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The influence of resistance training on antioxidant enzyme activity and oxidative stress

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Abstract
The generation of reactive oxygen species (ROS) is a perpetual biological process in eukaryotic cells. Oxidative stress has been defined as an imbalance between oxidants/antioxidants, prompting damage and death. An increase in macromolecule oxidation has been demonstrated after both aerobic and anaerobic exercise of sufficient intensity. The generation of reactive oxygen and nitrogen species (RONS) such as singlet oxygen, superoxide radical, hydroxyl radical, and peroxynitrite occurs as a consequence of normal cellular metabolism and is increased under conditions of physical stress such as prolonged exercise. There is some evidence that resistance exercise can also induce oxidative stress and generation of free radicals akin to those observed during prolonged aerobic and anaerobic exercise.

Keywords: adaptation, Skeletal muscle, free radicals

1. Introduction

Define of oxidative stress

The generation of reactive oxygen species (ROS) is a perpetual biological process in eukaryotic cells. Oxidative stress has been defined as an imbalance between oxidants/antioxidants, prompting damage and death (Halliwell and Gutteridge, 1989; Sies, 1991; Kirschvink *et al*., 2008) [15, 34, 22]. Organisms are constantly exposed to exogenous and endogenous ROS and reactive nitrogen species (RNS) such as nitric oxide and superoxide anions (Moslen, 1994; Niviere and Fontecave, 1995; Kowaltowski and Verseci, 1999; Kirschvink *et al*., 2008) [27, 28, 24, 23]. To block the injurious effects of ROS and RNS, organisms have evolved different antioxidative systems (enzymatic and non-enzymatic) (Cheeseman and Slater, 1993; Kirschvink *et al*., 2008) [7, 22].

Reactive oxygen and nitrogen species (RONS)

An increase in macromolecule oxidation has been demonstrated after both aerobic and anaerobic exercise of sufficient intensity (Bloomer *et al*., 2006) [4]. The generation of reactive oxygen and nitrogen species (RONS) such as singlet oxygen, superoxide radical, hydroxyl radical, and peroxynitrite occurs as a consequence of normal cellular metabolism and is increased under conditions of physical stress such as prolonged exercise (Sen, Packer, & Hanninen, 1994) [37]. In anaerobic exercise, however, other pathways of RONS generation exist (Bloomer & Goldfarb, 2004) [3] including ischemia reperfusion, xanthine and nicotinamide adenine dinucleotide phosphate oxidase production, prostanoid metabolism, phagocytic respiratory-burst activity, disruption of iron-containing proteins, and altered calcium homeostasis (Bloomer *et al*., 2006) [4]. The production of RONS via these pathways may result partly from eccentric muscle action, which causes muscle microtrauma and subsequent inflammation (McHugh *et al*., 1999) [25].

The relationship between resistance exercise and oxidative stress

There is some evidence that resistance exercise can also induce oxidative stress and generation of free radicals akin to those observed during prolonged aerobic and anaerobic exercise (Goldfarb *et al*., 2008; Hudson *et al*., 2008; Ramel, Wagner, & Elmadfa, 2004) [13, 16, 32]. For example, erythrocytes are subject to mechanical and oxidative stress during strenuous contractile work (Moore, Gioioso, Sills, & Mendelson, 1999) [26]; it has been shown that reactive oxygen species are released into the circulation by immune, endothelial, and muscle cells (Radak, Chung, & Goto, 2008; Valko *et al*., 2007) [31, 35] and impair normal erythrocyte function through lipid-membrane peroxidation.

Redox homeostasis

Exercise is associated with increases in both ATP requirements and aerobic and/or anaerobic metabolism, which result in higher levels of ROS and RNS (Davis *et al*., 1982; Jackson *et al*., 1985; Viguie *et al*., 1993; Inoue *et al*., 1993; Sen, 1995; Nojima *et al*., 2008) [10, 19, 38, 39, 40, 41]. Thus, when exercise is strenuous it causes oxidative stress and cell damage, but when done in moderation, it increases the expression of antioxidant enzymes (Gomez-Cabrera *et al*., 2008) [48]. The preventive effect of regular exercise is, at least partly, due to oxidative stress-induced adaptation. This response is systemic and includes enhancement of antioxidant systems and the reduction of oxidative damage due to changes in redox homeostasis (Radak *et al*., 2008) [31]. On the other hand, although training increases the antioxidant defence system of the organism, prolonged periods of training may cause disturbances in the oxidant/antioxidant equilibrium (Avellini *et al*., 1995; De Moffarts *et al*., 2005; Kirschvink *et al*., 2008) [22, 42, 43].

Skeletal muscle

Skeletal muscle is a highly specialized tissue with excellent plasticity in response to external stimuli such as exercise and training. The repetitive muscle contractions conducted during endurance training lead to a variety of phenotypic and

physiological responses. These responses include activation of mitochondrial biogenesis, fiber type transformation and angiogenesis. Together, they increase the muscle's capacity of aerobic metabolism and its resistance to fatigue. High muscle activity also involves a strong increase in reactive oxygen species (ROS) production. These unstable molecules and ions contain oxygen and are extremely reactive due to an unpaired electron. Among these oxygen intermediates are the free radicals superoxide, peroxide and the hydroxyl radicals and other highly reactive oxidants, such as singlet oxygen and hypochlorous acid. They promote oxidation reactions with other molecules, such as proteins, lipids and DNA and can thus be highly detrimental. However, recent research has demonstrated that ROS also have a beneficial role in promoting the adaptive responses of muscle to training (Dillard *et al*., 1978) [11].

Sources of ROS in Muscle

It has consistently been shown that muscle activity leads to a strong increase in ROS production (Powers *et al*., 2008) [30]. However, there is a large debate about the sources and the extent of ROS that these sources produce. Several potential producers of ROS have been identified in muscle cells which are likely to be activated by different stimuli. Among these are mitochondria, nicotinamide adenine dinucleotide phosphate (NADPH) oxidases (NOXs), phospholipase A2 (PLA2), xanthine oxidase (XO) and lipoxygenases. Some of these are discussed in more detail below. In addition to these intracellular sources, ROS has been shown to be produced from non-muscle sources. Strenuous exercise can elicit muscle injuries, which then lead to the activation of the neutrophils and macrophages via interferon (IFN), interleukin (IL) and tumor necrosis factor (TNF) (for more detailed information see reviews (Peake *et al*., 2004) [29].

Exogenous Antioxidants and Exercise

Apart from the endogenous antioxidants, which are obviously regulated by exercise, exogenous antioxidants such as vitamin C, E, and carotenoids are taken up with the food or are used as dietary supplements. The question therefore arises whether such supplements can be considered beneficial during exercise. To address this question, Ristow *et al* (2009) [45] investigated the effects of a diet supplemented with vitamin C and E on exercise-induced insulin sensitivity as measured by glucose infusion rates during a hyperinsulinemic, euglycemic clamp in previously untrained and pre-trained healthy young men. Interestingly, exercise was found to increase parameters of insulin sensitivity (including adiponectin) only in the absence of antioxidants in both previously untrained and pretrained individuals. This was paralleled by increased expression of ROS-sensitive transcriptional regulators of insulin sensitivity and ROS defense capacity, peroxisome proliferator-activated receptor (PPAR) and PPAR captivators PGC only in the absence of antioxidants. Molecular mediators of endogenous ROS defense (Mn-SOD, Cu, Zn-SOD and GPX) were also induced by exercise, and this effect was again blocked by antioxidant supplementation. The authors concluded that exercise-induced oxidative stress ameliorates insulin resistance and causes an adaptive response promoting endogenous antioxidant defense capacity and that supplementation with antioxidants may preclude these health-promoting effects of exercise in humans. It was demonstrated that exercise causes an activation of mitogen-activated protein kinases (MAPKs: p38, ERK 1 and ERK 2), which in turn activates nuclear factor B (NF-B) in rat gastrocnemius muscle and consequently the expression of important enzymes associated with defense against ROS (SOD) and adaptation to exercise—endothelial nitric oxide synthase (eNOS) and inducible nitric oxide synthase (iNOS) (Gomez *et al*., 2005; Ji *et al*., 2004) [14, 44]. The expression of these enzymes can be inhibited by allopurinol, an inhibitor of XO indicating also that the prevention of ROS formation causes an inhibition of an adaptive response. The authors therefore conclude that in all likelihood, antioxidant supplements should not be recommended before training as they interfere with muscle cell adaptation. Thus, physical exercise is considered a double-edged sword: when practiced strenuously it causes oxidative stress and cell damage; in this case application of antioxidants may be helpful. But when practiced in moderation, it increases the expression of antioxidant enzymes and thus should be considered an antioxidant (Gomez *et al*., 2005; Ji *et al*., 2004) [14, 44]. Supportive evidence for this assumption comes from studies on physical overtraining.

Role of free radicals in muscle adaptation to exercise

The idea of the deleterious effects of free radicals has been firmly entrenched in the minds of scientists for the past 30 years. However, there is now an appreciation that the reactive oxygen species generated during muscle contraction have a physiological role in the adaptation to exercise. In response to the free radical assault, the cell has developed a number of antioxidant defense systems such as superoxide dismutase, the peroxidases, the glutathione redox cycle with its associated constitutive enzymes, as well as glutathione itself, whose concentration is higher in the cell than that of glucose (Vina, 1990) [36]. Therefore the cell has become well equipped to deal with the normal production of reactive oxygen species. There is growing evidence that the continued presence of a small stimulus such as low concentrations of reactive oxygen species is in fact able to induce the expression of antioxidant enzymes and other defense mechanisms. The basis for this phenomenon may be encompassed by the concept of hormesis (Calabrese *et al*., 2005) [46], which can be characterized as a particular dose–response relationship in which a low dose of a substance is stimulator and a high dose is inhibitory. In this context radicals may be seen as beneficial, as they act as signals to enhance defenses, rather than as deleterious as they are when cells are exposed to high levels of these radicals. Recently the hormesis theory has been extended to the ROS-generating effects of exercise.

Material and methods

This article is review and the aims are the effect of resistance training on oxidative stress and antioxidant enzyme activity. The experiment 1 was conducted by (Azizbeigi *et al*., 2013) [2]. Twenty untrained men with no experience of resistance training or regular physical activity volunteered to participate in the study and were assigned to one of two homogeneous groups: Progressive resistance training (PRT, n = 10) and control (n = 10). The subjects followed a generally sedentary lifestyle and had not practiced formal physical exercise for more than 1 hr/week during the preceding 3 months (Azizbeigi *et al*., 2013) [2]. Subjects recorded all their physical

activities in 15-min intervals in activity diaries over a 4-day period before initiation of the study (Ainsworth et al., 2000) [1]. In addition, the groups were matched according to age, body mass, height, percentage body fat, body-mass index, and estimated 1-repetition-maximum (E1RM) values (Table 1) (Azizbeigi *et al*., 2013) [2].

Table 1: Physical Characteristics of Subjects at the Start of the Study, M ± SD (Azizbeigi *et al*., 2013) [2].

	Resistance-training group	Control group	*t*	*p*
Age (years)	21.2 ± 2.1	23.3 ± 2.5	−1.45	.157
Body mass (kg)	72.61 ± 3.86	71.37 ± 3.27	0.660	.517
Height (cm)	173 ± 3.67	176 ± 6.68	−1.25	.225
Body fat (%)	19.51 ± 6.12	18.54 ± 5.52	0.805	.431
Body-mass index (kg/m^2)	24.28 ± 2.17	23.09 ± 1.45	1.47	.158
E1RM chest press (kg)	33.5 ± 6.2	31.47 ± 7.2	0.943	.358

Note. E1RM = estimated 1-repetition maximum.

They were informed of the purposes and methods of the study before they provided written consent. None of the participants were taking any form of medication, nor did they have alcohol, smoking, or vitamin supplement habits. The subjects were warned against taking nonsteroidal anti-inflammatory drugs including aspirin and naproxen during the research project (Azizbeigi *et al*., 2013) [2]. The study design and experimental procedures were approved by the Regional Research Ethics Committee of Islamic Azad University, Central Tehran Branch. Subjects' height and weight (Seca, Mod 220, Germany) [47] were measured, and their percentage body fat was estimated estimated through measuring skinfold thickness (Lafayette, Mod 01127, USA). Skinfold thickness was measured at three sites—abdomen, suprailiac, and triceps—and percentage body fat was estimated using the equation of Jackson and Pollock (1985). PRT subjects underwent a whole day of weight training and safety precautions after the collection of pretraining blood samples. Session 1 included a general warm-up consisting of 3–5 min of low-intensity running, 5–10 min of stretching exercises, and four warm-up sets leading up to the final set to determine predicted maximum strength (see Table 2).

Table 2: Sets and Repetitions for Session 1, Familiarization and Maximum-Strength Protocol (Azizbeigi *et al*., 2013) [2].

Set	Number of repetitions
1: 50% of self-reported maximum	10
2: 50–60% of self-reported maximum	8–10
3: 70% of self-reported maximum	8
4: 80% of self-reported maximum	3
5: ≥85% of self-reported maximum	Maximum repetition but fewer than 3

This procedure was repeated for both upper body (UB) and lower body (LB) exercises. For both UB and LB exercises, the initial loads used during the warm-up sets were based on percentages of the participants' self-reported estimate of their 1-RM. The warm-up sets leading to the final maximum repetition attempt had a dual purpose of warming up the participant for the attempt and familiarizing him with the metronome-established cadence for the repetitions.

The experiment 2 was conducted by Elabed *et al*., (2014) [12]. Ten regional level judokas (mean age, 18.1 ± 1.7 years; mean body weight, 77.2 ± metricconverter- ProductID11.7 kg11.7 kg; mean height, 176.4 ± metricconverterProductID4.6 cm4.6 cm) were studied during the anaerobic exercise, and ten regional level judokas (mean age, 19.1 ± 1.4 years; mean body weight, 73.8 ± 1.4 kg; mean height, 176.1 ± 4.8 cm) were studied during the aerobic, and combined exercise. Judokas performed in random order a standard Wingate test on a cycle ergometer (Monark type 894E), used as the anaerobic exercise test (Elabed *et al*., 2014) [12]. 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 for duration of 30 minutes. The combined exercise consisted of both anaerobic and aerobic exercise, with the Wingate test performed first. For Standard Wingate test the participants were instructed to pedal as fast as possible for 30 seconds (Elabed *et al*., 2014) [12]. Then low-intensity aerobic exercise consisted of pedaling on a cycle ergometer at an intensity equal to 60% of maximal aerobic power for a duration of 30 minutes, and the combined exercise protocol consisted of both anaerobic and aerobic exercise. Specifically, the participants performed a warm up for 5 minutes followed by the performance of a standard Wingate test. At the conclusion of the Wingate test, participants 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 for a duration of 30 minutes. Blood samples were taken via Vacutainerrfrom an antecubital vein. All markers were analyzed using commercially available assay kits procured from Randox Laboratories (Randox Laboratories Ltd, placecountry-regionUK). SOD, GR, GPx, and TAS were measured using standard colorimetric assays. a-tocopherols was extracted with hexane from human plasma and then measured via high performance liquid chromatography (HPLC). For specimen preparation, first 100 µl of internal standard and 100 µl of plasma were mixed during 5seconds. The second step consisted of adding 200 µl of Ethanol and mixed during 30 seconds. 500 µl of Hexane was supplemented and mixed during 1 minute, and 450 µl of the upper layer were removed after centrifugation (4000 rpm to 4!C) for 8 min. 500 µl of Hexane were added in the sediment tubes and 450 µl 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 (Elabed *et al*., 2014) [12]. Solids were taken by 250 µl 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, placecountry-regionUK). MDA concentrations were measured by the following formula: sample = (Peak height sample ⇥ concentration of the calibrator)/ Peak height calibrator. Data were analyzed using

the software Statistica (Cityplace Stat Soft, country-region 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±SE (standard error of the mean). Two-way ANOVA was used to detect significant differences between the three types of exercise (Elabed *et al*., 2014) [12]. 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 three exercises at each post exercise recovery time, the t-test for independent samples was performed. Statistical significance was set at $p < 0.05$ (Elabed *et al*., 2014) [12].

Results and Discussion

The experiment 1 was conducted by (Azizbeigi *et al*., 2013) [2]. Before training, the daily energy intake of the PRT group consisted of 51% ± 3%, 31% ± 2%, and 18% ± 3%, respectively, for contributions from carbohydrate, fat, and protein, proportions not significantly different from those of the control group (48% ± 3%, 33% ± 2%, 19% ± 3%). Although individual and total macronutrient intake increased during training, there remained no significant difference between groups. Nutrient analysis of the dietary records of the PRT and control groups before and after the training period using independent t test are presented in Table 3. There was no significant difference between the PRT and control groups regarding percentage body fat. There was a significant interaction between group and time ($p = .014$) for SOD activity but no main effect for time, showing that SOD activity increased as a function of the PRT program and not simply time between tests. Similarly, MDA was significantly decreased with PRT ($p = .030$), but there was also a tendency for a main (time only) effect. Neither total antioxidant capacity nor GPx changed during the course of the study.

The experiment 2 was conducted by Elabed *et al*., (2014) [12]. Figure 1 shows the changes in TAS (Figure 1(a)) and MDA (Figure 1(b)) levels following aerobic anaerobic and combined exercise.

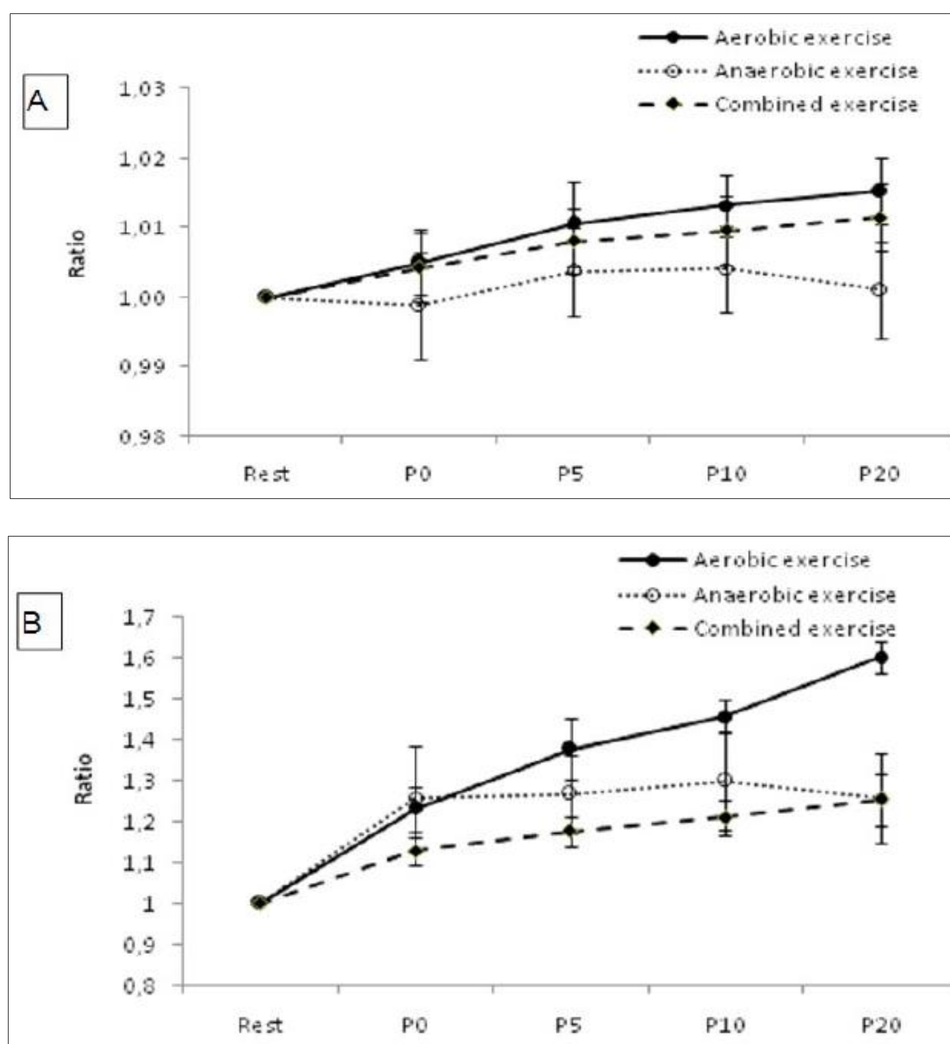

Fig 1. Plasma TAS (A) andMDA (B) levels before (Rest), immediately after (P0), and 5 (P5) and 10 (P10) and 20 (P20) minutes after aerobic, anaerobic, and combined exercise. Data are expressed as ratios to pre-exercise levels and are shown as means±SE. The average level of TAS in samples before anaerobic exercise was 1.797±0.037 mmol·l-1 and that in samples before aerobic exercise was 1.799±0.037 mmol·l-1 and that in samples before combined exercise was 1.803±0.022 mmol·l-1. And the average level of MDA in samples before anaerobic exercise was 1.64±0.27 µmol.l-1 and that in samples before aerobic exercise was 1.49±0.13 µmol.l-1 and that in samples before combined exercise was 1.74±0.08 µmol.l-1 (Elabed *et al*., 2014) [12]

No significant differences between the three types of exercise were detected (Elabed *et al*., 2014) [12]. After aerobic exercise, TAS level increased significantly ($p<0.05$) at 5 minutes of recovery (P5: 1.012 ± 0.01 $mmol \cdot l^{-1}$). Concerning the concentration of TAS, the ANOVA shows a non-significant effect of exercise [$F (2.18) = 1.27$; $p > 0.05$]. Secondly it shows a significant effect of recovery [$F (4.36) = 3.47$; $p < 0.05$]. Finally it shows a non-significant interaction of exercise vs recovery [$F (8.72) = 0.44$; $p > 0.05$]. Concerning the concentration of MDA, the ANOVA shows a non-significant effect of exercise [$F (2.18) = 3.03$; $p > 0.05$], a significant effect of recovery [$F (4.36) = 24.66$; $p < 0.001$] and a significant interaction of exercise vs recovery [$F (8.72) = 3.51$; $p < 0.01$]. Figure 1(b) shows the effects of exercise on MDA levels (Elabed *et al*., 2014) [12]. Immediately after aerobic anaerobic and combined exercise, MDA levels increased significantly ($P<0.05$). But after the aerobic exercise, MDA levels increased above the pre-exercise level ($P<0.05$). We found a significant difference ($P<0.05$) between anaerobic and aerobic exercise in terms of the change in MDA levels at P10 and P20, and a significant difference ($P<0.05$) between combined and aerobic exercise in MDA levels at 5, 10, and 20 minutes of recovery. The aerobic group had a higher MDA level ($P<0.05$) than the anaerobic and combined group (Elabed *et al*., 2014) [12].

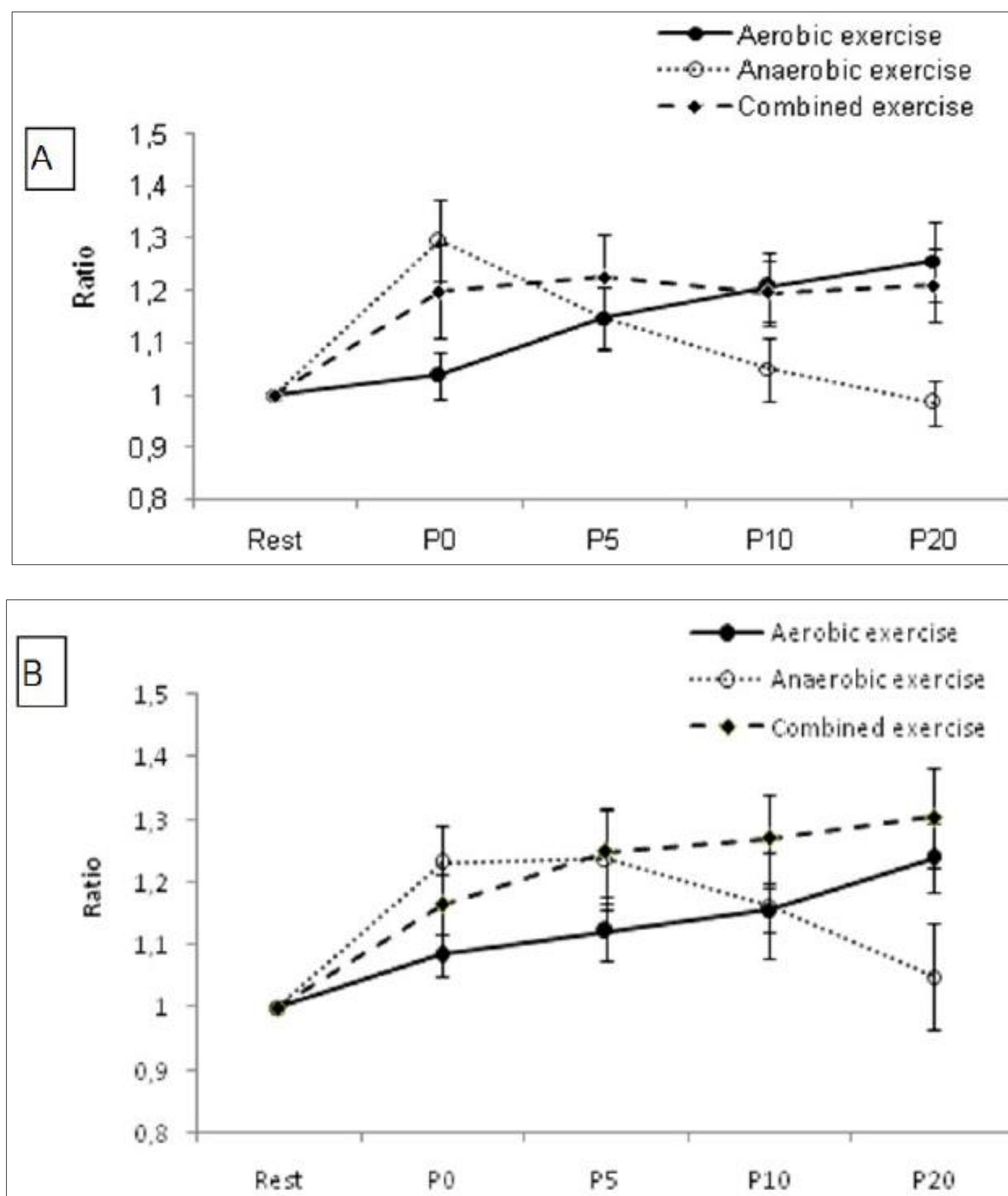

Fig 2. Plasma GPx (A) SOD (B) and GR (C) levels before (Rest), immediately after (P0), and 5 (P5) and 10 (P10) and 20 (P20) minutes after aerobic, anaerobic, and combined exercise. Data are expressed as ratios to pre-exercise levels and are shown as means±SE. The average levels of GPx, SOD and GR in samples before anaerobic exercise were respectively 36.5±7 U/g Hg, 1354.1±239.2 U/g Hg, 9±2.1 U/g Hg; and that in samples before aerobic exercise was 35.2±4.9 U/g Hg, 1313.08±115.9 U/g Hg, 9.8±1.3 U/g Hg, and that in samples before combined exercise were respectively 38.4±6.5 U/g Hg, 1338±161.7 U/g Hg, 10.5±2.2 U/g Hg.

The levels of GPx, SOD, and GR following the three types of exercise are shown in Figure 2(A, B, C). The ANOVA shows respectively, a significant interaction" exercise x recovery" for the three parameters [F (8.72) = 5.79; p < 0.001], [F (8.72) = 3.16; p < 0.01], and [FF (8.72) = 2.99; p < 0.01]. 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. In contrast, a significant increase was detected in samples taken after anaerobic exercise at P0 and P5. The GPx levels were returned to baseline values at P10. A significant difference was noted between aerobic and anaerobic exercise at P0, P10, and P20, and between aerobic and combined exercise at P0. A significant difference also appeared between anaerobic and combined exercise at P10 and P20. The anaerobic group had a higher GPx level than the aerobic group at P0, and combined exercise increased the levels of GPx more than aerobic exercise at P0. Concerning the comparison between anaerobic exercise and combined exercise, we note that combined exercise resulted in a greater GPx than anaerobic exercise at P10. For concentrations of SOD and GR, statistical analysis shows that aerobic exercise and combined exercise have SOD and GR concentrations significantly higher than those for anaerobic exercise at P20. We also note that the concentrations of these two parameters returning to basal values at P20 after anaerobic exercise and remain elevated after aerobic exercise and combined exercise after 20 minutes of recovery.

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