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PECULIARITIES OF THE EFFECT OF HIGH AIR TEMPERATURE ON CARBOHYDRATE-ENERGETIC METABOLISM OF THE HEART AND CORRECTION OF METABOLIC PROCESSES

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Abstract

Introduction: The fight against cardiovascular pathology is one of the paramount problems of medicine due to the significant spread of these diseases, often leading to premature old age and lethal outcomes. In this connection, we have undertaken a study of the influence of the unfavorable factor, high ambient air temperature, on functional shifts in the heart muscles.

Objective: Revealing the mechanism and regularities of the impact of unfavorable air temperature on the parameters of carbohydrate-energetic processes in the heart and activation of the adaptation process by the introduction of biologically active substances.

Methods: Experimental studies were performed on 98 white male rats weighing 180–200 g, divided into 7 groups. The first group served as the control and was under optimal temperature conditions. Group 2 animals under these conditions (group 1) received BAS; groups 3-6 were kept under high temperature conditions; and group 5 was kept under high temperature conditions and received BAS intratracheally. Group 7 animals, after 5 hours of exposure to high temperatures, were transferred for 1 hour to conditions of optimal air temperature. Experimental animals were kept under the canopy at 40.6 to 42.00°C in the summer period. Groups 3–6 in the dynamics for 1, 2, 3, and 4 hours conducted studies of energy processes in the heart and blood. Biochemical parameters after 3 hours of exposure to temperature factors were intragastrically administered by BAS. We studied the change of carbohydrate-energetic metabolism parameters under high temperature exposure.

Findings: The results of studies indicate changes in metabolic processes in the heart and blood tissue under the influence of high air temperature, accompanied by quantitative changes in the relationship between the main metabolic pathways of myocardial

carbohydrate-energetic metabolism and metabolic regulation with intragastric administration of bioactive substances (BAS), consisting of spirulina, acacia gum, and calcium carbonate.

Conclusions: The accumulation of end products of anaerobic glycolysis, inhibition of activity of redox enzymes of the tricarboxylic acid cycle, and respiratory enzymes inhibit the process of tissue respiration and oxidative phosphorylation when adding α-ketoglutaric acid substrate and rum-C succinate in the heart mitochondria of experimental animals, as revealed under exposure to high air temperature. The introduction of spirulina, acacia gum, and calcium carbonate affects the increase of adaptation processes on the state of carbohydrate metabolism, the activity of mitochondrial enzymes, and the intensity of respiration and oxidative phosphorylation in the heart muscles of animals.

Key words: heart, high fever, carbohydrate-energy metabolism, spirulina, acacia gum, calcium carbonate.

Introduction. Recent literature suggests that high air temperatures can not only cause pathological changes in various organs and systems but also influence the course and development of a number of pathological processes [7].

Under the influence of adverse temperature factors can change the reactivity of the organism, reducing its compensatory possibilities. Against the background of reactivity changes, unfavorable environmental factors (especially in production conditions) can influence the course and outcome of a variety of disease processes, usually having "non-occupational" etiologies (various cardiovascular system diseases, some infectious processes, etc.). [8].

The fight against cardiovascular pathology is one of the primary problems of medicine due to the significant spread of these diseases, often leading to premature old age and death [4, 5].

In this connection, we undertook a study of the influence of an unfavorable factor—high ambient air temperature—on the functional shifts in the heart muscles.

The aim of this work was to study the effect of high ambient air temperature on carbohydrate-energy metabolism in cardiac mitochondria.

Biochemical studies were carried out to elucidate a possible mechanism of the high temperature effect on the state of carbohydrate-energetic metabolism in the heart muscle. Functional state of myocardium was studied by determining lactate, pyruvate, glycogen, and activity of tricarboxylic acid cycle enzymes glutamate dehydrogenase (GDH), succinate dehydrogenase (SDH), malate dehydrogenase (MDH), and respiratory enzyme cytochrome oxidase (COX) in heart tissue and blood [1, 2, 5, 6, 7, 8].

Purpose of work. To reveal the mechanism and regularities of the influence of unfavorable air temperature on parameters of carbohydrate-energetic processes in the heart and the activation of the adaptation process by the introduction of biologically active substances.

Materials and methods. Experimental studies were carried out by determining the biochemical parameters of the heart by studying carbohydrate-energy metabolism (lactate, pyruvate, glycogen, activity of dehydrogenases in myocardium mitochondria - GDH, SDH, MDH, respiratory enzyme - cytochrome oxidase) under exposure to high air temperature. The investigations were performed according to the European Convention for the Protection of Vertebrate Animals Used for Experimental or Other Scientific Purposes (Strasburg, March 18,

1986, ETS N 123). All animals were kept in vivarium and laboratories for medical and biological research in hygiene at the Research Institute of SGPZ of the Ministry of Health of the Republic of Uzbekistan.

Experimental studies were performed on 98 white male rats weighing 180–200 g, divided into 7 groups. The first group served as the control and was under optimal temperature conditions. Group 2 animals under these conditions (group 1) received BAS; groups 3-6 were kept under high temperature conditions; and group 5 was kept under high temperature conditions and received BAS intratracheally. Group 7 animals, after 5 hours of exposure to high temperatures, were transferred for 1 hour to conditions of optimal air temperature. Experimental animals were kept under the canopy at 40.6 to 42.00°C in the summer period. Groups 3–6 in the dynamics for 1, 2, 3, and 4 hours conducted studies of energy processes in the heart and blood. Biochemical parameters after 3 hours of exposure to temperature factors were intragastrically administered by BAS.

Studies of carbohydrate-energetic metabolism parameters in the heart and blood included determination of lactate, pyruvate, and glycogen content in tissue and activity of succinate dehydrogenase (SDH) [7], glutamate dehydrogenase (GDH) [2], malate dehydrogenase (MDH), and cytochrome oxidase (COX) in mitochondria [1, 2, 5, 7, 8].

At the end of the corresponding periods of exposure to temperature factors, the animals were rapidly slaughtered by decapitation.

The traditional method of isolating mitochondria from cardiac muscle in 0.25 M sucrose with 0.001 M EDTA and 15 mm Tris-HCl.

Parameters characterizing the functional state of mitochondria (pH 7.5) were recorded on an LP-9 polarograph using a platinum electrode (pH-7.5). For the incubation medium, 170 mm sucrose, 0.6 mm EDTA, 35 mm KCl, 7 mm KH2PO, and 15 mm Tris-HCl pH-4 were used. The medium was suspended at the rate of 1 g per 10 ml of medium until a uniform suspension was formed. α-ketoglutaric acid and succinate were used as substrates. All procedures were performed in a cold room at 0–40 °C.

Biologically active substances (BAS) consisting of Spirulina (Spirulina phatensis)—250 mg, Acacia gum (Acacia gum)—6.175 mg, and Calcium carbonate—3.075 mg. Form of release - in the form of tablets. 1 tablet is crushed in a porcelain mortar to form a powder, and 20 ml of water is added to it. As a boltushka, it is administered intragastrically to animals using a syringe with a metal probe at a dose of 1 ml per 100 g of rat weight.

Results. To elucidate some aspects of the mechanism of the high air temperature effect, in particular, on the state of carbohydrate-energy metabolism, we studied the content of lactate, pyruvate, and glycogen, the activity of tricarboxylic acid cycle enzymes (GDH, SDH, and MDH), the respiratory enzyme cytochrome oxidase, as well as the oxidative phosphorylation of α-ketoglutaric acid and succinate substrates in the heart and blood mitochondria.

Data on the content of carbohydrate metabolites (glycogen, lactate, and pyruvate) in the heart tissue and blood of rats under the adverse effects of high temperatures are presented in Table 1.

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Table 1.

Peculiarities of the effect of high air temperature on carbohydrate metabolism in the blood and heart with intragastric administration of BAS to laboratory animals.

Groups and time frame of the study (hour)	Stats.	Lactate		Pyruvate		Glycogen	
		Blood mmol/l	**Heart mmol/l**	**Blood mmol/l**	**Heart mmol/l**	**Blood g/l**	**Heart mg/g**
Group 1 Control - under optimal temperature conditions	Msr±m	2,27±0,08	1,42±0,03	121,8±4,23	84,0±5,64	122,4±7,13	226,9±8,18
Group 2 Administration of BAS under optimal temperature conditions (21,4±1,400C)	Msr±m %	2,11±0,06 93,0	1,25±0,06 88,0	122,0±6,37 76,0	81,7±6,13 97,0	116,4±10,5 95,0	197,0±6,42 87,0
Group 3 High temperature (41.3±2.620C) for 60 min	Msr±m %	3,56±0,08 156,0	2,34±0,05 165,0	196,8±5,38 163,0	157,2±4,43 187,0	76,3±2,17 62,0	148,4±7,13 65,0
Group 4 2 hours after high temperature exposure (40.6±3.400C)	Msr±m %	4,07±0,7 177,0	2,41±0,07 170,0	211,3±7,13 173,0	172,6±7,53 205,0	81,3±6,15 66,0	162,6±7,84 72,0
Group 5 Administration of BAS under high temperature conditions (42.6±2.190C)	Msr±m %	3,17±0,05 110,0	1,98±0,04 140,0	149,8±6,34 123,0	112,6±6,42 134,0	98,6±3,82 80,0	187,0±9,34 83,0
Group6 4 hours after high temperature exposure (41.8±2.300C)	Msr±m %	3,38±0,08 149,0	1,96±0,07 138,0	186,3±5,27 153,0	102,0±7,5 121,0	71,4±5,13 58,3	181,3±6,84 80,0
Group 7 After 4 hours of exposure to high air temperature after a 1-hour transition to optimal temperature conditions (23.1±0.320C)	Msr±m %	2,03±0,06 89,0	1,27±0,04 89,5	132,2±4,65 108,0	98,2±4,15 117,0	106,2±4,15 87,0	202,4±7,14 89,0

*Note: reliability with respect to the control group with normal air temperature: * - $p<0.05$; ** - $p<0.01$; *** - $p<0.001$.*

As we can see from the table, in the myocardium of control rats (group 1) under the conditions of optimal temperature (21.4±1.100C), the content of lactate, pyruvate and glycogen in the heart tissue was 1.42±0.03 mmol/g, 84.0±5.64 μmol/g and 226.3±8.38 mg/g, respectively. Their blood levels were determined to be 2.27±0.03 mmol/l, 121.8±6.23 μmol/l, and 122.4±7.13 g/l.

Groups 3, 4, 5, and 6 animals were kept in conditions of high air temperature, and the studies were carried out for 60, 90, 120, and 180 min, respectively. In the blood and hearts of experimental animals (groups 3, 4, 5, and 6) under conditions of high air temperature, there was observed acceleration of anaerobic glycolysis, resulting in a significant increase of lactate and pyruvate levels in all terms of exposure, while glycogen concentration decreased, indicating a decrease in synthesis and increase in its breakdown, which is noted in acceleration of anaerobic glycolysis. Group 5 also received BAS. The metabolites of anaerobic glycolysis of carbohydrate metabolism in heart tissue and whole blood were determined at the end. Group 7 animals, after a 6-hour exposure to high temperatures, were transferred to conditions of optimal air temperature, and after 1 hour, biochemical parameters were studied.

In the hearts of animals in the experimental groups (groups 3, 4, and 5), the concentration of lactate increased to 165; 170; 140; 138%, respectively. After the transition from high to optimal temperature (group 7), the level decreased to 89.5%. Heart pyruvate concentration increased to 187–205 percent and blood pyruvate concentration also increased relative to control to 163–173%. When examining the amount of glycogen in the heart and blood, there was a decrease in it in the body.

Consequently, when animals were exposed to high air temperatures, there was an increase in lactate and pyruvate levels and a decrease in glycogen in whole blood and heart tissue. Changes in the concentration of carbohydrate metabolites were more pronounced in groups 3–4 of experiments.

The results of studies on the administration of biologically active substances when animals were exposed to high air temperatures indicate changes in metabolic processes in the heart, accompanied by quantitative changes in the relationship of carbohydrate metabolic pathways, the main energy substrates of the heart.

The obtained data on activity of tricarboxylic acid cycle dehydrogenases in heart muscle under conditions of exposure to high air temperature are presented in Table 2. As can be seen from the table, in the hearts and blood of the control groups (groups 1 and 2), activity of SDH significantly exceeded activity of other dehydrogenases.

In the study of the activity of redox enzymes under high temperature conditions for 180 min (groups 3-6) there was an inhibitory effect on the activity of SDH, GDH, and MDH in the mitochondria of the heart and whole blood of animals. Studies were carried out every 60, 90, 120, and 180 minutes when exposed to a high temperature factor. Thus, during all periods of the experiment, the activity of DMH in mitochondria of the heart and blood decreased to 67.4, 80%, and in the heart to 67; 56; 81.4%, respectively. Similar phenomena were observed in the activities of glutamate- and malate-dehydrogenases in blood serum and in the hearts of white rats. At the same time, inhibition of GDH was observed in all periods of

high temperature exposure. Especially sharp inhibition of activity was observed after 60 min of exposure. In cardiac mitochondria, inhibition of activity was observed at 90 and 120 min (47 and 50%, respectively). MDH activity also decreased in blood serum and heart during all periods of the experiments. At the same time, inhibition of activity was observed in cardiac mitochondria at 60 and 90 min of the experiment.

The changes in the activity of tricarboxylic acid cycle oxidative enzymes noted in our studies at different stages of exposure to physical factors may be due to the impact of cellular mechanisms.

Changes in the activity of tricarboxylic acid cycle oxidative enzymes when exposed to high air temperatures lead to disturbances in the functional state of intestinal mitochondrial membranes and inhibition of the activity of its enzymes, dehydrogenases and cytochrome oxidase.

So, when animals stay in conditions of high air temperature, a significant decrease in the intensity of the tricarboxylic acid cycle in the heart and blood serum is observed in them. When using biologically active substances in animals under conditions of high air temperature (groups 5 and 6), normalization of anaerobic glycolysis and redox processes is observed.

Thus, when exposed to high air temperature, inhibition of activity of tricarboxylic acid cycle enzymes - GDH, SDH and MDH in the heart mitochondria and accumulation of underoxidized products of anaerobic glycolysis (lactate, pyruvate) and increased glycogen cleavage and synthesis, which can lead to a deficit of energy supply in the heart, are observed.

Application of BAS in animals leads to strengthening of adaptation processes and the functional state of the intestine when animals stay in conditions of high air temperature, increasing the activity of tricarboxylic acid cycle enzymes.

Table 2

State of respiratory enzyme activity during exposure to high air temperatures and peculiarities of the effect of high air temperatures during intragastric administration of BAS on the activity of tricarboxylic acid cycle enzymes

Groups	**Research timeline**	**Stats.**	**COX**	**SDH**		**GDH**		**MDH**	
			Heart µmol/g.h.	**Blood mmol/l.h.**	**Heart µmol/g.h.**	**Blood mmol/l.h.**	**Heart µmol/g.h.**	**Blood g/l.h.**	**Heart mg/g.h.**
1	Control 22^0C	Msr±m	1,67±0,17	98,8±3,60	52,8±3,1	21,3±0,22	11,3±0,22	64,1±4,24	39,3±1,13
2	Control 22^0C + BAS	Msr±m %	1,73±0,06 103,4	96,7±4,18 98,0	53,9±2,36 102,0	21,9±0,21 102,0	11,5±0,77 101,0	69,7±5,06 108,0	36,4±0,91 92,0
3	41,3 After 1 hour	Msr±m %	1,18±0,05 70,0	64,3±2,94 65,0	33,5±2,41 67,0	13,8±0,32 65,0	7,4±0,13 66,0	48,4±2,4 75,0	23,4±1,16 59,0
4	40,6 After 2 hours	Msr±m %	1,09±0,08 62,0	67,8±4,15 67,4	29,6±2,2 56,0	15,4±0,38 72,0	5,34±0,21 47,0	53,3±3,42 83,0	21,0±0,78 53,0
5	42,0 After 3 hours + BAS	Msr±m %	1,29±0,06 77,0	78,6±3,61 80,0	37,6±1,93 81,0	17,3±0,34 81,0	5,67±0,35 50,0	43,6±3,7 68,0	27,3±1,11 69,0
6	40,7 After 4 hours	Msr±m %	1,13±0,07 67,0	74,2±4,47 75,0	33,5±5,1 63,4	18,4±0,51 86,0	6,11±0,3 54,0	31,8±4,11 50,0	27,6±0,88 70,0
7	23,4±0,32 After 5 hours of exposure after transition	Msr±m %	1,43±0,03	33,9±0,03	46,3±3,16	22,1±0,22	9,78±0,27	58,7±4,45	31,7±1,22

*Note: reliability in relation to the control group: * - p<0.05; ** - p<0.01; *** - p<0.001.*

Study of intensity of tissue respiration and oxidative phosphorylation in cardiac muscle mitochondria at high ambient temperature. When studying the intensity of cellular respiration and oxidative phosphorylation in cardiac mitochondria, we determined with the addition of endogenous substrates such as α-ketoglutaric acid and succinate (Tables 3, 4). The determination of oxidative phosphorylation with these substrates was performed in animals at different periods of exposure to an adverse environmental temperature factor.

We found significant changes in the rate of respiration and oxidative phosphorylation in mitochondria after the addition of the substrates α-ketoglutaric acid and succinate when exposed to high air temperature.

The results obtained using the α-ketoglutaric acid substrate at 5 h of high temperature exposure are presented in Table 3.

The table shows that in the control group of animals under the conditions of optimal air temperature, the intensity of tissue respiration in the heart mitochondria was determined at the level of 26.0±0, 82 μAO2/min, ADP phosphorylation rate (V3) - 36.0±1.11 μAO2/min, mitochondrial respiration rate after ADP depletion (V4) - 24.8±0.60 μAO2/min, ADP/O2 ratio - 1.58±0.06, respiratory control (DC) - 11.2 μAO2/min. Application of biologically active substances has a slight decrease, increase in the rate of oxidation in the medium by α-ketoglutaric acid substrate, respiration rate after phosphorylation of added ADP and efficiency of phosphorylation and respiratory control in the heart of animals under optimal air temperature conditions.

When animals were kept in conditions of high air temperature, the rate of cardiac mitochondrial respiration upon addition of α-ketoglutaric acid substrate V2 in all terms of the experiment (groups 3-6) decreased to 81.9-53.6%, the rate of ADP phosphorylation also decreased after adenosiphore depletion. After application of BAS under the above conditions (group 4 - temperature 42.60C) in animals after transitioning from high temperature (group 7) to optimum air temperature, respiration and phosphorylation rates were close to those of the control group.

When succinate substrate was added to heart mitochondrial suspension (groups 3, 4, 5, and 6), V2, V3, and V4 respiration rates and ADP/O2 ratios significantly decreased (Table 4).

Table 3

Effect of high air temperature on the intensity of tissue respiration and oxidative phosphorylation of α-ketoglutaric acid substrate in cardiac mitochondria and acceleration of processes with the introduction of BAS

Conditions, groups and terms of research	**V_2 µAO/min**	**V_3 µAO/min**	**V_4 µAO_2/min**	**Dк (V_3 - V_4)**	**ADP/0_2**
Group 1 Control at 21.4±1.40 M ± m	26,0±0,82	36,0±1,11	24,8±0,60	11,2	1,58±0,06
Group 2 Control + BAS at a temperature of 21.4±1.40 1 hour after BAS injection M ± m %	27,0±1,03 103,8	37,9±1,06 105,3	25,4±1,46 102,4	12,5 111,6	1,90±0,17 120,2
Group 3 at 41.3±1.62 1 hour after exposure to temperature M ± m %	19,2±1,76** 73,8	28,0±1,53*** 77,8	20,5±1,23** 82,6	7,5 66,9	1,18±0,03*** 74,7
Group 4 at a temperature of 40.5±1.40 2 hours after exposure to high temperature M ± m %	19,3±1,48** 53,6	28,3±1,84** 78,6	19,7±1,15** 79,4	8,6 76,8	1,15±0,02*** 72,3
Group 5 at a temperature of 42.6±2.12 after 3 hours + administration of BAS at					

high temperature M ± m %	21,3±0,80*** 81,9	24,3±0,88*** 67,5	20,0±1,06** 80,6	4,3 38,4	1,29±0,03*** 81,6
Group 6 at 41.8±2.30 4 hours after exposure to high temperature M ± m %	21,2±0,70*** 81,5	24,2±1,01*** 67,2	20,7±1,05** 83,6	3,5 31,3	1,36±0,05* 86,3
Group 7 5 hours after to optimum air temperature 23.1±1.20 air temperature 23.1±1.20 M ± m %	27,8±1,01 106,9	31,8±1,35* 88,3	22,8±0,95 91,0	9,0 81,8	1,76±0,07 111,4

Note: V2 - oxidation rate in medium with oxidation substrate; V3 - oxidation rate upon addition of ADP; V4 - respiration rate after phosphorylation of added ADP; Dk - respiratory control (V3 - V4); ADP/O2 - phosphorylation efficiency; reliability in relation to control (group #1): * $p<0.05$; ** $p<0.01$; *** $p<0.001$.

Table 4

Peculiarities of the effect of high air temperatures on the energy metabolism in the heart mitochondria and restoration of adaptation processes with the introduction of BAS

Groups and timing of studies	**Stats.**	**V_2 µAO/min**	**V_3 µAO/min**	**V_4 µAO/min**	**Dκ (V_3 - V_4)**	**ADP/0_2**
Group 1 Control Temperature - 21.4±1.40	M ± m	23,0±1,24	39,5±0,96	27,0±1,17	12,5	2,21±0,07

Group 2 Temperature - 21.4±1.40 Control + BAS 1 hour after injection	M ± m v %	29,2±1,08** 126,9	41,0±1,63 103,8	28,51±0,92 108,5	12,5 100,0	2,16±0,05 97,7
Group 3 1 hour after exposure Temperature - 41,3±1,62	M ± m v %	20,7±0,88 90,0	28,7±1,05*** 72,6	21,3±1,05** 78,9	7,4 59,2	1,47±0,08*** 66,9
Group 4 2 hours after exposure Temperature - 40,5±1,40	M ± m v %	19,0±0,97* 82,6	30,3±0,84*** 64,0	20,0±1,8*** 62,2	10,3 82,4	1,51±0,11*** 68,3
Group 5 + BAS injection after 3 hours Temperature - 42,6±2,12	M ± m v %	20,5±1,02 89,1	30,8±1,14*** 75,1	20,8±1,30** 77,0	10,0 80,0	1,70±0,09*** 76,3
Group 6 6 hours after exposure Temperature - 41,8±2,30	M ± m v %	20,8±0,79 90,4	29,3±1,31*** 74,2	21,3±1,20** 78,9	8,0 64,0	1,40±0,08*** 63,3
Group 7 after 7 hours of exposure and 1 hour after transition to the optimum temperature (23.1±1.20)	M ± m v %	26,0±1,46 113,0	40,8±1,68 103,3	26,3±1,05 97,8	14,5 116,0	2,01±0,08 91,0

Note: V2 - oxidation rate in medium with oxidation substrate; V3 - oxidation rate upon addition of ADP; V4 - respiration rate after phosphorylation of added ADP; Dk - respiratory control (V3 - V4); ADP/O2 - phosphorylation efficiency; reliability in relation to control (group #1): * $p<0.05$; ** $p<0.01$; *** $p<0.001$.

When biologically active substances consisting of spirulina, acacia gum, and calcium carbonate were applied at optimal air temperature, the intensity of oxidation at V_2, V_3, and V_4 increased respectively to 126.9; 103.9; and 108.5%.

When the animals went from a high temperature after a 4-hour exposure to the optimal temperature for 1 hour of the study, the intensity of tissue respiration and oxidative phosphorylation in the heart mitochondria increased and approached the level of the control group.

Conclusions:

1. Exposure to high air temperatures revealed accumulation of anaerobic glycolysis end products - lactate, pyruvate, and reduced glycogen levels and inhibition of activity of redox enzymes of the tricarboxylic acid cycle (GDH, SDH, and MDH) and respiratory enzyme - cytochrome-C in the heart mitochondria of experimental animals.

2. Based on the results of the study, it can be assumed that the effect of high temperature inhibits the processes of tissue respiration and oxidative phosphorylation in the mitochondria of heart muscle when the substrate α-ketoglutaric acid and succinate are added.

3. Studies of cardiac mitochondria have shown that under exposure to adverse temperature factors there are violations of the oxidative phosphorylation function, which are manifested by increased respiration and oxidative phosphorylation of substrates of the tricarboxylic acid cycle - α-ketoglutaric acid and succinate and a decrease leading to a decrease in the ADP/O ratio.

4. The application of biologically active substances (spirulina, acacia gum, and calcium carbonate) influences the increase of adaptation processes under high air temperature exposure on the state of carbohydrate metabolism, the activity of mitochondrial enzymes, the intensity of respiration and oxidative phosphorylation, and approaches to normalization of energy exchange in the hearts of animals.

Conflict of interest. All of the authors declare that there is no potential conflict of interest requiring disclosure in this article.

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