

# LIPID PEROXIDATION IN THE CORRECTION OF THEIR PREPARATION FIRUTAS

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## Abstract

One of the important aspects of studying the pathogenesis of various diseases is the study of the state of lipid peroxidation and the antioxidant system. The disruption of the stationary state of free-radical oxidation is considered one of the universal, non-specific indicators of damage and is characteristic of a wide range of diseases, including atherosclerosis, stress, neuroses, diabetes mellitus, inflammatory processes, autoimmune diseases, and others. (3,5,6,8,9,10).

**Keywords:** Experimental hepatitis with carbon tetrachloride administration, lipid peroxidation processes, lipid peroxidation products – diene conjugates, dien ketones, malondialdehyde, antioxidant system, toxic liver damage, treatment with phytin preparations.

## Introduction

### Research objective

The aim of the present work is to study the specific features of changes in lipid peroxidation and antioxidant system in experimental pathology and the mechanisms of pharmacological correction of these changes by domestic drugs.

When modeling pathological conditions by introducing various xenobiotics, the toxic damage of the liver with carbon tetrachloride, specific links in the violation of lipid peroxidation and the antioxidant system in the blood, liver of experimental animals were revealed.

The pattern of accumulation of primary products of lipid peroxidation, diene conjugates of unsaturated fatty acids, in organs and tissues with the highest content in the blood under the damaging effect of xenobiotics has been established. The organotropism of the accumulation of TBA-reactive products of lipid peroxidation has been revealed: the highest content of these products was found in liver tissues under the toxic effect of carbon tetrachloride.

A regular relationship between changes in lipid peroxidation (LPO) and the antioxidant system (AS) under the influence of hepatoprotectors has been established, resulting in a decrease in the content of LPO products and an increase in the activity of AS enzymes.

### Material and methods of research

The main objective of the conducted studies was to study the effect of the combined preparation of phytin – firtas in comparison with silibor on the processes of lipid peroxidation in experimental hepatitis. Hepatitis was reproduced in mongrel white rats by the introduction of



hepatotropic poison – carbon tetrachloride. Carbon tetrachloride is widely known as a hepatotoxic poison, causing liver damage in all the variety of its clinical, biochemical and morphological changes. The effects of the drugs were studied in comparison with the most commonly used bioflavonoid, silibor.

The effect of firutas and silibor on the parameters of the products of primary lipid oxidation – diene conjugates and dienketones in experimental hepatitis. Carbon tetrachloride  $\text{CCl}_4$  in the control series of experiments was subcutaneously injected in a volume of 0.8 ml per 100 g of animal weight for 4 days.

In the third and fourth series, the animals were orally administered 100 mg/kg of Firutas and 100 mg/kg of Silibor through a probe simultaneously with the hepatotropic poison for 10 days. The concentration of conjugated dienes and dien ketones was measured using a SF-46 spectrophotometer, respectively, at 232 and 273 nm. For this purpose, a lipid extract was prepared in a mixture of H-heptane-isopropyl alcohol (1:1) at a concentration of 0.5 mg/ml. The amount of peroxides was expressed in terms of optical density (D) per 1 mg of lipids.

The content of secondary products of lipid peroxidation, malondialdehyde, was determined using the method of I.D. Stalnaya and T.G. Garishvili (133) with the help of thiobarbituric acid (12).

The results of the studies showed that the toxic effect of carbon tetrachloride on the liver tissue of the control group of rats caused an increase in the processes of lipid peroxidation. In the control group, the concentration of conjugated dienes and dien ketones in the liver tissue increased by 34.7% and 37%, respectively, on the 10th day of the study. In the second treatment group, on the 14th day of the experiment, the concentration of primary lipid peroxidation products remained consistently high in the liver tissue of the control group.

These shifts, in turn, lead to an increase in the formation of free radical products of lipid peroxidation, which can contribute to the further progression of biological membranes and ultimately lead to the cytolysis of hepatocytes. The administration of the studied drugs simultaneously with carbon tetrachloride significantly reduced the levels of lipid peroxidation and prevented the pro-oxidant effects of the hepatotoxic poison.

In the treatment and prevention group of experiments, the administration of Firutas at a dose of 100 mg/kg simultaneously with carbon tetrachloride prevented the intensification of free radical lipid peroxidation processes and the increase in the concentration of primary products, such as diene conjugates, in the liver tissue. When Firutas was administered at a dose of 100 mg/kg, there was a decrease in the levels of dienes and dien ketones in the liver tissue by 18.9% and 30.2%, respectively, compared to the control group. In the therapeutic series of experiments (on the 14th day of the experiments), a dose of 100 mg/kg of Firutas caused a 22.1% decrease in the content of dienes and a 36.1% decrease in the content of dien ketones.

- **Effect of Firutas and Silibor on the content of conjugated dienes and dienketones in liver tissue in experimental hepatitis ( $M \pm m$ ). № Conditions of the experiment p Treatment and preventive group Treatment group Liver tissue D per/mg of lipid Liver tissue D per/mg of lipid Conjugated dienes Conjugated dienketones Conjugated dienes Conjugated dienketones**



№	Terms of the experience	N	Treatment and prevention group		Treatment group	
			Liver tissue D per mg of lipid		Liver tissue D per mg of lipid	
			Conjugated dienes	Conjugated dien ketones	Conjugated dienes	Conjugated dien ketones
1	Intact	8	1,180 ± 0,02 (1,160 – 1,200)	0,460 ± 0,02 (0,440 – 0,480)	0,873 ± 0,009 (0,864 – 0,882)	0,234 ± 0,008 (0,226 – 0,242)
2	Control, CCl4	7	1,590 ± 0,03 (1,560 – 1,620)	0,630 ± 0,018 (0,612 – 0,648)	1,309 ± 0,014 (1,295 – 1,323)	0,617 ± 0,02 (0,597 – 0,637)
3	Firutas, 100 mg/kg	8	1,290 ± 0,015 (1,275 – 1,305)	0,440* ± 0,012 (0,428 – 0,452)	1,020 ± 0,013 (1,007 – 1,033)	0,394 ± 0,012 (0,382 – 0,406)
4	Silibor, 100 mg/kg	8	1,320 ± 0,011 (1,309 – 1,331)	0,480 ± 0,016 (0,464 – 0,496)	1,073 ± 0,008 (1,065 – 1,081)	0,445 ± 0,014 (0,431 – 0,459)

- the results are not reliable for series 1 Thus, the results of the conducted studies showed that in the series of control experiments, the indicators of lipid peroxidation on the 14th day of the study were less pronounced than on the 10th day of the experiment. This suggests that on the 10th day of the experiment, there is a peak of clinical and biochemical manifestations, which then significantly decrease by the 14th day. The drug Firtas, which we studied, at a dose of 100 mg/kg, significantly reduced the content of conjugated dienes and dien ketones in the liver tissue, which indicates its antioxidant effect. The effectiveness of the drugs was clearly demonstrated when they were administered earlier, suggesting that prophylactic administration of the drugs prevents the pro-oxidant effects of carbon tetrachloride. Firtas showed a similar antioxidant effect to Silibor.

- Therefore, the search for new drugs, the identification of the hepatotropic properties of existing drugs, and the study of their effects on lipid peroxidation processes are of great importance for the rational pharmacotherapy of hepatitis of various etiologies.

### Conclusions

The coordination drug Firutas, in comparison with Silibor, reduces the content of conjugated dienes and dien ketones, malondialdehyde, and its fraction in liver tissue, which indicates the inhibition of lipid peroxidation processes and the antioxidant effect of the drugs. Firutas is superior to Silibor. In the treatment and prevention group, the drugs had a more pronounced hepatoprotective effect than in the treatment group.

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