IJDIAS International Journal of Discoveries and Innovations in Applied Sciences

| e-ISSN: 2792-3983 | www.openaccessjournals.eu | Volume: 1 Issue: 6

Peculiarities of the Action of Fosmetrine on Bioenergetic Processes in Mitochondria and Lipid Metabolism in RAT Liver

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Abstract:

Administration of 440 g/l (0.2 ml per 100 g body weight intraperitoneally) to male rats leads to a significant decrease in respiration rate of liver mitochondria in the 3rd metabolic state and to some activation in the 2nd metabolic state after 2 h of poisoning with their further partial normalization by 20 h. A decrease in the efficiency of oxidative phosphorylation of ADP in mitochondria correlates with the dynamics of phospholipid and β-lipoprotein content in the liver. Disturbances of bioenergetic processes in mitochondria precede the accumulation of total lipids in the liver. The decrease of cholesterol content in liver tissue is the result of disruption of its synthesis in the membranes of endoplasmic reticulum.

Keywords: Phosphomethrin, pesticides, mitochondria, liver, total lipids, phospholipids.

Introduction: Numerous researches directed on studying influence of industrial chemical and physical factors on an organism of the workers , early diagnostics of pathological processes and development of therapeutic and prophylactic methods are not reflected in works of domestic, foreign researches, devoted to studying mechanisms of formation and progression of liver lesion.

The present work was aimed at determining the relationship between the state of respiration and oxidative phosphorylation in liver mitochondria and parameters of fat metabolism in liver and blood serum during acute poisoning of male rats by Fosmetrin .

Aim of the investigation: the peculiarities of fosmethrin action on bioenergetic processes in mitochondrion and lipid metabolism in rat liver

Materials and methods of investigation: Rats were kept on a usual vivarium diet, fosmetrine was injected intraperitoneally in a dose of 0.2 ml per 100 g body weight. The investigation was performed 2 and 20 h after poisoning. Liver mitochondria were isolated by differential centrifugation according to a conventional scheme. Respiration rate and oxidative phosphorylation were studied by polarographic method. Total lipids were determined by turbidimetric method phospholipids by the amount of inorganic

ISSN 2792-3983 (online), Published under Volume: 1 Issue: 6 in November-2021

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phosphorus in lipid extract mineralizate; liver cholesterol, liver β-lipoproteins and blood serum were also studied.

Results of the study. The response of the liver mitochondrial respiratory chain to acute poisoning by Fosmethrin depends on the metabolic state of mitochondria. After pesticide injection (Table 1), oxygen uptake is slightly increased in the 4 metabolic state (controlled state) characterized by ADP deficiency. At the same time, mitochondria respiration rate in metabolic 3 state (active respiration state), accompanied by phosphorylation of ADP, was reduced more than 2 times (by 55 % compared with control) in 2 hours after administration of pesticide Fosmetrine and slightly restored in 20 hours (73 % of control).

The respiratory control value and the rate of ADP phosphorylation in rat liver mitochondria were significantly reduced 2 h after Fosmetrine administration and recovered slightly after 20 h.

The ADP:0 ratio characterizing the number of conjugation sites for phosphorylating succinate oxidation was decreased by one in the studied period after poisoning, indicating the disruption of ADP phosphorylation in one of the conjugation sites located after flavoprotein, probably, between cytochromes B and C (Sadikov A.U., Khamrakulova M.A.).

Damage of liver mitochondria in toxic hepatitis is accompanied by distinct changes in liver and serum lipid fractions. The content of total lipids in the liver (Table 2) increases 20 h after poisoning by 82% compared with control. As the content of phospholipids, cholesterol and β-lipoproteins in the liver is less than normal, the increase in total lipids can be attributed to the accumulation of neutral fats. Consequently, lipid accumulation in the liver is observed much later than mitochondrial damage. The content of phospholipids in the liver decreased after 2 hours after Fosmetrin injection by 28% and slightly normalized after 20 hours, although it remained lower (by 10%) than the control level. Decrease in quantity of phospholipids in the liver could not be a consequence of their increased excretion in the blood, because phospholipid level in blood serum didn't increase (12,8±1,1 vs. 14,9±1,0 mg% in control), but it could be explained by inhibition of phospholipid synthesis in the liver.

Table 1. Effect of Fosmetrin on respiration and oxidative phosphorylation in liver mitochondria of white rats (oxidation substrate - succinate) M ± m

Nature of experience	Respiratory rate, mcatoms 0/mg/min		Phospho-rylation rate, μmol ADF min	Breathing control	ADF : 0
	State 3	State 4			
Control	121,4±4,8*	39,4±1,0	236,5±12,3	3,0±0,2	2,0±0,0
Fosmethrin 2 h	55,2±5,5***	44,9±1,8 P < 0,05	57,5±5,1 P <0,001	1,2±0,1 P < 0,001	1,1±0,1 P < 0,001
Fosmethrin 20 h	86,4±16,3 4 P > 0,05	45,5±3,1 4 P > 0,05	99,5±30,0 4 P > 0,01	1,9±0,3 4 P < 0,05	1,1±0,1 4 P < 0,001

Table 2. Effect of Fosmetrine on the content of lipid fractions in the liver, M ± m

Indicator	Monitoring		Fosmethrin poisoning			
	mg/g liver	% total lipids	2 hours		20 hours	
			mg/g liver	% total lipids	mg/g liver	% total lipids

ISSN 2792-3983 (online), Published under Volume: 1 Issue: 6 in November-2021

Total lipids	60,3±2,9	100	54,9±2,8 P>0,2	100	109,7±11,6 P<0,001	100
Phospholipids	1,44±0,06	2,33±0,11	1,09±0,07 P<0,01	1,99±0,11 P>0,05	1,23±0,06 P<0,05	1,27±0,16 P<0,001
β-lipoproteins	0,79±0,45	1,26±0,66	0,37±0,25 P<0,01	0,75±0,46 P>0,05	0,97±0,57 P<0,3	0,97±0,58 P>0,2
cholesterol	4,95±0,18	8,55±0,54	4,32±0,18 P<0,05	7,92±0,18 P>0,3	4,05±0,36 P<0,05	3,96±0,45 P<0,001

The amount of β-lipoproteins in the liver decreases significantly at 2 h after poisoning and is completely restored after 20 h. However, serum β-lipoprotein concentration after 2 and 20 h was significantly elevated (by 170 and 162% respectively compared to control), which is inconsistent with the notion of impaired hepatic lipoprotein excretion under conditions of toxic hepatitis.

Conclusions.

1. The poisoning of rats with Fosmetrine was accompanied by a progressive reduction of liver cholesterol. The main part of liver cholesterol is contained in microsomal fraction, therefore, it is likely that the decrease of cholesterol content in the rat liver is due to damage of endoplasmic reticulum membranes by Fosmetrine.
2. The results obtained testify to a possible link between disorders of bioenergetic processes in mitochondria, damage of endoplasmic reticulum membranes and changes of lipid exchange in the liver in acute poisoning with pesticide Fosmetrin.

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ISSN 2792-3983 (online), Published under Volume: 1 Issue: 6 in November-2021