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Original Article

Effect of exercise intensity and oxygen consumption on blood lipid peroxidation

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Abstract
Background: It is well known that physical exercise is associated with an increase in reactive oxygen species production due to oxygen consumption. However, other factors related to the intensity of exercise could also be important in understanding the oxidative stress related to exercise. Thus, this study aimed to analyze the influence of exercise intensity on plasma lipid peroxidation levels, considering the same total amount of oxygen consumption. **Methods:** Twelve male trained subjects (aged 20.8 ± 2.6 years) were enrolled in this study. The procedures were divided into three distinct phases at intervals of 96 hours. Firstly, all subjects were submitted to a maximal oxygen capacity test on a cycloergometer to define the exercise intensity for subsequent tests. Then, an exercise protocol consisting of 20 minutes at 55-60% of maximal oxygen uptake and 15 minutes at 75-80% of maximal oxygen uptake was performed on a cycloergometer. Although the intensity and duration of the two exercises were different, the total amount of oxygen consumption was similar. A blood sample for MDA determination was collected before, immediately after, and one hour after each physical test. **Results**: Plasma MDA concentration increased immediately after both exercise tests and remained high one hour after both exercise protocols. The results showed that plasma MDA concentration was significantly higher in the exercise with higher intensity compared to the lower intensity exercise protocol. **Conclusion**: Our results suggest that lipid peroxidation induced by acute exercise depends not only on the total amount of oxygen consumed during physical exercise but also on the intensity of the exercise.
Keywords: VO_2, MDA, oxidative stress, cycloergometer.

Introduction

Although oxygen is indispensable for aerobic organisms' survival it can also be a deleterious chemical since is associated with several cell and tissue damage induced by reactive oxygen species (ROS) (Halliwell & Gutteridge, 1989). Despite the existence of antioxidant mechanism systems (Halliwell & Gutteridge, 1989; Radak, Chung, & Goto, 2008) some ROS escape to its action and induce damage in several macromolecules such as in DNA, proteins, and phospholipids, which can still be repaired or accumulate and cause attrition and decline in cell function (Cooke, Evans, Dizdaroglu, & Lunec, 2003; Halliwell & Gutteridge, 1989; Li, Jiang, Geng, Cao, & Zhong, 2008). When the imbalance between ROS production and the scavenging action of antioxidants is in favor of the former one, a state of oxidative stress exists (Fisher-Wellman & Bloomer, 2009; Lombard et al., 2005).

Considering the technical difficulty in directly measuring ROS production, acute plasma variations in biochemical parameters have been widely used to quantify the magnitude of oxidative stress damage induced by exercise. (Fisher-Wellman & Bloomer, 2009; Kawamura & Muraoka, 2018; Leeuwenburgh & Heinecke, 2001). For example, products of thiobarbituric acid reactions have been used as a marker of phospholipid damage (lipid peroxidation) induced by ROS in various conditions, including exercise (Fisher-Wellman & Bloomer, 2009; Tofas et al., 2019). It has been stated that due to the increased oxygen consumption during acute exercise, a rise in oxidants generation in different tissues occurs, leading to an upper state of oxidative stress (Kruk, Aboul-Enein, Kladna, & Bowser, 2019; Munoz Marin et al., 2010; Nikolaidis et al., 2007; Tofas et al., 2019). Almost 90% of the total oxygen is consumed by mitochondria and about 1% to 5% leads to ROS generation (Halliwell & Gutteridge, 1989). The metabolic challenge during physical exercise results in an elevated oxygen consumption that depends on the exercise intensity.

Therefore, it is expected that the higher the intensity of the exercise, the higher the ROS production. Several studies have investigated this hypothesis. (Ammar et al., 2020; Goto et al., 2003; Munoz Marin et al., 2010), although the relationship between intensity, duration, and volume with oxidative stress damage has not yet been well established. Indeed, the results from several studies are contradictory, which might be explained by considering the different exercise protocols used (Lu, Wiltshire, Baker, & Wang, 2021; Munoz Marin et al., 2010). However, different authors have mentioned that the greater the intensity and duration of exercise, the more intense the oxidative stress would be, which is following the postulated relation with oxygen consumption.

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Despite this, the effect of other variables related to different intensity modes, independent of oxygen consumption, is not yet clear. For example, type II muscle fibers are more activated in higher exercise intensities than in lower intensities, and it is also known that type II muscle fibers have a lower antioxidant capacity compared to type I fibers (Alves et al., 2020). Thus, it might be expectable that high intensity exercise increases the susceptibility to oxidative damage, despite the total volume of oxygen consumed. Considering all of this, this study aimed to evaluate the effect of exercise intensity on plasma lipid peroxidation concentration after two protocols with different duration but with the same total oxygen consumption.

Methods

Subjects

A group of 12 healthy male subjects, aged between 18 and 26 years (average = 21 yrs ± 2.6), with normal body mass (average = 64.7 Kg ± 7.8) and height (average = 173.2 cm ± 4.8), who were nonsmokers and not taking any medication, volunteered for the study. They were informed about the study and test procedures, as well as the possible risks and discomforts, and gave their written consent to participate.

Exercise protocol

Maximal test: To evaluate maximal oxygen consumption (VO2max), each subject performed a maximal test following a 5-minute warm-up by cycling (MEDIFIT 500U) at 50 watts. The workload during the test increased from an initial 50 watts to an additional 50 watts every minute until the subjects were unable to continue exercising.

The subjects were instructed to maintain the pedaling rate as close to 60 rpm as possible. When the pedaling rate fell to 55 rpm, subjects were verbally encouraged to pedal faster. If the subject could not return to the required rpm, the test was terminated. During the maximal tests, ventilatory parameters were continuously measured breath-by-breath using a metabolic analyzer SensorMedics 2900C system (USA). Heart rates were recorded continuously from four chest electrodes and monitored over an oscilloscope (Cardiovit, Switzerland). The criteria for achieving VO2max were evaluated based on maximum heart rate concerning age (220-age), VE/VO2 value close to 30 L.min-1, and respiratory exchange ratio (RER) greater than 1.15. All test results followed the criteria. Individual workloads equivalent to 55 to 60% of VO2max and 75 to 80% of VO2max were then calculated using an individual regression equation and VO2max.

Submaximal exercise between 55 to 60% of VO_2max

A week after performing the maximal test, the subjects completed a submaximal exercise of 20 minutes cycling between 55-60% of the VO2max, after a previous warm-up of 5 minutes by cycling (MEDIFIT 500U) at 50 watts. The workload during the test was continuously monitored breath-by-breath using a metabolic analyzer SensorMedics 2900C system (USA) to maintain the VO2 between 55 to 60% of the VO2max.

Submaximal exercise between 75 to 80% of VO_2max

Three days after the previous exercise had been performed, the subjects carried out a submaximal exercise of 15 minutes cycling between 75 to 80% of the VO2max, after a previous warm-up of 5 minutes by cycling (MEDIFIT 500U) at 50 watts. Once again, the workload during the test was continuously monitored breath-by-breath using a metabolic analyzer SensorMedics 2900C system (USA) to maintain the VO2 between 75 to 80% of the VO2max.

Blood sample collection and Malondialdehyde (MDA) assay

Before, after, and one hour after each submaximal exercise protocol, 3 mL of venous blood samples were taken into EDTAK3 vials and kept on ice until plasma separation. After centrifugation, products of the reaction to thiobarbituric acid (TBARs) were measured in ELISA microplates (Multiskan Ascent) according to Wills (1987). After completing the exercise, the subjects were kept at rest for one hour.

Statistical analysis

The data were processed using SPSS software. Means (±SD) were calculated for quantitative variables. After assessing the normality of the distribution and the equality of variances, a comparison between MDA concentrations obtained at different moments of each protocol was carried out using One-Way ANOVA and Bonferroni multiple comparisons a posteriori. The comparison between moments of both submaximal intensity exercise protocols was performed using a paired Student t-test. A Pearson test was used to determine the correlation between relative VO2max and plasma MDA concentration. A p-value less than 0.05 was considered significant.

Results

The average value of VO_2max achieved in the maximal test is shown in Table 1.

Figure 1 shows the blood MDA concentrations obtained at each moment of both submaximal exercise protocols. Compared to the resting condition, there was a significant increase in MDA concentration immediately after each submaximal exercise protocol ($F=8.820$, $p<0.05$ and $F=20.880$, $p<0.05$ for exercise performed at 55-60% and 75-80%, respectively). Blood MDA concentration remained elevated for at least one hour after the exercise finished.

Table 1 – Mean ± standard deviation values regarding the subject's maximal oxygen consumption (VO_2max), absolute (L/min), and relative values (mL/kg/min), achieved during the maximal test.

	VO_2max	
	Absolute (L/min)	Relative (mL/kg/min)
Mean ± SD	3,699 ± 0,431	57,51 ± 6,45

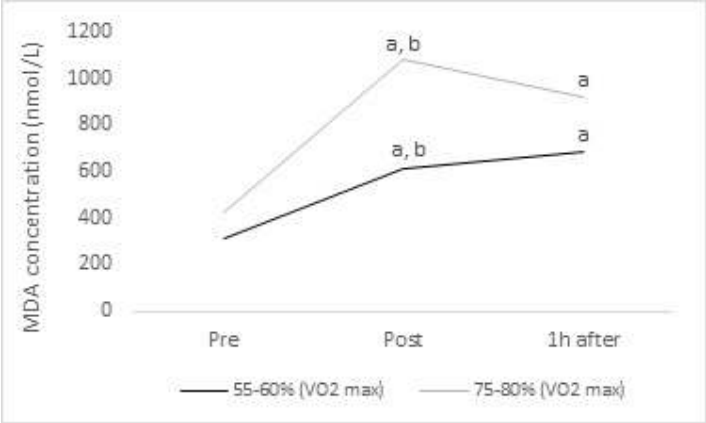

Fig. 1. Level of MDA concentration measured pre, post, and one hour after the exercise protocol (55-60% of VO_2max and 75-80% of VO_2max). Comparing values between different moments at the same exercise protocol, significant statistical differences when compared with rest values, $p<0.05$. [b] significant statistical differences when comparing values between different exercise protocols at the same moment, $p<0.05$.

The comparison between both exercise conditions reveals that the blood MDA concentration was significantly higher immediately after exercising at a superior intensity compared to the low-intensity situation ($t = -7.703$; $p < 0.05$). However, although one hour had passed since the exercise finished, the plasma MDA concentration remained elevated in the exercise performed between 75-85% of VO2max; the differences between both exercise conditions were not statistically significant.

Discussion

The effects of different modes of physical exercise (different durations, intensities, and volumes) on oxidative stress have been the focus of several authors (Ghiasi et al., 2015; Goto et al., 2003; Munoz Marin et al., 2010; Thirupathi et al., 2021), however, this is the first study which goal was to analyze the exercise intensity/duration defined by the total amount of oxygen consumption. MDA is a consequence of oxidative damage of lipids in cell membranes, and the alteration in MDA concentration has been used as an indicator of oxidative cell damage. First of all, this work showed that independently of the exercise intensity, MDA plasma concentration increased after exercise. Considering the different intensities for the same total amount of oxygen consumption, the results showed a significantly higher increase of MDA levels in the 75-80% of VO_2max test compared with the 55-60% of VO_2max test. These results demonstrate that ROS production and the related damage to macromolecules, such as membrane phospholipids, depends on the other factors related to the intensity of the exercise aside from the oxygen consumed during the exercise.

This criterion arose from the recognized importance of oxygen consumption increase with exercise intensity (Guyton & Hall, 2006) as well as mitochondria ROS production increase with oxygen consumption (Agarwal & Sohal, 1994; Jammes, Steinberg, Bregeon, & Delliaux, 2004; Nikolaidis et al., 2007). Our results show evidence that despite the same oxygen consumption, ROS production, and consequently MDA concentration, seem to be augmented with the increase of the exercise intensity. This result suggests that the role of mitochondria in ROS production during exercise is relevant but other factors must also have a meaningful contribution to the exercise-related oxidative stress.

Other researchers (Leeuwenburgh, Hansen, Holloszy, & Heinecke, 1999) suggest that mitochondria ROS production during exercise should not be faced as a direct relation with oxygen consumption, since the mitochondria respiration state depends on the ADP availability. Considering that during exercise the amount of ADP in mitochondria increases significantly due to the higher ATP consumption, it should be expected that mitochondria respiration is mainly in state 3 and the superoxide anion production is reduced (Leeuwenburgh et al., 1999). Britton Chance et. al. (1979) revealed that approximately 2% of the oxygen used by mitochondria is converted into free radicals only when the mitochondria are at the resting state, or state 4. Despite this, when the production of ATP from ADP is activated with a high electron flow into oxygen, the mitochondria is in State 3 and the proportion of oxygen converted into free radicals decreases to a tenth, comparatively to the resting state. Bearing this in mind, the main role of mitochondria usually attributed to the formation of free radicals during exercise should be reconsidered, so alternative sources of reactive oxygen species should be recognized.

Our results showed a significant increase in MDA concentration (nml/L) between the rest state (pre-exercise), post, and 1 hour after exercise, in both test protocols (55-60% of VO_2max and 75-80% of VO_2max). This increase in MDA concentration suggests that physical exercise induces oxidative stress that is following others studies (Ammar et al., 2020; Leaf, Kleinman, Hamilton, & Barstow, 1997; Liu et al., 2000; Nikolaidis et al., 2007). Despite our results showing statistically significant differences between MDA values post-exercise between the two intensities test protocols, after one hour MDA values were not statistically different. The results showed a different trend between tests. A slight decrease in MDA values in the 75-80% of VO_2max and a slight increase in 75-80% of VO_2max were observed. Despite this trend, 1 hour after the exercise test, the greater MDA concentrations in the 75-80% of VO_2max were observed.

Accordingly, the oxidative stress induced by exercise appears to depend on factors beyond total VO2 consumption. It is likely that mitochondrial respiration is not the only variable contributing to the rate of oxidative stress and lipid peroxidation. In fact, there are other factors that may explain oxidative stress induced by physical exercise, namely the specific recruitment of muscle fibers. Exercises with greater intensity preferentially stimulate fast muscle fibers (Guyton & Hall, 2006), which have been shown to have lower antioxidant capacity compared to slow muscle fibers (Alves et al., 2020; Powers, Ji, & Leeuwenburgh, 1999). This results in an inability to scavenge ROS. This might explain, at least partially, the increased oxidative stress observed in the higher exercise intensity bout of our study.

Additionally, muscular cell damage typically increases with exercise intensity (Ertel, Hallam, & Hillman, 2020; Powers et al., 1999). In addition, this damage is associated with increasing blood cytokine concentration, which enhances the immune response, provokes inflammation, and possibly causes extensive oxidative damage (Ammar et al., 2015; Fisher-Wellman & Bloomer, 2009; Pedersen & Hoffman-Goetz, 2000). Therefore, raised ROS production during exercise of greater intensity, due to the increased immunological response, could be another explanation for the higher MDA concentration obtained after the exercise performed at 75-80% compared to the exercise performed at 55-60%.

Thus, this suggests that the increased oxidative stress damage induced by exercise is associated with factors other than total VO2 consumption. Our findings revealed that, regardless of the cause of the greater MDA concentration, exercise intensity induces higher oxidative stress. Further studies are needed to identify the specific mechanisms involved in the increased oxidative damage induced by different exercise intensities.

Conclusion

This is the first study to analyze the effect of exercise intensity on lipid peroxidation while controlling for the total amount of oxygen consumption. Our results showed that acute physical exercise induces lipid peroxidation (plasma MDA levels) in both moderate and high-intensity conditions. Furthermore, our results indicate that lipid peroxidation induced by different exercise intensities depends on factors other than oxygen consumption. Oxidative damage increases with the intensity of the exercise, possibly due to inflammation and the type of skeletal muscle fibers used. Additional studies are needed to explore the mechanisms that contribute to the increase of oxidative damage with high-intensity exercise.

Conflict of interest declaration: None declared

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