

THE EFFECTS OF ACUTE LOW-VOLUME HIIT AND AEROBIC EXERCISE ON LEUKOCYTE COUNT AND REDOX STATUS

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RUNNING HEAD: HIIT and redox status

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Abstract

A single bout of exercise can result in inflammatory responses, increased oxidative stress and upregulation of enzymatic antioxidant mechanisms. Although low-volume high-intensity interval training (HIIT) has become popular, its acute responses on the above mechanisms have not been adequately studied. The present study evaluated the effects of HIIT on hematological profile and redox status compared with those following traditional continuous aerobic exercise (CET). Twelve healthy young men participated in a randomized crossover design under HIIT and CET. In HIIT session, participants performed four 30-sec sprints on a cycle-ergometer with 4 min of recovery against a resistance of 0,375 kg/kg of body mass. CET consisted of 30-min cycling on a cycle-ergometer at 70% of their VO_{2max} . Blood was drawn at baseline, immediately post, 24h, 48h and 72h post-exercise and was analyzed for complete blood count and redox status (thiobarbituric acid reactive substances, [TBARS]; protein carbonyls, [PC]; total antioxidant capacity, [TAC]; catalase and uric acid). White blood cells (WBC) increased after both exercise protocols immediately post-exercise (HIIT: 50% and CET: 31%, respectively). HIIT increased (+22%) PC post-exercise compared to baseline and CET ($p < 0.05$). HIIT increased TAC immediately post-exercise (16%) and at 24h post-exercise (11%, $p < 0.05$), while CET increased TAC only post-exercise (12%, $p < 0.05$) compared to baseline, and TAC was higher following HIIT compared to CET ($p < 0.05$). Both HIIT and CET increased uric acid immediately post- (21% and 5%, respectively, $p < 0.05$) and 24h (27% and 5%, respectively, $p < 0.05$) post-exercise and the rise was greater following HIIT ($p < 0.05$). There were no significant changes ($p > 0.05$) for TBARS and catalase following either exercise protocol. Low-volume HIIT is associated with a greater acute phase leukocyte count and redox response than low-volume CET,

and this should be considered when an exercise training program is developed and complete blood count is performed for health purposes.

Key words: High-intensity interval exercise, aerobic exercise, oxidative stress, immune system, recovery

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Figure 2. Thiobarbituric acid reactive species (TBARS) responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

Figure 3. Total antioxidant capacity (TAC) responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET). *Sig. vs. pre; †Sig. vs. CET.

Figure 4. Catalase responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

Figure 5. Uric acid responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET). *Sig. vs. pre; †Sig. vs. CET.

Introduction

Oxidative stress is defined as a shift in the balance between the production of Reactive Oxygen and Nitrogen Species (RONS, free radicals) and the antioxidant defenses, favoring the oxidants (Halliwell, 2001). RONS are produced at all times as part of normal cell metabolism, as well as the environment. During exercise the production of free radicals can increase up to 20-fold in comparison to resting states and even up to 100-fold in active muscle groups (Jackson, 2008). While exercising, the high energy demands of active muscles cause an increase of oxygen (O₂) consumption. This influx of O₂ in the mitochondrial electron transport chain makes for the large jumps in RONS production during exercise. If the amount of free radicals produced exceeds the amount that can be handled by the antioxidant defensive mechanisms, then we have what we call exercise-induced oxidative stress. It has been proposed that a single bout of exercise can result in an increase of oxidative stress and as a result in an upregulation of enzymatic antioxidant mechanisms (Michailidis et al. 2007). Redox changes are part of an exercise-induced inflammatory response that also includes the acute phase response, release of inflammatory mediators (cytokines), and immune cells mobilization and activation (Nieman et al., 2012, Brenner et al., 1999).

Regarding immune cells responses, an increase in leukocytes has been reported during and after short intense and longer submaximal exercise and resistance exercise (Nieman et al., 2007; Natale et al., 2003; Brenner et al., 1999), which may also trigger the increase in several cytokines (Nieman et al., 2006). Exercise-induced leukocytosis is characterized by a biphasic response in which the rise of leukocytes during and immediately after exercise is mainly due to an increase in both neutrophils and

lymphocytes, while the delayed rise peaking several hours post-exercise is mainly due to circulating neutrophils (Rowbottom and Green, 2000; Natale et al., 2003; McCarthy et al., 1991; Hansen et al., 1991). Additionally, the magnitude of the response seems to be exercise intensity- and duration-related (Rowbottom and Green, 2000; Hansen et al., 1991).

Low volume high-intensity interval training (HIIT) has become popular since it has been shown to result in increased aerobic and anaerobic performance (Burgomaster et al. 2005; 2006). HIIT is characterized by a combination of intense anaerobic exercise with less intense recovery periods and is considered an excellent way to maximize a workout that is limited on time. Several studies have reported positive effects of HIIT on mitochondrial biogenesis (Little et al., 2010), glucose metabolism (Laursen and Jenkins, 2002), body composition (Zhang et al., 2017) and muscle and bone mass (Nybo et al., 2010). However, there are not many studies that compared redox status and inflammatory responses following acute HIIT and traditional continuous aerobic exercise (CET). A rise in lipid and protein oxidation indices as well as in antioxidant status markers have been reported following acute HIIT (Baker et al., 2004; Bloomer et al., 2006; Bogdanis et al., 2013). HIIT and weight training exercise have been reported to result in similar upregulation of redox status markers (Bloomer et al., 2006). On the other hand, a CrossFit workout (performed in an interval fashion and at a high intensity), resulted in lower protein carbonyl and total antioxidant capacity and higher lipid peroxides compared to a treadmill bout (Kluszczewicz et al., 2015). The duration of both exercise sessions was 20 minutes and the intensity of the treadmill exercise bout was 90% of maximal heart rate. It is evident that both of these exercise protocols were intense without differentiating the anaerobic from the aerobic component and therefore

direct comparisons cannot be made. CET, usually characterized by low intensity, has been prescribed by exercise professionals as means to increase aerobic capacity and promote health and well-being. Increases in peak power outputs during exercise result in increased metabolic demands which in turn may compromise the integrity of skeletal muscle that could lead to early onset of fatigue and exhaustion and thus selection of exercise intensity should be done with caution in order to avoid undesirable outcomes.

As with redox status, not many studies have compared the effects of intense exercise and aerobic exercise on parameters related to immune cells changes such as white blood cell (WBC) count. In one of those, Mathes et al. found that a 4x30 sec all-out protocol resulted in significant perturbations of leukocytes, lymphocytes and neutrophils compared to high volume exercise (4x4 min at 90 – 95% VO_{2max}) (Mathes et al., 2017). Furthermore, HIIT and moderate intensity training effectively depressed apoptosis and promoted autophagy in CD4 lymphocytes (Weng et al., 2013). However, the latter study was a training study and no firm conclusion can be drawn if the acute response of WBC-related variables is different between HIIT and CET. Nevertheless, such information is important when an exercise training program is developed and complete blood count is performed for health purposes. Therefore, the purpose of this investigation was to evaluate the effects of low-volume HIIT on WBC count and redox status and compare them with the effects of CET. The total time of involvement with exercise during HIIT was two times less compared to that during CET.

Methods

Participants

Twelve young males (22.4 ± 0.5 y) volunteered to participate in this two-trial, crossover investigation. In the present study, females were not included to avoid possible sex-dependent variability in the results, as it has been shown that sex accounts for different outcomes in exercise-induced inflammatory, oxidative stress, and cardiovascular parameters in both animals (Pósa et al., 2013), and humans (Wiecek et al., 2017; Pepe et al., 2009). Additionally, the participants were about the same age, to avoid any age-related variability in the results of the study, since there is evidence that the response of oxidative stress biomarkers and antioxidants to exercise varies within different age groups (Deli et al., 2017; Barranco-Ruiz et al., 2017). Selection criteria included: a) absence of musculoskeletal and/or other health problems (cardiovascular disease or any other related health condition that could affect energy metabolism), b) no use of nutritional supplements, and medications before (≥ 6 months) and during the study, c) participants were non-smokers. The anthropometric and physiological characteristics of participants are shown in Table 1. The procedures were conformed to the Helsinki declaration of 1975 and were approved by the Human Participants Committee of the local University (ref #:799/10-12-13).

Study design

The participants reported to the laboratory three times in total. All measurements took place in the morning (08.00-10.00 a.m). At the first visit, anthropometric characteristics and maximal oxygen consumption (VO_{2max}) were measured. Body mass was measured to the nearest 0.5 kg with the participants lightly dressed and barefoot (Beam Balance 710, Seca, UK) and standing height was measured to the nearest 0.5 cm (Stadiometer

208, Seca, UK). Percent body fat was calculated from 7 skinfold measures (average of 2 measurements at each site), using a Harpenden calliper (John Bull, St. Albans, UK). Maximal oxygen consumption ($\text{VO}_{2\text{max}}$) was determined using a cycle ergometer (Monark 834E) test to exhaustion. The results of the initial maximal test were used to determine the exercise intensity of CET (70% $\text{VO}_{2\text{max}}$). During the second and third visit (a washout period of one week between the second and third visit was included), the participants performed in random order either a CET protocol, or a HIIT protocol.

Maximal oxygen consumption

Each subject started pedaling at 70 revolutions per minute (rpm) with no additional workload for 150 s. Work rate (300g) was then added incrementally every 120 s with the intent of reaching the subject's $\text{VO}_{2\text{max}}$ within 6 to 12 min (Lundgren, et al. 2001). Criteria used to determine $\text{VO}_{2\text{max}}$ were: i) participants' exhaustion, ii) a $<2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ increase in VO_2 with an increase in work rate, iii) a respiratory exchange ratio greater than or equal to 1.10, iv) a heart rate within 10 bpm of the theoretical maximum heart rate ($220 - \text{age}$). Respiratory gas variables were measured using a metabolic cart (Vmax29, Sensormedics, USA), which was calibrated before each test using standard gases of known concentration. Exercise heart rate was monitored by telemetry (Polar Tester S610TM, Electro Oy, Finland).

Submaximal Exercise

Submaximal CET bout was performed on the cycle ergometer for 30 min. Exercise intensity was set at 70% of the participant's VO_{2max} . The intensity of the exercise was checked through respiratory and heart rate measurements throughout the workout and adjustments were made so that participants were exercising at the prescribed intensity. In order to calculate the energy expenditure during the exercise, respiratory gas variables were measured for 30 min using a metabolic cart (Vmax29, SensorMedics, USA).

HIIT

The HIIT exercise bout involved the performance of four 30-sec sprints on a cycle ergometer (against a resistance of 0,375 kg/kg of body mass) interspersed with 4 min of recovery. In order to calculate the energy expenditure during the exercise, respiratory gas variables were measured for 30 min using a metabolic cart (Vmax29, SensorMedics, USA).

Dietary Analysis

To establish that macronutrient and antioxidant intake levels were similar in both conditions, participants were asked to record their diet for three consecutive days prior to participation in the first exercise condition (HIIT or CET) and replicate their diet for three days before the participation in the second exercise condition (HIIT or CET). Participants were provided with a written set of guidelines for monitoring dietary consumption and a record sheet for recording food intake. Diet recalls were analyzed

with ScienceFit Diet 200A (Science Technologies, Athens, Greece). The nutritional analysis is presented in Table 2.

Blood Sampling and Assays

Participants were asked not to engage in any intense physical activity and not to consume alcohol or caffeine products for at least 72h before reporting to the laboratory. Blood samples (20 mL) were drawn before the exercise, immediately post, 24, 48 and 72 hours post exercise from a forearm vein with participants in a seated position. A portion (1–2 mL) of the blood collected was used to determine the parameters of the complete blood count measured by an automatic hematology analyzer (Mythic 18 Orphee, Orphee Medical, Geneva, Switzerland). For plasma collection, a portion of blood was placed in separate tubes mixed with EDTA (20 uL/mL of blood) and centrifuged (1370 g, 10 min, 4°C) and the supernatant was transferred into Eppendorf tubes that were stored at -80°C for later measurement of protein carbonyls (PC), thiobarbituric acid reactive substances (TBARS) and total antioxidant capacity (TAC). Packed erythrocytes were diluted with distilled water (1:1 v/v), vortexed vigorously, and centrifuged (4000 g, 15 min, 4°C) for red blood cell lysate preparation and the resultant supernatant was transferred into Eppendorf tubes that were stored at -80°C for later analysis of catalase activity. Finally, another portion of blood was collected in plain tubes, left at room temperature for 20 min to clot, centrifuged (1370 g, 10 min, 4°C) for serum separation and the supernatant was transferred into Eppendorf tubes that were stored at -80°C for later determination of uric acid (UA). Capillary blood for lactate measurements was collected 3 minutes after the end of each exercise session.

TBARS were measured according to Keles et al. (2001). Protein carbonyls were analyzed according to procedures described by Patsoukis et al. (2004). Total antioxidant capacity (TAC) was measured as described by Janaszewska and Bartosz (2002). Catalase activity was measured as described by Aebi (1984). Hb was determined with a commercially available kit (Dutch Diagnostics BV, The Netherlands). UA was measured in a Clinical Chemistry Analyzer Z 1145 (Zafiropoulos Diagnostica, Athens, Greece) with commercially available kits (Zafiropoulos, Athens, Greece). Lactate was measured in a microphotometer (DR LANGE, Berlin) with commercially available kits (LCQ 140, DR LANGE, Berlin). Each variable was analyzed in duplicates on the same day. Inter- and intra-assay coefficients of variation for all assays ranged from 2.4% to 6.8% and from 2.8% to 7.1%, respectively. Spectrophotometric assays were performed on a Hitachi 2001 UV/VIS (Hitachi Instruments Inc., U.S.) in triplicates.

Statistical Analysis

Data normality was examined using the Kolmogorov–Smirnov test and was not found to differ significantly from normal. A 2x5 (condition x time) repeated measures ANOVA and a Bonferroni post-hoc test were used to analyze the data. SPSS was used for all analyses (SPSS Inc., Chicago, Ill.) The level of statistical significance was set at $p < 0.05$. The G*Power program (G*Power 3.0.10) was utilized to perform power analysis. With our sample size of 12 we obtained a statistical power greater than 0.80 at an α error of 0.05.

Results

Analysis of the diet records did not reveal any significant differences in the macro- and micro-nutrients between exercise trials (Table 2). The intensity of CET was $70.0 \pm 2.0\%$ VO_{2max} whereas mean power during HIIT was 412.5 ± 77.3 W. Mean energy expenditure during the 30 minutes of aerobic exercise was 348 ± 90 kcal whereas during HIIT was 200 ± 69 kcal. Post-exercise mean lactate values after the aerobic exercise and HIIT were 2.7 ± 0.4 mM and 15.3 ± 1.8 mM, respectively.

Redox status responses

PC increased significantly ($p < 0.05$) following HIIT immediately post-exercise compared to baseline and that increase was also significantly higher compared to CET (Figure 1). TBARS did not change significantly ($p > 0.05$) following neither type of exercise (Figure 2). TAC increased significantly ($p < 0.05$) following HIIT immediately post- and 24h post-exercise, while only immediately post-exercise following CET (Figure 3). TAC was significantly higher ($p < 0.05$) immediately post- and 24h post-exercise following HIIT compared to CET (Figure 3). Catalase did not change significantly ($p > 0.05$) following neither type of exercise (Figure 4). UA increased significantly ($p < 0.05$) following HIIT immediately post- and 24h post-exercise compared to baseline (Figure 5). UA responses 24h post-exercise following HIIT were significantly higher ($p < 0.05$) compared to CET.

Hematological responses

No significant results for condition or condition x time were found for red blood cell (RBC) count, hemoglobin or hematocrit (Table 3). A significant increase was found for mean corpuscular volume (MCV) following HIIT and mean corpuscular hemoglobin concentration (MCHC) following HIIT and CET (Table 3). Platelets increased after CET (Table 3).

WBC increased significantly ($p < 0.05$) following HIIT and CET immediately post-exercise compared to baseline with the increase in HIIT being significantly greater compared to the one of CET (Table 4). Lymphocytes (%) increased significantly ($p < 0.05$) following HIIT immediately post-exercise compared to baseline with the increase in HIIT being significantly greater compared to the one of CET (Table 4). Monocytes did not change significantly ($p > 0.05$) by either exercise protocol (Table 4). Granulocytes (%) decreased significantly ($p < 0.05$) following HIIT immediately post-exercise compared to baseline with HIIT inducing a decline of greater ($p < 0.05$) magnitude than CET (Table 4).

Discussion

To our knowledge this is the first study that compared the acute effects of HIIT and CET on hematological profile and redox status. Results revealed that low-volume HIIT results in greater perturbations in WBC count and redox status than CET. It has to be noted, however, that the absolute duration of the HIIT exercise bout was 15 times less than that of CET, and that energy expenditure was higher in CET compared to HIIT. Our intention was to compare HIIT and CET protocols usually employed for fitness development.

Redox status responses

A single bout of HIIT exercise that comprised of four 30-sec intense sprints induced a large rise in PC immediately post-exercise and that increase was significantly greater compared to CET. The results obtained by this study are different compared with those reported previously and revealed either no differences in lipid peroxidation and protein carbonylation following an intense HIIT bout (Bloomer et al., 2006) or lower protein and lipid oxidation and total antioxidant capacity in response to a CrossFit workout compared to an acute CET protocol (Kluszczewicz et al., 2015). However, our results coincide with those reported by Bogdanis et al. suggesting a rise in protein oxidation following a HIIT-type protocol (Bogdanis et al., 2013). The increase in PC in response to HIIT could be explained by several mechanisms. It is well known that intense exercise results in increased levels of catecholamines to fuel the muscles with energy from nutrient breakdown among other things (Wahrenberg et al., 1987). An increased autoxidation of catecholamines could be one mechanism of the elevated levels of protein carbonyls following HIIT (Zouhal et al., 1998). Another possible reason for the increased levels of PC could be the increased catabolism of purines that results in elevated levels of hypoxanthine and UA (Starling et al., 1996). The latter explanation can be supported by the uric acid responses in this study. UA was significantly higher immediately-post and 24 hours post-exercise following the HIIT protocol compared to CET. As mentioned previously, high intensity exercise increases the activation of the purine nucleotide system resulting in the formation of adenosine monophosphate which in turn, due to hypoxanthine activation, UA is formed (Starling et al., 1996). This may

explain the greater rise of UA following the HIIT protocol compared to CET which relies more on mitochondrial metabolism. Nevertheless, the response of UA in this study differs with the one of Baker et al. study where decreases in UA concentration were seen following a 30-sec high intensity exercise (Baker et al., 2004). Our protocol included four 30-sec cycling sprints and differences in the intensity and duration of the exercise bout could explain discrepancies between these two studies.

TAC responses were higher following the HIIT protocol. Previous results reported elevated levels of TAC (Bogdanis et al., 2013) whereas others found decrements in TAC a few hours after the end of an intense workout (Kluszczewicz et al., 2015). The elevated TAC levels in the present study could be explained by the significant increase of UA since it is known that this molecule represents almost 50% of the plasma antioxidant capacity. Catalase, an antioxidant enzyme found primarily in the RBCs, responses were not different due to time or protocol implying that the RBCs were not stressed due to exercise. That is further supported by the non-significant differences of the RBC count following either exercise protocol.

Hematological responses

It is well known that physical exercise results in a series of immune response changes (Keast et al., 1988; Fatouros and Jamurtas, 2016) whereas exercise of varying intensity and duration results in increased WBC (Nieman et al., 2007). Acute exercise can result in physiological changes in the quality and quantity of WBC affecting therefore the capacity to resist against common infections. Exercise-induced leukocytosis is accompanied by cytokines production after prolonged exercise (Nieman et al., 2012),

and blood leukocytes may produce IL-8, IL-10, IL-1ra (Nieman et al., 2006). In the present study, although cytokines were not measured, the observed leukocytosis is in accordance with previous results that show increases in total leukocytes and lymphocytes following aerobic exercise (Benoni et al., 1995; Nieman et al., 2012). In agreement with our results, all-out bouts of rowing exercise increased counts of leukocytes and lymphocytes but not monocytes (Nielsen et al., 1996) suggesting that intense exercise provokes significant short-term immune system perturbations immediately post-exercise. Perhaps this response is attributed to the increased catecholamine levels. Previous work with beta-adrenergic blockade has shown that after propranolol (a beta adrenergic blocker) administration during exercise the increase in neutrophils, leukocytes and lymphocytes was abolished (Ahlborg and Ahlborg, 1970). The exercise-induced leukocytosis response is transient since white blood cells returned to the resting values within 24 hours post-exercise. Platelet count appears to be elevated following moderate intensity CET (Warlow and Ogston, 1974) as well as prolonged (Lippi et al., 2014) and intense exercise (Belviranli et al., 2017). This response appears to be transient and platelets return to baseline within 24 hours post-exercise. These results are in accordance with those of previous studies regarding the aerobic and anaerobic types of exercise. One possible explanation for the increased levels of platelets could be the release of fresh platelets from the spleen, bone marrow or other stored places (Freedman et al., 1977; Schmidt and Rasmussen, 1984).

Conclusion

In conclusion, redox status and CBC responses following HIIT appear to be exacerbated compared to the ones following CET. Some of these variables are still elevated 24 hours post-exercise indicating a prolonged perturbation in metabolism following this type of exercise. These responses should be taken into consideration when an exercise training program is developed and complete blood count is performed for health purposes.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Conflict Of Interest

None declared.

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Table 1. Participants' personal characteristics.

Variable	Mean \pm SD
Age (years)	22.4 \pm 0.5
Weight (kg)	75.3 \pm 8.9
Height (m)	1.80 \pm 0.1
Body Fat (%)	10.8 \pm 4.1
VO _{2max} (ml/kg/min)	45.3 \pm 8.4
HR max (bpm)	185.2 \pm 7.1

VO_{2max}: Maximum oxygen consumption; HR: Heart rate.

Table 2. Nutritional analysis of the 3-day diet records prior to aerobic and high intensity interval training exercise protocol.

	CET	HIIT
Energy (kcal)	1772 ± 425	1766 ± 406
Protein (gr)	74.3 ± 24.1	71.9 ± 25.7
Carbohydrate (gr)	190.4 ± 56.9	193.3 ± 40.4
Fat (gr)	81.3 ± 21.7	76.8 ± 18.1
Vitamin A (IU)	4246 ± 2193	4443 ± 2895
Vitamin C (mg)	89.3 ± 36.4	87.6 ± 29.0
Vitamin D (mg)	109.1 ± 81.3	113.3 ± 65.0
Vitamin E (mg_RE)	8.7 ± 9.5	9.1 ± 9.9
Iron (mg)	14.3 ± 3.2	15.7 ± 4.3
Selenium (µg)	121.4 ± 33.6	115.0 ± 40.5

CET, continuous aerobic exercise; HIIT, high intensity interval training.

Table 3. Hematological response following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

Variable	Pre-exercise	Post-exercise	24 h	48 h	72 h
RBC (X10⁶/μl)					
HIIT	5,15 ± 0,67	5,26 ± 0,52	4,95 ± 0,46	4,93 ± 0,47	4,83 ± 0,47
CET	4,88 ± 0,50	5,3 ± 0,93	4,91 ± 0,56	4,94 ± 0,66	4,94 ± 0,66
Hemoglobin (g/dl)					
HIIT	14,46 ± 1,44	14,93 ± 1,20	14,14 ± 1,48	14,46 ± 2,06	13,59 ± 1,24
CET	13,89 ± 1,75	15,33 ± 2,80	14,06 ± 1,86	13,98 ± 1,80	13,85 ± 1,60
Hematocrit (%)					
HIIT	44,38 ± 4,43	45,8 ± 3,55	42,91 ± 4,16	42,84 ± 4,50	41,66 ± 3,54
CET	41,8 ± 4,71	46,04 ± 8,12	42,16 ± 5,35	42,33 ± 4,55	42,16 ± 4,42
MCV (fl)					
HIIT	86,68 ± 7,33	88,02 ± 7,48*	86,95 ± 7,43	87,05 ± 7,44	86,64 ± 7,68
CET	85,87 ± 7,96	87,12 ± 7,09*	86,00 ± 7,91	86,17 ± 8,20	85,89 ± 8,45
MCH (pg)					
HIIT	28,25 ± 2,61	29,81 ± 6,24	28,68 ± 2,93	28,55 ± 2,82	28,32 ± 3,28
CET	28,55 ± 3,17	29,03 ± 2,94	28,72 ± 3,17	28,47 ± 3,46	28,24 ± 3,42
MCHC (g/dl)					

HIIT	32,58 ± 1,00	34,02 ± 6,22*	32,94 ± 1,11	32,78 ± 1,64	32,63 ± 1,31
CET	33,19 ± 1,24	33,31 ± 1,29*	33,38 ± 1,73	33,03 ± 2,13	32,81 ± 1,22
Platelets (X10³/μL)					
HIIT	213,8 ± 66,0	233,8 ± 38,8	223,7 ± 44,9	214,1 ± 45,5	216,9 ± 54,6
CET	215,2 ± 50,9	260,5 ± 83,4*	225,7 ± 53,3	224,1 ± 47,0	216,0 ± 39,7

CET, continuous aerobic exercise; HIIT, high-intensity interval training. *Sig. vs. pre-exercise.

Table 4. White blood cells response following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

Variable	Pre-exercise	Post-exercise	24 h	48 h	72 h
WBC ($\times 10^3/\mu\text{L}$)					
HIIT	6,5 \pm 0.8	9,8 \pm 2,1 ^{*,†}	6,1 \pm 0.7	6,1 \pm 0.7	5,9 \pm 1.1
CET	5,8 \pm 0.7	7.6 \pm 1.5 [*]	5,9 \pm 0.8	6.0 \pm 0.9	6.0 \pm 1.1
Lymphocytes (%)					
HIIT	37,4 \pm 8,0	42,2 \pm 9,6 ^{*,†}	35,3 \pm 6,9	35,5 \pm 7,9	34,8 \pm 5,3
CET	35,6 \pm 7,7	36,7 \pm 6,5	34,8 \pm 8,1	34,5 \pm 6,7	33,4 \pm 5,5
Monocytes (%)					
HIIT	8,5 \pm 1,4	8,4 \pm 1,9	8,7 \pm 2,3	8,6 \pm 1,7	8,4 \pm 2,3
CET	9,9 \pm 3,5	9,2 \pm 2,3	9,8 \pm 3,7	9,8 \pm 4,7	9,7 \pm 4,1
Granulocytes (%)					
HIIT	54,1 \pm 8,1	49,4 \pm 10,2 ^{*,†}	56.0 \pm 7,5	56,1 \pm 8,3	56,8 \pm 5,3
CET	54,5 \pm 6.0	54,1 \pm 6,7	55,4 \pm 6,5	55,7 \pm 5,7	56,9 \pm 5.0

HIIT, high intensity interval training; CET, continuous aerobic exercise; *Sig. vs. pre-exercise; [†]Sig. vs. CET.

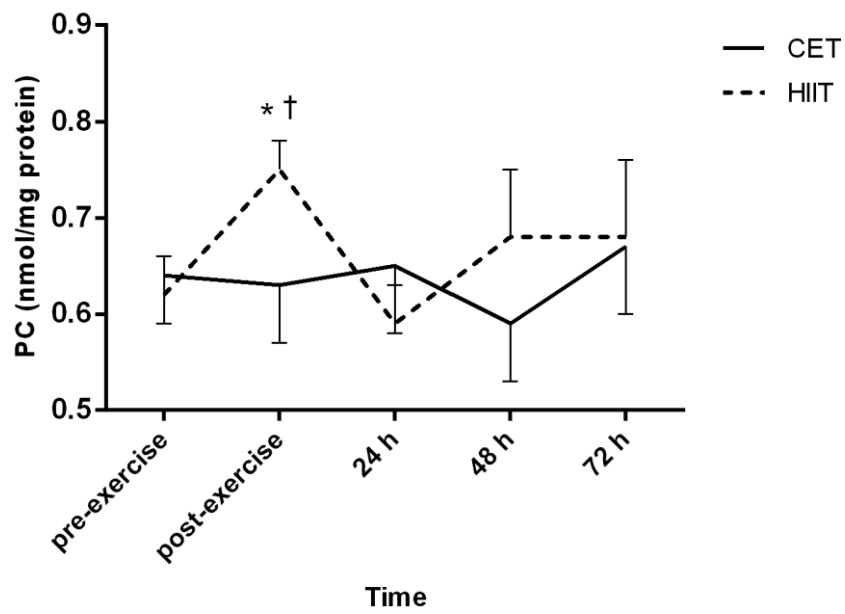


Figure 1. Protein carbonyls (PC) responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET). *Sig. vs. pre; †Sig. vs. CET.

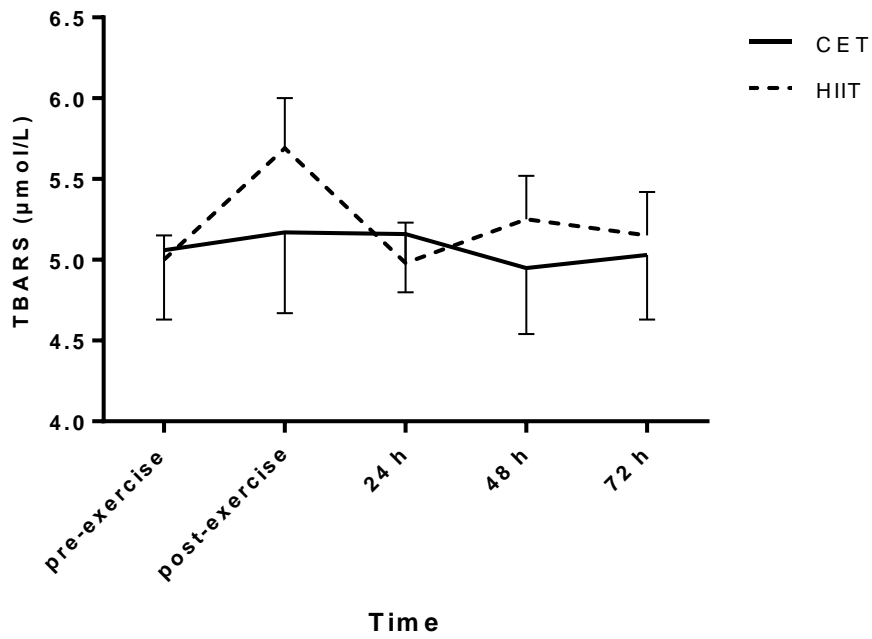


Figure 2. Thiobarbituric acid reactive species (TBARS) responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

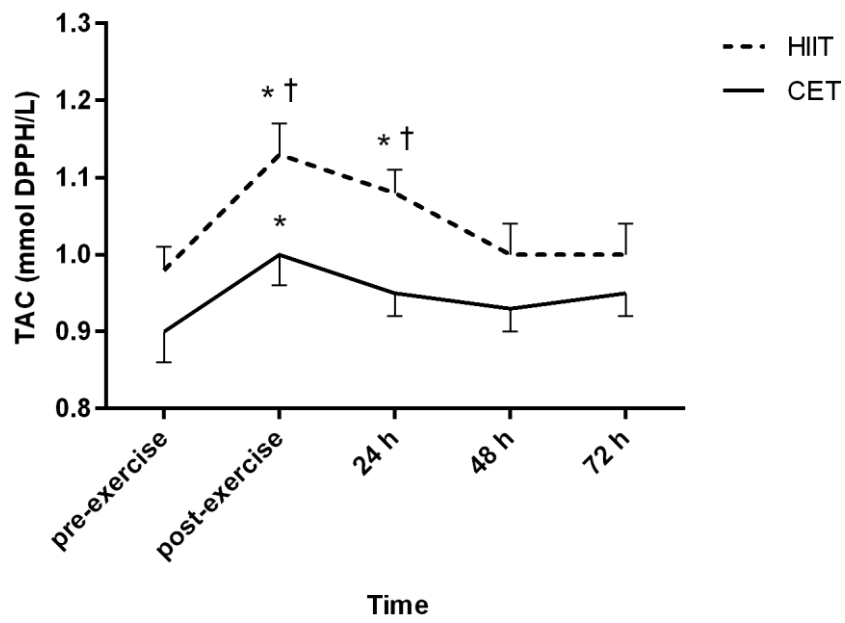


Figure 3. Total antioxidant capacity (TAC) responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET). *Sig. vs. pre; †Sig. vs. CET.

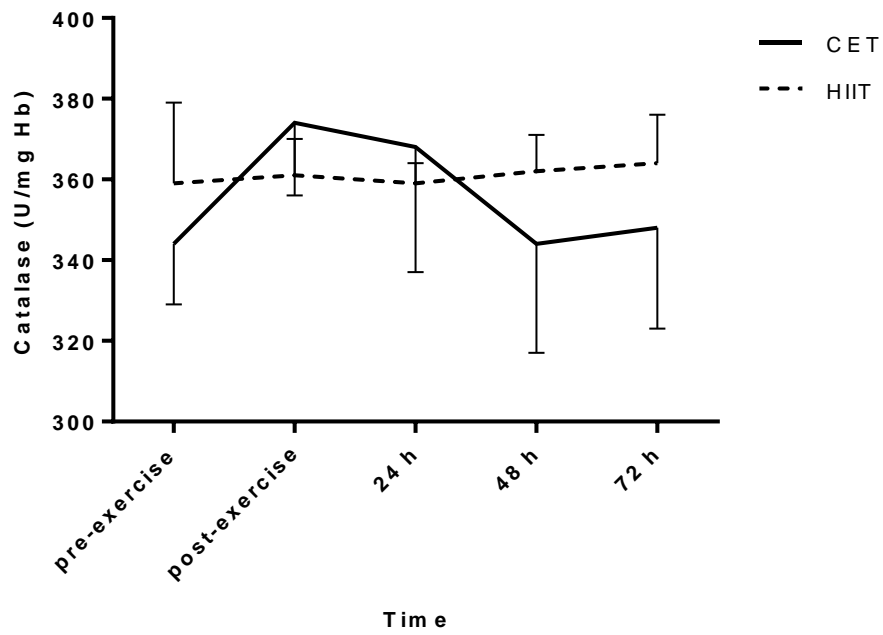


Figure 4. Catalase responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET).

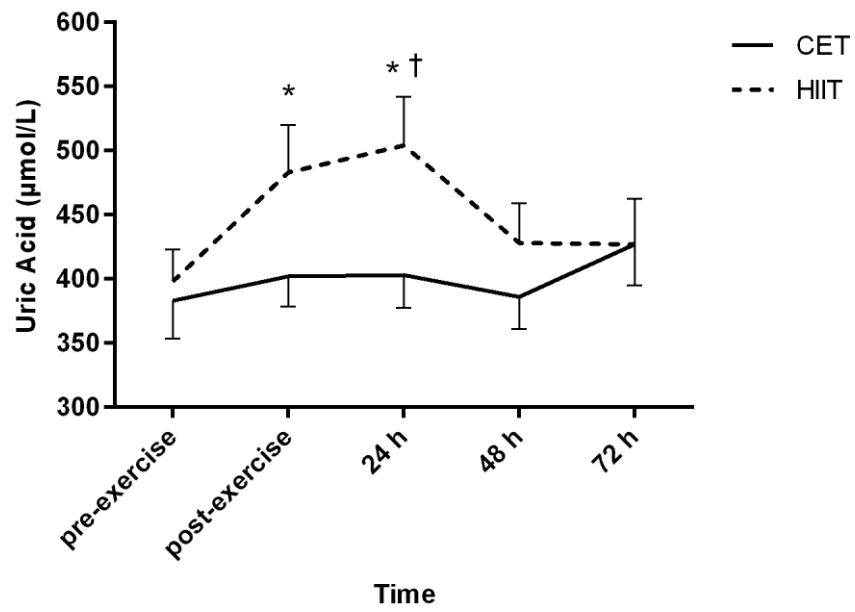


Figure 5. Uric acid responses following high intensity interval training (HIIT) and continuous aerobic exercise (CET). *Sig. vs. pre; †Sig. vs. CET.