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ORIGINAL ARTICLE

Six weeks of aerobic training improves VO_{2max} and MLSS but does not improve the time to fatigue at the MLSS

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Abstract The purpose of this study was to investigate the effects of a 6-week aerobic training period on the time to fatigue (t_{lim}) during exercise performed at the maximal lactate steady state (MLSS). Thirteen untrained male subjects (TG; age 22.5 ± 2.4 years, body mass 72.9 ± 6.7 kg and VO_{2max} 44.9 ± 4.8 mL kg^{-1} min^{-1}) performed a cycle ergometer test until fatigue at the MLSS power output before and after 6 weeks of aerobic training. A group of eight control subjects (CG; age 25.1 ± 2.4 years, body mass 70.1 ± 9.8 kg and VO_{2max} 45.2 ± 4.1 mL kg^{-1} min^{-1}) also performed the two tests but did not train during the 6-week period. There were no differences between the groups with respect to the VO_{2max} or MLSS power output (MLSSw) before the treatment period. The VO_{2max} and the MLSSw of the TG increased by 11.2 ± 7.2 % (pre-treatment = 44.9 ± 4.8 vs. post-treatment = 49.8 ± 4.5 mL kg^{-1} min^{-1}) and 14.7 ± 8.9 % (pre-treatment = 150 ± 27 vs. post-treatment = 171 ± 26 W), respectively, after 6 weeks of training. The results of the CG were unchanged. There were no differences in t_{lim} between the groups or within groups before and after training. Six weeks of aerobic training increases MLSSw and VO_{2max}, but it does not alter the t_{lim} at the MLSS.

Keywords Maximal lactate steady state · Fatigue · Aerobic training · Lactate threshold

Abbreviations

$[La^-]$	Lactate concentration
$\%HR_{max}$	Percentage of maximal heart rate
$\%VO_{2max}$	Percentage of maximal oxygen consumption
$\%W_{peak}$	Percentage of peak workload
CG	Control group
HR	Heart rate
HR_{max}	Maximal heart rate
MLSS	Maximal lactate steady state
W_{peak}	Peak workload
$MLSS_{post}$	Workload at the maximal lactate steady state after training
$MLSS_{pre}$	Workload at the maximal lactate steady state before training
$MLSS_w$	Workload at the maximal lactate steady state
RPE	Rate of perceived exertion
TG	Training group
t_{lim}	Time to fatigue in a constant power output exercise at the maximal lactate steady state
VO_2	Oxygen uptake
VO_{2max}	Maximal oxygen uptake

Introduction

Maximal oxygen uptake (VO_{2max}) has been historically used to evaluate aerobic status and endurance performance

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(Astrand 1976; Costil et al. 1973). However, individuals with similar VO_{2max} values can show different levels of endurance performance (Costil et al. 1973; Hagberg and Coyle 1983) because performance can be influenced by other variables, such as running economy, anaerobic threshold (e.g., blood and gas exchange thresholds), maximal lactate steady-state power output (MLSSw), and velocity at VO_{2max} (Basset and Howley 2000; Midgley et al. 2007).

The $MLSS_w$ is defined as the highest work load that can be sustained for an effort lasting for 30–60 min without continuous blood lactate ($[La^-]$) accumulation, and this value is considered the gold standard for evaluating the anaerobic threshold and predicting aerobic performance (Beneke and Duvillard 1996; Denadai et al. 2004; Faude et al. 2009). The MLSS delineates the margin between low- to high-intensity exercise (Billat et al. 2003) and could be considered the upper boundary of moderate to heavy exercise intensity and the lower boundary of the severe exercise intensity (Billat et al. 2003; Svedahl and MacIntosh 2003; Jones et al. 2010). Even though both $[La^-]$ and oxygen uptake (VO_2) remain stable at MLSS (Baron et al. 2008), heart rate (HR) increases over time without reaching maximum values (Lajoie et al. 2000; Baron et al. 2008; De Barros et al. 2011). On the other hand, exercise intensity above MLSS lead to increases in $[La^-]$ and oxygen uptake (up to VO_{2max}) and also to a significant reduction in exercise tolerance (Billat et al. 2003; Svedahl and MacIntosh 2003).

Interestingly, the MLSS occurs at a similar power output to the critical power (Jones et al. 2010), which represents the highest rate of oxidative metabolism that can be sustained without a progressively and significantly increasing contribution of anaerobic energy turnover. Although competitions are not performed at a fixed intensity, the $MLSS_w$ can be used to predict performance in several sports, such as running, cycling, swimming, and rowing (Beneke 1995; Beneke et al. 2000; Billat 1996; Harnish et al. 2001; Van Schuylenbergh et al. 2004) because the $MLSS_w$ can be associated with the subjects' ability to maintain an elevated percentage of VO_{2max} ($\%VO_{2max}$) or the highest sustainable rate of aerobic ATP resynthesis (Midgley et al. 2007).

Recently, Baron et al. (2008) and Fontana et al. (2009) investigated performance during exercise until fatigue at the $MLSS_w$. Baron et al. (2008) showed that the time to fatigue (t_{lim}) in trained individuals at the $MLSS_w$ was 55.0 min on a cycle ergometer, while Fontana et al. (2009) found a t_{lim} of 37.7 min on a cycle ergometer and 34.4 min on a treadmill in moderately trained individuals. In both studies, exercise interruption occurred in the absence of any key physiological parameters reaching maximum (e.g. oxygen uptake, heart rate) and these interruptions were associated with an integrative central regulation to prevent catastrophic failure and cell damage (St Clair Gibson and Noakes, 2004), and an increase in the rate of perceived exertion (RPE) (Crewe et al. 2008). The difference in t_{lim} reported by Baron et al. (2008) and Fontana et al. (2009) cannot be explained by the difference in exercise intensity because all subjects exercised at the $MLSS_w$, despite the differences in participants training status.

The $MLSS_w$ has been evaluated before and after aerobic training in some studies (Carter et al. 1999; Ferreira et al. 2007; Gobatto et al. 2001; Philp et al. 2008) and under different conditions, such as different environmental temperatures (De Barros et al. 2011), hydration statuses (Moquin and Mazzeo 2000), oxygen partial pressures (Friedmann et al. 2004), and phases of the menstrual cycle (Dean et al. 2003). Studies have also examined individuals of different ages (Mattern et al. 2003) and have evaluated different exercise modes (Beneke et al. 2001).

Carter et al. (1999) and Philp et al. (2008) found an increase in running speed and a similar HR at the MLSS after 6 or 8 weeks of aerobic training (continuous and/or intermittent exercises near at $MLSS_w$). Despite this similarity, Carter et al. (1999) observed no changes in the $[La^-]$ at the MLSS, whereas Philp et al. (2008) found an increase in the $[La^-]$. Nevertheless, none of these authors studied the effects of aerobic training on the t_{lim} for exercise at the $MLSS_w$.

The t_{lim} has been reported to increase after training associated with a $[La^-]$ reduction through exercise at the same $\%VO_{2max}$ (Burgomaster et al. 2005; Markov et al. 2001; Messonnier et al. 2006; Spengler et al. 1999). The post-training decrease in $[La^-]$ during exercise at the same $\%VO_{2max}$ depends on the ratio of $\%VO_{2max}$ to MLSS (e.g., 70 % VO_{2max} can represent 100 % of the MLSS before and 90 % of the MLSS after training) (Gass et al. 1991). Coyle et al. (1988) found a relationship between the time to fatigue and the anaerobic threshold. In this study, although the subjects exercised at the same $\%VO_{2max}$, the authors found differences in t_{lim} for groups with high and low lactate threshold intensities. If after training there is a reduction in the ratio of $\%VO_{2max}$ to MLSS, different metabolic responses (Gass et al. 1991) and exercise tolerance (Coyle et al. 1988) can be expected. Based on these data, we hypothesize that when the exercise intensity is set at the $MLSS_w$, $[La^-]$ and the t_{lim} should be similar, irrespective of training status. Therefore, the purpose of this study was to investigate the effect of 6 weeks of aerobic training on the t_{lim} at the MLSS.

Methods

Subjects

This study was approved by the local Ethics Committee (EC# 153/08). A total of 21 untrained healthy male subjects participated in the study. The participants were randomly divided into two groups: the training group (TG, $n = 13$) and the control group (CG, $n = 8$). The mean (±SD) age, body mass, and VO_{2max} were 22.5 ± 2.4 years, 72.9 ± 6.7 kg, and 44.9 ± 4.8 mL kg^{-1} min^{-1}, respectively, for the TG and 25.1 ± 2.4 years, 70.1 ± 9.8 kg, and 45.2 ± 4.1 mL kg^{-1} min^{-1}, respectively, for the CG.

Procedures

All volunteers underwent a series of tests and evaluations and performed a test until fatigue at the MLSS twice, once before and once after the 6-week training period (either TG or CG) (9.9 ± 0.9 visits to the laboratory). All tests and the aerobic training were performed on a mechanically braked cycle ergometer (Monark Ergomedic E-824E) calibrated before each test, according to the manufacturer's specifications.

The participants were asked to drink 500 mL of water 2 h prior to the beginning of the exercise sessions (Convertino et al. 1996). They were asked not to drink any alcohol or caffeine-containing beverages and not to perform any strenuous physical activity for at least 24 h prior to the experiments. They were also asked to maintain the same diet for the last meal of the evening and for the morning on the days of the experiments. There was a minimum of 48 h of rest between all exercise sessions and 72 h before the test until fatigue. The experiments were always performed at the same time of the day (±1 h) to avoid circadian effects and in a climate-controlled room (21.8 ± 0.5 °C and RH 64 ± 5 %). The water intake was ad libitum and all subjects started the tests euhydrated (urine specific gravity <1,030 mg L^{-1}).

Measurements of body composition, VO_{2max}, and MLSS ($MLSS_{pre}$) were conducted prior to the start of the treatments. The two groups performed constant exercise until fatigue at the $MLSS_{pre}$ power output. The TG subjects then performed 6 weeks of aerobic training, while the CG subjects were instructed not to change their typical daily activities. At the end of the treatment period (training or control), all evaluations performed previously were repeated, and the participants then exercised until fatigue at the post-treatment MLSS ($MLSS_{post}$) (Fig. 1).

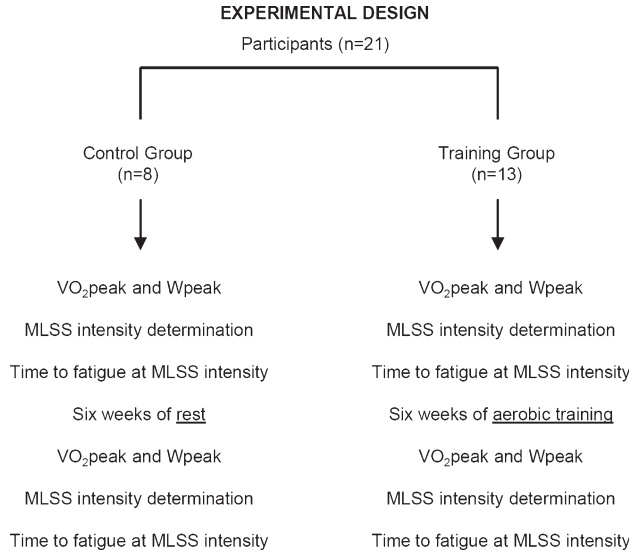

Fig. 1 Experimental design

Evaluations before and after treatment

Participant body mass and height were measured using a digital scale and a stadiometer (Filizola®), and the body composition was estimated by the seven skinfold method according to Jackson and Pollock (1978). The skinfold evaluation was always performed by the same researcher with a skinfold caliper (Lange®).

A progressive exercise-to-fatigue test was performed to determine the VO_{2max} (Balke and Ware 1959) before and after the treatments. The test started at 50 W, and the workload increased at a rate of 25 W 2 min^{-1}. The cadence was maintained at 50 rpm. At the end of each stage, the RPE (Borg 1982) was evaluated. Before each test, the participants rested in a seated position on a chair (5 min) while the equipment used to measure O_2 uptake and the HR was set up (Polar Team System, Finland). The HR was sampled at every 15 s and recorded at every minute and ventilation variables were measured, breath-by-breath, using a gas analyzer ($K4b^2$; Cosmed®, Italy) calibrated before each test according the manufacturer's recommendations using a standard concentration gas mixture and a 3-L calibration syringe.

At least two of the following criteria had to be met to determine the VO_{2max}: (1) no increase in the VO_2 or HR despite increased exercise intensity; (2) an RPE greater than 17 on the Borg scale; (3) a respiratory exchange ratio greater than 1.10; (4) the maximum blood LA concentration of 8 mM, and (5) a heart rate in excess of 90 % of age predicted maximum (220-age) (ACSM 2009). The peak power output (W_{peak}) was estimated as described in detail elsewhere (Kuipers et al. 1985). The highest VO_2 and HR were considered to be the VO_{2max} and the subject's maximal heart rate (HR_{max}), respectively.

The participants performed two to five 30-min exercise sessions at constant pre-set intensities to determine the MLSS at 60 rpm (De Barros et al. 2011) with a minimum of 48 h between each experimental situation. In the initial

evaluation, the first power output corresponded to 60 % of the W_{peak} obtained during the VO_{2max} test. In the final evaluation, we increased the initial workload by 15–20% of the $MLSS_{pre}$ for the TG (Carter et al. 1999; Philp et al. 2008), while the same initial workload was used for the CG. If the [La^-] remained stable or decreased toward the end of the 30 min of exercise during the first trial, the power output for the subsequent trial was increased until a steady [La^-] could no longer be maintained. By contrast, if the [La^-] increased continuously over 30 min or the exercise was interrupted due to the subject's fatigue during the first trial, the power output was reduced for the subsequent trial. The $MLSS_w$ was determined with a precision of 15 W.

During the MLSS tests, blood samples (30 μL) were collected from the earlobe for [La^-] analysis prior to the beginning of the exercise and at 5-min intervals until the end of the test. The highest power output at which the [La^-] increased less than 1 mM during the last 20 min of exercise was defined as the $MLSS_w$ (Beneke 2003; Heck et al. 1985). Blood was stored at −20 °C in tubes containing 60 μL NaF (1 %) and later analyzed in duplicate for lactate concentration using an electro-enzymatic analyzer (YSI 1500 STAT®, Yellow Springs, Ohio, USA). The intraclass correlation coefficient (ICC) found for MLSS determination was 0.85. The HR was recorded every minute, the RPE (Borg scale) every 5 min, and ventilation variables by breath-by-breath. The means of the values recorded from the 10th- to the 30th-min of exercise were considered the [La^-], HR, and VO_2 at the MLSS.

Exercise-to-fatigue at the MLSS

All subjects exercised until fatigue at the $MLSS_{pre}$ before the treatment period and at the $MLSS_{post}$ after the treatment period. In all tests, fatigue was considered to be the individual's inability to maintain a pedal frequency of 60 rpm or the subject stopping the exercise. A verbal encouragement was provided during the latter minutes of the exercise. Blood samples were collected before the onset of exercise, at every 10-min period during exercise, and at the onset of fatigue for the [La^-] analysis. The VO_2 and HR were continuously evaluated, and the RPE was measured every 5-min period. Body mass variation and urine gravity were evaluated before and after the tests to assess the hydration state (Armstrong 2000).The participants were not informed of any physiological variables, including the exercise time until the end of the study. The ICC found for t_{lim} at MLSS determination was 0.73.

Treatment trials

The TG participants performed 6 weeks of aerobic training, three times per week, at the $MLSS_{pre}$ power output. The first training session duration was 24 min, and the duration increased to 39 min over the 6-week period (increase of 3 min per week). Each training session took place under the supervision of a researcher. The CG participants were instructed to maintain their normal daily activities and not to undertake any type of physical training during the study period.

Statistical analyses

A two-way mixed ANOVA with repeated measures was used to analyze the results obtained for the two groups before and after treatment (groups vs. pre/post-treatment) and a three-way mixed ANOVA with repeated measures was used to analyze the results overtime obtained for the two groups before and after treatment (groups vs. pre/post-treatment vs. time) followed by the Student–Newman–Keuls post hoc. To analyze RPE results obtained for the two groups before and after treatment the Wilcoxon test for paired and Mann–Whitney test for non-paired data were used, and to compare results overtime, the Friedman test for paired and Kruskal–Wallis test for non-paired data were used. All results were presented as the mean ± SD, except for the results for the RPE, which were presented as the median. The significance level used was $p < 0.05$.

Results

All subjects from the TG completed 6 weeks/18 sessions of aerobic training (total training time of 567 min at the MLSS power output-mean intensity of 79 % HR_{max} and median 12 of RPE). Before the treatment period, there were no differences between groups with respect to body mass, VO_{2max}, W_{peak}, or HR_{max}. After the treatment period, the TG showed significant increases of 11.2 ± 7.2 % in VO_{2max} (range 4.3–23.6 %) and 14.7 ± 8.9 % in W_{peak} (range 7.1–37.5 %) ($p < 0.01$). At the end of the treatment period, the TG had significantly higher VO_{2max} and W_{peak} values ($p < 0.01$) than the CG (Table 1). There was no difference in the percent body fat between the groups before or after the treatment period.

There were no significant differences in exercise power output, [La^-], RPE, or VO_2 at the $MLSS_{pre}$ between the TG and the CG. After the training period, there was an increase in exercise power output at the $MLSS_{post}$ only for the TG ($p < 0.05$). However, there was no alteration in the [La^-], HR, VO_2, or RPE at the $MLSS_{post}$ in either group (Table 2). When the exercise power output at the MLSS was normalized by W_{peak} (%W_{peak}) or by %VO_{2max}, no difference was observed between the groups at either time point (pre- and post-treatment).

Table 1 Body mass, maximal oxygen uptake (VO_{2max}), peak power output (W_{peak}) and maximal heart rate (HR_{max}) pre- and post-treatments for control ($n = 8$) and training group ($n = 13$)

	Group	Pre	Post
Bodymass (kg)	Control group	70.1 ± 9.8	70.7 ± 9.3
	Training group	72.9 ± 6.7	72.3 ± 6.1
Bodyfat (%)	Control group	14.2 ± 6.3	14.0 ± 6.5
	Training group	15.1 ± 6.0	14.0 ± 5.3
VO_{2max} (mL kg^{-1} min^{-1})	Control group	45.2 ± 4.1	43.8 ± 4.9
	Training group	44.9 ± 4.8	49.8 ± 4.5*,#
W_{peak} (W)	Control group	216 ± 23	218 ± 20
	Training group	219 ± 31	252 ± 28*,#
HR_{max} (bpm)	Control group	185 ± 12	181 ± 14
	Training group	187 ± 8	183 ± 8

Results are shown by mean ± SD

* $p < 0.01$ when compared with pre for the same group

# $p < 0.01$ when compared with post-treatment

Table 2 Power output ($MLSS_w$), lactatemia, heart rate (HR), rate of perceived exertion (RPE) and percentage of maximal oxygen uptake ($\%VO_{2max}$), heart rate ($\%HR_{max}$) and power output ($\%W_{peak}$) at maximal lactate steady-state pre- ($MLSS_{pre}$) and post- ($MLSS_{post}$) treatments

	Group	$MLSS_{pre}$		$MLSS_{post}$	
$MLSS_w$ (W)	Control group	139 ± 22	(105–165)	137 ± 19	(105–165)
	Training group	150 ± 27	(105–201)	171 ± 26*,#	(135–225)
$\%W_{peak}$ (%)	Control group	64 ± 7	(53–73)	63 ± 6	(53–71)
	Training group	69 ± 10	(53–80)	68 ± 6	(57–78)
Lactatemia (mM)	Control group	5.4 ± 1.4	(3.8–7.8)	6.4 ± 1.8	(3.6–9.3)
	Training group	6.4 ± 1.7	(3.9–9.4)	6.4 ± 1.4	(3.7–8.4)
HR (bpm)	Control group	155 ± 14	(124–170)	162 ± 14	(139–177)
	Training group	158 ± 14	(136–184)	157 ± 12	(141–180)
$\%HR_{max}$ (%)	Control group	83.0 ± 6.3	(70–90)	86.5 ± 3.0	(82–90)
	Training group	84.7 ± 7.5	(74–96)	83.8 ± 7.1	(74–98)
$\%VO_{2max}$ (%)	Control group	76.4 ± 13.2	(54.0–89.7)	75.1 ± 5.1	(69.8–83.0)
	Training group	73.2 ± 9.6	(61.9–88.7)	71.9 ± 4.2	(78.0–83.0)
RPE	Control group	15	(11–18)	16	(12–19)
	Training group	15	(9–20)	14	(10–17)

Results are shown by mean ± SD and (range)

* $p < 0.01$ when compared with pre for the same group

# $p < 0.05$ when compared with post-treatment

No significant difference was observed between the t_{lim} values at the MLSS before and after training for either group (Fig. 2).

There was an increase in the [La^-] at the onset of exercise, followed by a steady state and a reduction at fatigue in all experimental settings (Fig. 3a). The VO_2 increased at the beginning of exercise and remained stable during the test in the CG, whereas it increased in the TG at fatigue (Fig. 3b). At the $t_{lim}VO_2$ reached 82.9 ± 10.7 and 80.3 ± 5.1 % VO_{2max} for TG (pre and post) and 80.4 ± 13.6 and 75.0 ± 9.0 % VO_{2max} for CG. The HR (Fig. 3c) and RPE (Fig. 3d) increased continually during exercise. At the t_{lim} RPE reached a maximum values in all experimental situations.

Discussion

The main finding of the present study was that the t_{lim} did not increase and [La^-] did not decrease after aerobic training at the MLSS, although the absolute exercise power output was higher in the post-training. These results are in

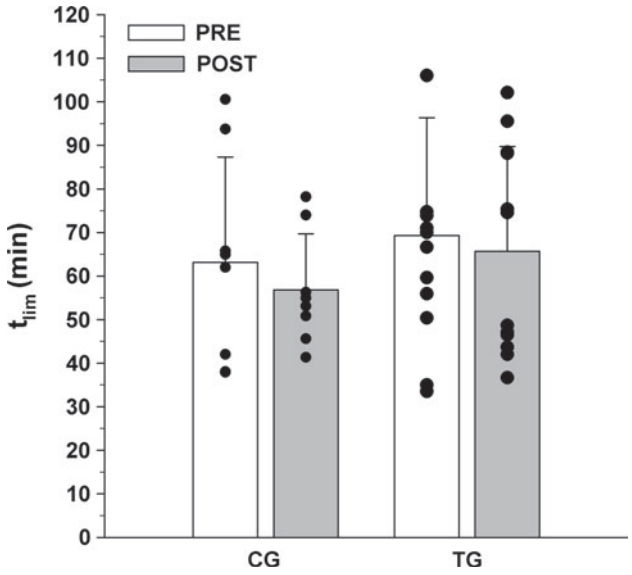

Fig. 2 Time to fatigue (t_{lim}) at maximal lactate steady state before ($MLSS_{pre}$) and after ($MLSS_{post}$) the treatment

agreement with our initial hypothesis. This study is the first in which the t_{lim} at the MLSS was evaluated before and after aerobic training. In previous studies, the t_{lim} at the same $\%VO_{2max}$ was increased after training, and the t_{lim} was associated with a reduction in [La^-]. These results suggest that the exercise power output set at the MLSS was the main factor for the t_{lim} maintenance. In the present study, the $MLSS_w$ and the VO_{2max} increased after the training period in the TG. Therefore, we believe that the use of the MLSS as exercise power output was effective in maintaining the same exercise tolerance, irrespective of training status. Using well-trained individual subjects, Baron et al. (2008) observed a t_{lim} at the MLSS (55.0 ± 8.5 min). However, shorter t_{lim} were found by Fontana et al. (2009), who reported that moderately trained individuals exercised for 37.7 ± 8.9 min on a cycle ergometer and for 34.4 ± 5.4 min on a treadmill at the MLSS. Despite that different ergometers were used, Fontana et al. (2009) achieved similar metabolic, cardiovascular responses and t_{lim}.

Previous studies suggest an increase in t_{lim} and a reduction in [La^-] for the same $\%VO_{2max}$ after training (Burgomaster et al. 2005; Markov et al. 2001; Messonnier et al. 2006). This result suggests that when the effort is relative to $\%VO_{2max}$, different individuals may exercise at intensities above, below, or at the anaerobic threshold, which will most likely lead to different metabolic and cardiovascular responses (Gass et al. 1991) and can alter the exercise tolerance (Coyle et al. 1988). In the present study, the exercise power output set at the $MLSS_w$ caused a similar physiological stress that results in no differences in the t_{lim} before and after training.

Exercise tolerance is limited by several factors, such as energy reserves, cardiovascular demands, acid–base profiles, hydration status, and environmental and body temperatures. In the present study, no volunteer reached the maximal HR or VO_2, and most likely the volunteers did not stop the exercise because of energy depletion (Sahlin and Seger 1995) or a high body temperature (Baron et al. 2008). Baron et al. (2008) investigated the main factor associated with fatigue at the MLSS and could not find any physiological response (HR, respiratory variables, [La^-], pyruvate concentration or pH) that explains the interruption of exercise.

These results reinforce the hypothesis that the fatigue occurs because of an integration of the various factors that lead the subjects to reduce the exercise intensity (when allowed by the exercise protocol) or interrupt the exercise (St Clair Gibson and Noakes 2004). Thus, fatigue has been associated with an increase in the RPE (Tucker et al. 2006; Crewe et al. 2008; Baron et al. 2008; Tucker 2009) that results from an increase of in the central motor commands to locomotor and respiratory muscles (Marcora 2009) and the integration of the afferent feedback of various physiological systems involved in the exercise (St Clair Gibson and Noakes 2004; Tucker et al. 2006; Crewe et al. 2008; Tucker 2009).

Training elicited adaptations in the aerobic capacity, which allowed subjects to perform exercise at a greater workload without continuous lactate accumulation. At this new MLSS power output ($MLSS_{post}$), the [La^-], $\%VO_{2max}$, HR, and $\%HR_{max}$ were maintained, indicating that both physical and cardio-respiratory demand and adrenergic activity were similar to those observed under the pre-training conditions in the t_{lim} test; these responses can explain the similar RPE responses and t_{lim}. An increase in VO_2 upon exercise interruption was observed after training only in the TG ($MLSS_{post}$), although the $\%VO_{2max}$ did not differ between the groups, and no volunteer reached the VO_{2max}.

The increase in the $MLSS_w$ found in the present study is in agreement with the results of Carter et al. (1999) and Philp et al. (2008), who also observed a higher $MLSS_w$ after aerobic training periods of 8 and 6 weeks, respectively. Other studies have also observed an increase in the $MLSS_w$ after a training period in rats (Ferreira et al. 2007; Gobatto et al. 2001).

The maintenance of the [La^-] at the MLSS is in agreement with the results of Carter et al. (1999), who did not find any alteration in the [La^-] at the MLSS before and after 6 weeks of aerobic training. Ferreira et al. (2007) and Gobatto et al. (2001) found similar results in rats. However, Philp et al. (2008) observed an increase in the [La^-] at the MLSS after 8 weeks of aerobic training. Their study was the only study to show an alteration in the [La^-] after a

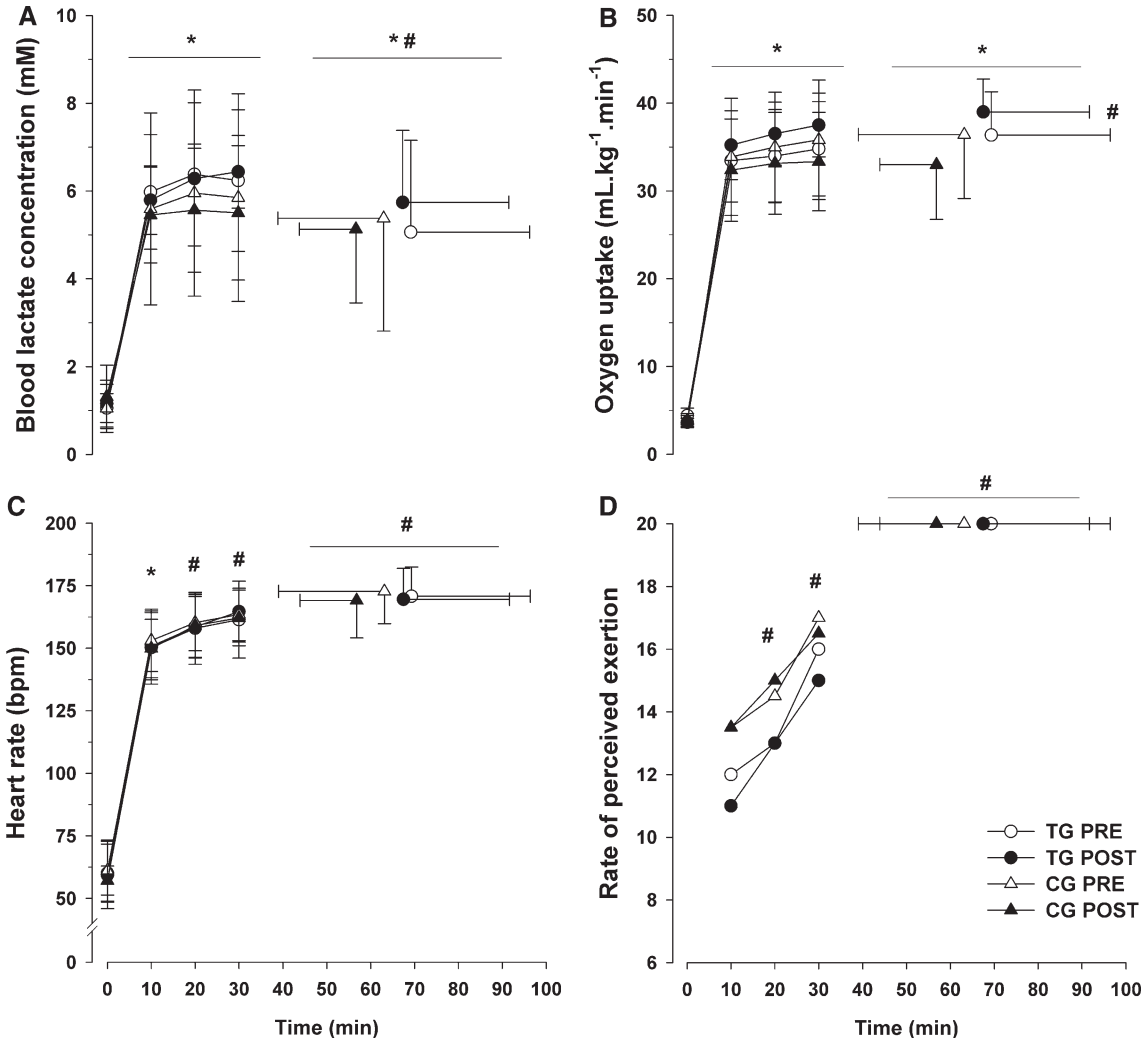

Fig. 3 Blood lactate concentration (**a**), oxygen uptake (**b**), heart rate (**c**) and rate of perceived exertion (**d**) at maximal lactate steady state in control (CG) and training group (TC) pre- and post-treatments. $^{*}p < 0.05$ when compared with rest; $^{\#}p < 0.05$ when compared with previous measure

training period at the MLSS. Previous studies indicated that [La^{-}] at the MLSS changes according to the exercise type and the ergometer and that [La^{-}] at the MLSS is related to the quantity of muscle mass involved in the exercise (Beneke et al. 2001). The [La^{-}] increase at the MLSS found by Philp et al. (2008) may be related to changes in lactate production, the oxidation rate, and body muscle mass that result from training adaptations.

Dubouchaud et al. (2000) found an increase in the expression of monocarboxylate transporters (MCTs) after a period of aerobic training. This increase in MCT expression may facilitate lactate transport out of and into adjacent cells or other places in the body for oxidation (Brooks 2007). Therefore, during exercise, the lactate produced by active muscle cells can be used as an immediate source of energy for adjacent tissues (active muscles or less active tissues), the heart (Brooks 2007), and even for the brain (Gladden 2004). After aerobic training, the maintenance of the [La^{-}] at the MLSS with an increase in the $MLSS_w$ indicates a lower [La^{-}] for the same absolute exercise power output, possibly due to a lower production rate and/or a higher removal rate for this metabolite (Bergman et al. 1999; Messonnier et al. 2006). However, previous studies have shown the maintenance of the [La^{-}] (Carter et al. 1999; Ferreira et al. 2007; Gobatto et al. 2001) or an increase (Philp et al. 2008) in the [La^{-}] at the MLSS, but these results cannot be explained only by an increase in oxidation and/or a lower production of lactate during exercise because the MLSS is the highest steady state between the production and removal of lactate (Heck et al. 1985).

No alteration in the HR at the MLSS was observed after the training period in either group, and these results are in accordance with the findings of Carter et al. (1999) and

Philp et al. (2008). Moreover, there was no change in the HR in other studies that evaluated the response to a training period using different methods to estimate the anaerobic threshold (Hurley et al. 1984; McMillan et al. 2005; Zapico et al. (2007). De Barros et al. (2011) did not find any differences in the HR at the MLSS for exercises performed in hot (40 °C) and temperate (22 °C) environments. HR maintenance at the MLSS after training may indicate a similar demand on both the cardio-respiratory system and adrenergic activity. Mendenhall et al. (1994) and Greiwe et al. (1999) did not find any differences in the concentration of catecholamines for the same relative exercise intensity after an anaerobic training period (10 days and 10 weeks, respectively).These results indicate that the HR may be a good parameter for controlling training intensity because it does not change with increased exercise power output at the MLSS after a period of training. Therefore, after a training period, an individual must increase their absolute exercise power output to maintain a steady HR.

No alterations in the RPE at the MLSS were observed after the training period for either group despite the increase in the $MLSS_w$ for the TG. Because there was no alteration observed in the HR or $\%VO_{2max}$ at the MLSS, one can expect the maintenance of RPE at the same level. We did not find any study in which the RPE at the MLSS was evaluated after a training period. Similar to our study, Hurley et al. (1984) did not find RPE differences at an exercise intensity corresponding to an $[La^-]$ of 2.5 mM before and after a 12-week aerobic training. Furthermore, in the present study, the RPE increased over time in both groups before and after the treatment period, although the exercise power output was held constant. Additionally, the increase in RPE was observed during 30- (De Barros et al. 2011) and 60-min exercises (Lajoie et al. 2000), and exercises until fatigue (Baron et al. 2008; Fontana et al. 2009) at the MLSS.

Conclusion

Based on the results of the present study, 6 weeks of aerobic training at the MLSS increases the $MLSS_w$ and the VO_{2max} although it did not alter the t_{lim} at the MLSS. The maintenance of the t_{lim} before and after training was related to similar HR, $\%VO_{2max}$, $[La^-]$ and RPE responses. The exercise power output set at the MLSS elicited similar metabolic, cardiovascular, and perceptual responses, irrespective of training status.

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Conflict of interest The authors declare that they have no conflicts of interest.

Ethical standard This study was performed in accordance with Brazilian ethical standards and was approved by the Ethics Committee of the Federal University of Minas Gerais (EC# 153/08).

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