

# Performance, Metabolic and Oxidant/Antioxidant Response Were Improved By Hyperbaric Oxygen in High Performance Runners

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QV312,QU125,QV325 }

**Abstract- Background:** There are no studies that evaluate the effects of an hyperbaric session (HBO) on the athletes of different performance levels. **Methods:** 34 endurance runners were divided into 2 groups: experimental (HBO session, n=22) and control (without HBO session, n=12). Two scaled tests (5x2000m) were carried out, one 4 days before an HBO session and the 45 minutes after (2.5 ATA, 75 minutes). The metabolic, hematological and oxidant/antioxidant responses were determined before and after this session. **Results:** A significant increase in the anaerobic threshold (AT, 4mmol/l lactate) and maximum test velocity was observed only in the experimental group. The high performance runners showed a greater increase of AT and decrease of glucose, lactate, triglycerides, TBARS and GPx activity during the HBO session, compared with low performance runners. Urea significantly decreased in both groups. **Conclusions:** One HBO session showed a greater improvement in the performance as well as metabolic and oxidant/antioxidant response in high performance runners.

**Keywords-** hyperbaric oxygen, performance, anaerobic threshold, marathon, oxidative stress.

## I. INTRODUCCION

The principal problem for elite athletes is their recovery after intense training. Among the recovery methods currently being discussed is the use of hyperbaric oxygen (HBO) sessions (Delaney and Montgomery, 2001; Staples et al. 1999). The scarce data available on the effect of an HBO session on the physical performance of sedentary individuals or trained athletes in normobaric conditions is contradictory, ranging from an increase (Cabric et al. 1991; Staples et al. 1996; Gardette et al. 1999) to no change (Hoffman et al. 1990; Webster et al. 1998; McGavock et al. 1999; Hodges et al. 2003). The scarce data available on the effect of an HBO session on the physical performance of sedentary individuals or trained athletes in normobaric conditions is contradictory, ranging from an increase (Cabric et al. 1991; Staples et al. 1996; Gardette et al. 1999) to no change (Hoffman et al. 1990; Webster et al. 1998; McGavock et al. 1999; Hodges et al. 2003). It seems improbable that reserve oxygen from HBO affects performance, as was assumed in some studies (Staples et al. 1996; Cianci, 2004). The metabolic effects of an HBO session are unclear. According to recent data,

carbohydrate and lipid oxidation is not feed during an HBO session (Stellingwerff et al. 2005) but in different studies an inhibition of glucoytic enzymes was observed after an HBO session (Weglicki et al. 1966; Hodges et al. 2003), which according to the authors can stimulate the use of lipids as a source of energy, resulting in a decrease of lactate production. A recent study shows that during an HBO session there was an increase in the concentration of erythropoietin (EPO), followed by an exponential decrease in the same (Balestra et al., 2006). This hormone improves the performance of elite athletes and has been used as a doping in high performance sports. It is possible that negative effects can be induced by an HBO session by the oxidative stress (OS) that may result from a drastic increase in the concentration of oxygen in tissues, the latter having been detected by the increased concentration of peroxidation products in the blood. There are contradictory reports in this sense, ranging from an absence of changes (Muth et al. 2004) to an increase in this parameter (Bader et al. 2006). Furthermore, studies measuring the antioxidant response have also shown contradictory results (Dennog et al. 1999; Muth et al. 2004; Bader et al. 2006). It is obvious that there is still too little data to have a clear idea about the real effects of an HBO session on the performance and metabolic response of athletes, much less about the differences in such effects between athletes of different performance. The fact that anaerobic threshold (4 mmol/l of lactate), which was determined by the lactate response during a graded maximal exercise testing under a steady lactate state, correlated with the competitive velocity in a long distance runners (Yoshida, 1989; Sjödin and Jacobs, 1981; Mader, 1991) permits it to be used as a parameter of physical performance in runners. The aim of this study was to evaluate the effect of an HBO session on the anaerobic threshold in runners of different performance compared with control group as well as the metabolic, hematological and oxidant/antioxidant response during the session.

## II. METHODS

### 1) Subjects

Of the 34 runners who completed the study, all were in good health, were not under any pharmacological treatment, and had at least 8 years of continuous training. Written informed consent was obtained from each participant and the protocol was approved by the institution's ethics committee. The study was carried out in Mexico City (2200m), where all study participants were born and had been trained. These

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athletes were randomly divided into experimental (n = 22) and control (n = 12) groups. After completing the study protocol, the runners of experimental group were categorized based on their anaerobic threshold (AT) as high performance and low performance subgroups (Table 1). 27 runners of 34 had previously run in a marathon and their average time was presents in this Table 1.

## 2) Test protocol and training

The same graded maximal running test (scaled test) of 5x2000m was carried out by all athletes on a stadium track, lasting 40-60 minutes depending on the performance level. The individual intensity of running in the test protocol was determined in this test conducted 1 to 2 months before the present study, at which time at least three of five measurements of lactate for each athlete were within the 2 - 6 mmol/l range of lactate. There are three main reasons for using this protocol: 1) the duration of each scale in the test (minimum 6 minutes) allows for a steady state of lactate to be reached in the blood; 2) this test is part of the regular evaluation of the runners; 3) up to 10 athletes can be evaluated during one hour after an HBO session. We were able to increase the accuracy of the test by a previous adaptation period, standardized warm-ups, control of the velocity of the runners each 400m, and strict control of rest periods of 3 minutes between each run (5 minutes before ultimate run). The scaled test was carried out in the experimental group 4-5 days before the HBO session and 45±15 minutes after the same. For the control group the same two tests were conducted with a difference of 4-5 days (at the same time of day and in the same way as with the experimental groups), but without any HBO session. The entire study was conducted during a period of three weeks. The study was conducted in the middle of the general training, when the running distance was at the highest level for all runners.

## 3) HBO session, sampling and measurements

The HBO session was carried out in the morning (7-10am) 1-2 hours after a light breakfast (bread and juice) in the multiplaza chamber (2.5 ATA, 100% O<sub>2</sub>, 10 min. pressurization + 50 min. + 15 min. depressurization). Capillary blood samples were taken 10 minutes before and immediately after the HBO session to determine metabolic, hematological and oxidant/antioxidant response in serum and erythrocytes. During the scaled test, blood samples were taken from the ear lobe within 1-2 minutes after finishing each 2000 m run and at 7 minutes of recovery after the last stretch. The average pulse was measured with an electronic pulse meter during the test run and the first 7 minutes of recovery. In the control group two capillary blood samples were taken with a difference of 2 hours (at the same time period as experimental group). The runners of the control group had the same light breakfast as the athletes of HBO group. Blood samples were processed on the same day and hematological parameters were determined immediately with QBC (Becton Dickinson) equipment. Metabolic parameters were assessed with a routine procedure

(RANDOX). Thiobarbituric acid reactive substance (TBARS, nmol/ml) and the velocity of TBARS production (TBARS-VP, nmol/ml/ min) induced by the Fenton reactive were measured in serum according to a modified method (Hicks et al., 1995). The total serum antioxidant capacity (AOC) was calculated in relative units as the opposite of the TBARS-VP  $[100-(TBARS-VP)/1.5]$  that reflected principally non-enzymatic AOC because principal antioxidant enzymes are in erythrocytes. The GPx and SOD activity were measured in erythrocytes with RANDOX procedure. The results are presented in U/grHb.

## 4) Statistical analysis

All values represent the mean ± SD. Statistical analysis of data included comparison of test variables before and after the protocol period, within and between groups by the Student t-test. The Pearson's correlation coefficient was calculated for evaluation of the relation between two variables.

## III. RESULTS

### a. Data from the scaled test

The data of scale test for all runners before and after the HBO session was presented in Table 2. Whereas there was no significant change found in measured parameters in the control group, a significant increase in AT and maximum test velocity (V<sub>m</sub>) was found in the experimental group, which corresponds to the increase in performance in the latter runners. There was no difference in the basal levels of AT or V<sub>m</sub> between the experimental and control group, but the levels of these parameters after the HBO session were greater in the experimental group (p<0.001). There were no differences between the experimental and control groups in the other parameters. The cardiovascular load (maximum pulse) found by the test was similar in both groups. The greater performance of the runners corresponds to a lower Lm/V<sub>m</sub> ratio (lower lactate at greater velocities). During the short time period of the protocol (one week) and with a similar cardiovascular load, the metabolic adaptation to training is minimal and the level of Lm/V<sub>m</sub> is determined only by the change in lactate level. If we compare change of AT ( $\Delta$  AT) in the experimental group during the study with the basal level of Lm/V<sub>m</sub>, there is not any correlation. However, there was a significant negative correlation between  $\Delta$  AT and Lm/V<sub>m</sub> after the HBO session (r = 0.62, p<0.05) (Fig. 1a) and between  $\Delta$  AT and  $\Delta$ Lm/V<sub>m</sub> (r = -0.74, p < 0.001) (Fig. 1b). Given that the cardiovascular load during test is similar, the increase in AT most probably was determined by the decrease in lactate production after the HBO session. The performance of the runners measured by AT varies in HBO and control groups (Table 1). The scale test data on the low and high performance subgroups is presented in Table 3.  $\Delta$ AT was 2.2 times greater in the HP than the LP subgroup (p=0.021). This parameter was significantly greater in the HP and LP groups than the control group (p=0.001 and 0.052, respectively). Whereas the Lm/V<sub>m</sub> showed a significant decrease in the HP group after the HBO session, there was a tendency to an increase

(14%) in the LP group, resulting in a significant difference in the  $\Delta Lm/Vm$  ( $p=0.015$ ) between these two groups. This data demonstrates that the increase in AT as well as the decrease in  $Lm/Vm$  was greater in the HP runners. There was no difference in response between HP y LP runners of control group

*b. Metabolic, hematological and oxidant/antioxidant response*

In the Table 3 basal blood parameters of all runners are presented. No significant differences were found in the basal values of any blood parameters between the groups, which permitted us to express the changes after an HBO session in percentages (Fig. 2). A decrease was observed in the four main metabolic parameters only in the experimental group, being significant in glucose, lactate and urea (Fig.2a). These changes were determined principally by changes in HP subgroup (Fig.2b). In the LP group the only significant decrease was in the level of urea. That is, on the one hand the metabolic response was greater in the HP group, and on the other hand the change in urea does not depend on the performance level of the runners. There was no significant change in the CK activity, cholesterol, total protein, hemoglobin, hematocrit or platelets of any group, which confirms that there was no significant change in plasmatic volume during the HBO session. There was a tendency to an increase in granulocytes and a significant increase in agranulocytes as a result of the HBO session (Fig.3a) only in the experimental group. This increase in white blood cells (WBC) count in the experimental group was determined principally by the response in the LP subgroup (Fig.3b). It is logical that the effect of intense exercise is greater for low performance runners in both muscular tissue and the immune response. Regarding the oxidant/antioxidant response during an HBO session (Fig.4), there was a significant decrease in the experimental group in the level of TBARS and AOC ( $p=0.028$  and  $0.008$ , respectively), whereas the change in GPx bordered on significance ( $p=0.056$ ) and no change was observed in the SOD activity (Fig.4a). The level of TBARS after the HBO session (Fig.4) was significantly less than that found in the control group ( $p<0.05$ ). In the Table 4 was presented data of oxidant/antioxidant response in HP and LP subgroups. There were differences in basal levels of AOC ( $p<0.01$ ) and GPx ( $p<0.05$ ) in this subgroups. Given that there was a significant decrease in TBARS and GPx in the HP subgroup but not the LP subgroup (Fig.4b), the oxidant/antioxidant response of the experimental group in these parameters was determined mainly by the results of the HP subgroup. A significant decrease of total serum nonenzymatic AOC was observed in the LP subgroup, whereas there was only a tendency to such a decrease in the HP subgroup.

#### IV. DISCUSSION

*a. Effects on performance*

There are few studies about the effects of an HBO session on the performance and metabolic response of athletes. One study with two athletes showed a decrease of ventilation,

$VO_2$  and lactate concentration during the submaximum exercise of 40 minutes after the HBO session (2 ATA, 70 min.) (Banister et al. 1970). In another study with untrained women evaluated on the treadmill, there was an increase of  $VO_{2max}$  (14.4%) and physical performance (12.8%) 30 minutes after an HBO session (2.8 ATA, 60 min.), an effect that gradually diminished until disappearing six hours later (Cabric et al. 1991). There are other reports of an increase in maximum velocity and  $VO_{2max}$  observed in trained runners 30 minutes after an HBO session (2.8 ATA, 60 min.) (Staples et al. 1996) and an increase of maximum velocity for 4 out of 9 runners found after an HBO session (2.2 ATA, 3x30 min.) in a 5km race (Prather and Wilson, 2004). Still another study reported that 12 sessions of training during one month conducted under HBO conditions (1.6 ATA, 90 min.) increased physical performance in trained weight lifters as well as in trained cyclists compared with the control group (Gardette et al. 1999). Contrarily, such effects were not found in other studies: 1) in untrained individuals who were evaluated in cycloergometer (2.0 ATA, 60 min.) (Webster et al. 1998); 2) in 12 trained runners (2.5 ATA,  $O_2$ , 90min.) (McGavock et al. 1999); 3) in 10 runners (Hodges et al. 2003); and 4) in cyclists (1.5 ATA, unspecified length of the session and time elapsed before testing after exercise of 70%  $VO_{2max}$ ) (Hoffman et al. 1990). Owing to the differences in test and HBO session protocols, performance of the individuals, types of exercise, and measured parameters, it is difficult to compare these studies. In the current study a significant increase was observed in AT and Vm after an HBO session, and this increase depended on performance: higher performance corresponded to a significantly greater increase in AT. This result coincides with data (Welch, 1982; Linossier et al. 2000) on the effect of normobarichyperoxia on AT: an increase was found in individuals with high physical performance. Although the results of the current study contradict another report on an HBO session (Webster et al. 1998), it is difficult to compare, as steady state lactate levels were not achieved in the blood during the latter study. Lactate levels were measured at the end of a short scaled test (7 minutes) with a final lactate level of 5mmol/l and 10mmol/l at 5 minutes of recovery. Another study with untrained individuals found no decrease in maximum test lactate level or increase in  $VO_{2max}$  after an HBO session (Hodges et al., 2003) and a short (10 minutes) scaled test that included a change of velocity every minute. There was no lactate control during the exercise or recovery period. We suppose that these differences in the performance of athletes and the test protocols determined the distinct results. The negative correlation between  $Lm/Vm$  after HBO session and  $\Delta AT$  in the experimental group of the current contribution shows that the runners with a lower lactate per km of maximum velocity responded in a scaled test with the greatest increase in AT, indirectly confirming that the decrease in lactate production is responsible for the increase in AT. In various studies an inhibition of glycolytic enzymes was observed during and after an HBO session (



Weglicki et al. 1966; Cabric et al. 1991). In this case, there must have been a stimulation of the use of lipids as a source of energy to compensate for the decrease in glycolysis, a conclusion reached in other studies on normobaric hyperoxia (Hughes et al. 1968; Welch et al. 1974) as well as hyperbaric hyperoxia (Cabric et al. 1991; Weglicki et al. 1966). Thus the greater increase in performance of the HP subgroup compared to the LP subgroup could be determined by the greater capacity of the former to utilize lipids as a source of energy. The possibility of an increase in the elimination of metabolic waste can be discarded, as there was a 20 to 30% decrease in blood flow in different organs, including the kidney (Muhvich et al. 1992). That is, this change in blood flow would tend to decrease such elimination. In this study a significant decrease in urea was observed in all runners of the experimental group during and after an HBO session, reflecting a decrease in protein catabolism. Under conditions of diminished glycolysis, this decreased protein catabolism can be accounted for in two ways: 1) an increase in the participation of lipids in the energy supply, which would diminish the necessity of protein participation, and/or 2) a decrease in the energy demand because of the elevated concentration of oxygen in all tissues. The latter explanation has been put in question by various researchers (Webster et al. 1998; Weglicki et al. 1966), who show that the basal metabolic level does not change during an HBO session. The significant decrease in glucose and lactate observed in the HP subgroup (Fig.2) can be explained by the greater participation of lipid metabolism in the energy supply. The WBC count increased after the HBO session only in the LP group, in granulocytes as well as agranulocytes. Several studies have demonstrated that hyperbaric oxygen decreases adhesion of leukocytes (Labrousche et al. 1999), increasing their concentration in the blood flow. Hence, it is logical that there is less WBC response in high versus low performance athletes.

#### *b. Oxidant/antioxidant response*

It is supposed that hyperbaric oxygen causes an increase in oxidative stress (OS) in trained athletes due to an increment in ROS generation (Bader et al. 2006). In the current study significantly greater basal levels of TBARS, non-enzymatic AOC and GPx activity were found in the HP subgroup than the LP subgroup, which confirmed the greater basal level of OS and antioxidant capacity in elite runners. This probably reflects the greater volume and intensity of training in the former subgroup. GPx activity is directly related to peroxidation processes. In this study hyperbaric oxygen did not change SOD activity, which coincides with data from another study (Dennog et al. 1999). Surprisingly, an HBO session provoked a significant decrease in serum TBARS and GPx activity in erythrocytes only in the HP group. This result can be principally attributed to the greater basal level of non-enzymatic AOC and GPx in the HP than LP group, as the reduction of blood flow in all organs during the HBO session rules out the possibility that this is related to the elimination of metabolic waste. Contrarily, this decreased circulation should contribute to an increased

concentration of TBARS in the blood. The similar response of TBARS, GPx, total nonenzymatic AOC and the majority of the

## V CONCLUSIONES

The improvement in the AT of trained runners as a result of hyperbaric oxygen depended on their performance level, with a greater increase in AT corresponding to a greater performance. The metabolic and oxidant/antioxidant response during an HBO session was significant only in the high performance subgroup, resulting in a decrease in the majority of parameters, which confirms the importance of the performance level of participants in relation to these responses. It can be assumed that the performance improvement of the HP group was determined by the decrease in lactate production, probably due to the stimulation of the use of lipids as a source of energy.

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TABLES OF ARTICLE –PERFORMANCE, METABOLIC AND OXIDANT/ANTIOXIDANT RESPONSE IS IMPROVED BY HYPERBARIC OXYGEN IN ELITE RUNNERS”

Table 1. Data of runners

group s	subgro ups	N	sex (m+f)	age years	weight kg	AT range km/h	maratho n minutes
HBO	HP	12	8 + 4	29.5 ± 9.1	63.6 ± 6.9	14.7-18.9	170 ± 21
	LP	10	7 + 3	32.6 ± 8.7	65.8 ± 7.2	12.1-14.0	219 ± 35
Contr ol	HP+LP	5+7	8 + 4	32.8 ± 9.5	65.3 ± 4.8	12.2-18.3	192 ± 39

HP- high performance, LP – low performance

Table 2. Scaled test parameters before and after the HBO session in experimental and control groups. \* - p<0.05; \*\* - p<0.01 compared to initial level

	HBO group		control group	
	before	after	before	after
AT	14.5 ± 2.1	15.2 ± 2.2**	14.4 ± 2.1	14.5 ± 2.1
Vm	16.2 ± 2.5	16.8 ± 2.5**	15.9 ± 2.0	16.1 ± 1.9
Lm	7.0 ± 2.2	6.8 ± 2.1	7.6 ± 1.8	7.4 ± 1.8
L7rec	6.8 ± 1.8	6.6 ± 1.6	7.4 ± 1.8	7.5 ± 2.1
Lm/Vm	0.43 ± 0.11	0.41 ± 0.12	0.47 ± 0.12	0.46 ± 0.11
Pm	177 ± 17	176 ± 14	174 ± 19	176 ± 19
P7rec	105 ± 11	103 ± 9	107 ± 11	104 ± 9
P(AT)	167 ± 10	163 ± 11	165 ± 13	163 ± 14

Table 3. Basal blood parameters in experimental and control groups

Parameters/ Groups		HBO	C
Glu	mmol/l	4.4 ± 0.7	4.2 ± 0.8
TG	mmol/l	1.4 ± 0.6	1.4 ± 0.4
lactate	mmol/l	2.0 ± 0.2	1.9 ± 0.3
urea	mmol/l	4.9 ± 1.1	5.8 ± 0.9
cholesterol	mmol/l	5.1 ± 0.6	4.8 ± 0.9
proteins	g/dl	6.8 ± 0.6	7.2 ± 0.8
CK	u/l	64 ± 24	79 ± 34
HCT *	%	49.9 ± 3.2	48.9 ± 3.0
hemoglobin	g/dl	16.4 ± 1.0	16.2 ± 1.5
PLT	1000/μl	253 ± 69	266 ± 33
granulocytes	1000/μl	3.7 ± 0.8	4.1 ± 0.6
agranulocytes	1000/μl	2.3 ± 0.5	2.4 ± 0.8
TBARS	nmol/ml	11.6±9.6	9.8±3.2
AOC	units	56.1 ± 7.6	58.3±7.6
GPx	U/grHb	49 ± 27	57 ± 11
SOD	U/grHb	987±127	991±84

\* - the elevated HCT levels result from the altitude (2200m) of Mexico City

Table 4. Oxidant/antioxidant response in HP and LP subgroups after HBO session. \*-p&lt;0.05

		HP		LP	
		before	after	before	after
TBARS	nmol/ml	14.9 ± 11.1	7.3 ± 2.9*	7.2 ± 4.2	7.4 ± 4.5
AOC	units	64 ± 8.1	60 ± 10.6	57 ± 2.8	50 ± 6.1*
GPx	U/grHb	64 ± 27.4	49 ± 30.3*	38 ± 19.7	38 ± 12.5
SOD	U/grHb	987 ± 127	1006 ± 110	977 ± 82	939 ± 110

FIGURES FOR ARTICLE “PERFORMANCE, METABOLIC AND OXIDANT/ANTIOXIDANT RESPONSE IS IMPROVED BY HYPERBARIC OXYGEN IN ELITE RUNNERS” (has vinculum with excel archive)

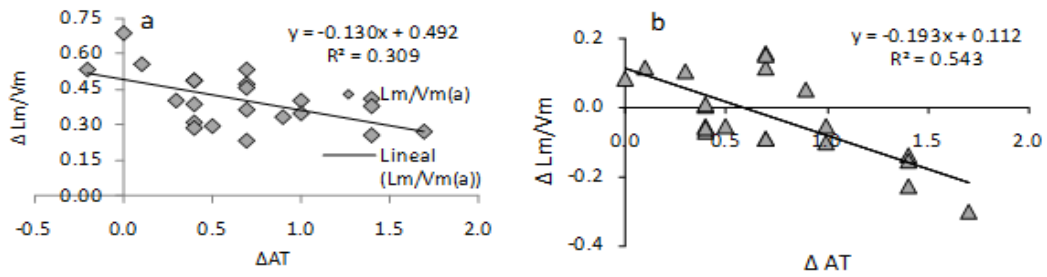


Fig 1. Relation between Δ AT and Lm/Vm after HBO session (a) or ΔLm/Vm (b) in all runners of experimental group

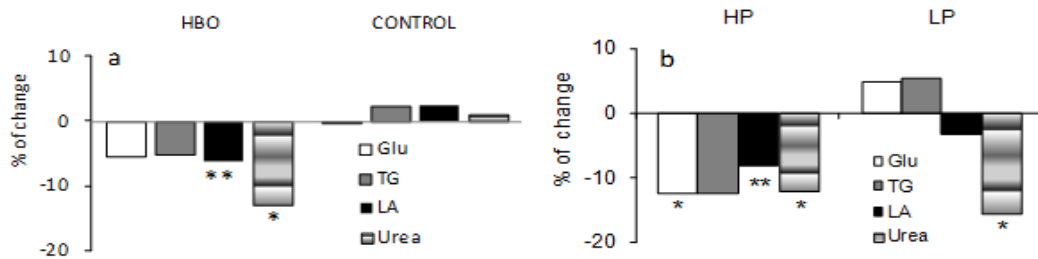


Fig. 2. Percentage of changes in metabolic parameters en HBO y control groups (a) and performance differences (b). Glu – glucose, TG – triglycerides, LA – lactate, HP – high performance, LP – low performance. \* - p<0.05; \*\* - p<0.01

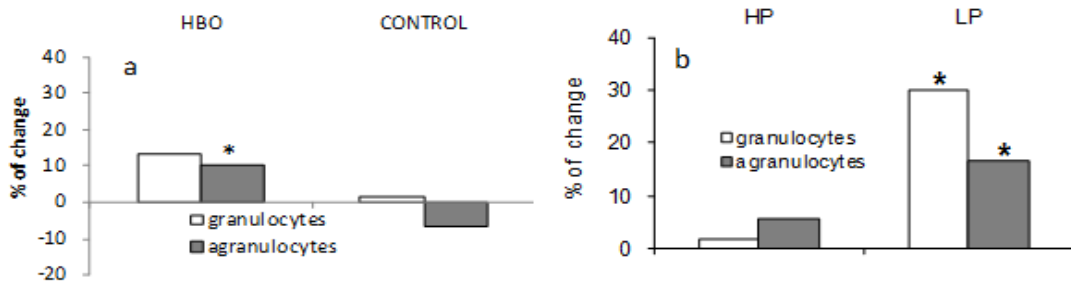


Fig 3. Percentage of changes in different WBC in experimental and control groups (a) and performance differences (b). HP – high performance, LP – low performance. \* - p<0.05

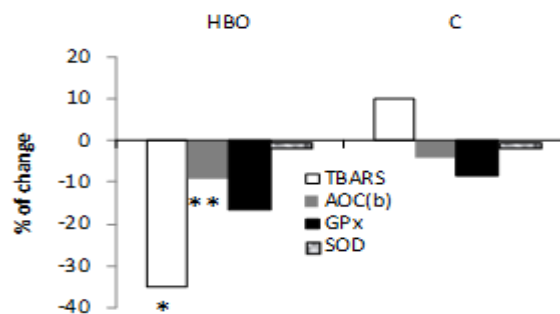


Fig.4. Oxidant/ antioxidant response of experimental and control groups. \* - p<0.05, \*\* - p<0.01