in the processes of aminoacid and protein metabolism. It is known that these enzymes are widely spread in tissues, and that in normal conditions they show very low activity in serum (plasma). However, in stress condition and also due to the influence of different pollutants the damages of tissues occur, particularly in liver and heart, causing the liberation of transaminases into circulation, increasing their concentration and acticity (HENRY *et al.*, 1974).

The treatment with Cd increased lipid peroxidation (LP) contents in the blood, suggesting that the Cd induced oxidative stress (Table 2). Several studies have reported that lipid peroxidation resulting from oxidative damage is the primary mediator of cadmium toxicity ((SARKAR *et al.* 1998, STOHS *et al.* 2000). Indeed, free radical-induced peroxidative damage to membrane lipids has long been regardrd as a critical initiating event leading to cell injury (CHOW, 1991).

In comparison to the acute studies (CASALINO *et al.*, 2002; OGNJANOVIĆ *et al.*, 2003), two chronic studies that examined Cd-induced oxidative stress reported either no change in lipid peroxidation in either liver or kidney (SHAIKH *et al.*, 1999), or an increase in lipid peroxidation in the liver (WAISBERG *et al.*, 2003). In low or insignificant TBARS increase measured in chronic cadmium intoxication when antioxidant enzyme activity is reduced is probably due to the activation of other defence factors.

Oxidative stress is defined as a disturbance in the prooxidant-antioxidant balance in favor of the former and has been suggested to be a relevant factor in aging as well as in the pathogenesis of a number of injury and diseases states, such as heart attack, diabetes, and cancer (YU, 1994). Recently, it has been suggested that Cd^{+2} may induce oxidative damage in different tissues by enhancing lipid peroxidation and inhibiting the enzymes involved in the removal of certain activated oxygen species (STOHS and BAGCHI, 1995). The defense mechanisms the budy has against oxidative stress consist of enzymatic (e.g. SOD, CAT, GSH-Px, GST and GR) and nonenzymatic (e.g. glutathione, ascotbic acid, α-tocopherol, coenzyme Q, β-carotene and uric acid) components, as well as repair systems (HALLIWEL and GUTTERIDGE, 1999).

Other authors have reported that exposure to cadmium, in addition to causing lipid peroxidation, stimulated endogenous defences such as increases in antioxidant enzyme activities and in liver glutathione concentration (GUPTA *et al.*, 1998; SHAIKH *et al.*, 1999). It is known that Cd induces oxidative stress by producing superoxide anions and nitric oxide (KOIZUMI *et al.*, 1996) and

it is reasonable to expect an increased activity of SOD (KOSTIĆ *et al.* 1993a, SARKAR *et al.* 1998).

The nonenzymatic antioxidant glutathione of the blood of control and experimental group is shown in Table 3. The obtained results show that GSH content in rats acutally exposed to Cd was significantly increased ($p<0.05$) Acute Cd-induced toxicity may be due to the exhaustion of GSH stores and the increase in oxidative stress (RANA and VERMA, 1996). Glutathione is the most abundant low-molecular weight thiol in mammalian cells. As such, it plays a mayor role in cellular detoxification processes by providing the cell with multiple defenses against reactive oxygen species and also against their toxic products. GSH also has a high affinity for heavy metals and, as a result, constitutes the first line of defense against Cd toxicity (SINGHAL *et al.*, 1987). GSH is an antioxidant and can also form complexes with Cd to altre Cd distribution and excretion (RANA and VERMA, 1996). CHEN and CHERIAN (1992) have demonstrated that cadmium produced dose- and time-dependent increases in intracellular glutathione concentration. As for other heavy metals, oxidative strass by mercury and lead (STOHS *et al.*, 2000) stimulated glutathione synthesis in kidney and liver, respectively. Several protective agents, including glutathione and metallothionein, as well as vitamin E, play an important role in detoxification of endogenous and exogenous compounds (OGNJANOVIĆ *et al.*, 2003).

CONCLUSIONS

Our results suggest that cadmium induced oxidative stress has an important role in the pathogenesis of anemia. Cadmium induces oxidative injuries in erythrocytes, their destruction and haemolysis, decreases haematocrite values, haemoglobin concentration and total red blood cell count. These parameters can be taken as sensitive indicators of cadmium toxicity. Cadmium causes alterations in carbohydrate and protein metabolism demonstrated as hyperglycemia and increased activity of transamonases in plasma. From presented results it can be concluded that Cd induced oxidative damage in erythrocytes causes appearance of anemia, loss of membrane function by enhancement of LP concentration as well as alteration of the concentration of GSH in rat blood.

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KADMIJUMOM INDUKOVANE PROMENE U HEMATO-BIOHEMIJSKIM PARAMETRIMA, LIPIDNOJ PEROKSIDACIJI I SADRŽAJU GLUTATIONA U KRVI PACOVA

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I z v o d

Kadmijum (Cd^{+2}) je široko rasprostranjen toksičan metal koji može izazvati oksidaciona oštećenja u krvi remeteći prooksidaciono-antioksidacionu ravnotežu. *Wistar* albino pacovi (stari 3 meseca), tretirani su pojedinačnim dozama $CdCl_2$ (0.4 mg Cd/kg i.p.) i žrtvovani posle 24 časa. Hematološki parametri, i to: broj eritrocita (RBCs), hematokritska vrednost (Ht) i koncentracija hemoglobina (Hb) značajno su smanjeni u krvi tretiranih pacova. Aktivnost enzima alanin aminotransaminaze (ALT) i aspartat aminotransaminaze (AST) u plazmi, kao i koncentracija glukoze u krvi značajno su povećani kod životinja tretiranih kadmijumom u poredjenju sa kontrolnim vrednostima. Tretman sa kadmijumom, takodje je doveo do povećanja lipidne peroksidacije (LP) i redukovanog glutationa (GSH). Sve to ukazuje da je kadmijum izazvao oksidacioni stres u krvi pacova. Dobijeni rezultati ukazuju da je Cd indukovao promene u hemato-biohemijskim parametrima, kao i u sadžaju LP i GSH u krvi tretiranih pacova.

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