Experimental and Therapeutic Medicine

The Effects of Acute Exercise on Liver Function and Blood Redox Status in Heavy Drinkers

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Abstract

Background: Excessive exposure to alcohol can induce oxidative stress, resulting in several diseases. Exercise training has been reported to prevent and/or improve a number of health problems through several mechanisms, including improvement in redox status. It has also been suggested that exercise training could help individuals with alcohol use disorders reduce alcohol intake but research on this field is limited. Objectives: The purpose of the present study was to investigate the effects of acute exercise of moderate intensity on liver function and blood redox status in heavy drinkers. Methods: Seventeen heavy drinkers (age: 31.6 ± 3.2 yrs; BMI: 27.4 ± 0.8 kg/m2) (EG) and 17 controls (age: 33.5 ± 1.3 yrs; BMI: 26.1 ± 1.4 kg/m2) (CG) that did not exceed moderate alcohol use, underwent one trial of acute exercise of moderate intensity (50-60% of the heart rate reserve) for 30 minutes on a cycle ergometer, after overnight fast, smoking and alcohol abstinence. Blood samples were obtained before and immediately after exercise for later determination of indices of liver function and blood redox status. Results: EG had significantly higher (p < .05) baseline γ-GT levels compared to CG. Exercise, resulted in significantly higher γ-GT levels (p < .005) only in EG. No significant difference in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) at baseline was found between the two groups. After exercise, AST increased significantly (p < .001) in both groups, while ALT increased significantly (p < .01) only in EG. Baseline GSH were significantly lower (p < .05) and remained lower after exercise in EG and there was a trend for higher (p = .07) baseline TBARS that remained elevated post exercise in EG compared to CG. There were significantly increased post-exercise TAC (p < .01) and UA (p < .05) levels in CG whereas TAC (p = .06) and UA (p = .08) increased and approached significance post exercise in EG. No significant differences in the levels of total bilirubin and PC were found at the baseline or post-exercise between the two groups. Conclusions: The present study indicates that even though heavy drinkers may be prone to oxidative stress, their exercise-induced antioxidant response is similar to the one of individuals that do not drink heavily.

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Letter to the Editor

Editor
Experimental and Therapeutic Medicine

Dear Editor

I am submitting the manuscript entitled: **The Effects of Acute Exercise on Liver Function and Blood Redox Status in Heavy Drinkers** for consideration for publication at Experimental and Therapeutic Medicine. The authors of the manuscript are: Kalliopi Georgakouli; Eirini Manthou; Ioannis G. Fatouros; Chariklia Deli; Yiannis Koutedakis; Yannis Theodorakis; Athanasios Z. Jamurtas. All of them have contributed significantly towards data collection and analysis and writing of the present manuscript. Furthermore, all of the authors have given me the authority for the submission of the manuscript for review. In addition, I would like to let you know that the manuscript is not under consideration for publication by any other journal and the material will not be submitted elsewhere for publication, either in part or in whole, without the written consent of the General Editor.

Thank you very much.

Yours sincerely,

A.Z. Jamurtas, Ph.D.

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The Effects of Acute Exercise on Liver Function and Blood Redox Status in Heavy Drinkers

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Background: Excessive exposure to alcohol can induce oxidative stress, resulting in several diseases. Exercise training has been reported to prevent and/or improve a number of health problems through several mechanisms, including improvement in redox status. It has also been suggested that exercise training could help individuals with alcohol use disorders reduce alcohol intake but research on this field is limited. Objectives: The purpose of the present study was to investigate the effects of acute exercise of moderate intensity on liver function and blood redox status in heavy drinkers. Methods: Seventeen heavy drinkers (age: 31.6 ± 3.2 yrs; BMI: 27.4 ± 0.8 kg/m^2) (EG) and 17 controls (age: 33.5 ± 1.3 yrs; BMI: 26.1 ± 1.4 kg/m^2) (CG) that did not exceed moderate alcohol use, underwent one trial of acute exercise of moderate intensity (50-60% of the heart rate reserve) for 30 minutes on a cycle ergometer, after overnight fast, smoking and alcohol abstinence. Blood samples were obtained before and immediately after exercise for later determination of indices of liver function and blood redox status. Results: EG had significantly higher (p < .05) baseline γ-GT levels compared to CG. Exercise, resulted in significantly higher γ-GT levels (p < .005) only in EG. No significant difference in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) at baseline was found between the two groups. After exercise, AST increased significantly (p < .001) in both groups, while ALT increased significantly (p < .01) only in EG. Baseline GSH were significantly lower (p < .05) and remained lower after exercise in EG and there was a trend for higher (p = .07) baseline TBARS that remained elevated post exercise in EG compared to CG. There were significantly increased post-exercise TAC (p < .01) and UA (p < .05) levels in CG whereas TAC (p = .06) and UA (p = .08) increased and approached significance post exercise in EG. No significant differences in the levels of total bilirubin and PC were found at the baseline or post-exercise between the two groups. Conclusions: The present study indicates that even though heavy drinkers may be prone to oxidative stress, their

exercise-induced antioxidant response is similar to the one of individuals that do not drink heavily.

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1. Introduction

Many epidemiological studies have shown that there is a dose-response association between alcohol use and the risk of several diseases and mortality. It has been reported that light to moderate alcohol use has beneficial effects on many aspects of health, especially on cardiovascular outcomes (1, 2). On the contrary, heavy alcohol drinking is thought to be responsible for hundreds of thousands deaths per year worldwide, causing many diseases, being a precursor to injury and violence and often leading to alcohol use disorders (alcohol abuse and dependence) (3, 4).

Oxidative stress is an imbalance between oxidants and antioxidants in favor of the oxidants, resulting in reversible redox modification of molecules involved in cellular signaling pathways, and damage of biological molecules (lipids, proteins and DNA) (5). Oxidative stress is responsible for the development of several pathological conditions and it can be induced by numerous factors, including alcohol (6). Excessive chronic alcohol use may lead to impaired redox status through both increased production of reactive oxygen species (ROS) and impaired antioxidant defense mechanisms (7), and is associated with the pathogenesis of alcohol-related diseases, such as alcohol liver disease, alcoholic cardiomyopathy and cancer (7, 8). Many studies have shown that acute exercise can increase oxidative stress in humans (9-13). Exercise-induced oxidative stress activates signalling pathways that increase the expression of antioxidants and also are responsible for exercise adaptations (11, 13). These adaptations are influenced by various factors, including the training volume, intensity, frequency and mode of exercise (13).

Although not well established, exercise is a promising non-pharmaceutical intervention for alcohol intake reduction or cessation in heavy drinkers and individuals with alcohol use disorders (AUDs) (14-16). In the last 40 years a small number or studies have investigated the effect of exercise on alcohol intake in individuals with AUDs (15, 16); however, only one recent study has investigated the physiological

responses to acute exercise in this population. Jamurtas and his colleagues (17) examined the effects of low intensity exercise on alcohol urge, and the levels of β-endorphin (β-E), lactic acid and hematological parameters (complete blood count - CBC). The results showed that pre-exercise levels of β-E were significantly lower in alcoholic patients, while exercise led to significantly ($p < .001$) increased β-E levels only in alcoholic patients. Lactic acid and hematological parameters assessed through the CBC did not differ between the two groups, while exercise led to significantly increased lactic acid, red blood cells, hemoglobin and hematocrit in both groups. Moreover, there was a 17% decrease in alcohol urge in alcoholic patients. The results from this study indicate that a bout of low intensity exercise affects the endogenous opioids in alcoholic patients. Greater increases in β-E levels as a response to exercise of different type and/or higher intensity has been observed in other special populations (18). Since chronic excessive exposure to alcohol leads to decreased β-E production that may be responsible for negative reinforcement (19), a greater increase in β-E levels after exercise could lead to significantly reduced alcohol urge. Therefore, exercise could be used as a healthy alternative to alcohol intake. The effects of acute and chronic exercise of different intensities and types on alcohol urge and health status in individuals with AUDs, as well as the physiological mechanisms involved should be investigated.

Since there is gap in the literature on the acute effects of exercise on metabolism and redox status of individuals with AUDs, the purpose of the present study was to investigate the effect of acute exercise of moderate intensity on indices of liver function and redox status of heavy drinkers. This is a preliminary step in describing the responses to exercise of individuals that drink heavily in order to develop exercise training programmes that aim at alcohol abuse cessation and health improvement.

2. Materials and methods

2.1. Subjects

Seventeen heavy drinkers (age: 31.6 ± 3.2 yrs; BMI: 27.4 ± 0.8 kg/m^2) (experimental group - EG) and 17 controls that did not exceed moderate alcohol use (age: 33.5 ± 1.3 yrs; BMI: 26.1 ± 1.4 kg/m^2) (control group - CG) participated in this study. All subjects were sedentary and the level of physical activity was assessed by the International Physical Activity Questionnaire (IPAQ). Subjects in the two groups were also matched for number of cigarettes smoked per day.

Individuals exceeding the limits for drinking at low risk for developing an AUD according to the National Institute on Alcohol Abuse and Alcoholism (more than 14 drinks per week or more than 4 drinks per occasion for men, more than 7 drinks per week or more than 3 drinks per occasion for women) were identified as heavy drinkers (4). Moreover, the Alcohol Use Disorders Identification Test [AUDIT (20)] was also used in order to identify individuals with AUDs. An AUDIT score between 8 and 15 indicates hazardous alcohol drinking, a score between 16 and 19 indicates harmful alcohol drinking, and a score of 20 or above indicates alcohol dependence (21). Six heavy drinkers had a score between 8 and 15, five heavy drinkers had a score between 16 and 19, and six heavy drinkers had a score of 20 or above (total AUDIT score: 17.65 ± 1.25).

Subjects were informed about the study protocol, the associated risks and benefits and they signed an informed consent form. Before proceeding to other measurements, medical history was reviewed and a resting electrocardiogram (ECG) was performed in order to detect any heart abnormalities and contraindications to exercise. The procedures were in accordance with the 1975 Declaration of Helsinki and ethics approval was received from the University of Thessaly review board. Exclusion criteria included serious health problems, physical disabilities or any other medical condition that contraindicate safe participation in exercise, history of drug abuse other than alcohol and age over sixty.

2.2. Experimental design

Subjects reported in the lab after an overnight fast, alcohol and smoking abstinence. Subjects' anthropometric and physiological characteristics were measured prior to exercise and thereafter underwent one trial of acute exercise of moderate intensity (50-60% of the heart rate reserve - HRR) for 30 minutes on a cycle ergometer (Monark Ergomedic 874E, Monark AB, Vansbro, Sweden). Heart rate (HR) was monitored during exercise by short-range telemetry (Polar RC3 GPS HR, Polar Electro, Kempele, Finland). Blood samples were collected prior to and immediately after exercise for later determination of indices of liver function [γ-glutamyl transferase (γ-GT), aspartate aminotransferase (AST) and alanine aminotransferase (ALT)] and blood redox status [reduced glutathione (GSH), catalase activity, uric acid (UA), total antioxidant capacity (TAC), total bilirubin, thiobarbituric acid-reactive substances (TBARS) and protein carbonyl (PC) levels].

2.3. Blood sampling and handling

Blood samples (15 ml) were drawn from a forearm vein and in order to obtain plasma, a portion of blood was placed in separate tubes, mixed with EDTA (20 µL/mL of blood) and centrifuged at 1370 g for 10 min at 4°C. The supernatant was aliquoted and stored at -80 °C for later determination of TAC, TBARS and PC levels. For red blood cell lysate preparation, packed erythrocytes were diluted with distilled water (1:1 v/v), vortexed vigorously, and centrifuged at 4000 g for 15 min at 4°C. The supernatant was also aliquoted and stored at -80 °C for later analysis of catalase activity and GSH levels. Finally, another portion of blood was collected in separate tubes containing clot activator, left at room temperature for 20 min to clot, and centrifuged at 1370 g for 10 min at 4°C in order to obtain serum. The supernatant was aliquoted and stored at -80 °C for later determination of UA, total bilirubin, γ-GT, AST and ALT.

2.4. Methods

Each variable was analyzed in duplicates on the same day. Samples went through only one freeze-thaw cycle.

Assays in plasma: TAC determination was based on the scavenging of 1,1-diphenyl-2-picrylhydrazyl, according to Janaszewska and Bartosz (22). TBARS levels were measured according to Keles et al. (23). PC levels were measured according to Patsoukis et al. (24).

Assays in red blood cell lysate: Catalase activity was determined according to Aebi (25) and GSH according to Reddy et al. (26). Hb in red blood cell lysate was also determined with a commercially available kit (Dutch Diagnostics BV, The Netherlands), in order to estimate the final levels of GSH and catalase activity.

Assays in serum: UA, total bilirubin, γ-GT, AST and ALT were measured in a Clinical Chemistry Analyzer Z 1145 (Zafiropoulos Diagnostica, Athens, Greece) with commercially available kits (Zafiropoulos, Athens, Greece).

Intra-assay coefficients of variation for GSH, catalase, TAC, UA, total bilirubin, TBARS, PC, γ-GT, AST and ALT were 2.21, 3.38, 2.44, 2.75, 3.81, 2.10, 1.53, 1.15, 1.76 and 2.19, respectively.

2.5. Statistical analysis

Two-way (time x group) repeated measures ANOVA was conducted to examine differences in indices of liver function and blood redox status. If a significant interaction was obtained, pairwise comparisons were performed through simple contrasts and simple main effects analysis using Bonferroni adjustment. Moreover, independent t-test was conducted to examine if there were any differences in the baseline values of anthropometric and physiological parameters. The level of statistical significance was set at $p < .05$. The statistical programme used was SPSS version 18.0 (SPSS Inc., USA). Data are presented as mean ± SEM.

3. Results

3.1. Anthropometric, physiological and other characteristics

WHR and systolic blood pressure (SBP) values were significantly higher ($p < .05$) in EG compared to CG, whereas AUDIT score was much higher ($p < .05$) in EG than CG (Table I). The other anthropometric and physiological characteristics did not differ between groups (Table I).

3.2. Liver function variables

γ-GT: There was a significant main effect of time ($p < .05$), a significant main effect of group ($p < .05$) and a time x group interaction ($p < .05$) for γ-GT. EG had significantly higher ($p < .05$) baseline γ-GT levels compared to CG. Exercise, resulted in significantly higher γ-GT levels ($p < .005$) only in EG (Figure A a).

AST: No significant main effect of group or time x group interaction was observed for AST; however, there was a significant main effect of time ($p < .001$). Pairwise comparisons showed that AST significantly increased ($p < .001$) after exercise in both groups (Figure A b).

ALT: No significant main effect of group was observed for ALT; however, a significant main effect of time ($p < .05$) and a time x group interaction ($p < .05$) was observed. Pairwise comparisons showed that ALT increased significantly ($p < .01$) after exercise only in EG (Figure A c).

3.3. Redox status variables

GSH: No significant main effect of time or time x group interaction for GSH was observed; however, there was a significant main effect of group ($p < .05$), with EG exhibiting significantly lower GSH levels than CG before and after exercise (Figure B a).

Catalase: There was no significant main effect of group or time x group interaction for catalase; however, there was a trend ($p = .07$) for main effect of time. Pairwise comparisons showed increased post-exercise catalase levels in EG (Figure B b).

TAC: There was no significant main effect of group or time x group interaction for TAC; however, there was a significant main effect of time ($p < .005$), with significantly increased ($p < .01$) post-exercise TAC levels in CG and a trend for increased ($p = .06$) post-exercise TAC levels in EG (Figure C a).

UA: There was no significant main effect of group or time x group interaction for UA; however, there was a significant main effect of time ($p < .05$), with significantly increased ($p < .05$) post-exercise UA levels in CG and a trend for increased ($p = .08$) post-exercise UA levels in EG (Figure C b).

Total bilirubin: No significant main effect of time, group or time x group interaction for total bilirubin was detected.

TBARS: No significant main effect of time or time x group interaction for TBARS was observed; however, there was a main effect of group ($p = .06$). Pairwise comparisons showed that there were increased baseline ($p = .08$) and post-exercise ($p = .06$) TBARS levels in EG compared with CG (Figure D).

PC: There was no significant main effect of time, group or time x group interaction for PC.

4. Discussion

4.1. Liver function

To the authors' knowledge this is the first study to investigate the effect of acute exercise on liver function and blood redox status in heavy drinkers. It is well documented that chronic excessive exposure to alcohol can lead to liver inflammation that may eventually impair liver function. A hypothesis of this study was that heavy drinkers would exhibit higher baseline levels of liver enzymes (mainly of γ-GT and ALT) and that exercise would lead to a further increase (mainly in AST).

γ-GT is a common index used in medical practice for detection of liver malfunction or bile ducts. Heavy alcohol drinking can also result in increased γ-GT levels. The results showed that there were increased baseline γ-GT levels in heavy drinkers compared to controls. It is most likely that this finding is related to heavy drinking. Another factor that could, independently of alcohol intake, lead to increased γ-GT levels is cigarette smoking (27); however, heavy drinkers and controls had the same smoking habits and therefore most probably the alcohol intake influenced the γ-GT levels.

AST and ALT are commonly used for detection of inflammation and viral infections of the liver. AST is present in the liver and other tissues including the skeletal muscles, with increased levels of AST indicating muscular inflammation. ALT is mainly found in the liver and in smaller amounts in other tissues such as kidneys and skeletal muscles, thus increased levels of ALT are mainly attributable to liver inflammation (28). Baseline levels of AST and ALT were within the normal limits for men and women in both groups. It has been suggested that increased body mass index (BMI) is an important contributing factor of increased liver enzyme levels in men (29); however, no significant difference in BMI between heavy drinkers and controls was found in the present study. On the other hand, significant increased waist to hip ratio (WHR), which indicates increased visceral fat levels, was evident in heavy drinkers. We believe that differences in physiological (e.g. BMI, systolic BP) and biochemical parameters (e.g. γ-GT and ALT) that were found at the baseline are the result of heavy drinking.

Concerning liver enzyme responses to exercise, γ-GT and ALT levels increased significantly only in EG whereas AST levels increased in both groups after exercise. It is known that exercise can result in transient increases in liver enzymes in healthy individuals, depending on the intensity, duration and type of exercise performed (30, 31). Heavy drinkers may be more prone to increased liver inflammation after moderate intensity exercise due to increased oxidative stress.

Finally, total bilirubin levels did not differ between groups and did not change after exercise. An increase in bilirubin levels at rest can indicate a number of liver function problems, while in healthy individuals can be detected after intense exercise due to hemolysis. Therefore, it is assumed that moderate intensity exercise did not cause hemolysis in heavy drinkers, despite decreased blood GSH levels that could render erythrocytes more susceptible to lipid peroxidation and consequently to hemolysis (32).

Taken all together, findings from the present study indicate that heavy drinking may have resulted in liver inflammation in men that was enhanced by acute exercise. Although acute exercise may trigger increased liver inflammation in heavy drinkers, exercise training could lead to decreased levels of liver enzymes. Previous studies in clinical populations have shown that exercise can ameliorate metabolic abnormalities. It has been found that aerobic exercise training might decrease liver enzyme levels in patients with liver diseases that are not caused by alcohol (33, 34). Training studies to examine the chronic effects of exercise on liver enzymes in individuals with AUDs are needed.

4.2. Redox status

Excessive exposure to alcohol may result in oxidative stress by both increasing ROS production and decreasing antioxidant defense mechanisms (7, 35), and is also thought to be involved in the pathogenesis of alcohol-related diseases (7, 8). On the other hand, acute exercise results in increased production of ROS and also enhances antioxidant defense mechanisms (11, 13). Based on these, we hypothesized that heavy drinkers could be more susceptible to oxidative stress compared to individuals that do not exceed moderate alcohol use, and that exercise could lead to changes in indices of blood redox status in both groups, with heavy drinkers experiencing greater increases in oxidative stress after exercise. The results of this study indicate differences in redox status between heavy drinkers and healthy control

with the former exhibiting lower glutathione and higher TBARS, an index of lipid peroxidation.

GSH is a major cellular thiol antioxidant with many functions that protect cells against oxidative stress and its consequences. Excessive exposure to alcohol can lead to GSH depletion and decreased antioxidant activity (36-38). It has been found that chronic depletion of cytosolic GSH can lead to decreased levels of mitochondrial GSH (39). Alcohol is thought to contribute to GSH depletion in mitochondria of hepatocytes by producing oxidative agents and also by inhibiting the mitochondrial glutathione transporter (transport of GSH from the cytosol into mitochondria) (6, 40, 41). Mitochondrial GSH may be of greater importance for hepatocyte survival than cytoplasmic GSH because its depletion can result in increased production of H2O2 in mitochondria, causing oxidation of cytoplasmic proteins and affecting cell signaling (42). However, impaired redox status also influences changes in erythrocytes and can lead to decreased levels of blood GSH. It has been reported that individuals with alcohol-related liver diseases exhibit low levels of blood GSH (36-38). Although heavy drinkers that participated in this study did not exhibit greater than normal levels of γ-GT, blood GSH levels were significantly lower than the ones of controls. Findings from the present study indicate that heavy drinkers and individuals with AUDs without clinical signs of liver dysfunction may experience lower blood GSH levels than individuals that do not exceed moderate alcohol use.

Impaired redox status can lead to DNA damage, protein modification and lipid peroxidation. Blood redox status indices usually reflect the overall status of the body and we would expect to observe differences between the two groups. Baseline and post-exercise levels of TBARS, which is an index of lipid peroxidation, tended to be higher in heavy drinkers than controls in this study. This result has been reported before in alcoholics and suggests that alcohol abuse results in enhanced lipid peroxidation which in turn leads to increased fragility of cell membranes (6, 37, 43).

Exercise-induced antioxidant response was found to be higher in healthy controls than heavy drinkers, as indicated by post-exercise changes in TAC and UA. Heavy drinkers may not respond well to exercise-induced oxidative stress due to lower antioxidant defenses (35). However, it cannot be concluded whether this antioxidant response in heavy drinkers would increase some hours after exercise. Changes in these indices in more time points after exercise should be examined.

Oxidative stress can alter membrane permeability and lead to hemolysis (44); however, exercise did not lead to hemolysis in heavy drinkers regardless of the increased oxidative stress. This could be explained by the intensity of the exercise used in the present study and also by the fact that antioxidant responses to exercise were increased in similar fashion to the one of the controls.

4.3. Conclusion

Taken together, it is concluded that excessive alcohol intake results in low baseline GSH and increased γ-GT and TBARS levels. Acute aerobic exercise increases the responses of liver enzymes in heavy drinkers whereas the elevated antioxidant responses following the aerobic bout of exercise in heavy drinkers are somewhat attenuated compared to healthy controls. More post-exercise time points could provide a better understanding of the way individuals with AUDs respond to exercise. Finally, since exercise training has been proposed as a useful and safe strategy in the treatment of AUDs, research should focus on training exercise interventions aiming at alcohol use reduction that would prevent or ameliorate alcohol-related liver damage.

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Figure Legends

Figure A: γ-GT (a), AST (b) and ALT (c) levels before and immediately after a trial of acute exercise in heavy drinkers (EG) and control group (CG). *Significantly different from pre-exercise value at the same group ($p < .05$); #Significantly different from CG at the same time point ($p < .05$).

Figure B: GSH (a) and catalase (b) levels before and immediately after a trial of acute exercise in heavy drinkers (EG) and control group (CG). *Significantly different from pre-exercise value at the same group ($p < .05$); #Significantly different from CG at the same time point ($p < .05$).

Figure C: TAC (a) and UA (b) levels before and immediately after a trial of acute exercise in heavy drinkers (EG) and control group (CG). *Significantly different from pre-exercise value of TAC ($p < .05$) and UA ($p < .05$) for CG; significantly different from pre-exercise value of TAC ($p = .06$) and UA ($p = .08$) for EG.

Figure D: TBARS levels before and immediately after a trial of acute exercise in heavy drinkers (EG) and control group (CG). #Significantly different from CG ($p = .08$) at baseline; significantly different from CG ($p = .06$) after exercise.

Table

Table I: Anthropometric, physiological and other characteristics of the subjects (mean ± SE).

Variable	EG	CG
Age (yrs)	31.6 ± 3.2	33.5 ± 1.3
Height (cm)	175.1 ± 1.9	170.3 ± 2.1
Weight (kg)	84.3 ± 3.4	76.4 ± 4.9
BMI (kg/m^2)	27.4 ± 0.8	26.1 ± 1.4
WHR	0.90 ± 0.03[a]	0.82 ± 0.02
Systolic BP (mm Hg)	122.1 ± 2.7[a]	111.7 ± 3.8
Diastolic BP (mm Hg)	80.4 ± 1.9	77.4 ± 2.4
Rest HR	66.2 ± 1.7	65.3 ± 1.4
Exercise HR	128-139	126-138
IPAQ	1322.9 ± 386.9	1340.7 ± 139.0
AUDIT score	17.7 ± 1.3[a]	2.6 ± 0.4
Cigarettes/day	10.7 ± 2.0	10.9 ± 3.5

[a]Significantly different from control group (CG) ($p < .05$). EG: Experimental Group; BMI: Body Mass Index; WHR: Waist to Hip Ratio; BP: Blood Pressure; HR: Heart Rate; IPAQ: International Physical Activity Questionnaire; AUDIT: Alcohol Use Identification Test.

Figure A: -GT (a) levels before and immediately after a trial of acute exercise

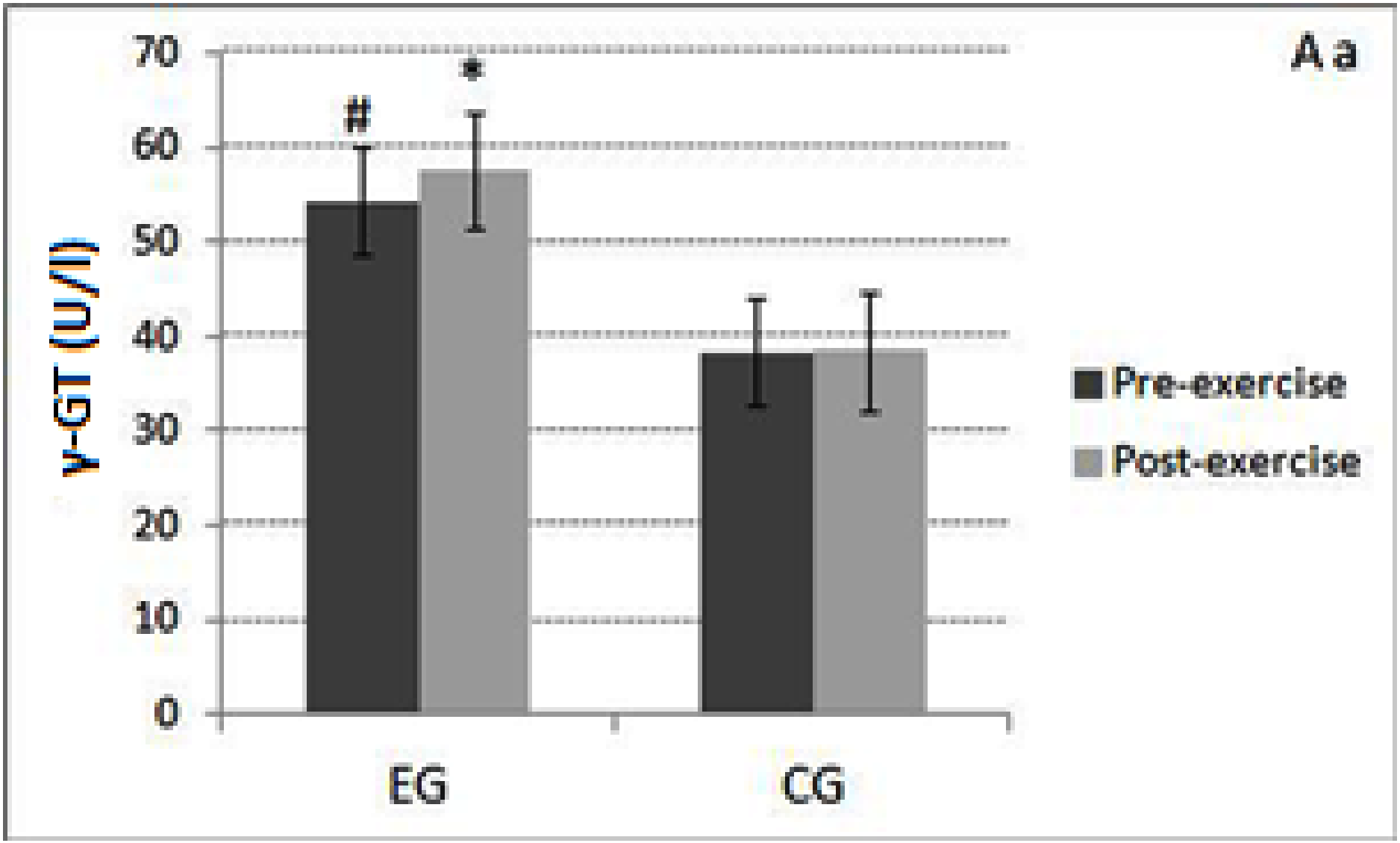

Figure A: AST (b) levels before and immediately after a trial of acute exercise

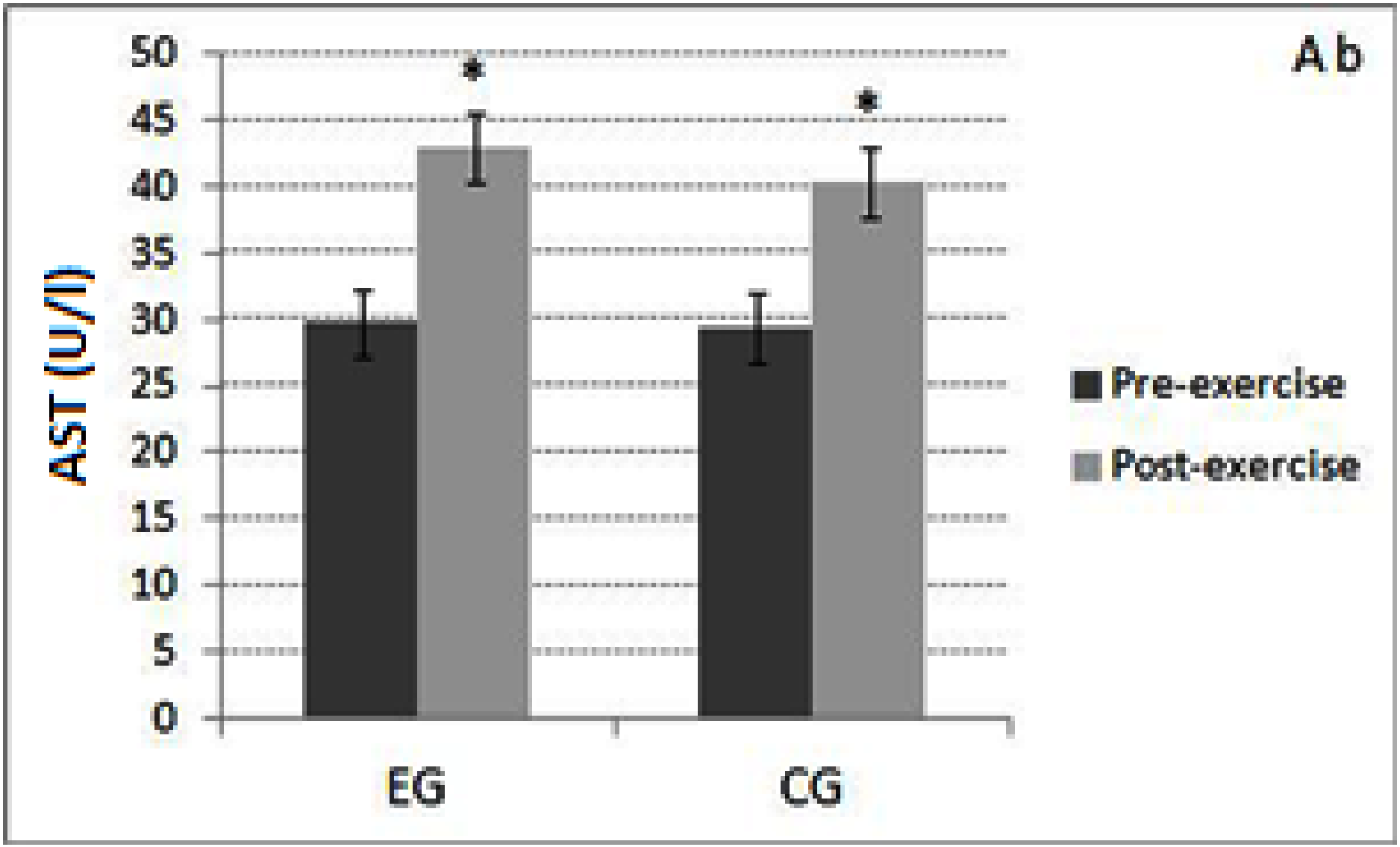

Figure A: ALT (c) levels before and immediately after a trial of acute exercise

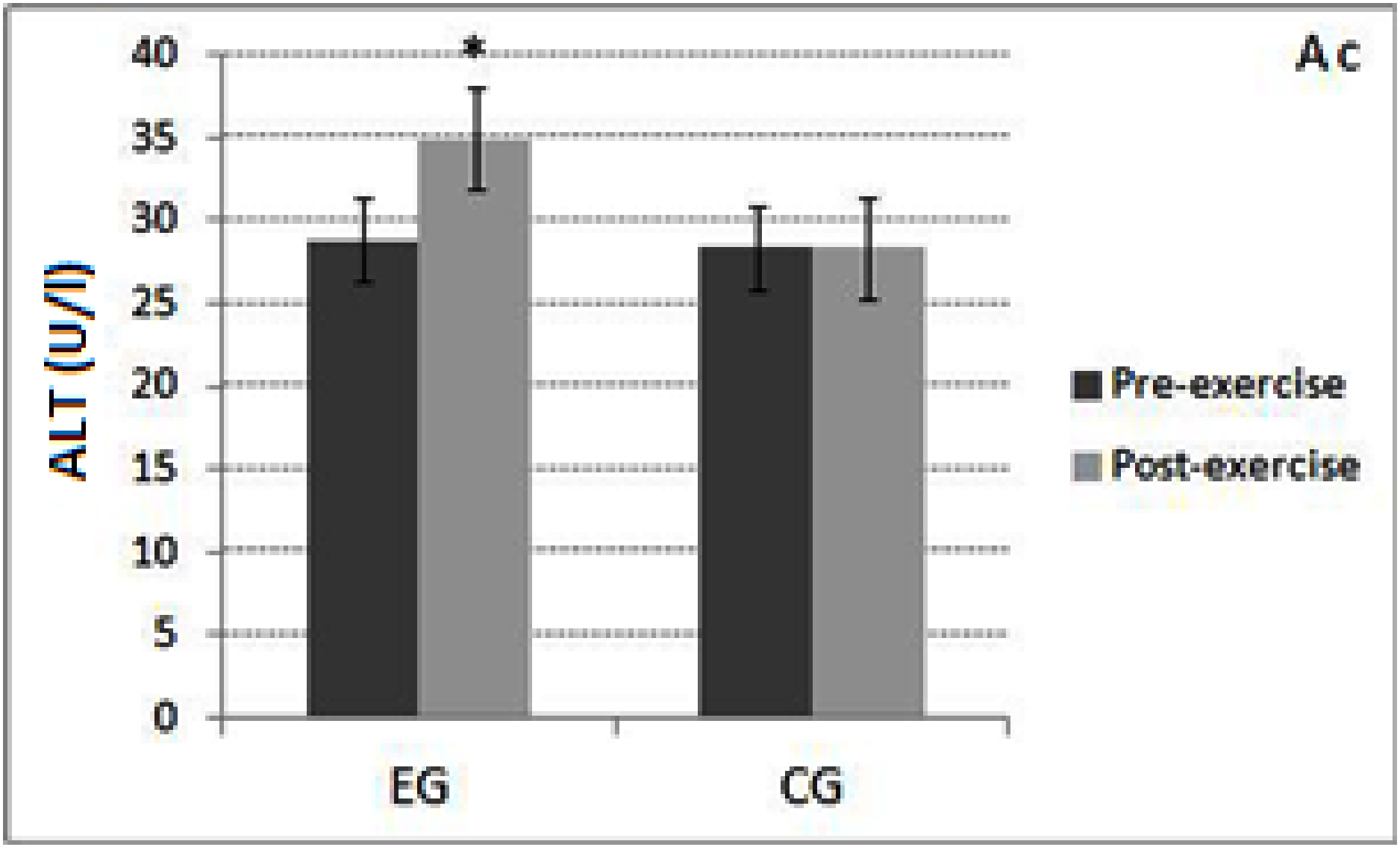

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Figure B: GSH (a) levels before and immediately after a trial of acute exercise

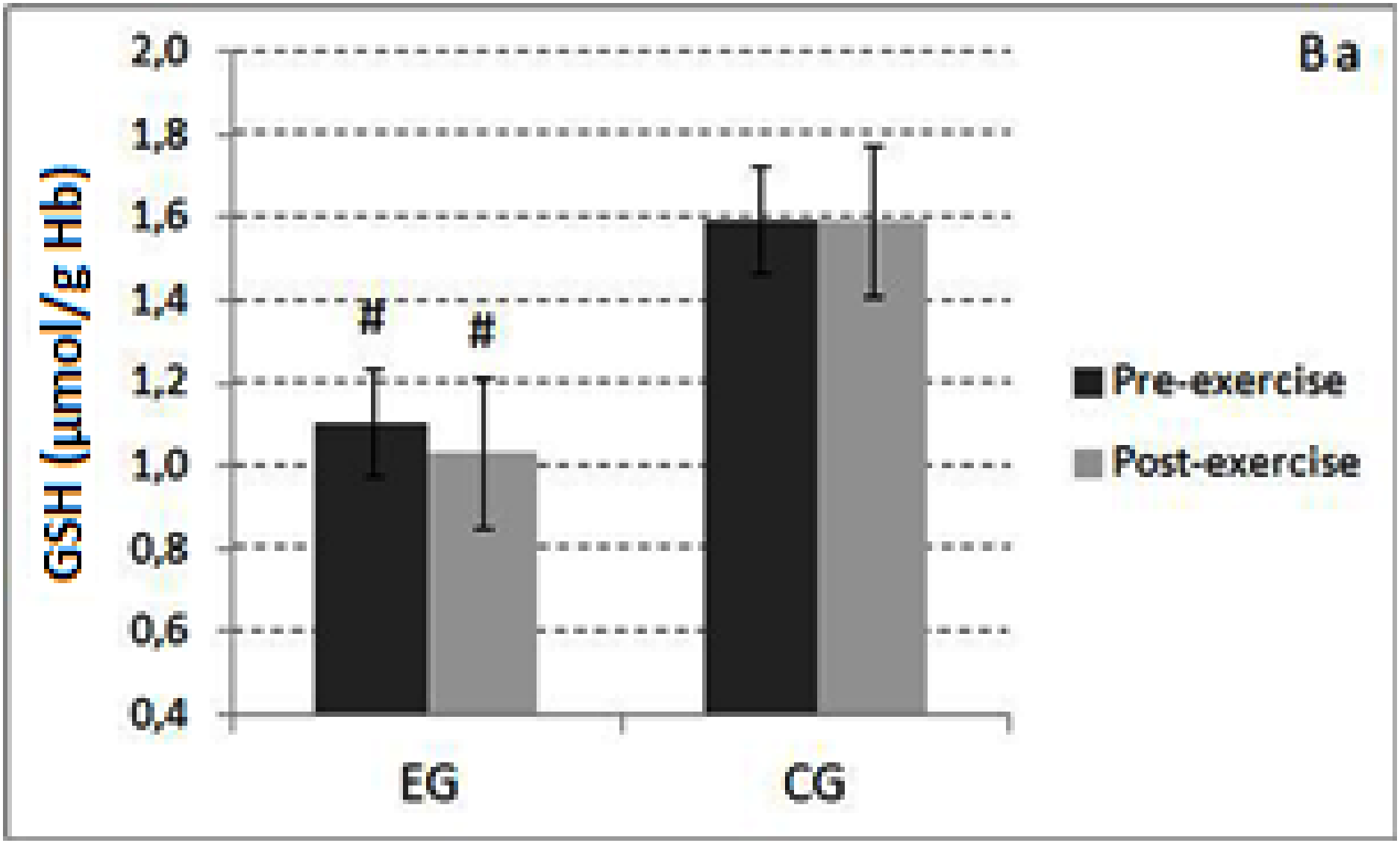

Figure B: Catalase (b) levels before and immediately after a trial of acute exercise

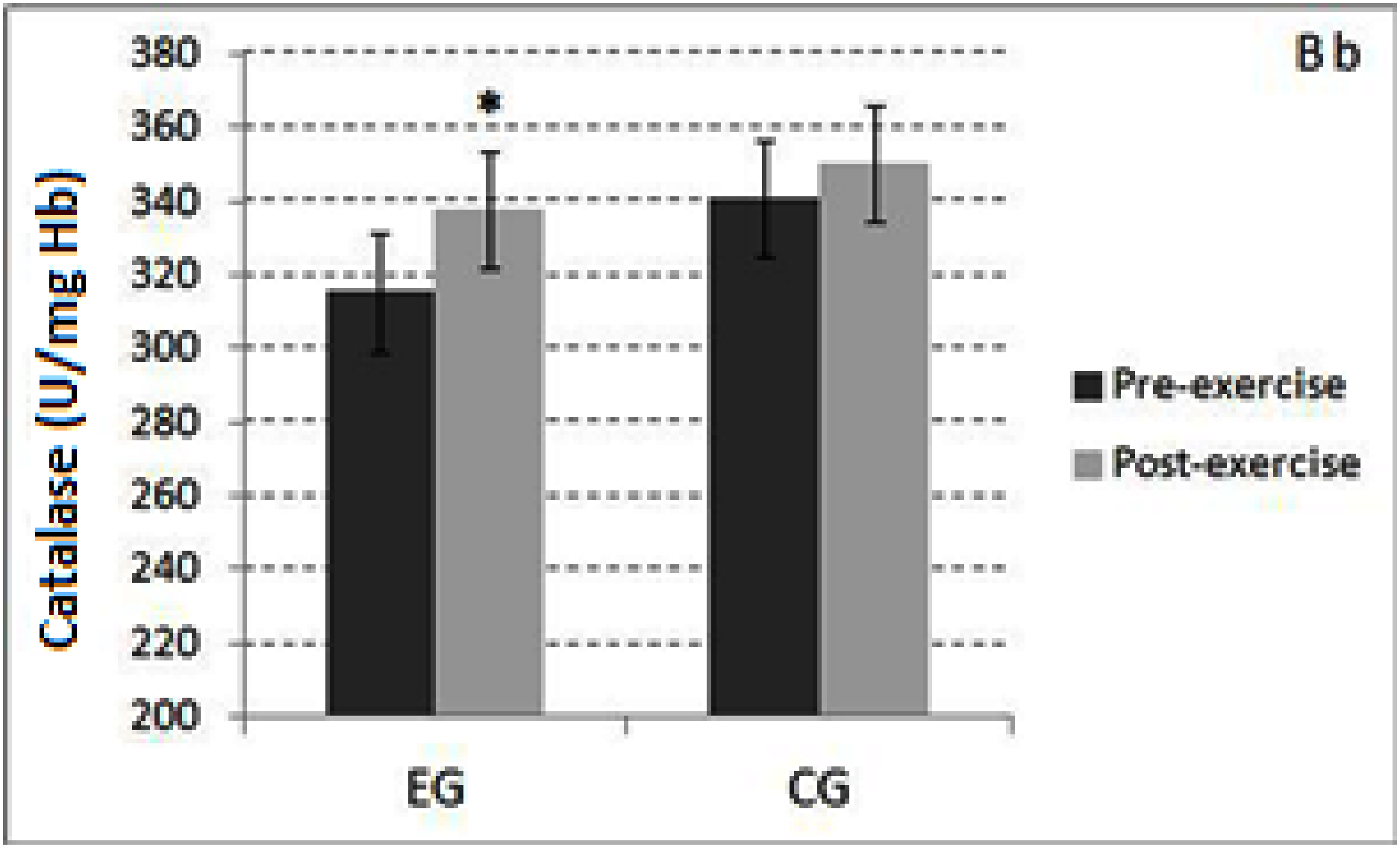

Figure C: TAC (a) levels before and immediately after a trial of acute exercise

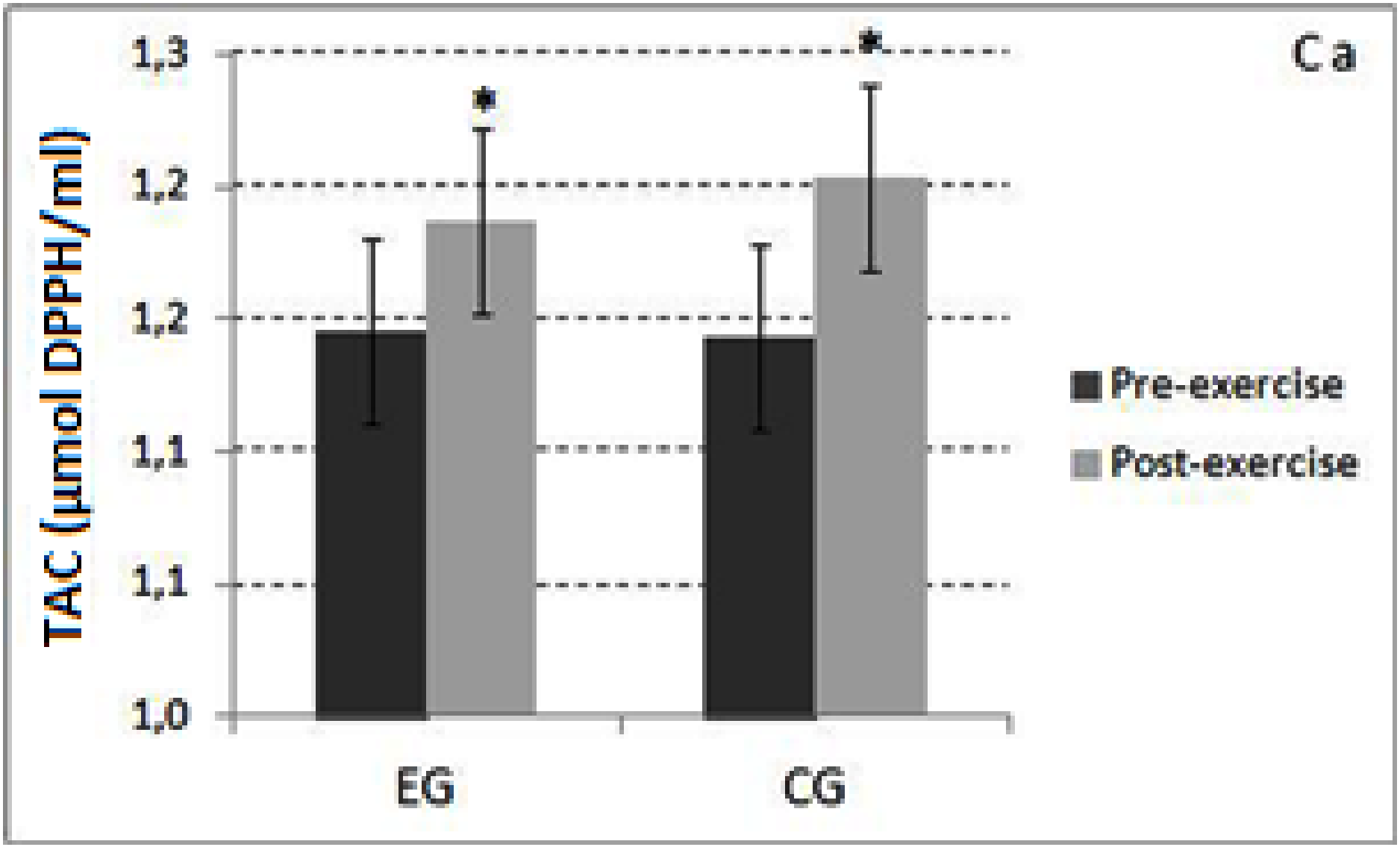

Figure C: Uric Acid (b) levels before and immediately after a trial of acute exercise

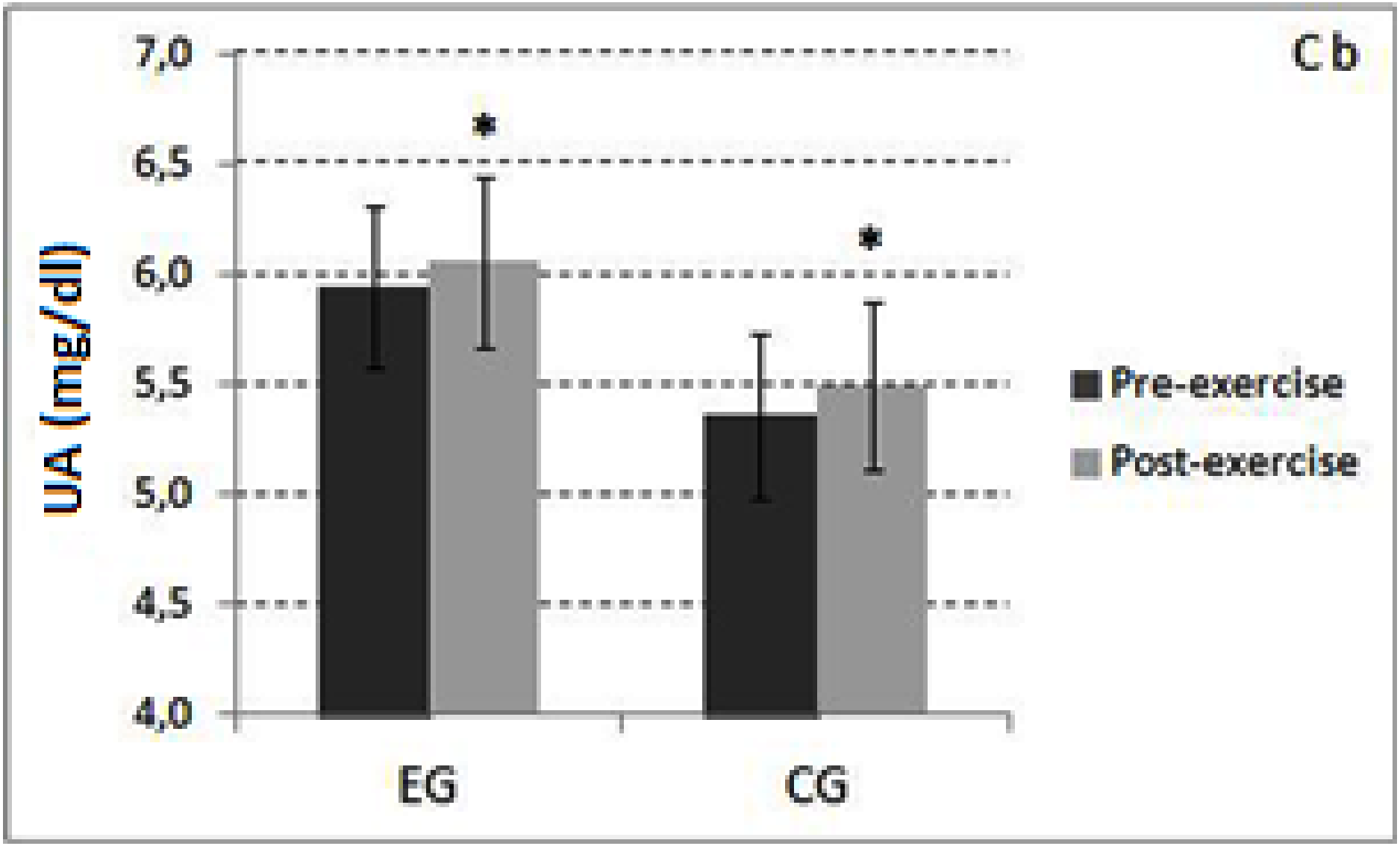

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Figure D: TBARS levels before and immediately after a trial of acute exercise