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INFLUENCE OF THE ORGANOSELENIUM COMPOUND SELENOPYRAN ON THE ANTIOXIDANT SYSTEM OF LABORATORY ANIMALS WITH TOXICOSIS CAUSED BY CADMIUM COMPOUNDS

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Abstract

The article investigates the effect of an organoselenium compound on the antioxidant status of the body of laboratory animals when they are administered a cadmium compound. Cadmium compounds are widely known eco-pollutants, the toxic effect of which is due to their ability to stimulate free radical processes in the body of animals and humans. The purpose of this work was to identify aspects of cadmium toxicity due to its thiol specificity and features of the short-term adaptation of the antioxidant system to the administration of a toxicant in rats at the stages of ontogenesis. At the same time, the effectiveness of the organic selenium-containing compound was evaluated. In the tissues, the accumulation of lipid peroxidation products, as well as the content of selenium in the blood plasma, liver, and testicles of animals, were evaluated. In the course of the research, it was found that the prophylactic administration of selenopyran to experimental animals reduced the manifestation of toxicosis caused by cadmium. A similar effect of the drug is due to a decrease in the degree of damage to cell membranes and intracellular structures by free radical oxidation products.

Key words*: cadmium, diene conjugates, free radical oxidation, glutathione peroxidase, glutathione reductase, malondialdehyde, selenopyran, selenoprotein, superoxide dismutase.*

INTRODUCTION

In the formation of resistance to free radical damage, an important role belongs to the functioning of the enzymatic link of antioxidant protection. In turn, the structure of antioxidant enzymes provides for the presence of a large number of sulfhydryl groups, which determine both the spatial conformation of proteins and which are part of the active center.

Cadmium is a heavy metal capable of rapidly reacting with sulfhydryl groups in an aqueous medium, as well as replacing zinc in the active sites of enzymes. Cadmium compounds are widely known eco-pollutants, the toxic effect of which is due to their ability to stimulate free radical processes in the body of animals and humans (Voloshin et al., 2010; Gavryushina et al., 2021).

It is known that the microelement selenium largely mitigates the toxic effects of cadmium (Boryaev et al., 2015; Kravchenko et al., 2007).

The purpose of this work was to identify aspects of cadmium toxicity due to its thiol specificity and features of the short-term adaptation of the antioxidant system to the administration of a toxicant in rats at the stages of ontogenesis. At the same time, the effectiveness of the organic selenium-containing compound selenopyran (SP), 9-phenyl-symmetric octahydroselenoxanthene, was evaluated.

MATERIALS AND METHODS

The experiment was carried out on male rats of the Wistar breed. Three groups of animals were formed, receiving an isocaloric semi-synthetic diet. All groups of animals consisted of 2 subgroups: adults of sexual maturity (age over 90 days) and young adults in the period of puberty (age from 56 to 63 days).

The first group is the control. In the last 4 days of the experiment, the animals of the second group were intragastrically injected with an aqueous solution of $CdCl_2$ at a dose of 4 mg/kg of live weight.

Animals of the third group within 7 days intragastrically received an oily solution of selenopyran at a dose of 0.04 mg/kg of live weight, and $CdCl_2$ was administered similarly to the second group, against the background of the introduction of selenopyran.

In tissues (blood, liver, intestinal mucosa, myocardium, aorta), the accumulation of lipid peroxidation products was assessed by the content of diene conjugates (DC) and malondialdehyde (MDA), the activity of the main antioxidant defense enzymes (superoxide dismutase - SOD, glutathione peroxidase - GPO and glutathione reductase - GR) . The content of selenium in the blood plasma, liver and testicles of animals was studied.

Biometric processing of research results was carried out by the method of variation statistics for the reliability of the difference between the compared indicators using the Microsoft Office Excel software.

RESULTS AND DISCUSSIONS

The introduction of cadmium affected the function of the main life support systems of the body. Kidney damage was detected (in young and adult animals, the plasma creatinine content increased two times compared to the control group), and in the animals of the third group, this figure did not exceed the control value.

A significant increase in the activity of aminotransferases in the second group (by 30%) and bilirubin indicated liver damage, and in young animals to a greater extent than in adults. Selenopyran did not prevent an increase in alanine aminotransferase activity, however, aspartate aminotransferase activity and plasma bilirubin concentration remained at the control level.

In young animals, the accumulation of diene conjugates with the introduction of cadmium significantly increased in plasma (6.15±0.292 nmol/ml) and liver (72.2±0.86 nmol/g) compared with the control group (4.31±0.439 and 64.1±2.37, respectively).

In the second group, the administration of cadmium increased the content of malondialdehyde in the liver and aorta by 30 and 50%, respectively.

The introduction of selenopyran did not significantly reduce the degree of accumulation of diene conjugates in the blood and tissues.

In adult animals, the accumulation of diene conjugates in plasma, erythrocytes, liver, and intestines significantly decreased (compared to the control group) with cadmium administration, but tended to increase in the aorta and myocardium.

The accumulation of malondialdehyde in adult animals, both with the introduction of only cadmium, and against the background of selenopyran, significantly increased only in the myocardium (compared to the control).

The activity of antioxidant defense enzymes under cadmium load changed ambiguously.

Thus, the activity of glutathione reductase (Figure 1) in young animals remained at the control level in erythrocytes, myocardium and aorta, and significantly decreased in the liver and intestines, while in adults there was a significant decrease in the activity of the enzyme in the aorta (three times compared with the control), while no changes were observed in other organs.

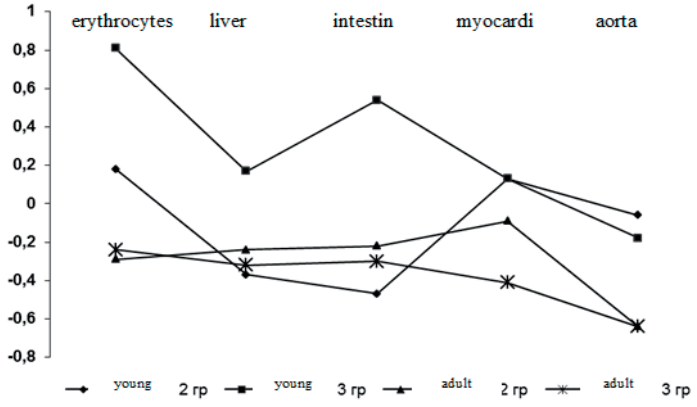

Figure 1. Changes in the activity of glutathione reductase in the organs of young and adult rats with experimental cadmium toxicosis (relative units to control values)

In young animals with toxicosis caused by cadmium, there was an increase in the activity of superoxide dismutase in the intestine, while in adults - in the intestine, liver and aorta, and in erythrocytes there was a significant decrease compared to the control.

The introduction of selenopyran against the background of cadmium loading led to an increase in the activity of glutathione reductase (Figure 1) in the liver and erythrocytes in young animals, and in adult rats in the myocardium and aorta. In adult animals, an increase in the activity of superoxide dismutase in erythrocytes and a decrease in this indicator in the intestine and myocardium were found compared with the second group.

The activity of glutathione peroxidase (Figure 2) in young animals of the second group in the liver and intestines decreased significantly in relation to the control, and increased in the myocardium.

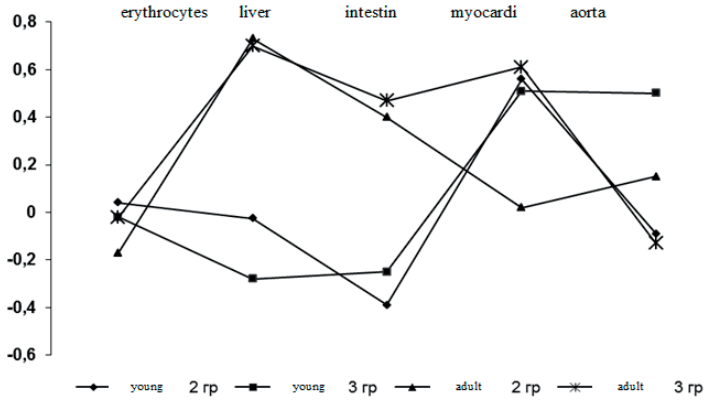

Figure 2. Changes in the activity of glutathione peroxidase in the organs of young and adult rats with experimental cadmium toxicosis (relative units to control values)

On the contrary, adult animals showed a significant increase in enzyme activity in the liver and intestines, and no changes in other tissues. The activity of this enzyme in adult and young animals correlated with changes in the content of selenium in the liver - a decrease in the concentration of selenium in the liver in young animals, and an increase in adults (Table 1).

Table 1. The content of selenium in the organs of young and adult rats with experimental cadmium toxicosis

Groups	Plasma, ng/ml		Liver, ng/mg		Testicles, ng/mg	
	young	adults	young	adults	young	adults
1	185±16.3	400±24.5	0.912±0.077	0.562±0.042	0.081±0.009	0.082±0.003
2	182±11.5	375±30.7	0.488*±0.068	0.635±0.065	0.061*±0.003	0.117*±0.006
3	287*±25.4	369±23.3	0.819*±0.118	0.698*±0.044	0.090*±0.007	0.147±0.036

* - differences with control are significant P<0.05

It is known that glutathione peroxidase is the main selenoprotein in the liver of mammals; therefore, the observed difference in enzyme activity is probably associated with its quantitative content in cells, and not with functional damage to macromolecules. The use of selenopyran against the background of a cadmium load increased the content of selenium in the liver in young individuals to the level of control animals, and in adults - by 24.2%, compared with the control, which did not correlate with changes in the activity of glutathione peroxidase. Apparently, in this case, slow metabolization of selenopyran in liver cells takes place, which corresponds to the short duration of the experiment, or its deposition, since the release of selenium from the molecule would lead to an increase in the synthesis of selenoproteins.

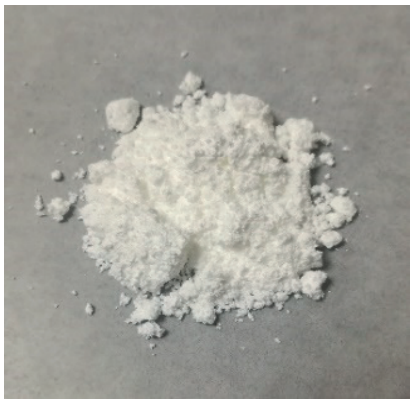

Figure 3. Cadmium hydroxid

It is known that the testes are the target organs of toxicosis caused by cadmium (Figure 3), and cadmium significantly affects the content of the trace element selenium in them (Table 1), the most important component of enzymatic antioxidant protection. At the same time, immature and adult individuals react differently. If the concentration of selenium in young individuals under a cadmium load in the testicles sharply decreased (by 25%), then in adults the opposite picture was observed (increase by more than 40%).

With the introduction of selenopyran, both in young and adult animals, the content of selenium in the testes increased, and this contributed to a decrease in the toxic effect of cadmium.

CONCLUSIONS

Thus, the prophylactic administration of selenopyran to experimental animals reduced the manifestation of toxicosis caused by cadmium.

A similar effect of the drug is apparently due to a decrease in the degree of damage to cell membranes and intracellular structures by free radical oxidation products.

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