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ACUTE REPEATED WINGATE STYLE HIIT EXERCISE IN HYPOXIC CONDITIONS DOES NOT ALTER OXIDATIVE STATUS IN UNTRAINED MEN

AKUTNA PONAVLJAJOČA SE WINGATE HIIT VADBA V HIPOKSIČNIH POGOJIH NE SPREMENI OKSIDATIVNEGA STANJA PRI NETRENIRANIH MOŠKI

ABSTRACT

Both high-intensity interval training and training in hypoxic conditions have gained popularity in recent years due to their cardiorespiratory and metabolic benefits. Recent studies focus on the combined effects of both factors in detail. But there is still a scientific gap in the literature on the effects caused and its magnitude in response to this type of training. Therefore, this study aims to investigate the acute effects of Repeated Wingate Style HIIT applied in hypoxic and normoxic conditions on oxidant and antioxidant indicators of recreationally active men. Sixteen participants were randomly assigned to normoxic or hypoxic groups and then they were completed a wingate style 30s*4rps*4 min (sprint time * repeats * recover between sprints) HIIT on normoxic and hypoxic conditions (2500m, FiO₂: 0.130) in the normobaric environment. The normality assumption calculated using a Shapiro-Wilk test to ensure the normal distribution of the quantitative for all data ($p < 0.05$). To analyze differences in baseline data between groups an Independent Sample t-test was performed. The Two-way analysis of variance was used in repeated measurements to reveal the effects of training under hypoxia and normoxia conditions (time, time \times group interactions) oxidant and antioxidants. The activities of blood oxidant and antioxidant did not change significantly after training in the hypoxic and normoxic group. As a result, repeated Wingate style HIIT applied in hypoxic and normoxic conditions, did not change both MDA ($p=0.79$), which is an indicator of oxidant stress, and SOD ($p=0.46$), CAT ($p=0.26$), and GSHPX ($p=0.17$), which are general indicators of antioxidant defense.

Keywords: high intensity interval training, hypoxia, oxidative status

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IZVLEČEK

V zadnjih letih sta zaradi svojih kardiorespiratornih in presnovnih koristi postala priljubljena tako visokointenzivna intervalna vadba (HIIT) kot vadba v hipoksičnih pogojih. Nedavne študije se podrobno osredotočajo na kombinirane učinke obeh dejavnikov. Zato je namen te študije raziskati akutne učinke ponavljajoče se Wingate HIIT vadbe, uporabljene v hipoksičnih in normoksičnih pogojih na oksidativne in antioksidativne kazalnike netreniranih oseb. 16 udeležencev je bilo naključno razporejenih v normoksično ali hipoksično skupino. V normobaričnem okolju so nato v normoksičnih in hipoksičnih pogojih (2500 m, FiO₂: 0,130) opravili Wingate HIIT vadbo, sestavljeno iz štirih 30-sekundnih šprintov, med vsakim šprintom pa so imeli udeleženci štiri minute pasivnega počitka. Aktivnosti oksidacijskih in antioksidativnih encimov v krvi se po treningu v hipoksični in normoksični skupini niso bistveno spremenile. Zato pri netreniranih posameznikih takoj po ponovljivi Wingate HIIT vadbi, ki se je izvajala v hipoksičnih ali normoksičnih pogojih, niso bile ugotovljene pomembne spremembe aktivnosti MDA ($p=0.79$), SOD ($p=0.46$), CAT ($p=0.26$) in GPX ($p=0.17$).

Ključne besede: visoko intenzivna intervalna vadba, hipoksija, oksidacijsko stanje

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<https://doi.org/10.52165/kinsi.28.3.154-165>

INTRODUCTION

It is frequently reported that the high intensity interval training (HIIT) method is effective in improving athletic performance and some health-related physical fitness parameters. HIIT is characterized by high intensity, short term exercise periods, and absolute rest or active recovery is applied between these periods (Tjønnå et al., 2008; Gillen & Gibala, 2014). HIIT provides fast and effective adaptation, it also shortens exercise time. In addition, it has been reported that it has positive effects on the cardiovascular system and metabolic functions (Shing, Webb, Driller, Williams, & Fell, 2013; Barker, Day, Smith, Bond, & Williams, 2014). In this regard, Repeated Wingate Style HIIT exercises have been frequently practiced recently.

Training under hypoxic conditions is also frequently used to improve physical performance (Møller, Loft, Lundby, & Olsen, 2001; Furman et al., 2018). Training in hypoxic conditions is preferred as a method for elite athletes in their preparation for the season (Rodríguez, Murio, & Ventura, 2003; Rodríguez et al., 2007). Due to the thought that training at an altitude is more effective than sea level (Czuba et al., 2011; Czuba et al., 2014).

In this context, the application of the HIIT method, which has been reported to have positive effects on health and performance in recent years, under hypoxic conditions, which have also been reported to have similar characteristics, has gained popularity (Warnier et al., 2020). Although HIIT has become a popular training modality for athletes, as well as for the general population, there is still little information on oxidative stress related to short-term training. Therefore, little is known about the effects of HIIT on oxidative stress (OS) markers, and on the magnitude of some oxidative markers (de Souza et al., 2020)

OS is caused by an imbalance between oxidation, reduction reactions. The imbalance is caused by increased production of reactive oxygen species (ROS) that exceeds the tissue's antioxidant capacity. The excess of ROS favors oxidative reactions and, consequently, cell damage (Powers & Jackson, 2008; Parker, Trewin, Levinger, Shaw, & Stepto, 2018; Zuo, Zhou, Pannell, Ziegler & Best, 2015). These events, along with post-exercise inflammatory responses, may result in muscle damage (Parker, Trewin, Levinger, Shaw, & Stepto, 2018). The degree of muscle damage and inflammation are proportional to the intensity of the exercise (Demicine et al., 2010).

The fact that acute exercise induces oxidative cell damage and thereby contributes to systemic oxidative stress has been initially established almost four decades ago by Dillard et al. (1978). However, it is important to note that exercise modulates oxidative stress in a dose-dependent

manner (Goto et al., 2003). In particular, the oxidative stress response magnitude does seem to be mainly related to the relative exercise intensity (i.e., higher intensity, higher exercise-related oxidative stress) and to a lesser extent the exercise duration (Johnson, Padilla & Wallace, 2012). Indeed, both acute (Magalhães et al., 2004; Pialoux et al., 2009; Faiss et al., 2013) as well as long-term hypoxic exposures (Joanny et al., 2001; Askew, 2002; Dosek, Ohno, Acs, Taylor, & Radak, 2007) have been shown to augment oxidative stress.

Some authors who evaluated the effects of HIIT on markers of oxidative stress and muscle damage have presented, for the most part, evidence of the existence of intercalated recovery periods between sessions (Demicine et al., 2010; Bogdanis et al., 2013; Songstad et al., 2015), but there is still a scientific gap in the literature on the effects caused and its magnitude in response to this type of training that uses consecutive short sessions with recovery periods. Thus, the objective of this study was to evaluate the effects of short-term HIIT under hypoxic and normoxic conditions on markers of oxidative stress and antioxidants.

METHODS

Participants

Sixteen recreationally active men (age: 22.50 ± 2.54 -year, bodyweight: 69.96 ± 6.4 kg, height: 173.43 ± 0.06 cm, BMI: 23.19 ± 1.43 kg/m², VO₂: 49.14 ± 3.10 ml/kg/min) voluntarily participated in this study. The exclusion criteria for study were drugs and medicines intake as well as suffering from some illness and smoking habit. The participants were those who did not exercise regularly and did not engage in high intensity exercise twenty-four hours before the study. Participants continued their normal dietary routines during the study and were warned not to take any antioxidant containing vitamin tablets. In the first interview with the participants, the purpose, procedures, and risks of the study were informed and written consent was obtained. The study procedures followed the principles outlined in the Declaration of Helsinki and were approved by Ankara University, Non interventional Clinical Ethics Committee (09-381-15).

Study Design

Sixteen participants were randomly assigned to normoxic or hypoxic groups and then they were completed a wingate style $30s * 4rps * 4$ min (sprint time * repeats * recover between sprints) HIIT on normoxic and hypoxic conditions (2500m, FiO₂: 0.130) in the normobaric environment. The hypoxic conditions were provided with Everest Summit II-Altitude

Generator (Hypoxico, USA). Normoxia altitudes were determined based on the the altitude at which test sessions were performed in Ankara, Turkey (890 m).

Repeated Wingate Style HIIT

Wingate style of HIIT program was performed on a Monark894E (Monark Exercise AB, Vansbro, Sweden) cycle ergometer. Following a 5 min warm up at 60 watts with 5 s. sprints without resistance on the second and third minutes, participants performed 4 × 30-s Wingate “all-out” sprints with 4 min of passive rest (de Magalhaes & Church, 2006; Debevec et al., 2015). The test was automatically initiated by Monark test software when participant reached ≥ 150 rpm during unloaded pedaling and subsequent instant application of load corresponding to 7.5% of body weight (de Sousa et al., 2017).

Biochemical analyses

Peripheral venous blood samples obtained before and immediately after exercise. Malondialdehyde (MDA) levels in the samples were measured by the thiobarbituric acid reactive substances (TBARS) method (Dahle, Hill, & Holman, 1962). MDA levels were expressed as nmol/mL. Glutathione peroxidase (GSHPX) activity was measured by following changes in NADPH absorbance at 340 nm (Paglia & Valentine, 1967). Catalase (CAT) activity was determined by measuring decrease of H₂O₂ absorbance at 240 nm (Aebi, 1974). In the activity calculations (IU-international unit), extinction coefficients of uric acid, H₂O₂ and NADPH were used for XO, CAT and GSHPX, respectively. Superoxide dismutase (SOD) activity was measured by the method based on nitro blue tetrazolium (NBT) reduction rate (Durak, Canbolat, Kavutçu, Öztürk, & Yurtarslani, 1996). One unit for SOD activity was expressed as the enzyme protein amount causing 50% inhibition in the NBT reduction rate. All spectrophotometric analyses were made by the UV-visible spectrophotometer (Unicam Helios alpha, England).

Statistical analysis

Mean and standard deviations or 95% confidence intervals for baseline scores, malondialdehyde, superoxide dismutase, catalase, and glutathione peroxidase for both groups were demonstrated. The normality assumption was calculated using a Shapiro-Wilk test to ensure the normal distribution of the quantitative for all data ($p < 0.05$). To analyze differences in baseline data between groups an Independent Sample t-test was performed. The Two-way analysis of variance was used in repeated measurements to reveal the effects of training under

hypoxia and normoxia conditions (time, time \times group interactions) oxidant and antioxidants. The minimum number of participants needed in the study was determined using G-Power software version 3.1.9.6 (Dusseldorf, Germany). Sixteen participants were included in the study by calculating the sample size for double-sided hypothesis testing with 80% power, effect size:0.4, and 0.05 error level. The separation of the participants included in the study into Normoxic and Hypoxic groups was carried out using the 2-block randomization method (Random Allocation Software). Statistically significant value of the study was considered as $p < 0.05$ in all calculations. For effect sizes, partial eta-square (partial η^2) was evaluated as .01 low impact power, .06 average impact power, .14 and above large impact power.

RESULTS

Table 1. Biochemical characteristics of high intensity interval training under hypoxic and normoxic conditions.

	Test	Normoxia	Hypoxia	F(df)	Time	Group	Time \times Group
		Mean \pm SD with 95% CI	Mean \pm SD with 95% CI	Sig.			
MDA (nmol/mL)	pre	263.12 \pm 60.22	263.12 \pm 21.41	F	,069	,129	,251
	post	260.60 \pm 28.42	271.14 \pm 19.24	p	,797	,725	,624
SOD (U/mL)	pre	2449.82 \pm 799.13	1930.16 \pm 1183.74	F	,555	,961	,003
	post	2410.94 \pm 1209.72	2110.53 \pm 1073.80	p	,469	,343	,955
CAT (IU/mL)	pre	32848.50 \pm 8687.06	29691.75 \pm 5232.79	F	1,35	,094	1,932
	post	28731.00 \pm 6605.41	30057.75 \pm 6150.66	p	,264	,764	,186
GSHPX (IU/mL)	pre	5.73 \pm 1.31	6.42 \pm 1.68	F	2,01	,225	,297
	post	6.81 \pm 1.95	6.81 \pm 1.95	p	,178	,642	,594

Malondialdehyde (MDA), Superoxide dismutase (SOD), Catalase (CAT) and Glutathione peroxidase (GSHPX). Values are given as mean \pm SD. A 2-way analysis of variance with repeated measure (group \times time) was used to assess training-main effects' statistical significance ($p < 0.05$).

Figure 1-4. Pre-test and post-test values in normoxia and hypoxia groups.

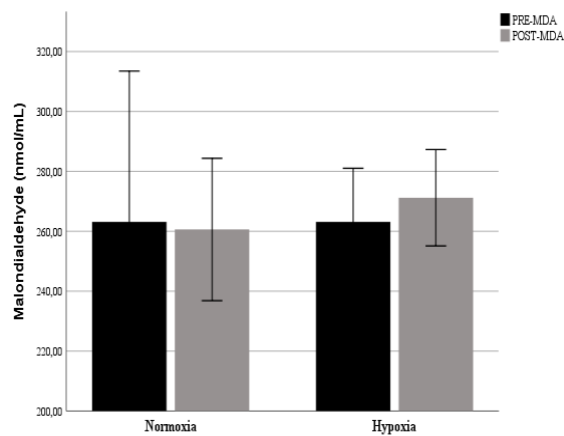


Figure 1. Results of Malondialdehyde pre and post test responses after High Intensity Interval training for both conditions (Normoxia and Hypoxia). All data executed as mean with SD for errors bar.

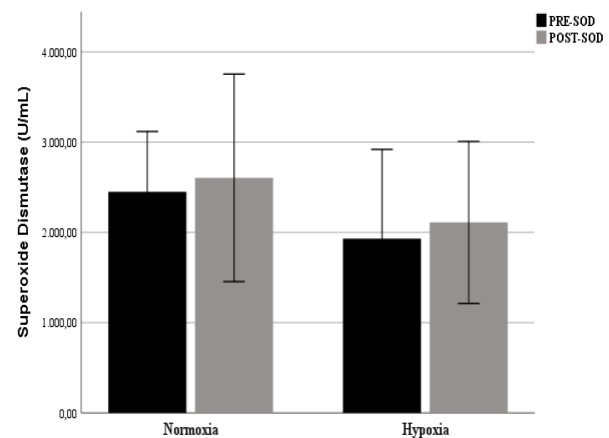


Figure 2. Results of Superoxide Dismutase pre and post test responses after High Intensity Interval training for both conditions (Normoxia and Hypoxia). All data executed as mean with SD for errors bar.

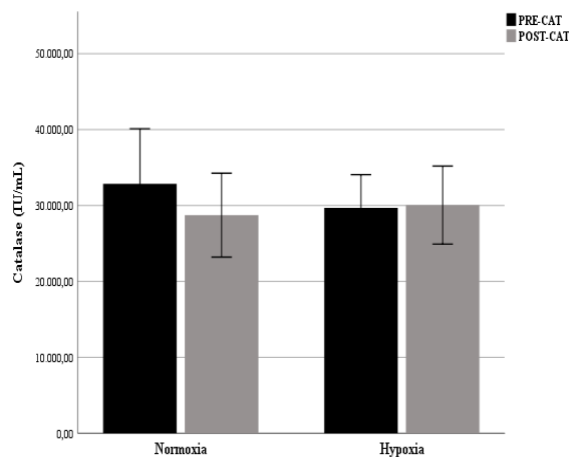


Figure 3. Results of Catalase pre and post test responses after High Intensity Interval training for both conditions (Normoxia and Hypoxia). All data executed as mean with SD for errors bar.

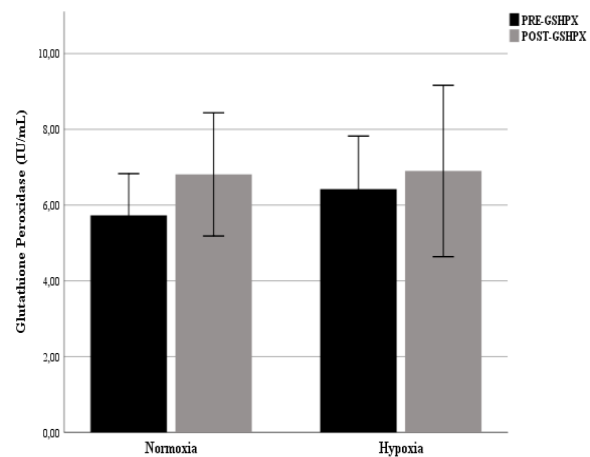


Figure 4. Results of Glutathione pre and post test responses after High Intensity Interval training for both conditions (Normoxia and Hypoxia). All data executed as mean with SD for errors bar.

Normoxic and hypoxic conditions acquired biochemicals data results are shown in table 1. The results revealed no significant difference between group baseline biochemicals results, MDA $t = 0.51$, $p = 0.98$, SOD $t = 1.03$, $p = 0.32$, CAT $t = 0.88$, $p = 0.39$ and GSHPX $t = -0.93$, $p = 0.38$. Pre-Post test statistical comparison of anti-oxidant results revealed no significant main time effects were found for both conditions. The main effects of within-subject time contrast under both conditions were on MDA $F = 0.069$, $p = 0.797$, partial $\eta^2: 0.005$ low effect size, SOD $F = 0.555$, $p = 0.469$, partial $\eta^2: 0.038$ low effect size, CAT $F = 1.353$, $p = 0.264$, partial $\eta^2: 0.088$ average effect size and GSHPX $F = 2.01$, $p = 0.178$, partial $\eta^2: 0.125$ average effect size. There were no significant differences for each group. On the other hand there were no significant Time \times group interaction effects within a subject comparisons MDA $F = 0.251$, $p = 0.624$, partial $\eta^2: 0.018$ low effect size, SOD $F = 0.003$, $p = 0.955$, partial $\eta^2: 0.000$ low effect size, CAT $F = 1.932$, $p = 0.186$, partial $\eta^2: 0.121$ average effect size and GSHPX $F = 0.297$, $p = 0.594$, partial

η^2 : 0.021 average effect size. Furthermore, there were no significant main effects of the group when the group effect took into account between-subject effects ($p > 0.05$).

DISCUSSION

In this study, it was observed that repeated Wingate style HIIT for 30s*4rps*4 min, which is a high intensity exercise type applied in hypoxic and normoxic conditions, did not change both MDA, which is an indicator of oxidant stress, and SOD, CAT, and GSHPX, which are general indicators of antioxidant defense.

To our knowledge, this is the first investigation to compare the acute effects of repeated Wingate style HIIT in hypoxia vs HIIT in normoxia on oxidative stress and antioxidant status. As a result of the study, it was determined that repeated Wingate style HIIT training in both hypoxic and normoxic conditions did not significantly change MDA, which is an indicator of oxidant stress. It is considered that since the oxidant stress did not change, there is no significant change observed in antioxidant indicators.

In general, it is reported in the literature that antioxidant markers tend to increase after exercise. Also, there are statements that this situation is not affected by the intensity, scope, type of exercise and the population applied (de Sousa et al., 2017). Although there is a consensus that excessive free radical production due to exercise occurs mostly on active skeletal muscles (Vina et al., 2000; Jackson, 2005), it is known that exercise causes systemic oxidative damage. In addition, since taking a biopsy during and after exercise will be contrary to the nature of the exercise, most studies in this field are focused on post exercise plasma oxidative stress markers (Pialoux et al., 2009). In this regard, a study found an increase in MDA as a marker of lipid peroxidation due to oxidative stress after a single intense exercise session (Sureda et al., 2009). In another similar study, it was reported that the membrane stiffness of erythrocytes increased due to oxidative stress after a high intensity exercise (Ajmani et al., 2003). Membrane stiffness is an indicator of high lipid peroxidation. However, not all studies have shown increases in MDA levels especially in response to exercise training.

Some studies show that there is no significant change in oxidant and antioxidant indicators after an acute exercise in sedentary individuals, but after six months of regular exercise, acute high oxidant and antioxidant responses to moderate and high intensity exercise are detected in these individuals. They explained that this occurs because regular training stimulates genes that include the regulation of antioxidant enzymes in redox sensitive signal transduction pathways (Shin, Lee, Song, & Jun, 2008). Accordingly, the fact that no significant changes were observed

in the oxidant and antioxidant response to exercise in both methods in our study may be since the participants were recreationally active. However, on the contrary, lower plasma MDA levels have been reported in trained individuals after an exhausting treadmill exercise compared to the control group (Niess, Hartmann, Fuchs, Poch, & Speit, 1996). Miyazaki et al. found a reduction in oxidative stress markers in trained individuals after high intensity endurance training (Miyazaki et al., 2001). However, these conclusions still remain controversial. Ferrer et al. reported that CAT and GSHPX activities decreased after a single intense exercise session. (Ferrer, Tauler, Sureda, Tur, & Pons, 2009) Aquil et al. (2005) observed an increase in CAT activity in erythrocytes after a single high intensity exercise. On the other hand, Cases et al. reported that CAT, GSHPX, and SOD increased in lymphocytes after single long-term cycling and swimming (Cases et al., 2006).

Groussard et al. (2003) reported that SOD activity decreased after a one time anaerobic exercise. However, not all studies suggest that SOD activity decreases after exercise. It has been reported that SOD activity did not change after eight weeks of moderate intensity aerobic exercise. In another study of skiers, it was reported that the level of SOD decreased after a high intensity exhausting treadmill workout. In another study, high erythrocyte SOD activity was reported in sprinters after a speed training (Urso & Clarkson, 2003).

Similarly, there are studies showing that the response of CAT and GSHPX activity to exercise is variable. It has been reported that erythrocyte CAT activity decreases after a supramaximal exercise in trained cyclists. In another study, it was reported that erythrocyte CAT activity did not change after sprint exercise in sprinters, similar to our results. It has been reported that GSHPX, which was reported to be activated to neutralize hydrogen peroxide formed as a result of higher oxygen consumption during exercise, increased after a single HIIT exercise but did not change after endurance exercise in runners (Urso & Clarkson, 2003). As can be seen, MDA, SOD, CAT, and GSHPX activities in response to exercise are variable.

Wozniak et al. (2001) observed an increase in erythrocyte SOD and CAT activities 4, 10, and 18 days after exercise, in which they examined the effect of exercise applied under high altitude (2000 m) conditions on oxidant and antioxidant markers of ten skiers and ten rowers. The reasons why the findings of this study differ from our study may be that it was performed under hypobaric hypoxic conditions and that longer term antioxidant responses were examined. Bailey et al. (2001) on the other hand, reported that a 60-minute exercise program applied in simulated hypoxic conditions significantly increased the serum lipid peroxidase level,

accompanied by a decrease in antioxidant enzyme activity. A study reported that the plasma MDA levels of swimmers decreased immediately after the swimming test was applied in hypoxic conditions (4800 m) (Gonzalez et al., 2005). It is considered that the higher altitude and the fact that the study was carried out on trained athletes is the reason why the findings are different from our study. It should be noted that in our study, participants were exposed to hypoxic conditions for about 15 minutes in a training session. Pialoux et al. (2006) reported that MDA levels of cyclists decreased after 13 days of training at high altitudes (2500-3000m). In this regard, it is considered that being in a hypoxic environment for a longer time or exercising for a longer time may affect oxidant and antioxidant responses.

The present study has several limitations. One of them is that the nutritional standardization of the participants could not be achieved. The different dietary habit of the participants is most important a limitation of our study. Because it is known that dietary patterns can affect oxidant and antioxidant markers in the blood. In future studies, applying a similar study to individuals with nutrition standardization and/or training may affect the results. In addition, since it remains unclear how different exercise protocols affect the redox status in cells and tissues, the acute and chronic effects of different exercise protocols can be investigated by providing different altitudes and accordingly varying hypoxic conditions.

CONCLUSION

This study found no significant change in MDA, SOD, CAT, and GSHPX activities immediately after Repeated Wingate Style HIIT in untrained individuals. Hypoxic and normoxic conditions did not affect this result. It is clear from the above summarized studies that altered physical activity level differentially affects systemic oxidative stress and antioxidant capacity levels. Oxidant and antioxidant markers responses to exercise emerge in a very complex way, which makes it difficult to reach a clear conclusion about its effects.

Conflicts of interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

This study was supported by the Ankara University Scientific Research Projects Coordinatorship with the project code 14A0672001.

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