

How to Cite:

Ikramova, M. M., Topilova F. M., & Tojiboev, Q. T. (2022). The effect of herbal tea on the energy metabolism of mitochondria in liver damage. *International Journal of Health Sciences*, 6(S1), 4447–4453. <https://doi.org/10.53730/ijhs.v6nS1.5917>

The Effect of Herbal Tea on the Energy Metabolism of Mitochondria in Liver Damage

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Abstract---In our scientific work, the effect of the tincture “world of phytochoyi”, made from natural herbs, which has a high efficiency, on the energy metabolism of mitochondria, was observed in the case of injury to the liver of toxic hepatitis animals. Inflammation of the liver depends on the chemical structure of the hepatotoxic substance, the period and amount of exposure. The deposition of geliotrin in the seeds of ostrich (*Heliotrorium lasiosarum*) into the grains of the spike plant calls for toxic hepatitis. In the conducted studies, it was found that the tincture “world of phytochoyi” brings the energy metabolism in the liver mitochondria of animals infected with heliotrin hepatitis closer to the indicator in the liver mitochondria of healthy animals.

Keywords---Mitochondria, oxidative phosphorylation, tissue, geliotrin, metabolism.

Introduction

The respiratory and oxidative phosphorylation system in mitochondria is one of the most important factors determining the energy state of the adenine nucleotides of Huayra. When analyzing the oxidative strength of mitochondria, we first drew attention to their description, which corresponds to a certain activity of tissue. In a calm state, the main part of the mitochondria in the activity of mitochondria in the conditions of in vivo is in the 4 state of the Chans. This condition means that mitochondria are well supplied with oxygen and substrates. But in this case, the respiratory activity will be reduced, since it is associated with the phosphorylation process, in tissues in a calm state, the main background of adenine nucleotides used in the transport of intracellular energy will be in the form of ATP.

An increase in the activity of the cell leads to an increase in energy expenditure and ATF to paralysis. The formation of a phosphate akceptor in the form of an ADF leads to an increase in the respiratory activity of mitochondria (respiratory control), and this process continues until the cell consumes the energy of its macroergic phosphorous bonds and does not bring ADF to mitochondria. This same process can be checked by the method of polyarography in mitochondria, which are separated from the tissues. Bunda is learned from the sharp acceleration of breathing of mitochondria when a certain amount of ADF is added to the measuring environment. Each added ADF is taken into account by the time it goes to its phosphorescent oxidation.

With complete addition of phosphate actin to mitochondria, mitochondria passes from a phosphorylated oxidation state (V3) to a state of peace, that is, a state of slowing breathing (V4). The ratio of the phosphorylated oxidation state to the oxidation state indicates the normal operating state of the mitochondria, and it is called the respiratory coefficient according to the Chans (Nokch V3/V4). One of the indicators that determines the state of activity of mitochondria is the coefficient of ADF/o. The higher the size of ADF/o, the less oxygen is expended on forrestation, respectively, the higher the useful coefficient of action of mitochondria in terms of Energy Reserve in intracellular metabolic processes

Materials and Methods

Between the skins of animals, a solution of 5 mg of geliotrin in a physiologic solution was administered once a week for two months, relative to the weight of 100 g. The tincture "world phytochoyi", prepared from medicinal herbs growing in Central Asia by the people's Medical Center "Zeynul" under the Institute of plant chemistry of the Academy of Sciences of Uzbekistan, was given with food from 7 g per kg of weight per day for a month during the last month of geliotrin administration. Essentsiale forte was sent under the skin from 7 mg.

Respiratory and oxidative phosphorylation of mitochondria was determined by the polyarography method. To do this, 1 ml of measuring media (sucrose 250 mm, KCl 50 mm, KH_2PO_4 5 mm, pH 7,4) is poured into the polyarography ontainer and one of the oxidation substrates (suctsinate from 10 mm, or glutamate) is added on it. After it, mitochondria (0,05-0,06 ml; 3-4 mg of whitewash) are added to the polyarography yacht. Mitochondria begin to breathe (v2 state), that is, mitochondria begin to consume oxygen in the measuring environment, between 30-40 seconds, 200 μm ADF was placed on the polyargraphy collar, as a result of which suddenly the breathing of mitochondria sharply accelerated (V3 state). This means that ADF added to mitochondria begins to form ATF by joining with Fn in the measurement medium, and this is called an oxidized phosphorylated state.

Results and Discussions

We the phosphorylation of glutamate (V₃) slightly decreased (11,7%), peaceful oxidation (V₄) increased by 55,4% in 0,5 months after the introduction of Geliotrin into the body, resulting in a decrease in the concentration of Chans boyichv breathing by 43,3%, the coefficient of ADF/O decreased by 9,9%. In mitochondria under the influence of heliotrin 4 oxidation of the metabolic state is evidence of a

violation of the activity of the cell in a calm state, that is, a decrease in the synthesis of ATF and a decrease in heat dissociation. The oxidation of glutamate (V_{DNF}) under the action of Dinitrophenol decreased by 16,9%. This allows electrons to be seen from NAD-related substrates as they decrease their transfer of molecular oxygen across the respiratory chain. However, serious changes in the oxidation of succinate in mitochondria in various metabolic states of the animal liver, which received heliotrin in these conditions, were practically not observed.

Table 1
Changes in energy metabolism in liver mitochondria, injured by heliotrin
($m \pm m$; $n = 10$)

Pointers	Respiratory rate, nanogrammm atom O_2 / minute mg protein					
	Glutamate			Succinate		
	Healthy Animals	With Hepatitis Animals	%	Healthy Animals	With Hepatitis Animals	%
Duration of the experiment: 0,5-month						
V_2	13,2 $\pm$ 1,1	19,3 $\pm$ 1,5	146,2	29,2 $\pm$ 3,0	32,2 $\pm$ 2,8	110,3
V_3	52,3 $\pm$ 3,1	46,2 $\pm$ 2,7	88,3	105,5 $\pm$ 4,1	102,0 $\pm$ 3,8	96,7
V_4	12,8 $\pm$ 1,2	19,9 $\pm$ 1,9****	155,4	30,2 $\pm$ 3,2	32,4 $\pm$ 3,0	107,3
V_{DNF}	77,7 $\pm$ 4,1	64,6 $\pm$ 3,8	83,1	220,5 $\pm$ 5,6	219,6 $\pm$ 5,2	99,6
Respiration rate _{CH}	4,09 $\pm$ 0,17	2,32 $\pm$ 0,10***	56,7	3,49 $\pm$ 0,13	3,14 $\pm$ 0,20	90,0
ADF/0	2,84 $\pm$ 0,11	2,56 $\pm$ 0,09	90,1	1,90 $\pm$ 0,009	1,86 $\pm$ 0,08	97,9
1-month						
V_2	12,5 $\pm$ 1,2	18,8 $\pm$ 1,6	150,4	29,4 $\pm$ 3,0	30,0 $\pm$ 3,1	102,0
V_3	52,0 $\pm$ 3,3	43,4 $\pm$ 3,0*	83,4	108,6 $\pm$ 5,2	96,4 $\pm$ 4,2	88,7
V_4	12,7 $\pm$ 1,4	19,0 $\pm$ 1,7****	149,6	29,7 $\pm$ 3,4	30,2 $\pm$ 3,4	101,7
V_{DNF}	75,9 $\pm$ 4,4	61,6 $\pm$ 3,7***	81,1	218,8 $\pm$ 5,3	208,8 $\pm$ 4,6	95,4
Respiration rate _{CH}	4,09 $\pm$ 0,14	2,29 $\pm$ 0,12***	56,0	3,65 $\pm$ 0,12	3,21 $\pm$ 0,10	87,9
ADF/0	2,86 $\pm$ 0,12	2,50 $\pm$ 0,10*	87,4	1,88 $\pm$ 0,08	1,72 $\pm$ 0,09	91,5
1,5 – month						
V_2	12,8 $\pm$ 1,6	18,6 $\pm$ 2,6	145,3	29,2 $\pm$ 2,2	30,9 $\pm$ 2,5	105,8
V_3	51,4 $\pm$ 2,9	41,0 $\pm$ 2,1**	79,7	103,0 $\pm$ 3,4	87,0 $\pm$ 3,3**	84,4
V_4	13,1 $\pm$ 1,0	18,5 $\pm$ 2,9****	141,2	29,0 $\pm$ 2,3	31,0 $\pm$ 2,7	106,9
V_{DNF}	74,3 $\pm$ 3,7	58,7 $\pm$ 3,7**	79,0	210,6 $\pm$ 4,7	198,0 $\pm$ 3,8*	94,0
Respiration rate _{CH}	3,92 $\pm$ 0,10	2,22 $\pm$ 0,22***	56,6	3,54 $\pm$ 0,12	2,80 $\pm$ 0,13***	79,1
ADF/0	2,78 $\pm$ 0,0	2,48 $\pm$ 0,11**	89,2	1,86 $\pm$ 0,07	1,63 $\pm$ 0,07*	87,6

	9					
2 – month						
V ₂	12,4±2,0	17,2±2,0	138,7	28,5±2,3	27,2±2,4	95,4
V ₃	51,9±4,4	37,8±2,3**	72,8	102,2±4,1	69,9±3,1***	68,4
V ₄	12,5±2,2	17,4±2,1*	139,2	28,7±2,4	27,3±2,6	95,1
V _{DNF}	76,1±4,9	55,0±3,6**	72,3	214,0±6,8	158,6±4,0****	74,1
Respiration rate _{CH}	4,14±0,13	2,17±0,12***	52,4	3,56±0,13	2,57±0,14****	72,2
ADF/O	2,80±0,10	2,26±0,09**	80,7	1,85±0,07	1,50±0,06****	81,1

The continuation of the introduction of heliotrin into the body led to the acceleration of violations of energy metabolism in the mitochondria of the liver. As for the 1 month of the experiment, the phosphorylated oxidation of glutamate in liver mitochondria decreased by 16,6%, while the oxidation of V₄ status increased by 1,5%, the respiratory coefficient and ADF/o coefficient by Chance decreased by 1,44 and 12,6%. The decrease in oxidation under the action of Dinitrophenol reached 18,9%. By this period, the phosphorylated oxidation of succinate in the mercury mitochondria of animals receiving heliotrin also decreased by 11,3%, the respiratory coefficient by chance by 12,1%, the coefficient of ADF/o by 8,5%. So by this period, the heliotrin began to show its negative effect on the phosphorylated oxidation of succinate, which is much more resistant to various effects on the mitochondria of the liver.

Changes in glutamate oxidation in liver mitochondria when going to 1,5 months of the experiment remained at the level of the changes observed in the previous 1 month: a decrease in phosphorylated oxidation was 20,3%, an increase in oxidation in the V₄ state was 41,2%, a decrease in the respiratory index and ADF/o coefficient according to the By this period, the changes in the oxidation of succinate were slightly accelerated, that is, the oxidation of phosphorylates, the decrease in the respiratory index and the coefficient of ADF/o by chance reached 15,6; 20,9 and 12,4%.

The violation of the respiration and oxidative phosphorylation of the liver mitochondria also began to increase when the duration of the heliotrin entering the body reached 2 months. The phosphorylation of glutamate, the respiratory index by chance and the decrease in the ADF/o coefficient were 27,2; 47,4 and 19,3%. Especially significantly changed the oxidation of succinate: phosphorylated oxidation decreased by 31.6%, the respiratory index by chance by 27,8%, the coefficient of ADF/o by 18,9%. If the decrease in the oxidation of glutamate in the action of Dinitrophenol during this period amounted to 27,7%, the decrease in the oxidation of succinate reached 25,9%.

If, in 0,5 months of the experiment, the oxidation of glutamate in V₂ and V₄ cases in the liver mitochondria of animals with hepatitis increased by 26,7 and 41,3% compared to the norm, the oxidation of phosphorylated by 20,4%, the respiratory index by Chance decreased by 41,3%, the coefficient of ADF/o decreased by 10,8%, in the respiratory index and ADF/O coefficients decreased by 17,4; 31,4

and 6,8%, and there was a significant improvement in the convergence of healthy animals to indicators in pig mitochondria.

As the duration of phytochoy, which was sent to the body by animals with hepatitis, increased, the oxidation of glutamate in various metabolic states and the synthesis of ATF began to approach the indicators in healthy animals. In 1 month of the experiment, the oxidation of glutamate in V_2 and V_4 cases in the liver mitochondria of animals with hepatitis increased by 26,4 and 40,6% in comparison with the norm, the oxidation of V_3 cases, the respiratory index by chance and the coefficient of ADF / o 22,3; In fitochoi recipients, oxidation in V_2 and V_4 cases increased only by 6,2 and 9,8%, oxidation in V_3 cases, respiratory indication and ADF/o coefficient by Chance was determined to decrease only by 9,9; 17,2 and 5,0%, respectively.

When the duration of sending phytochoy to the body of animals with hepatitis was 2 months, glutamate oxidized and oxidized phosphorylation indicators in various metabolic states approached the indicators in the liver mitochondria of healthy animals. The oxidation of glutamate under the action of Dinitrophenol in the liver mitochondria of hepatitis animals decreased by 0,5% in 1 and 2 months of the experiment compared to the norm by 21,0; 22,7 and 27,7%, while in animals with phyto-acquired hepatitis by 10,4; 4,4 and 1,4% with a large decrease in sohlom equated to the indicators

When the transfer of phytochoy to animals with hepatitis reaches 2 months, the oxidation of glutamate in mitochondria under the influence of Dinitrophenol reaches the norm. Our next experiment was to determine what changes would occur in the oxidation of succinate in liver mitochondria when phytochondria were given to animals with hepatitis. In 0,5 months of the experiment, the oxidation of succinate in V_2 and V_4 cases in the liver mitochondria of animals with hepatitis slightly increased compared to the norm, but V_3 decreased by 12,7%, resulting in a decrease in the respiratory index and ADF/o coefficient by chance to 18,1 and 12,4%.

In animals receiving phytochoy, however, oxidation in the V_2 and V_4 status was only slightly reduced compared to the control indication, and the increase in V_3 led to the convergence of oxidized phosphorylation indicators to the control indicators. As the duration of phytochoy introduced into the body of animals with hepatitis increases, in liver mitochondria, the oxidation of succinate in various metabolic States has also accelerated its convergence with the indications in healthy animals. In 1 month of the experiment, the oxidation of suctsinate in V_2 and V_4 cases in the liver mitochondria of animals with Hepatitis does not change in relation to the norm, but in V_3 , the respiratory index

and the ADF/o coefficient according to Chans decreased to 23,8; 28,0 and 18,0%, while in animals receiving phytochoy approached the When the duration of phytochoi introduced into the body of animals with hepatitis reaches 2 months, the oxidation and oxidative phosphorylation of succinate reached the norm. In 0,5, 1 and 2 months of the experiment, the oxidation of succinate under the influence of Dinitrophenol in the liver mitochondria of hepatic animals decreased by 26,0;

36,7 and 35,9%, while in phytochondria only by 13,3; 6,6 and 2,3%, respectively, the indicators in the liver mitochondria of healthy animals decreased.

Conclusion

Hence, prolonged administration of heliotrin into the body simultaneously reduces the oxidation of FAD-dependent substrates and ATF synthesis along with NAD-dependent substrates. Respiratory and oxidative phosphorylation of the liver mitochondria were restored during long-term treatment of hepatitis animals with phytochoy, equal to those in the liver mitochondria of healthy animals.

References

- Ganitkevich V.Y. (2015) The role of mitochondria in cytoplasmic Ca^{2+} cycling. // Exp. Physiol. - V.88. - N1. - P.91-97.
- Grattagliano I., Vendemiale G., Lauterfubryg B. H. (2009). Reperfusion injury of the liver: role of mitochondria and protection by glutathione ester. // J Surg. Res. -V. -P. 2-8.
- Drogovoz T.V., Borodina T.V., Derimedved L.V. Alternatives to the choice of hepatoprotectors in the conditions of toxic hepatitis and frequent resection of the liver. PROVISOR jur. 2008. №18 Internet materials.
- Karimov X.Ya., Inoyatova F.X., Dadajonova Sh.N., (2008). Section of glutathione in the mechanisms of natural detoxification // Pathology- №1-p.3-7
- Karimov X.Ya., Inoyatova F.X. (2011). Oxidative processes in the liver during toxic lesions. Directory guide. Tashkent pp.50-76.
- Katikova O.Yu., Katin Ya.V. Tishikin V.S.(2012.). Hepatoprotective action of drugs of plant origin // Eksperim. and klin. formology №1-p.41-43
- Karimov X.Ya., Inoyatova F.X., Askarov X.A. (2006). Composition of perekisnogo oxidation of lipids in the combined lesions of the pancreas and biscuits. Med.jurn.Uzbekistana. №5-6.
- Lenaz. G. (1998). Role mitochondria in oxidative stresses and ageing. // Biochim. Biophys. Acta. V. 1366. P. 53-67.
- Lee H ,Wie V. (2010.) Mitochondrial role in life and death of the cell. // J. Biomed , science. -N. 7.- P. 2- 15.
- Madesh M., Balasubramanian K. A. (1997). Activation of liver mitochondrial phospholipase A2 by superoxide. // Arch. Biochem. Biophys. V. 346, iss. 2. P. 187-192.
- Mitochondria as a source of reactive oxygen species during reductive stress in rat hepatocytes. Dawson. T.I., Gores G J., Nieminen A. L., Herman B., Lemasters J.J. (2001). // Am.J.Physiol.v.264.p. c 961-e.967
- Mayer K.P. (2011). Hepatitis and the aftermath of hepatitis. Moscow: Nauka,- 423 p.
- Samartsev V.N., Chezganova S.A., Polishchuk L.S. (2017). Temperature dependence of rat liver mitochondrial respiration during uncoupling of oxidative phosphorylation by fatty acids, influence of inorganic phosphate. // J. Biochemistry. - Moscow, - T.68. Issue 6.-.759-768 p.
- Tojiboev Q.T., Ikramova M.M. (2019). From biochemistry and molecular biology experimental works. Tashkent. 207 p.p.

Tojiboev Q.T., Ikramova M.M. (2021). Effect of herbal tea on the change of adenine nucleotides in liver injury. // International journal of Aquatic Science. India. V 12, Issue 3. 2997-3004p.