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The Effect of a Sauna on Blood Oxygen Transport and the Prooxidant–Antioxidant Balance in Untrained Subjects

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Abstract—The effect of sauna on blood oxygen transport and the prooxidant–antioxidant state was studied in 18- to 22-year-old men. The course of heat treatment was performed once a week for five months, i.e., twenty procedures in total. A sauna procedure consisted of two exposures for 5 and 10 min at a temperature of 85–90°C and humidity of 10–15%. In young men, dry-air bath exposure resulted in respiratory alkalosis, increased pO_2, and a decreased affinity of hemoglobin to oxygen in the venous blood, which increased the transportation of O_2 to tissues. A single sauna procedure was associated with the development of oxidative stress, which was expressed as an enhancement of free radical processes and a decrease in antioxidant defense. Oxidative stress intensity decreased after the course of heat treatment. An elevation of nitric oxide formation could modify oxygen-dependent processes in the body.

Keywords: sauna, oxygen, lipid peroxidation, antioxidants, nitric oxide

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Dry-air baths are widely used as a therapeutic and prophylactic procedure. This is an efficient method for improving physical and mental capacities, activation of processes of repair and resistance to occupational and stress loads, immune resistance of the body, and an elevation of the functional capacities of blood circulation [1, 2]. However, baths substantially influence the human body, primarily, the cardiovascular, respiratory, and nervous systems [3]. An increase in oxygen consumption has been shown after both a single sauna procedure and a seven-day course. These increases were from 0.237 ± 0.05 to 0.407 ± 0.20 L/min after a 30-min exposure to a temperature of 80.1°C and from 0.236 ± 0.04 to 0.331 ± 0.10 L/min after a seven-day course [4]. There are only a few data on the effects of dry-air baths on the redox balance, gaseous constitution of the blood, and prooxidant–antioxidant balance. A gradual increase in pO_2 with the maximum 1 h after the sauna treatment and an elevation of oxygen saturation of hemoglobin, measured using the oxymetrical approach, immediately after sauna treatment were observed [5]. Oxidative stress, expressed as increases in the levels of hypoxanthine and xanthine in the blood plasma, was detected after dry-air baths [6].

Experimental study on the mechanisms for the transportation of oxygen in the blood at a high environmental temperature in animals demonstrated a decrease in hemoglobin affinity for oxygen considering the real values of temperature, pH, and pCO_2 and, a shift of the curve of oxyhemoglobin dissociation to the right [7]. An increase in lipid peroxidation processes and an attenuation of the activity of the antioxidant system during hyperthermia, which directly correlated with the rate of the shift of the curve of oxyhemoglobin dissociation to the right, have been reported [8]. Close functional interaction was observed between hemoglobin affinity for oxygen and intensity of lipid peroxidation in tissues. It has been found that the direction and strength of the changes in lipid peroxidation and activity of the antioxidant system under these conditions depend on the oxygen-binding properties of the blood [9]. However, oxygen binding after dry-air baths remains poorly studied.

In the present study, we examined the effects of a sauna on the transportation of oxygen in the blood and the prooxidant–antioxidant balance in untrained subjects.

EXPERIMENTAL

We studied the effects of sauna on the transportation of oxygen in the blood and the prooxidant–antioxidant balance in untrained 18- to 22-year-old subjects who were included in the basic medical group according to their state of health and physical development. A course of thermal treatment consisted of 20 procedures, which were performed once a week for five months. Each procedure consisted of two consecutive exposures of 5 and 10 min to a temperature of 85–90°C and humidity of 10–15%. Between the trials, the subjects were placed under the conditions of room temperature, i.e., 20–21°C, for 5 min. After restoration of the outflow, 8 mL of blood was sampled from the cubital vein using a precooled syringe washed with heparin. All experimental

manipulations with the subjects were made upon their informed consent and were approved by the Committee on Biomedical Ethics of Grodno State Medical University.

Temperature was measured in the left axillary region using an MT 1831 electrical thermometer (Microlife). The oxygen tension (pO_2), blood oxygen saturation (SO_2), oxygen content (C_vO_2), contents of hemoglobin (Hb) and methemoglobin (MetHb), blood oxygen capacity (OC), carbon dioxide tension (pCO_2), and pH in the samples were measured at a temperature of 37°C using a Synthesis-15 gas analyzer (Instrumentation Laboratory). The acid–base state of the blood was estimated using the Siggaard–Andersen nomograms on the basis of the following indices: actual and standard excesses of buffer bases (ABE/SBE) and concentrations of bicarbonate (HCO_3^-), total carbon dioxide (TCO_2), and standard bicarbonate (SBC). Hemoglobin affinity for oxygen was calculated using a p50 index, which corresponded to pO_2, measured at the level of 50% hemoglobin saturation with oxygen. This index was assayed spectrophotometrically at a temperature of 37°C, pH 7.4, and pCO_2 = 40 mmHg and denoted a $p50_{stand}$. Then, the actual p50 ($p50_{act}$) was calculated for actual values of pH, pCO_2, and temperature using the equations suggested by Severinghaus [10]. The location of the curve of oxyhemoglobin dissociation was calculated using the p50 values and Hill's equation.

The content of diene conjugates was measured by the intensity of ultraviolet absorption at a wavelength of 233 nm [11]. The content of malondialdehyde was assayed by the intensity of the pink color of trimethin complex at a wavelength of 540 nm [11]. The level of Schiff bases was measured by the intensity of fluorescence of a chloroform extract at excitation and emission wavelengths of 344 and 440 nm, respectively [12]. The content of α-tocopherol was assayed by the intensity of fluorescence of a heptane extract at excitation and emission wavelengths of 295 and 326 nm, respectively [13]. The catalase activity was measured spectrophotometrically at a wavelength of 410 nm using the hydrogen peroxide capacity for forming a stable colored complex with molybdenum salts [14]. The total level of nitrites in the plasma was estimated spectrophotometrically at a wavelength of 540 nm in reaction with the Griss reagent [15].

The normality of the distribution of the data was tested using the Shapiro-Wilk test. The data that fit the normal distribution are presented as the mean ± standard deviation. In this case, the significance of differences between groups was calculated using Student's *t* test for dependent variables. The data that did not fit the normal distribution are presented as the median (25 percentile–75 percentile), and the significance of differences between groups was calculated using Wilcoxon's test for paired comparisons.

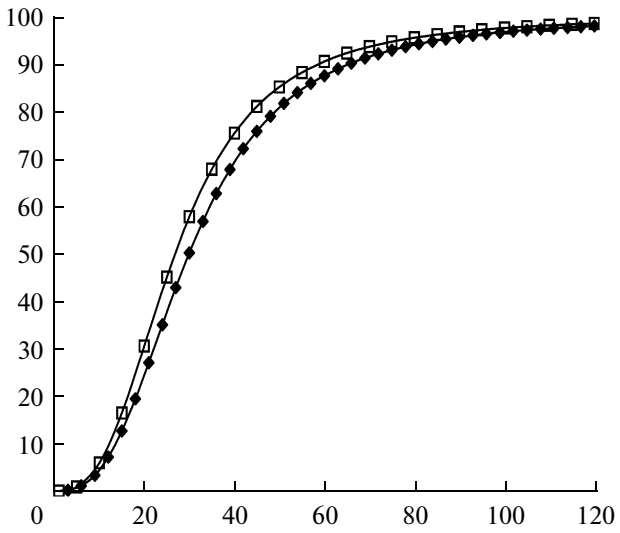

Fig. 1. The oxyhemoglobin dissociation curve at the actual pH, pCO_2, and temperature before (open squares) and after (solid diamonds) a sauna session at the start of the course. The ordinate shows the oxygen saturation of the blood in percent. The abscissa shows the oxygen tension in the venous blood in mmHg.

RESULTS

After two consecutive sauna exposures, the body temperature of the subjects increased by 2.55°C ($p < 0.001$) and 2.6°C ($p < 0.003$), respectively. The body weight decreased by 0.89% ($p < 0.002$) and by 0.76% ($p < 0.003$) at the start and the end of the course, respectively. The data on the oxygen transport function of the blood are presented in Table 1. The first procedure was followed by a pH increase by 1.2% ($p < 0.001$). We observed decreases in pCO_2 by 26.7% ($p < 0.001$), total carbon dioxide concentration by 13.3% ($p < 0.001$), bicarbonate concentration by 11.6% ($p < 0.001$), the actual excess of buffer bases by 18.3% ($p < 0.003$), and the standard excess of buffer bases by 42.5% ($p < 0.001$). At the end of the course, we observed similar changes in the indices of the acid–base state of the blood; however, they were smaller. The final procedure resulted in an alkaline shift of pH by 1.1% ($p < 0.005$), as well as a decrease in partial CO_2 tension by 24.5% ($p < 0.010$), a total decrease of the carbon dioxide concentration by 11.1% ($p < 0.011$), and a decrease of the bicarbonate concentration by 10.03% ($p < 0.013$).

At the start of the course, the sauna led to the oxygen content in the venous blood increasing by 133.7% ($p < 0.001$); pO_2, by 132.1% ($p < 0.001$); hemoglobin content, by 18.5% ($p < 0.001$); and blood oxygen capacity, by 17.4% ($p < 0.001$). The level of blood oxygen saturation and the methemoglobin content increased by 100.6 and 22.2%, respectively, ($p < 0.001$). Thermal treatment resulted in an increase in the p50 value at the standard and actual pH, pCO_2, and temperature by 5.8 and 11.6% ($p < 0.001$), respectively, as compared to the initial value that reflected a shift of the curve of oxyhemo-

Table 1. Effect of sauna on the indices for the transportation of blood in oxygen in untrained subjects

Index	Start of course			End of course		
	sauna		Wilcoxon's test	sauna		Wilcoxon's test
	before ($n = 16$)	after ($n = 16$)		before ($n = 11$)	after ($n = 11$)	
pO_2, mmHg	28.00 (24.50–32.00)	65.00* (54.50–68.00)	0.001	29.00 (27.00–37.00)	51.00* (36.00–57.00)	0.004
CvO_2, mL/L blood	9.05 (8.35–12.10)	21.15* (19.40–23.70)	0.001	9.50 (8.00–12.90)	17.20* (13.70–17.50)	0.003
Hb, g/L	140.50 (134.00–151.50)	166.50* (154.00–185.00)	0.001	135.00 (118.00–142.00)	144.00* (137.00–147.00)	0.026
SO_2, %	47.10 (41.05–61.00)	94.50* (92.55–95.55)	0.001	49.00 (43.60–69.20)	87.90* (66.40–90.60)	0.004
OC, %	19.25 (18.75–21.10)	22.60* (21.00–25.05)	0.001	18.36 (16.05–19.31)	19.58* (18.63–19.99)	0.026
MetHb, %	0.90 (0.60–1.05)	1.10* (1.00–1.20)	0.003	1.00 (0.90–1.20)	1.20* (1.20–1.20)	0.043
$p50_{act}$, mmHg	26.60 (25.95–27.24)	29.68* (28.96–30.65)	0.001	27.20 (26.80–28.28)	30.57* (30.03–31.15)	0.002
$p50_{stand}$, mmHg	26.44 (26.03–27.51)	27.96* (27.70–28.77)	0.001	27.10 (26.76–28.30)	28.37* (27.33–29.28)	0.016
pCO_2, mmHg	52.95 (49.50–57.75)	38.80* (37.50–40.45)	0.001	52.70 (43.90–59.00)	39.80* (38.30–42.00)	0.010
pH, unit	7.354 (7.342–7.379)	7.442* (7.424–7.453)	0.001	7.350 (7.318–7.398)	7.432* (7.417–7.439)	0.005
ABE, M	3.55 (3.00–5.45)	2.90* (2.20–3.60)	0.003	3.80 (2.10–4.30)	2.90 (1.80–3.10)	0.120
HCO_3^-, mM	30.20 (28.90–31.00)	26.70* (25.65–27.15)	0.001	29.90 (28.20–30.60)	26.90* (25.80–27.10)	0.013
TCO_2, mM	32.05 (30.50–32.50)	27.80* (26.85–28.40)	0.001	31.50 (29.50–32.40)	28.00* (27.20–28.40)	0.011
SBE, mM	4.00 (3.15–6.10)	2.30* (1.30–2.95)	0.001	3.60 (2.60–4.50)	2.40* (1.00–2.70)	0.041
SBC, mM	26.65 (26.00–27.55)	26.95 (26.25–27.50)	0.889	26.70 (24.90–27.20)	26.20 (24.50–27.00)	0.415
T, °C	36.30 (36.05–36.45)	38.85* (38.60–39.50)	0.001	36.3 (36.10–36.40)	38.9* (38.80–39.20)	0.003

Note: See "Experimental" for the abbreviations here and in Table 2. *The difference from the initial level is statistically significant.

globin dissociation to the right (Fig. 1). At the end of physiological hyperthermia, we observed increases in the oxygen content in the venous blood by 81.1% ($p < 0.003$); pO_2, by 75.9% ($p < 0.004$); and the contents of hemoglobin and methemoglobin, by 6.7 ($p < 0.026$) and 20.0% ($p < 0.043$), respectively. We also found that blood oxygen saturation increased by 79.4% ($p < 0.004$) and oxygen capacity increased by 6.6% ($p < 0.026$). The values of the $p50_{stand}$ and $p50_{act}$ increased by 4.7 ($p < 0.016$) and 12.4% ($p < 0.003$), respectively, which reflected a shift of the curve of oxyhemoglobin dissociation to the right. Thus, the indices for the transportation of oxygen in blood in young men at the end of the course demonstrate smaller changes in pO_2, CvO_2, KE, SO_2, and hemoglobin as compared to the first exposure.

The level of total nitrites in the plasma increased after the procedure at the start and end of the course by 20.1 and 17.1%, respectively (Fig. 2), which demonstrated an increased production of NO in the body after sauna treatment.

The data on lipid peroxidation processes and activity of the antioxidant system after the sauna at the start and end of the course are presented in Table 2. At the start of the course, the contents of diene conjugates increased in the plasma and erythrocytes by 33.3% ($p < 0.016$) and 26.3% ($p < 0.002$), respectively. The levels of malondialdehyde in the plasma and erythrocytes increased by 48.4% ($p < 0.028$) and 30.5% ($p < 0.002$), respectively. The contents of Schiff bases increased in the plasma and erythrocytes by 38.7% ($p < 0.039$) and 55.1% ($p < 0.001$), respectively. The enhancement of free radical processes was associated with a simultaneous decrease in the level of α-tocopherol in the plasma by 7.8% ($p < 0.004$) and inhibition of catalase activity in erythrocytes

by 14.9% ($p < 0.002$). At the end of the course, we found that the contents of malondialdehyde in erythrocytes increased by 6.9% ($p < 0.010$) and Schiff bases in the plasma increased by 92.9% ($p < 0.003$), whereas the catalase activity decreased in erythrocytes by 9.2% ($p < 0.016$). Thus, the final sauna procedure resulted in smaller changes in the levels of diene conjugates, malondialdehyde, and Schiff bases in erythrocytes as compared to the first sauna treatment, which indicates attenuation of the prooxidant–antioxidant imbalance.

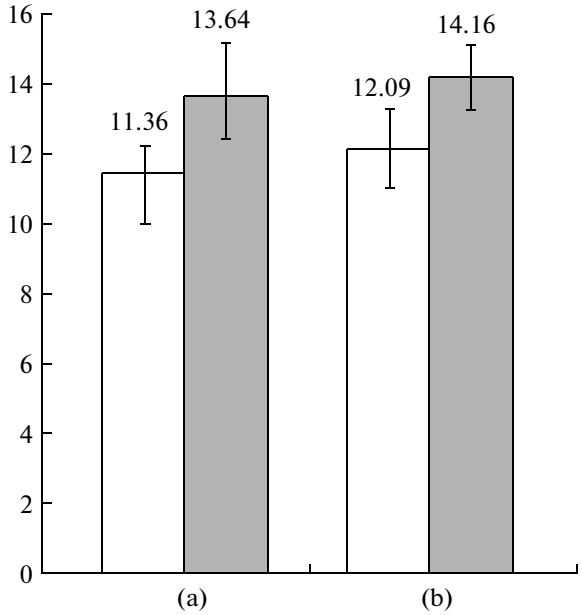

Fig. 2. The total nitrite content of blood in the subjects before (open bars) and after (solid bars) a sauna session. The ordinate shows the total nitrite content in μM. The abscissa shows the moment of measurement: (a) at the start of the course and (b) at the end of the course.

DISCUSSION

An important factor mediating the effect of taking a sauna is inspiration of hot air followed by the development of reflex respiratory adaptation and changes in the blood circulation, which prevent excessive growth of internal body temperature and impairment of homeostasis [16]. In our study, modification of the acid–base state of the blood reflects the development of respiratory alkalosis caused by a decrease in the carbon dioxide content due to lung hyperventilation associated with an increase in the body temperature in dry-air baths [4, 17]. An elevation of the body temperature of 2–3°C results in an increase in lung ventilation, which is associated with a decrease in pCO_2 and an alkaline shift of pH. Then, accumulation of underoxidized metabolic products in the blood leads to exhaustion of the mechanisms of compensation of the acid–base state, which results in metabolic acidosis [18, 19]. These changes in the acid–base state at different stages of overheating represent a complex two-phase course of its modification. Our data demonstrate that hyperventilation mediates elevated excretion of carbon dioxide from the body, which shifts the pH towards more alkaline values; however, decreases in the bicarbonate level and the actual and standard base excesses indicate that respiratory

Table 2. Effect of sauna on processes of lipid peroxidation and factors of antioxidant defense in untrained subjects

Index		Start of course			End of course			
		sauna		Wilcoxon's test	sauna		Wilcoxon's test	*t* test
		before ($n = 13$)	after ($n = 13$)		before ($n = 11$)	after ($n = 11$)		
Diene conjugates, units/mL	Plasma	1.20 (1.04–1.46)	1.60* (1.26–1.94)	0.016	1.44 (0.76–1.56)	1.42 (1.34–1.60)	0.534	–
	Erythrocytes	14.44 (11.16–15.36)	18.24* (16.56–19.32)	0.002	10.80 (8.76–12.12)	11.04 (8.88–13.20)	0.790	–
Malondialdehyde, μM	Plasma	1.86 (1.54–2.63)	2.76* (1.92–3.78)	0.028	2.23 ± 1.41	2.31 ± 1.32	–	0.639
	Erythrocytes	26.92 (18.97–28.46)	35.13* (29.49–38.46)	0.002	26.15 (15.13–26.67)	27.95* (21.79–31.02)	0.010	–
Schiff bases, units/mL	Plasma	152.30 (88.56–215.00)	211.19* (186.50–224.00)	0.039	136.76 (55.42–247.97)	263.80* (119.12–318.87)	0.003	–
	Erythrocytes	244.76 (203.36–297.06)	379.61* (341.60–428.27)	0.001	252.31 (28.39–375.52)	198.74 (48.74–334.81)	0.859	–
α-tocopherol, μM	Plasma	27.28 (26.66–29.34)	25.15* (20.69–26.67)	0.004	22.36 ± 2.31	23.21 ± 2.84	–	0.064
Catalase, mM H_2O_2/(min *g*) Hb	Erythrocytes	6.58 (6.12–7.26)	5.60* (5.47–5.91)	0.002	6.86 (5.58–7.33)	6.23* (4.68–6.70)	0.016	–

Note: * The difference from the initial level is statistically significant, $p < 0.05$.

alkalosis is partially compensated due to metabolically mediated acidotic processes.

The indices for the transportation of oxygen in blood in subjects prior to sauna treatment indicate hypoxemia, because we used the blood samples from the cubital vein, which collects blood from the corresponding region. The values of pO_2 and oxygen saturation were substantially higher as compared to the initial values due to the effects of several factors. Body hyperthermia under the conditions of dry-air baths is known to induce an elevation of oxygen consumption [4]. Simultaneous growth of O_2 tension in alveolar air promotes an increase in blood oxygenation. Under the conditions of heat stress, development of hypovolemia associated with increased perspiration and an elevation of the hemoglobin content are involved in those processes [20]. Ohishi et al. [21] have reported that a growth of rectal temperature to 39.5°C is accompanied by an alkaline pH shift, an increase in pO_2, and a decrease in pCO_2 in the venous blood. However, these changes were not related to significant intensification of lung ventilation, which indicated the involvement of additional factors in the elevation of oxygen tension. A high external temperature leads to the redistribution of the blood flow, which is expressed in a substantial increase in the peripheral area and a decrease in the splanchnic area. This results in the redistribution of the oxygen supply, including a decrease in the transportation of oxygen to the tissues and organs with high levels of metabolism and O_2 consumption, which is followed by the development of tissue hypoxia [22]. Under these conditions, lung hyperventilation and redistribution of the blood flow associated with a decrease in O_2 delivery to visceral organs with a high level of metabolism, together with other factors, cause an elevation of pO_2 in the venous blood, as was observed after taking a sauna in the present study.

Growth of the body temperature in dry-air baths due to the inefficacy of heat emission results in the activation of oxidation reactions [23]. The data on the indices of the prooxidant–antioxidant balance demonstrate an enhancement of free radical processes and a decrease in antioxidant defense after taking a sauna. These changes are induced by relatively insufficient oxygen delivery to the internal organs and, as a consequence, its increased utilization via the oxygenase pathway [24]. This leads to the activation of lipid peroxidation. After the course of heat procedures, changes in the oxygen-binding properties of the blood and free radical lipid oxidation are smaller as compared to those observed after a single treatment. This reflects mobilization of the adaptive resources of the body due to regular heat treatment [25].

In the body, hemoglobin's affinity for oxygen significantly influences oxygen diffusion from alveolar air to the blood and, then, from the capillaries to the tissues [26]. A 1-mmHg increase in p50 results in a 3.2-mmHg elevation of the arterial–venous difference, which increases tissue oxygenation. Under the conditions of this unchanged O_2 supply, heart output decreases as much as 5.8% per unit of p50 fluctuation [27]. After replacement of 80% of the circulating blood volume with a blood substitute consisting of polymerized hemoglobin with a higher p50 value, such as 54.2 mmHg, a substantial increase in pO_2 and O_2 consumption in tissues was observed [28]. Estimation of the oxygen supply of the skeletal muscle using Krogh's model demonstrates that growth of p50 from 26 to 39 mmHg is associated with a 7% increase in O_2 consumption [29].

Hemoglobin oxygenation is an exothermic reaction; therefore, oxyhemoglobin dissociation requires heat absorption. An increase in the blood temperature decreases hemoglobin affinity for oxygen. This effect of the temperature on the interaction of hemoglobin with oxygen is specific for most of the different types of hemoglobins. Samaja et al. [27] note that, although hemoglobin affinity for oxygen is estimated usually at 37°C, it is important to consider the body temperature and its gradient within the body, because they cause fluctuations of p50 from 8.7 to 36.5 mmHg during temperature growth from 15 to 42°C.

The attenuation of hemoglobin affinity for oxygen is an important factor for compensating the oxygen deficit under various pathologies and is responsible for the adaptation to hypoxia [30]. When the value of p50 is higher than the physiological level it probably promotes optimal oxygen transportation to the tissues under normoxia or moderate hypoxia, which are observed when taking a sauna. However, taking into account the physiological meaning of the *S*-like curve of oxyhemoglobin dissociation, we suppose that its shift to the right will make conditions of blood oxygenation in the capillary of the pulmonary circulation worse. In the body, the direction of a shift of the curve of oxyhemoglobin dissociation is closely related to the other components of the system of oxygen transport [31]. Insufficiency in their compensatory responses results in hypoxia. In the body, the shift of the curve of oxyhemoglobin dissociation after a sauna should be considered to be a form of the functioning of one of the elements of the system of oxygen transportation and estimated in its relationship with the other body systems, such as thermoregulation, blood circulation and others. Our data show that, after dry-air baths, the increased body temperature leads to the redistribution of the circulation to the prevalence of the peripheral blood flow and a decrease in O_2 delivery to tissues and organs with a high level of metabolism. These processes load the hematic component of the oxygen transportation system in order to provide appropriate oxygenation of the tissues and attenuate hemoglobin affinity for oxygen.

The oxygen-binding properties of hemoglobin determine not only the rate of O_2 delivery to the tissues and tissue pO_2 but also the efficiency of the functioning of the antioxidant system. Hyperthermia modifies these properties so as to respond to the tissue demands for oxygen, which substantially influences the antioxidant defense of the body [32]. When antioxidant mecha-

nisms cannot normalize cellular generation of active oxygen species, tissues accumulate products of free-radical reactions, which indicates the development of oxidative stress [33]. A shift of the curve of oxyhemoglobin dissociation rightwards indicates an increased release of oxygen in tissues [34]. Insufficient use of oxygen in tissues results in an increase in the level of molecular O_2 in the blood, which induces progression of oxidation reactions and promotes generation of free radicals [32]. This significantly strains antioxidant mechanisms, which cannot prevent oxidative stress under these conditions [30].

In volleyball players subjected to intense physical load, a compensatory–adaptive response which provided more effective delivery of oxygen to tissues expressed as an increase in p50, as well as an accumulation of diene conjugates and TBA-reactive products, was observed [35]. Under the conditions of dry-air baths, O_2 transportation to tissues increased. The O_2 fraction used for oxygenase processes also grew. However, long-term heat treatment was associated with a decrease in the content of lipid peroxidation products, which suggests enhancement of the efficiency of O_2 utilization. An increase in body temperature during muscle activity is very important for providing working capacities, because it allows increasing the heart rate, conductivity and excitability of the nervous tissue, accelerating metabolic processes in the phase of contraction and relaxation in the skeletal muscles, decreasing the tone of smooth muscles, and activating enzymatic reactions and metabolism as a whole according to the vant Hoff–Arrhenius law [36]. We suppose that the effects of thermal sauna treatment and overheating during muscle work have similar mechanisms.

Sauna heat treatment results in increased formation of NO, which is involved in the vascular thermoregulatory response. Being a free radical, this molecule directly participates in the maintenance of a prooxidant–antioxidant balance and influences the oxygen transport function in the blood [30, 32]. Interaction of NO with hemoglobin results in the formation of different compounds, including nitrosyl hemoglobin, *S*-nitrosohemoglobin, and methemoglobin, that play the role of allosteric regulators of the functional activity of hemoglobin at the level of its specific tetramers and the whole pool of hemoglobin [37]. During the cycle of erythrocyte movement in the circulation, hemoglobin consecutively reacts with NO, which modulates its structural transitions from the *R* to the *T* state. In the capillaries of the pulmonary circulation, these modifications may serve as an additional mechanism providing blood oxygenation, whereas in the microcirculatory stream of the systemic circulation, they optimize blood desaturation and oxygen supply to the tissues [30]. It is supposed that the mechanism that improves the state during cardiomyopathy is the increased production of endothelial NO induced by repeated sauna treatment [38]. The NO increase found at the start of the course represents the involvement of the elevated expression of endothelial NO-synthase in thermoregulation and oxygen-dependent processes in the body, whereas a smaller growth of NO at the end of the course reflects the elevated adaptive capacity of the body.

CONCLUSIONS

Thus, dry-air baths in untrained subjects result in changes in the acid–base state and oxygen transportation function of venous blood, which are expressed in respiratory alkalosis, a pO_2 increase, and a decrease in hemoglobin's affinity for oxygen, all increasing oxygen supply to the tissues. Single sauna bathing causes the development of oxidative stress, which is expressed in an enhancement of free radical processes and weakening of the antioxidant defense. Oxidative stress indices decrease after the course of heat treatment. The increased NO generation may modify oxygen-dependent processes in the body, such as blood oxygen transportation and the prooxidant–antioxidant balance.

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