

# Kinetics of Oxidative Stress Markers at Rest and in Response to Aerobic and Acute Exercise in Judokas Men

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**Abstract:** *The purpose of this study was to compare oxidative modifications following aerobic and acute exercise in judokas men. Judokas performed in random order a low-intensity aerobic exercise bout on a cycle ergometer. Blood samples were analyzed for total antioxidant status, malondialdehyde, glutathione peroxidase, superoxide dismutase, glutathione reductase, and  $\alpha$ -tocopherols. The main results of this study were (a) aerobic and combined exercise had a significant effect on TAS (b) lipid peroxidation indicated by the levels of MDA showed a significant increase after aerobic and combined exercise (c) aerobic and combined exercise induced an increase in GPx, SOD and GR concentrations. Our results suggest different responses within the antioxidant system dependent on the intensity and duration of exercise.*

**Keywords:** Antioxidant, Oxidative stress, Aerobic exercise, Combined exercise.

## 1. Introduction

Oxidative stress is a situation where an imbalance exists between prooxidants and antioxidants, in such a manner that the production of prooxidants (i.e., free radicals) overwhelms antioxidant defense, often leading to oxidation of lipids, proteins, DNA and other molecules which can alter cell function (Bloomer, 2008). To defend themselves against the harmful effects of free radicals, the body has anti-radical systems composed of antioxidants which work in conjunction with antioxidants consumed through dietary means, both natural and synthetic.

Indeed, since the early work of Davies et al. 1986 showing that exercise increases the generation of free radicals in the body, numerous studies have demonstrated in humans and several animal species that acute exercise induces oxidative stress (Bloomer, 2008; Leeuwenburgh et al 2001); and disturbances in intracellular homeostasis occur with an imbalance between prooxidants and antioxidants. The chain of electron transport within mitochondria, the phenomenon of ischemia-reperfusion injury, and local inflammation have all been identified as major sources of production of free radicals during exercise Jenkins et al 1993; Bloomer et al 2004). It is well described that regular exercise training allows for adaptations to the antioxidant defense system which allow for attenuation in acute exercise-induced oxidative stress (Ji, 1999). However, what is less well described is the type of acute exercise that actually results in the greatest increase in oxidative stress. Indeed, studies on the effects of aerobic and combined exercise on oxidative stress and antioxidant status are rare and present divergent results. Fisher-Wellman and Bloomer (2009) provide a review of investigations comparing the effects of aerobic and anaerobic exercise on oxidative stress markers (Alessio et al 2000; Bloomer et al 2005; Inal et al 2001, Marzatico et al 1997).

In their studies, Alessio et al (2000) and Bloomer et al (2005) showed that the aerobic exercise induces a greater increase in protein carbonyl than anaerobic exercise. Furthermore, Inal et al (2001) and Marzatico et al (1997) demonstrated that both acute aerobic and anaerobic exercise increase the activities of antioxidant defense enzymes. Studies on the effects of combined exercise on antioxidant defense and oxidative stress are extremely rare, and to our knowledge, no studies exist comparing combined aerobic-anaerobic exercise with one form of exercise in isolation. Therefore, it was our aim to compare the effects of aerobic exercise alone or in combination on markers of antioxidant activity and oxidative stress. We used a sample of trained male judokas as test subjects, as these men are familiar with the exercise tests employed, and would represent the conditioning level of many well-trained athletes. Moreover, the study of judo athletes is of particular interest because the sport is in constant evolution and growth.

## 2. Materials and Methods

### 2.1 Subjects

Ten judokas (mean age,  $19.1 \pm 1.4$  years; mean body weight,  $73.8 \pm 1.4$  kg; mean height,  $176.1 \pm 4.8$  cm) were studied during the aerobic (trial 1), and combined exercise (trial 2). The combined exercise protocol was mixed between trial 1 and anaerobic exercise (as described below). Judokas trained six days per week, two sessions of 1.5 hours each per day, from Monday to Friday. All judokas regularly practiced judo for the preceding six years prior to participating in the study. No subject used dietary supplements containing antioxidants, or medications during the study period.

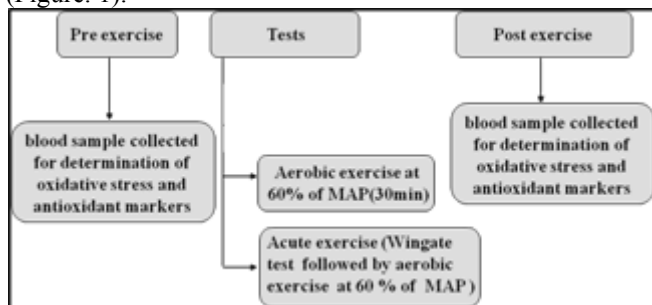
### 2.2 Anthropometric Measurements

Subjects' height was measured using to the nearest 5 mm with a stadiometer and the body mass of each subject was measured to the nearest 100 g with a calibrated electronic

scale (Tanita, TBF 300-A).

### 2.3 Experimental Protocol

All tests were performed at the same time of day (around 08:00 hours) in an attempt to avoid chrono-biological effects. Judokas performed in random order a standard Wingate test (30-second maximal effort sprint cycle test) on a cycle ergometer (Monark type 894E), used as the anaerobic exercise test. The load imposed was 75g/Kg of body mass. As the aerobic exercise test, a low-intensity aerobic exercise bout on a cycle ergometer was performed at intensity equal to 60% of maximal aerobic power (MAP) for a duration of 30 minutes. The combined exercise consisted of both anaerobic and aerobic exercise, with the Wingate test performed first. Prior to each test the subject performed a warm up for 5 minutes at 25 watts using a cycle ergometer (Figure. 1).



**Figure 1:** Schematic illustration of experimental protocol  
MAP: maximal aerobic power

### 2.4 Standard Wingate test

The subjects were instructed to pedal as fast as possible for 30 seconds. The resistance load was adjusted to the pre-determined level, which is about 75 g/kg body mass.

### 2.5 Low-intensity aerobic exercise

The maximum aerobic power (MAP) was first determined by a progressive continuous cycle test. During this test, the cadence was set at 75rpm and the power at the first level was set at 75 Watt (W), with increments of 30 W. The duration of the first stage of testing was 4 min and that of the following stages, two min. The test was terminated when the subject could no longer maintain the imposed cadence, despite encouragement. The value of MAP is the steady power to the final level of the test. Once the MAP was obtained, this value was used for the low-intensity aerobic exercise test, which consisted of pedaling on a cycle ergometer at intensity equal to 60% of maximal aerobic power (MAP) for a duration of 30 minutes

### 2.6 Combined exercise

The exercise test protocol consisted of both anaerobic and aerobic exercise. Specifically, the subject performed a warm up for 5 minutes followed by the performance of a standard Wingate test. At the conclusion of the Wingate test, subjects recovered for 3 minutes, and then performed a low-intensity aerobic exercise bout on a cycle ergometer at an intensity equal to 60% of maximal aerobic power (MAP) for a

duration of 30 minutes.

### 2.7 Blood Collection and Analysis

Blood samples were taken via Vacutainer® from an antecubital vein. Samples were immediately centrifuged for 5 minutes at 4000 rpm at a temperature of 4° Celsius in order to obtain plasma. Plasma was stored in multiple aliquots at -80°C to be used for the measurement of antioxidant and oxidative stress biomarkers. Prior each exercise test a 5mL blood sample was taken. Additional blood samples were taken immediately, 5min, 10min, and 20min following each exercise test. Assays were performed in the laboratory of Pharmacology, Faculty of Medicine of Sfax. All markers were analyzed using commercially available assay kits procured from Randox Laboratories (Randox Laboratories Ltd, UK).

SOD, GR, GPx, and TAS were measured using standard colorimetric assays.  $\alpha$ -tocopherols was extracted with hexane from human plasma and then measured via high performance liquid chromatography (HPLC). Because of their reducing properties, tocopherols are susceptible to oxidation; hence for the importance of ensuring the stability conditions: The levy was realized at fasting and was collected on dry tubes or anticoagulant (lithium heparin or EDTA), after that they were quickly transported to the laboratory of pharmacology to be centrifuged (4°C) at 3000 rpm for 10 minutes. The plasma from these samples was aliquoted into three tubes and was preserved (-80°C) away from light.

For specimen preparation, first 100  $\mu$ l of internal standard and 100  $\mu$ l of plasma were mixed during 5seconds. The second step consisted of adding 200  $\mu$ l of Ethanol and mixed during 30seconds. 500 $\mu$ l of Hexane was supplemented and mixed during 1 minute, and 450  $\mu$ l of the upper layer were removed after centrifugation (4000 rpm to 4°C) for 8 min. 500  $\mu$ l of Hexane were added in the sediment tubes and 450  $\mu$ l of the upper layer were removed after centrifugation (4000 rpm to 4°C) for 8 min and finally evaporated to dryness under a stream of nitrogen at room temperature. Solids were taken by 250  $\mu$ l of methanol and finally centrifuged under the same conditions after shaking 30 seconds, and then analyzed using the HPLC method.

Malondiadehyde (MDA), a measure of lipid peroxidation, was analyzed using a commercially available Malondiadehyde HPLC procedure (Randox Laboratories Ltd, UK). The first step in determining MDA is a sample preparation with derivatisation. The derivatisation reagent transforms MDA into a fluorescent product. Afterwards, the pH is optimized with addition of a reaction solution. 20  $\mu$ l of the supernatant is injected into the HPLC system. The separation via HPLC follows an isocratic method at 30°C using a reversed phase column. One run lasts 4 minutes. The chromatograms are recorded by a fluorescence detector. The quantification is performed with the delivered plasma calibrator; the concentration is calculated via integration of the peak heights by the external standard method. MDA concentrations were measured by the following formula:  $\text{sample} = (\text{Peak height sample} \times \text{concentration of the calibrator}) / \text{Peak height calibrator}$ .

## 2.8 Statistical Analyses

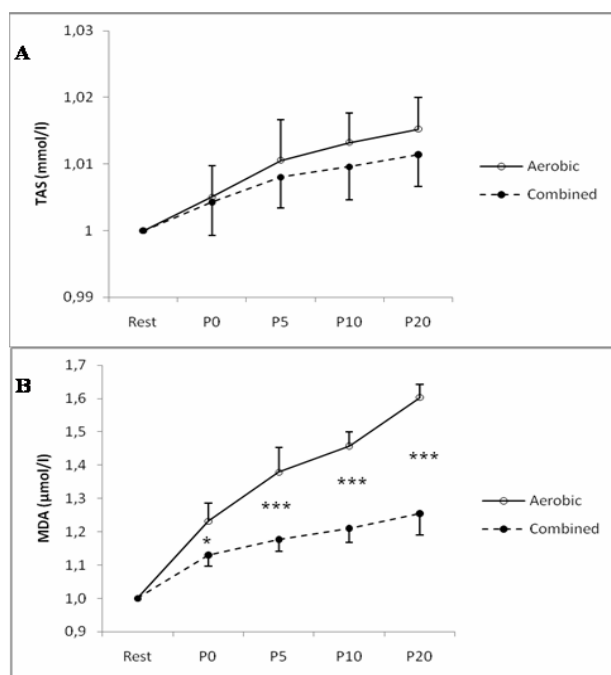
Data were analyzed using the software Statistica (StatSoft, France). Because of the large inter-individual variations in the measured data, we calculated the ratios of post-exercise to pre-exercise values. Values are reported as mean $\pm$ SE (standard error of the mean). Two-way ANOVA was used to detect significant differences between the two types of exercise. Comparisons between pre-exercise and post-exercise within one type of exercise were performed by one way ANOVA with the LSD test. To compare between the two exercises at each post exercise recovery time, the t-test for independent samples was performed. Statistical significance was set at  $p < 0.05$ .

## 3. Results

### 3.1 Oxidative stress markers: Total antioxidant status and malondialdehyde

Figure 2 shows the changes in TAS (Fig. 2 A) and MDA (Fig. 2 B) levels following aerobic and combined exercise.

No significant differences between the two types of exercise were detected. After aerobic exercise, TAS level increased significantly ( $p < 0.05$ ) at 5 minutes of recovery (P5:  $1.82 \pm 0.02$  mmol $\cdot$ l $^{-1}$ ) and at 20 minutes after combined exercise (P20:  $1.82 \pm 0.02$  mmol $\cdot$ l $^{-1}$ ).

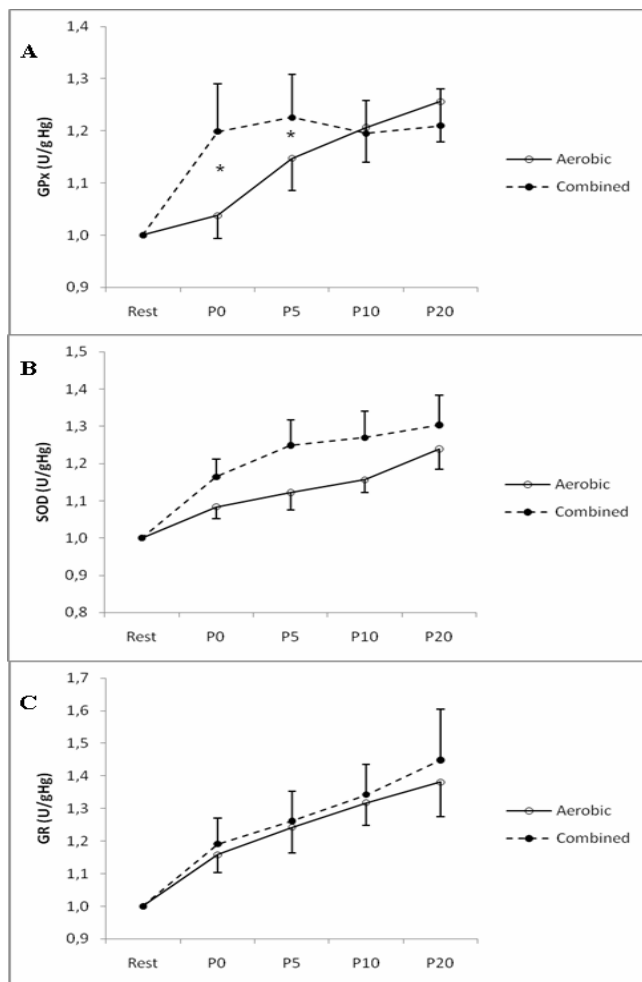


**Figure 2:** Plasma TAS (A) and MDA (B) levels before (Rest), immediately after (P0), and 5 (P5) and 10 (P10) and 20 (P20) minutes after aerobic and combined exercise. Data are expressed as ratios to pre-exercise levels and are shown as means $\pm$ SE. The average level of TAS in samples before aerobic exercise was  $1.8 \pm 0.03$  mmol $\cdot$ l $^{-1}$  and that in samples before combined exercise was  $1.8 \pm 0.02$  mmol $\cdot$ l $^{-1}$ . And the average level of MDA in samples before aerobic exercise was  $1.49 \pm 0.13$   $\mu$ mol $\cdot$ l $^{-1}$  and that in samples before combined exercise was  $1.74 \pm 0.08$   $\mu$ mol $\cdot$ l $^{-1}$ .

Concerning the concentration of TAS, the ANOVA shows a non-significant effect of exercise [ $F(1, 9) = 0$ ;  $p = 0.945$ ]. Secondly it shows a significant effect of recovery [ $F(4, 36) = 5.78$ ;  $p = 0.001$ ]. Finally it shows a non-significant interaction of exercise vs recovery [ $F(4, 36) = 0.11$ ;  $p = 0.979$ ]. Concerning the concentration of MDA, the ANOVA shows a non-significant effect of exercise [ $F(1, 9) = 0.35$ ;  $p = 0.567$ ], a significant effect of recovery [ $F(4, 36) = 32.53$ ;  $p < 0.001$ ] and a significant interaction of exercise vs recovery [ $F(4, 36) = 6.29$ ;  $p < 0.001$ ]. Table 1 shows the effects of exercise on MDA levels after aerobic and combined exercise. After aerobic exercise MDA levels increased significantly ( $P < 0.001$ ) at P0 ( $1.82 \pm 0.16$   $\mu$ mol/l). But after the combined exercise, MDA levels increased above the pre-exercise level ( $P < 0.05$ ). We found a significant difference ( $P < 0.05$ ) between aerobic and combined exercise in terms of the change in MDA levels at 0, 5, 10, and 20 minutes of recovery. The aerobic group had a higher MDA level ( $P < 0.05$ ) than the combined group.

### 3.2 Enzymatic antioxidants

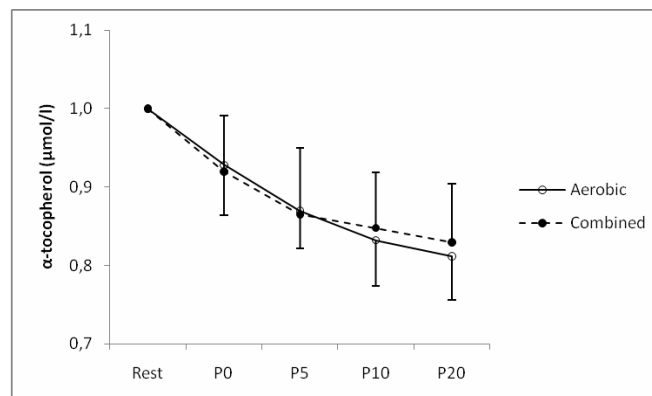
The levels of GPx, SOD, and GR following the two types of exercise are shown in Figure 3 (A, B C). The ANOVA shows respectively, a non significant interaction "exercise x recovery" for the three parameters [ $F(4, 36) = 1.39$ ;  $p = 0.257$ ], [ $F(4, 36) = 0.85$ ;  $p = 0.506$ ], [ $F(4, 36) = 0.04$ ;  $p = 0.997$ ]. After aerobic exercise, a significant increase ( $P < 0.05$ ) in GPx level was apparent at P5, while for the combined exercise it increased at P0 (Table 1). A significant difference was noted between aerobic and combined exercise at P0 and P5. The combined exercise increased the levels of GPx more than aerobic exercise at P0 and P5. The SOD concentrations were significantly increased ( $p < 0.01$ ) just after the end of combined exercise (P0:  $1540.1 \pm 120$  U/gHg) and significantly increased ( $p < 0.05$ ) at P5 after aerobic exercise (P5:  $1462.9 \pm 147.5$  U/gHg). After combined exercise the GR concentrations were increased significantly ( $p < 0.01$ ) at P5 ( $12.6 \pm 2.1$  U/gHg), and significantly increased ( $p < 0.05$ ) at P0 after aerobic exercise (P0:  $10.2 \pm 1.7$  U/gHg). We also note that the concentrations of these two parameters remain elevated after aerobic exercise and combined exercise after 20 minutes of recovery.



**Figure 3:** Plasma GPx (A) SOD (B) and GR (C) levels before (Rest), immediately after (P0), and 5 (P5) and 10 (P10) and 20 (P20) minutes after aerobic and combined exercise. Data are expressed as ratios to pre-exercise levels and are shown as means $\pm$ SE. The average levels of GPx, SOD and GR in samples before aerobic exercise was 35.2 $\pm$ 4.9 U/g Hg, 1313.08 $\pm$ 115.9 U/g Hg, 8.8 $\pm$ 1.3 U/g Hg, and that in samples before combined exercise were respectively 38.4 $\pm$ 6.5 U/g Hg, 1338 $\pm$ 161.7 U/g Hg, 10.5 $\pm$ 2.2 U/g Hg.

### 3.3 Non-enzymatic antioxidant ( $\alpha$ -tocopherol)

After combined exercise, a significant decrease was noted ( $p < 0.05$ ) in concentrations of  $\alpha$ -tocopherol at P20 (Fig. 4), while this decline appeared at P5 for aerobic exercise. No significant difference was found between the types of exercise in terms of  $\alpha$ -tocopherol levels.



**Figure 4:** Title:  $\alpha$ -tocopherol levels before (Rest), immediately after (P0), and 5 (P5) and 10 (P10) and 20 (P20) minutes after aerobic and combined exercise. Data are expressed as ratios to pre-exercise levels and are shown as means $\pm$ SE. The average levels of  $\alpha$ -tocopherol in samples before aerobic exercise was 7.8 $\pm$ 0.9  $\mu\text{mol.l}^{-1}$  and that in samples before combined exercise was 7 $\pm$ 1  $\mu\text{mol.l}^{-1}$ .

## 4. Discussion

The main results of this study were (a) aerobic and combined exercise had a significant effect on TAS, while aerobic exercise induced an increase in TAS at 5, 10, and 20 minutes of recovery, (b) lipid peroxidation indicated by the levels of MDA showed a significant increase after aerobic and combined exercise, with a significant difference between the aerobic and combined exercise at 0, 5, 10, and 20 minutes of recovery, (c) aerobic and combined exercise induced an increase in GPx, SOD and GR concentrations, and (d)  $\alpha$ -tocopherol concentrations were lower after the two tests and no significant difference was observed between the two types of exercises. Regarding the choice of judokas as subjects, we selected men of the same age with a similar physical condition, as these factors may have influenced the markers of oxidative stress in the present design. We also chose to perform all exercise tests on a cycle ergometer in order to minimize learning. Results from the present study indicate that TAS concentration was significantly elevated at P5 for aerobic exercise and at P20 for combined exercise. Some authors (Michailidis et al 2007, Kyparos et al 2009) have reported that aerobic exercise causes an increase in TAS. Furthermore, an increase in uric acid (a primary contributor of TAS) after endurance exercise has been noted (Liu et al 1999; Neubauer et al 2008; Schippinger et al 2009). Concerning the combined exercise, our results are in disagreement with Ascensao et al (2008) who demonstrated that a football match causes an increase in TAS concentrations 30 minutes from the end of a game. As others have noted, they attributed the increase in TAS to an increase in uric acid. It seems necessary therefore, to include a measure of uric acid in future investigations. Our results showed a significant increase in MDA immediately post both aerobic and combined exercise with a significant difference between the aerobic and combined exercise. In a similar way, Baker et al (2004) noted an increase in both lipid hydroperoxides and MDA immediately post a single sprint exercise. This increase of lipid peroxidation can be explained by an increased production of free radicals during and/or

following exercise in the judokas. In contrast to our results, Surmen-Gur et al (1999) found no change in MDA after 20 maximal contractions. This may be explained by the fact that the exercise used in the Surmen-Gur et al (1999) study was not intense enough to cause a significant increase in free radicals. These findings suggest that the volume and intensity of exercises may play an important role in the overall oxidative stress response to sprint exercise.

Aerobic exercise studies performed in trained subjects (Child et al 1998) are in agreement with our results. In contrast, other authors (Niess et al 1996; Margaritis et al 1997) have reported no changes in indices of lipid peroxidation following aerobic exercise. This discrepancy of results may arise from the diversity of assays used to evaluate products of lipid peroxidation in circulation, but also in reference to trained subjects, a lack of identification regarding their specific training status could confound the results. Our data show that the combined exercise causes a significant increase in MDA levels. These results are in agreement with the work of Thompson et al (2003) and Ascensao et al (2008), but are in disagreement with the findings of Svensson et al (1999) and Bloomer et al (2006). This discrepancy probably results from the specificity of training loads applied and physiological characteristics of subjects studied. It is likely that the increase in MDA after the combined exercise is attributed to the combination of radicals produced during both the Wingate test and submaximal aerobic exercise. Our results showed an increase in the level of GPx, SOD and GR at the end of the two exercises. Increasing concentrations of antioxidant enzymes may occur in response to an increased production of free radicals, in an attempt to counteract the radical production and minimize oxidative damage. Regarding aerobic exercise, our results are in agreement with several studies in the literature (Ji et al 1993; Inal et al 2001) who showed that aerobic exercise, whether running or swimming, increases the activity of antioxidant enzymes (SOD, GPx, GR). Ji et al (1993) explains these findings by the fact that the response mechanism of antioxidants may take place after 5 minutes of effort in response to an increase in free radicals. However, this response is proportional to the intensity of exercise and not specifically to the production of free radicals (Criswell et al 1993). Contrary to these findings, Miyazaki et al (2001) found no change in CAT, GPx or SOD in athletes, results that may be explained by the training status of the athletes involved in this investigation. Regarding combined exercise, our results are agreement with the work of Marzatico et al (1997), which demonstrated that SOD and GPx are increased immediately after intermittent exercise (6 \* 150m sprints). The increase on the level of the enzymatic antioxidant may be explained by the increase of the oxygen consumption, acidosis, catecholamines and xanthine oxidase activity. To our knowledge, no study has examined the effect of a combined exercise on GR activity. The relative paucity of data related to the effect of exercise of mixed intensity on GR activity merits attention and further study. Regarding  $\alpha$ -tocopherol concentrations, a decrease was noted after the two tests and no significant difference was observed between the two types of exercises. However our results concerning aerobic exercise are in disagreement with other studies (Liu et al 1999; Mastouldis et al 2001). The same applies for

combined exercise, with our results in discordance with the work of Kingsley et al (2005) and Ferrer et al (2009). The absence of differences in the effects of the two exercises studied on  $\alpha$ -tocopherol concentrations might be explained by possible changes in the diet of subjects studied during the entire experimental period. Our failure to control dietary intake is a limitation of this work and we suggest that future studies include the careful monitoring of dietary intake in order to detect changes in the vitamin status.

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