The influence of acute exposure to moderate altitude upon the oxidants/antioxidats balance and upon the effort capacity

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Abstract: The experiment aimed to dissociate the effects of acute hypoxic stress from the hypoxic stress associated with physical stress caused by exercise and the implications for the oxidants/ antioxidants balance. The induced sympathoadrenal activation can lead indirectly to acute hypobaric hypoxia and oxinit oxidative stress, in addition to the direct effect of the hypobaric stress. In the case of the hypoxic stress associated with physical stress the effect of the hypobaric stress and physical stress interferes directly on the oxinit oxidative stress and the indirect effects produced on the one hand by physical stress and activation sympathoadrenal and the sympathoadrenal activation on the other hand which generates oxinit oxidative stress.

Key words: - ceruloplasmin, hydrogen donors, hypobaric hypoxia, malondialdehyde, oxidants/ antioxidants balance.

I INTRODUCTION

Acute exposure to altitude is characterized by a wide array of individual variability, depending on physical effort, physical condition, ambient temperature, etc.. Due to the fact that mountain climbing is done back carrying weights of 10-15 kg, the mountain (altitude) sickness phenomenon occurs sooner, in comparison with plane (air) sickness. Mountain sickness occurs at varying altitudes, smaller in the Alps compared to the Andes and the Himalayas.

Altitude sickness syndrome develops in 8-24 hours after ascent and disappears in 4-8 days. Pulmonary edema does not occur in those who climb gradually to higher altitude and in those who do not exercise during the first days after exposure to high altitude. [1], [2].

Higher than 4000-5300 m appear more obvious disorders, known as altitude sickness. They are caused by hypoxia and, at first their manifestation is good disposition, euphoria, followed in a few hours by an intense feeling of fatigue, headache, nausea, drowsiness, vomiting, sensory disturbances, decreased mental performance (memory and thinking disorders), disorders of motor acts, respiratory disorders (periodic breathing, high altitude pulmonary edema, pulmonary hypertension), renal disorders (high altitude diuresis), circulatory disorders (cerebral edema). Severe disorders arising from the presence of fasciculations, followed by seizures and coma, which suggests that the person has not adapted to high altitude hypoxia. If the climb continues, syncope and even death can occur [3], [4].

II PROBLEM FORMULATION

A. What is the influence on rats of the continued acute exposure to moderate hypobaric hypoxia, corresponding to the altitude of 1,500 m, have on the maximum aerobic capacity, made under normobaric normoxia?
B. What is the influence on rats of the continued acute exposure to moderate hypobaric hypoxia, corresponding to the altitude of 2500 m, have on the maximum aerobic capacity, made under normobaric normoxia?
D. What is the influence on rats of the continued acute exposure to moderate hypobaric hypoxia, corresponding to the altitude of 1500 m, have on the O / AO balance?

E. What is the influence on rats of the continued acute exposure to moderate hypobaric hypoxia, corresponding to the altitude of 2500 m, have on the O / AO balance of rats?
F. Does the exposure to acute moderate hypobaric hypoxia, corresponding to the altitude of 1500 and 2500 m, produce lasting effects on the aerobic exercise capacity under normobarism?

III PROBLEM SOLUTION

A. Material and methods

A.1 Conditions

The researches have been conducted on white Wistar rats, male, weighing 160-180 g, purchased from the biobase of the University of Medicine and Pharmacy "Iuliu Haţieganu", Cluj-Napoca. The animals were kept in the vivarium under appropriate and standardized hygienic conditions in the biobase of the Department of Physiology – from the UMF "Iuliu Haţieganu": the ambient temperature of 22±1oC, standardized food and water "ad libitum", with a light-dark cycle of 12 hours and they were left for a week to acclimatize before starting the experiment [5].

A.2 Lots

For the experiment we used 4 groups of white, male Wistar, rats, weighing 180-200g:

- group I (no. = 10) - animals exposed 48 hours at a simulated altitude of 1500 m (pO2 in the air - 132 mm Hg)
- group II (no. = 10) - animals exposed 48 hours at a simulated altitude of 2500 m (pO2 in the air - 117 mm Hg)
- group III (no. = 10) - animals exposed 48 hours at a simulated altitude of 1500 m (pO2 in the air - 132 mm Hg) and subjected pre and post exposure to effort under normobaric normoxia conditions, immediately and after 48 hours;
- group IV (no. = 10) - animals exposed 48 hours at a simulated altitude of 2500 m (pO2 in the air - 117 mm Hg) and subjected pre and post exposure to effort under normobaric normoxia conditions, immediately and after 48 hours.

A.3 Methods

a) Exposure to hypoxia

The simulated exposure was performed in the hypobaric chamber with a vacuum pump 0016 KB D which belongs to the Physiology Faculty of the Medicine and Pharmacy University „Iuliu Haţieganu" from Cluj-Napoca, designed by engineer Pop Tiberiu-Vasile and built in 2004 by SC Elserv SRL.

The hypoxia was interrupted daily for 1-2 hours in order to feed the animals and clean their cages.

b) Exploring of the effort capacity

The swimming test was done in a thermostatic water pool at the temperature of 32oC. The time was kept from the moment the animals were introduced in the pool till they were exhausted the moment the swimming stopped (submersion, floating tendency, clinging to the pool edges). A 5% loading was done based on the initial weight of the animals.

c) Determination of the oxidants/ antioxidants balance (O / AO balance)

To determine the O / AO balance venous blood was sampled from the retro-orbital sinus pre and post exposure to hypobaric hypoxia and effort, blood which was use to dose:

- oxidative stress parameters

Free MDA, bound MDA and total MDA [6], [7] .

- antioxidant parameters

- hydrogen donor ability (Janaszewska method in [8])
- Ceruloplasmin (Ravin method 1961 [9]).

1.A.1 **Experimental Work Programs**

The work programs for groups I and II are presented in Fig. 1 and those for groups III and IV in Fig. 2

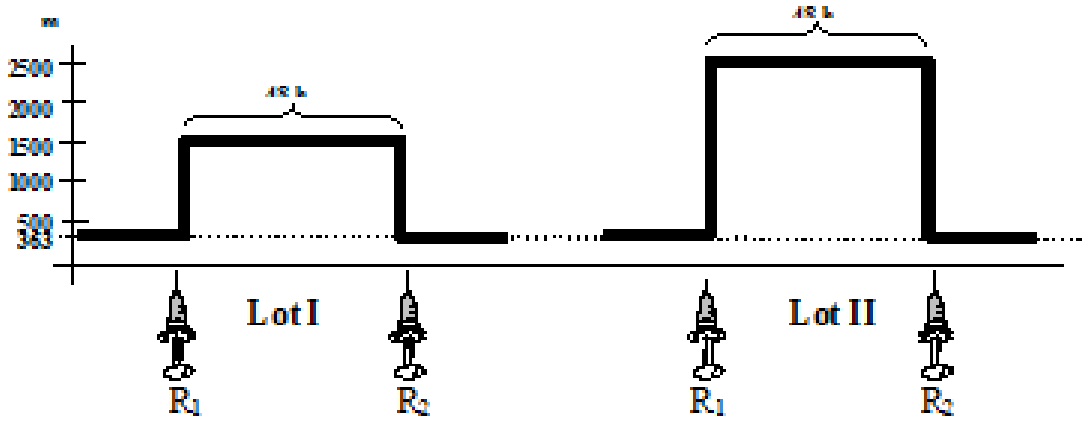

Fig.1 Experimental programs for exposure to acute hypobaric hypoxia of groups I and II
R_1 - pre-exposure sampling, R_2 – post-exposure sampling. Cluj-Napoca = 363 m

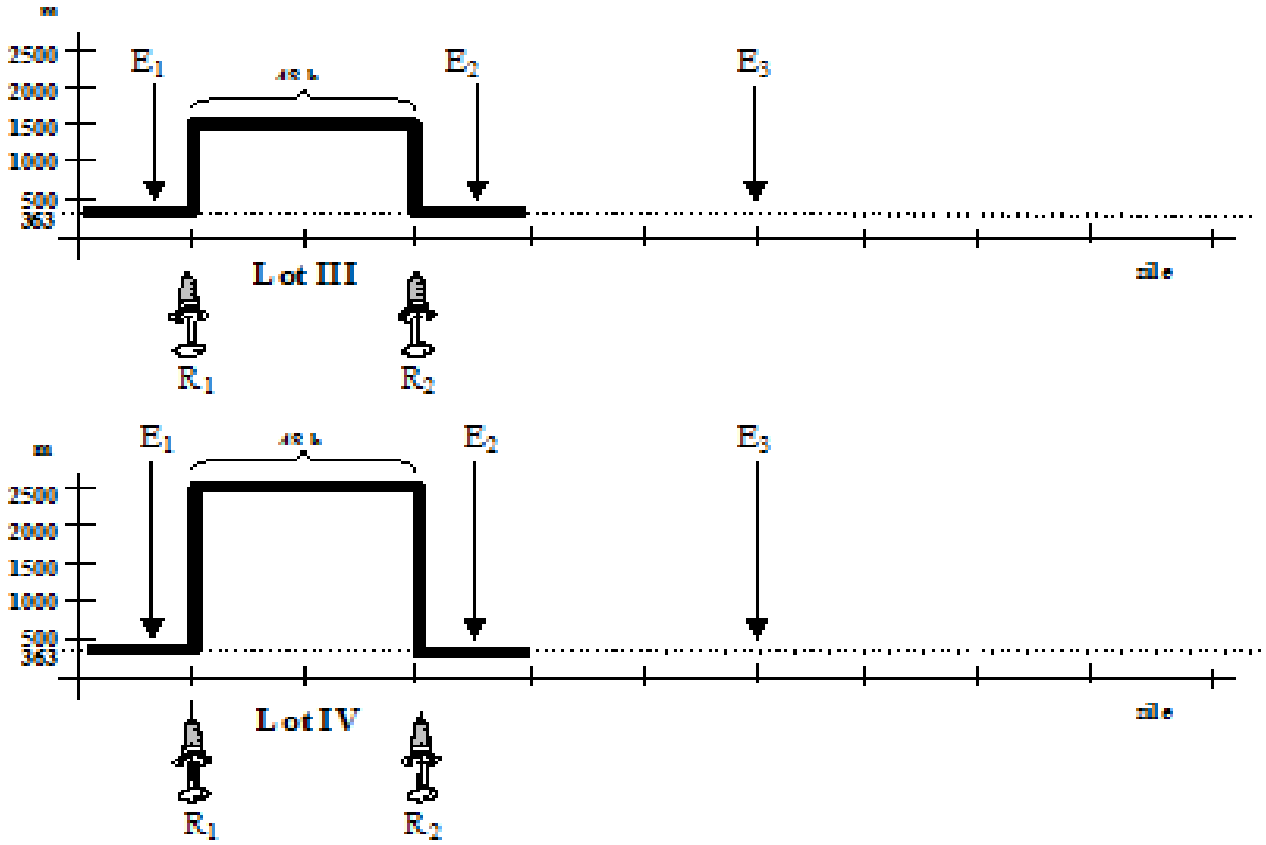

Fig. 2 Experimental programs for exposure to acute hypobaric hypoxia of groups III and IV
R_1 - pre-exposure sampling, R_2 – post-exposure sampling. E_1 – physical effort under normobaric conditions pre-exposure to hypobaric hypoxia; E_2 - physical effort under normobaric conditions 48 h post-exposure to hypobaric hypoxia Cluj-Napoca = 363 m

1.A.2 **Statistical processing**

Two aspects were followed:

- the descriptive analysis of the data
- analyzing the significance of the difference between the average values – when comparing two groups, test „t" Student was used for unpaired samples; we used a 95% confidence interval and we considered significant the results for which $p < 0,05$.

IV. RESULTS

a) The exposure to moderate hypobaric hypoxia corresponding to the altitude of 1500 m for 48 hours did not affect significantly the maximum aerobic effort capacity for group III under normobaric conditions (Fig.2 and 3).

b) Exposure to moderate hypobaric hypoxia corresponding to the altitude of 2500 m for 48 hours increases significantly the maximum aerobic capacity for group IV under normobaric conditions the increases are 2, 6 times. (Fig 2 and 3).

c) The control groups (I and II), the exposure to moderate hypobaric hypoxia corresponding to the altitudes of 1500 m and 2500 m, for 48 hours, causes post exposure significant increases of the free, bound and total MDA in comparison to the initial values; the increases higher in group I as compared to group II, LPX and total MDA, and higher in group II as compared to group I, for free MDA (Fig.4).

d) The groups subjected to effort, the exposure to moderate hypobaric hypoxia corresponding to the altitudes of 1500 m and 2500 m, for 48 hours, post exposure causes significant increases in free, bound and total MDA in comparison with the initial values (groups III and IV); the increases are higher in group III as compared to group IV (Fig.4).

e) In the control groups, the exposure to moderate hypobaric hypoxia corresponding to the altitude of 1500 m and 2500 m for 48 hours, post exposure causes a significant increase of DH in groups I and II and a significant increase of CRP in group I (Fig. 5 and 6).

f) The groups subjected to effort, the exposure to moderate hypobaric hypoxia corresponding to the altitudes of 1500 m and 2500 m for 48 hours, post exposure and effort determines under normobaric conditions, significant increases in DH and CRP in groups III and IV, the increases being higher in group IV as compared to group III (Fig. 5 and 6).

g) Effect of the acute hypobaric hypoxia on the moderate aerobic effort capacity under normobaric conditions is of short duration (Fig. 3).

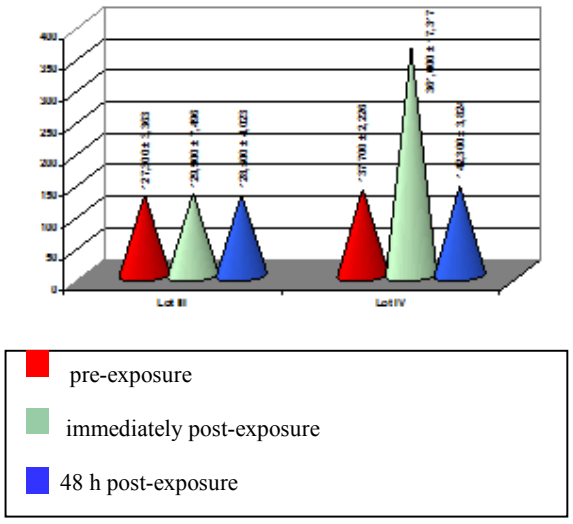

Fig. 3 Maximal effort capacity of the studied groups. Values

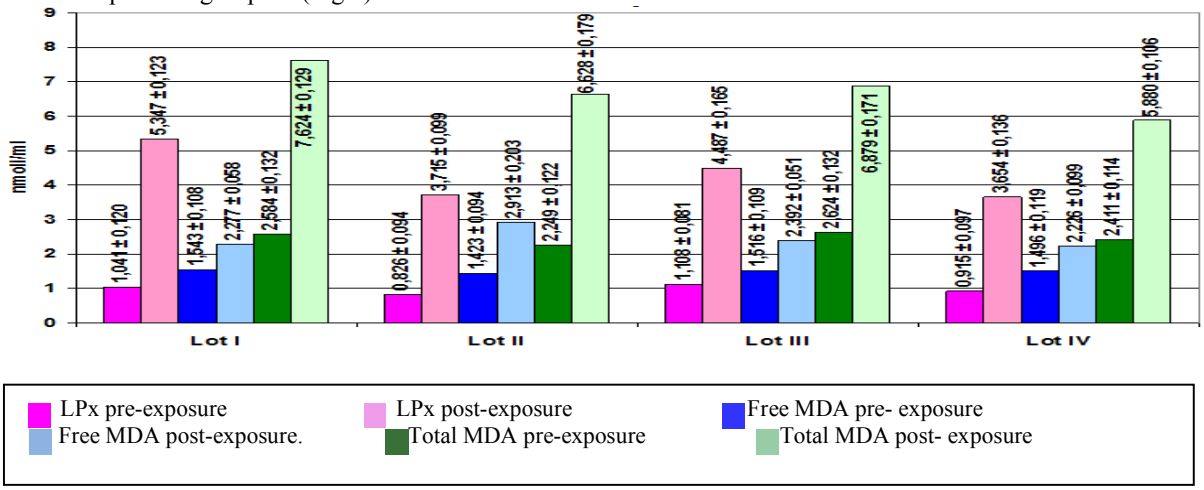

Fig. 4 LPx, free MDA and total MDA

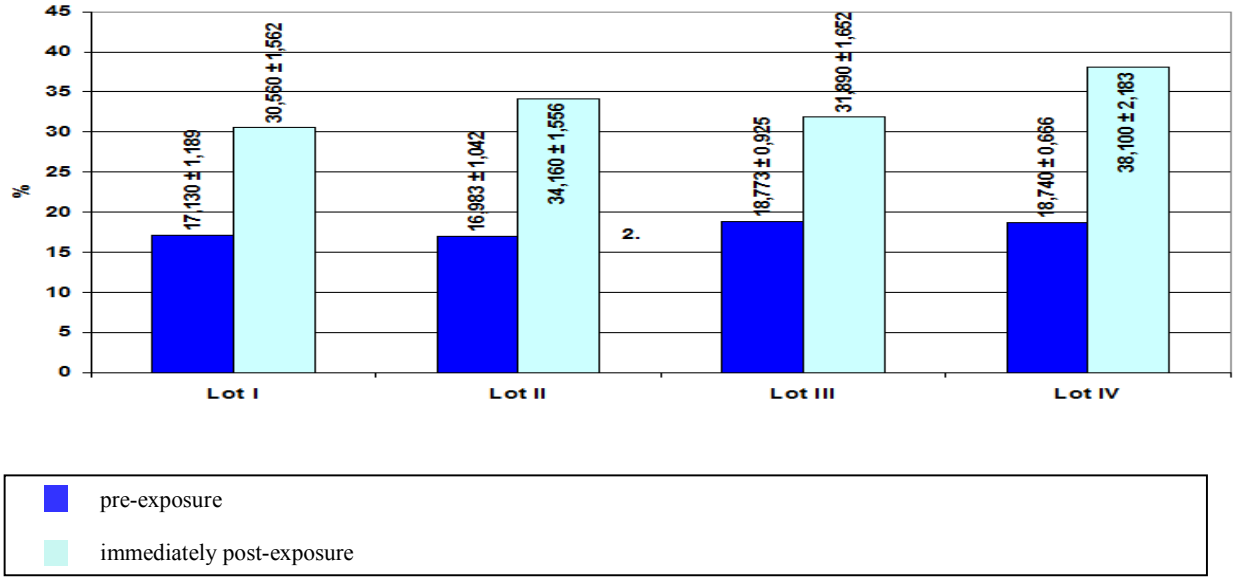

Fig. 5 Hydrogen donors

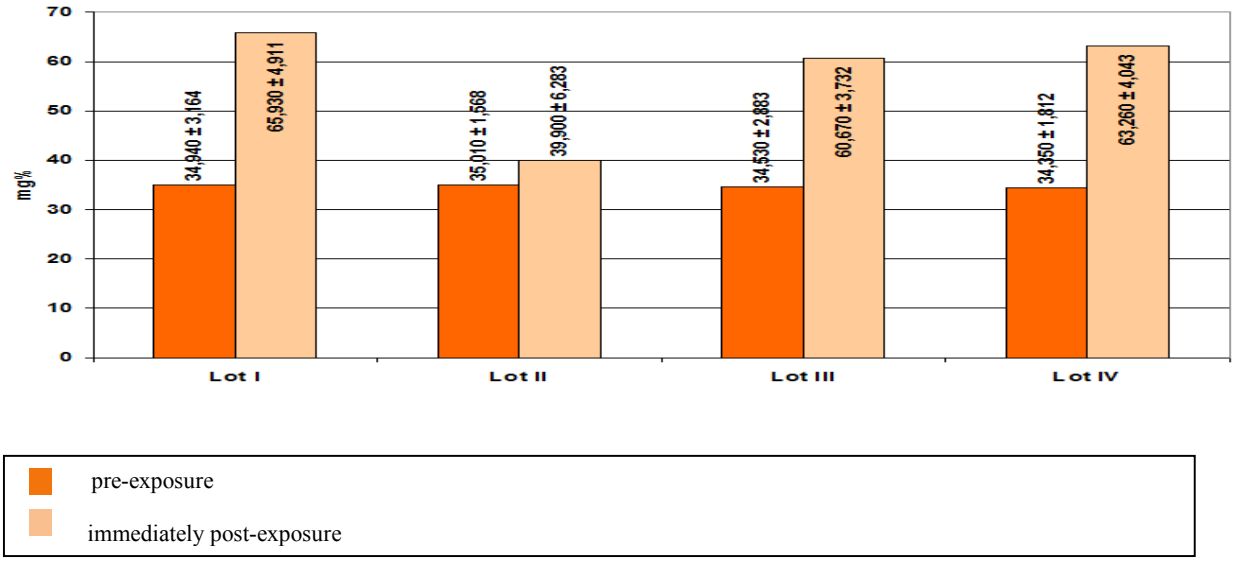

Fig. 6 Ceruloplasmin

The mobilization and release of catecholamines (CA) is regulated by nervous factors (sympathetic centers) and humoral factors (e.g. hypoxia through low partial O_2 pressure in the blood). Adrenaline (A) is released during short moderate effort and noradrenaline (NA) during intensive and prolonged effort and fatigue. CA have the following roles in the body:

- to accelerate neuromuscular impulse transmission increasing the muscle contractility-
- to mobilize the energy sources of carbohydrates and lipids;

- to activate the cortex, the spinal cord, the reticular formation, increasing the psychoneuromotor and intellectual performance
- to increase the cardiovascular activity.

The sympathoadrenal hyperactivity during intense effort on hypersensitive subjects can have negative effects both on their health and on their performance.

In such cases it is used psychotherapy or non-doping medication.
AC dosages allow specifying the degree of psychological training for competition and the dynamic tracking of training improvement and performance.

Increased urinary excretion of amines during hypobaric hypoxia is in agreement with some literature data on the activation sympathoadrenal during acute or chronic hypoxia through decompression or at altitude and some data on the level increase of plasma serotonin. At rest there have not been found differences in the sympathetic muscle activity depending on the type of training [10]. During short efforts, the sympathoadrenal system activation is faster and bigger; during prolonged effort, well-trained athletes undergo less changes [11].

Untrained subjects show an increased sympathetic activation. Other recent data confirm the increased plasma concentration of A and NA during effort; NA response under effort and training depends both on the absolute and relative to that effort [12]. The results obtained by Koski indicate the attenuation of plasma NA activation in the trained subjects. It should be emphasized that in the trained subjects occurs balanced pluriglandular activation, both at rest and during exercise [13].

During sub-maximal effort performed at altitude (4540 m) the CA level in the blood increases more than at sea level. At 1800 m plasma NA level increases significantly post effort [14]. In women, in contrast to men, there are no training effects (Wingate test) on plasma CA profile [15]. Endocrine responses during exercise are therefore modulated by the intensity and physical loading duration and also by many physical factors: the initial level at rest, the degree of fatigue, the psychological motivation, the endogenous biorhythms the hormone elimination rate (Table I) [16].

Table I.

Catecolamins modifications under acute hypobaric hypoxia conditions

Species	Hypobaric hypoxia conditions	Modification	Authors
human	Intense cronic hypoxia (4560 m)	↑ blood and urinary CA ↑ VMA elimination	Cunningham
human	Moderate cronic Hypoxia (3658 m)	↑ urinary CA in symptomatics ↓ urinary CA in asymptomatics	Honn s.c.
human	Moderate cronic hypoxia (3850 m)	↑ NA elimination, lack of response MSR	Pace quoted by Cunningham
human	Cronic hypobaric hypoxia (over 3000 m)	↑ CA urinary eliminations (incresed levels in the people living at altitude compared to those living in the plain)	Sharma
goat	Cronic hypoxia	↑ plasma NA	Maher
Rats/guinea pigs	Moderate cronic hypoxia (2200 m)	↑ urinary A and NA; in the first days moderate production from the ADM (adrenal medulla)	Groza

Obs. The differentiated eliminations: A at simulated altitude, NA gradually during ascents to high altitude. The sympathoadrenal system response does not appear during gradual solicitation. The sympathoadrenal reaction to hypoxia is amended after 3 days of exposure [17].

Currently, it is known that during effort the catecholamines (CA) increase about 2-6 times, and are involved in the rapid adaptation to effort both in the initial stage and stage of stable adaptation and restitution. catecholamines are involved in the effort in several ways:

- mobilization of energy sources;
- activation of brain structures: the somatomotor, reticular formation, limbic system, with a role in enhancing performance;

•activation of the cardiovascular and respiratory;
• changes in oxidative metabolism of the skeletal muscle and myocardium.

CA-induced changes upon the muscle oxidative metabolism are done through the activation of β-adrenergic receptors and through them the mitochondrial ROS production increases.

Lipoperoxidation induced by stress during strenuous physical effort can be attributed to the CA-Fe^{2+} complexes [18]. CA causes an increase in the mitochondrial lipoperoxidation in the presence of NADPH, when NA + Fe^{2+} or A + Fe^{2+} complexes from ferritin. Intense effort is associated with local acidosis, which induces Fe^{2+} release. Also, the acidosis causes damages biomembranes, the distortion of the enzymes containing Fe and its release. Moreover, the autoxidation of adrenaline to adrenochrom is associated with the formation of $O_2^{\bullet -}$ in the heart during ischemia/reperfusion. β-blockers (e.g .propranolol) reduce plasma markers of oxidative stress in humans who make a high intensity effort [19].

The sympathoadrenal system activation with release of catecholamines - adrenaline (A) and noradrenaline (NA) - takes place in the initial phase, in the stable adaptation phase and also at rest after making the effort.

The older data are summarized in table II

Table II

The response of the sympathoadrenal system under effort

Indicator	Sympathoadrenal response	Observations
Adrenal medulla	A and NA depletion	Experiments on rodents
CA level in the blood	Increases	Determinations on athletes also.
Urinary CA excretion	Increases (more intense for untrained subjects and under anaerobic efforts)	Multiple information from different categories of athletes
VMA urinary excretion	Increases	-
Cardiovasculat tests	Sympathoadrenal activation	-
Electrodermal activity	Activation	-
Biocurrents of the sympathetic vegetative nerves	Intensification	Wallin method

(according to [20], [21], [22])

The CA modifications under effort and training are summarized in table III.

Table III.

The CA response under acute effort and training

Hormone	*Modifications under acute effort*	Modifications under training	Significance under physical training
Adrenaline	Reduced modifications during short periods of light effort. Increase along with the increase in effort intensity and duration.	Increases less for the same absolute effort rate	The glycemia increases through hepatic and muscle glycogenolysis Increase in muscle blood flow, heart contractility,and cardiac frequency. Increase in O_2 consumption
Noradrenaline	Relative increase stressed at the same time with the increase in the effort rate.NA produces by the sympathetic neurons and the adrenal medulla.	Increases less for the same absolute effort rate	The blood pressure control , the cardiac frequency, the heart contractility and vasomotricity .

(according to[21], modified)

During intense effort, but without depletion, the sympathoadrenal system activation predominates, followed after a latency by the activation of hypothalamic-pituitary-adrenal (corticosteroids act permissively upon the CA and other hormones such as: GH hormone, glucagon, the antidiuretic hormone,the thyroxine.[23], [24].

During moderate prolonged exercise, prevails the action of glucocorticoids and the endocrine activation of above-mentioned hormones is maintained.

During the competitive effort, characterized by the endocrine-metabolic and psychoemotional activation prevails the sympathoadrenal system stimulation.

The research observed the maximum aerobic capacity of animals under conditions of normobaric normoxia, after an acute exposure to hypobaric

hypoxia corresponding to the altitude of 1500 and 2500 m and the influence of hypoxia on the O / AO balance. The altitudes were chosen because at these altitudes, in many European countries exist mountain resorts for leisure and performance sports. Exposure duration of 48 hours was chosen taking into in account comments from the literature regarding the early adaptation to hypobaric hypoxia and the level increase of 2,3 DPG, during the effort at 60 min and with maximal values at 48 hours, during human ascent to high altitudes [1], [25], [26].

The increase in the maximal aerobic capacity during exercise for untrained animals, but exposed to moderate hypobaric hypoxia, could be explained by:

•the mobilization of stored energy substances, under the influence of the ergotropic effects of the sympathetic nervous system [22], [27], [28] (Fig. 7 and 8);

• poliglobulia produced by the mobilization of haematids deposits (splenocontraction) and the improvement of O_2 intake to tissues under the influence of the sympathoadrenal activation;

• the increase of 2.3 DPG intraerythrocytic concentration, which moves the dissociation curve of HbO_2 to the right and reduces Hb affinity for O_2, as a result of intraerythrocytic pH followed by the release of large amounts of O_2 required for cellular oxidation (Edwards 1971, Neville, 1977, Lacroix, 1980, quoted by [17]).

This experiment aimed at dissociating the effects of acute hypoxic stress from the hypoxic stress associated with physical stress caused by exercise and the implications for the O / AO balance. The sympathoadrenal activation induced by the acute hypobaric hypoxia can lead *indirectly* to oxinit oxidative stress (Fig. 8), in addition to the *direct* effect of the hypobaric stress. In the case of the hypoxic stress associated with physical stress interferes the direct effect of hypobaric stress and physical stress on the oxinit oxidative stress and the indirect effects produced on the one hand by the physical stress and the sympathoadrenal activation and the sympathoadrenal activation on the other hand, which generates oxinit oxidative stress (Fig. 8).

The persistence of the aerobic capacity increase effect up to 72 hours after returning to the normoxic conditions has been also noticed in untrained animals by Giurgea, who highlighted the energizing effect of hypobaric hypoxia for 48 hours, corresponding to the altitude of 2000 m, where the performances increase at the same time the intraerythrocytic 2.3 DPG increases [17], [30], [31].

The results provide further arguments related to the biochemical SON under conditions of physical effort under normoxia for hypoxic animals, in which the AO defense capacity (H donors and the ceruloplasmin) increases significantly from the pre-exposure values [].

The immediate favourable effects of acute and moderate hypobaric hypoxia upon the aerobic effort capacity, which were showed experimentally, could be used to improve sportive performance after only 48 hours stay at a moderate altitude of up to 2500 m.

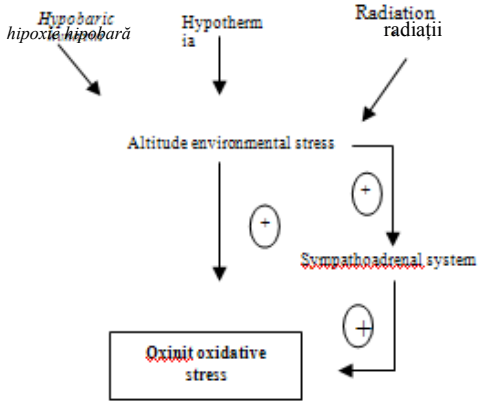

Fig. 7. Exposure to moderate acute and its effects upon hypobaric hypoxia oxinit oxidative stress

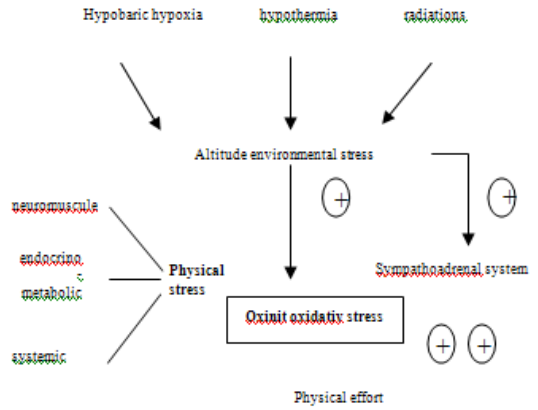

Fig. 8. Exposure to moderate acute hypobaric hypoxia, physical effort under normobaric normoxia conditions and its effects upon the oxinit oxidative stress

V. CONCLUSIONS

a) The acute exposure for 48 hours to moderate hypobaric hypoxia corresponding to the altitude of 2500 m influences favourably the maximal aerobic capacity under normobaric normoxia in rats and causes a moderate increase in OS indicators, together with an increase in AO.

b) The acute exposure to moderate hypobaric hypoxia, corresponding to the altitude of 2,500 m causes post exposure and post effort under normobaric normoxia, a lesser increase in the OS indicators and a significant increase in the AO indicators compared with the exposure to 1500 m.

c) The acute exposure to moderate hypobaric hypoxia corresponding to the altitude of 1500 m does not affect maximum aerobic effort capacity under normobaric normoxia in rats and causes significant increases in the OS indicators simultaneously with the increase in DH and CRP.

d) The acute hypobaric hypoxia for 48 hours corresponding to the altitude of 2500 influences favorably the aerobic effort capacity under normobaric normoxia conditions.

e) The favourable effect of the acute moderate hypobaric hypoxia corresponding to the altitude of 1500 and 2500 m on the aerobic effort capacity is not maintained over time.

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