©Journal of Sports Science and Medicine (2014) **13,** 73-77
http://www.jssm.org

Research article

Lactate Kinetics during Multiple Set Resistance Exercise

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Abstract
Intensive exercise like strength training increases blood lactate concentration [La]. [La] is commonly used to define the metabolic stress of an exercise and depends on the lactate production, transportation, metabolism, and elimination. This investigation compared multiple set training of different volumes to show the influence of exercise volume on [La]. Ten male subjects performed 3 sets of resistance exercises within 4 separate sessions: Arm Curl with 1 or 2 arms (AC1 or AC2), and Leg Extension with 1 or 2 legs (LE1 or LE2). Each set was performed at a standard velocity and at a previously determined 10RM load. Blood lactate samples were taken immediately before and after each set (pre1, post1, pre2, post2, pre3, post3). Maximum [La] was significantly higher after LE2 (6.8 ± 1.6mmol·L^{-1}) and significantly lower after AC1 (2.8 ± 0.7mmol·L^{-1}) in comparison with the other exercise protocols. There was no difference between AC2 (4.3 ± 1.1mmol·L^{-1}) and LE1 (4.4 ± 1.1mmol·L^{-1}). Surprisingly, [La] decreased during the 3rd set (for AC exercise), and during both the 2nd and 3rd sets (for LE exercise) and increased only during the recovery phases. In contrast to our expectations, blood [La] decreased during the 2nd and 3rd exercise sets and further increased only during recovery phases. However, from the increases observed following the first set, we know that lactate was produced and transported to the blood during our exercise protocol. We speculate that lactate is taken up and metabolized by distal muscle fibres or organs. In addition, as the decreases occurred within a short period of time, blood volume shifts and/or the muscle-to-blood gradient may account for the rapid decreases in [La].

Key words: Muscle-to-blood lactate gradient, metabolism, strength training.

Introduction

Strength exercises are usually characterized by high energy demand and restricted blood flow during time under tension (TUT). Thus, due to the hypoxic environment experienced by the exercising muscle, anaerobic energy metabolism plays an important role during resistance exercise. As a result, lactate production is increased in the working muscles, especially in type II muscle fibers, and consequently blood lactate concentration [La] increases. [La] depends on production, transportation, metabolism, and elimination of lactate and is commonly used to estimate lactate production/elimination in muscle (Beneke et al., 2011; Dotan, 2012). Although measurements of [La] were performed in investigations of hormonal responses to strength training (Kraemer and Ratamess, 2005; Lin et al., 2001; Vingren et al., 2008), neither transportation nor metabolism of lactate have been specifically investigated. However, [La] has been documented to assess metabolic demands resultant from different exercise protocols (Skidmore et al., 2012), i.e. various additional loads (Buitrago et al., 2012a; Kang et al., 2005; Thornton and Potteiger, 2002), movement velocities (Buitrago et al., 2012b; Gentil et al., 2006; Hunter et al., 2003), exercise volumes (1-set vs. multiple-sets) (Haddock and Wilkin, 2006), rest intervals (Ahtiainen et al., 2005; Denton and Cronin, 2006; Ratamess et al., 2007), and exercise order (Bellezza et al., 2009). Special conditions in the muscle lead to a specific metabolic situation during resistance exercises as intramuscular pressure exceed blood pressure and, as a consequence, blood flow is interrupted (Longhurst and Stebbins, 1997; Miles et al., 1987; Walloe and Wesche, 1988). As the muscle to blood lactate gradient, and therefore lactate transportation, is influenced by blood flow, it can be expected that different transportation rates occur during and between exercises in the course of an exercise protocol. We hypothesized that [La] would only slightly increase or stagnate during an exercise session and the increase in between the sets would decrease over the course of exercise.

Until now, no investigation has focused on how [La] develops in the course of resistance exercise with respect to different volume or exercise structure. However, such information would give an indication of the duration and level of metabolic stress experienced by the muscles and would provide information regarding the physiological processes of production, transportation, metabolism and elimination of lactate. Therefore, the aim of this study was to measure [La] over the course of multiple set resistance exercise protocols. Furthermore, we aimed to compare the time course of [La] accumulation in different muscle groups. We hypothesized that alterations in [La] would depend on muscle volume, and that [La] would not rise linearly, in the progression of a 3 set resistance exercise session, due to a suppressed ability of muscle to clear lactate during exercise.

Methods

Subjects

Ten male healthy subjects (22.6 ± 2.0 years, height: 1.80 ± 0.05 m, weight: 73.5 ± 9.3 kg) with at least two years of strength training experience participated in the study. Participants were informed about the design and possible risks of the study and gave written informed consent to participate in this study. The investigations were done in accordance with the declaration of Helsinki and the Ethi-

Received: 03 June 2013 / Accepted: 11 September 2013 / First Available (online): 05 December 2013 / Published (online): 20 January 2014

cal Committee of the University.

Design

Prior to the main experiments, subjects were familiarized with the experimental testing procedures and 10 repetition maximum (RM) was determined for Leg Extension (LE) and Arm Curl (AC) exercises (10 RM LE2: 103 ± 12kg; LE1: 51 ± 6kg; AC2: 66 ± 13kg; AC1: 33 ± 7kg). The 10 RM was determined as described by Baechle and Earle (2008). Velocity and range of motion (ROM) in all testing procedures were standardized by Biofeedback (Biofeedback 2.3.1, digimax): 2 seconds for concentric and eccentric phase each and 90° to 170° knee joints for LE exercise and from 170°-90° in elbow joints for the AC exercise. For the protocols where only one leg or one arm was exercised, half of the weight that was determined for both arms or legs was assigned. After determination of the 10 RM, it was tested again, to ensure that exhaustion occurred at the completion of 10 repetitions. Previous studies showed, that this kind of testing is a reliable method to determine additional load for training (Abernethy et al., 1995; Brown and Weir, 2001). Subjects performed four exercise protocols in a randomly chosen order: Leg Extension with one leg (LE1), Leg Extension with both legs (LE2), Arm Curl with one arm (AC1), Arm Curl with both arms (AC2). Each protocol consisted of 3 sets with the same muscle group with a 3 min rest period between each set. Before each protocol a warm up, consisting of 10 repetitions at 30% 10RM was performed. Sets were performed with a standardized velocity and ROM equal to those described in the 10 RM testing procedure. To ensure sufficient time under tension (TUT), the load during the 10 RM sets was adjusted for the next set, if ROM or movement velocity could not be maintained during the last repetitions of a set. On the experimental day the subjects were instructed to have to maintain identical dietary practices prior to each testing situation and all tests were conducted at the same time of the day, with at least 2 days between each testing condition.

Blood lactate samples were always taken before training at rest (R) and immediately before (pre) and after (post) each of the three sets (pre1, post1, pre2, post2, pre3, post3), as well as 2, 4 and 6 min (2´, 4´, and 6´) after the last set. Each sampling point involved the collection of 20 µl of blood from the earlobe and each sample was directly analyzed with EBIO plus (Eppendorf, Wesseling, Germany). Each sample was analyzed in duplicate and the mean was calculated for subsequent statistical analyses.

Statistical analyses

The normal distribution was checked by the Kolmogorov-Smirnov test ($p \leq 0.05$). As we were interested in the differences in [La] from one value to the subsequent value, changes were analyzed by dependent t-test. The level of significance was $p \leq 0.05$ (*) and $p \leq 0.01$ (**). For the comparison of absolute changes in [La] (Δpost-pre) during exercise bouts, between the sets 1, 2 and 3, ANOVA repeated-measures with Tukey post-hoc test was calculated. Therefore the Δpost-pre values of the first, second or third set of all four interventions (LE1; LE2; AC1; AC2) were summed and compared. For the comparison of absolute changes in [La] (Δpost-pre) during exercise bouts of different interventions (LE1; LE2; AC1; AC2), ANOVA repeated-measures with Tukey post-hoc test were calculated. All data are presented as means with their associated standard deviations (mean ± SD).

Results

The temporal profile of [La] response was similar in each of the four sessions (Figure 1A and 1B). However, the change in [La] (Δpost-pre) was altered with exercise progression (Δpost1-pre1: 0.3 ± 0.3 mmol·L^{-1}; Δpost2-pre2: -0.2 ± 0.5 mmol·L^{-1}; Δpost3-pre3: -0.6 ± 0.5 mmol·L^{-1}). The decrease in set 3 was significantly higher at LE2 than in all other interventions (LE: $p = 0.036$; AC2: $p = 0.004$; AC1: $p = 0.002$) (Figure 2). Between the sets (during recovery) the increase of [La] became significantly lower in training progression (Δpost1to pre2 > Δpost2 to pre3; $p = 0.003$). Maximum [La] was significantly higher after LE2 (6.8 ± 1.6 mmol·L^{-1}) and significantly lower after AC1 (2.8 ± 0.7 mmol·L^{-1}) compared with the other interventions, which are not significantly different (AC2: 4.3 ± 1.1 mmol·L^{-1}; LE1: 4.4 ± 1.1 mmol·L^{-1}).

Discussion

As expected, maximum [La] increased with increased muscle volume and exercise progression. Thus LE2 caused the highest [La] and AC1 the lowest [La]. Contrary to our expectations of a slight increase or stagnation of [La] over the whole intervention/during exercise and recovery, a decrease was observed during set 2 and 3. Increases in [La] were only present during set 1 and during rest periods. Furthermore, in the course of multiple sets of the same muscle group, the decline of [La] during exercise became larger and the increase of [La] between the sets became significantly lower as the exercise protocol progressed.

The results suggest a lactate efflux from trained muscles into the blood during the first set and recovery. Our results show that as [La] increases over the course of training, the lactate gradient between trained muscles and blood decreases, resulting in a lower efflux. Furthermore, vascular occlusion during TUT reinforces a lower lactate gradient between muscle and blood, as it prevents blood of a lower lactate level to flow in to the muscle. After finishing contractions, the increased blood flow in the now relaxed muscle leads to a higher gradient and an increased efflux of lactate. Nevertheless, the results show a decrease of [La] during sets 2 and 3. Besides the reduced efflux due to vascular occlusion, transportation to other tissues and elimination of lactate may explain the observed results. Several investigations dealing with recovery from short term exercise, or even during continued, prolonged exercise, find a net lactate uptake from the blood by resting muscles or by other muscles that are exercising at low to moderate intensity (Brooks, 2000; Gladden, 2000; Gladden, 2004; Richter et al., 1988). Different studies, in particular by Brooks and his laboratory, have established the view of shuttle mechanisms being important for a distribution of lactate as an energy

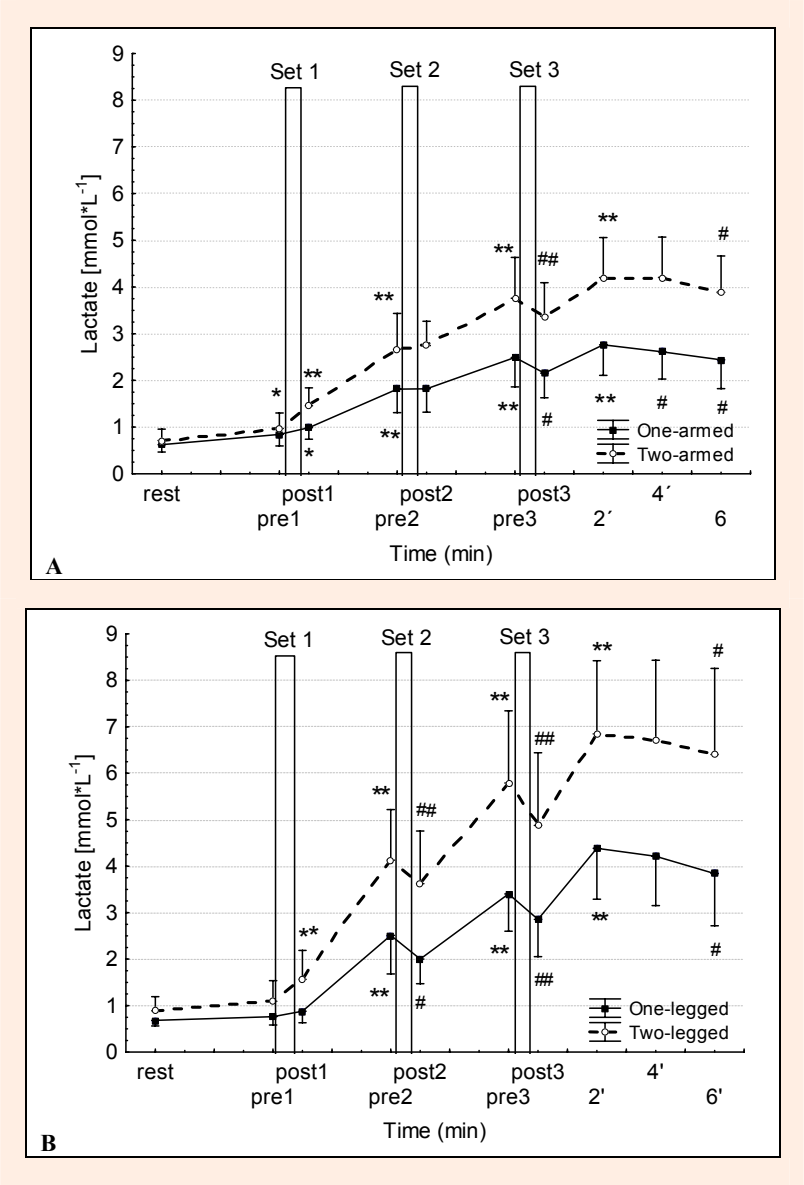

Figure 1A and 1B. Blood lactate concentration (mean ± SD) in exercise progression (set 1, 2 and 3 - white bars) of leg or arm exercise (one legged: LE1; two legged: LE2; one armed: AC1; two armed: AC2). * = higher value than sample point before; (* = $p \leq 0.05$; ** = $p \leq 0.01$); # = lower value than sample point before(# = $p \leq 0.05$; ## = $p \leq 0.01$)

substrate for an aerobic metabolism (Brooks, 1986; 1991; 2000; 2002; Brooks and Hashimoto, 2007). We observed quite large decreases (greater than 1 $mmol \cdot L^{-1} \cdot min^{-1}$) during just one exercise set. Blood volume shifts could explain the quite large [La] decreases in this short period of time. Although up to now no literature is available in this context, previous publications do attribute elevations in hormone concentrations in the blood to plasma volume reductions also (Kraemer and Ratamess 2005). However, previous studies showed that active recovery, such as moderate regeneration activity in between strength bouts does not influence [La] (Mohamad et al., 2012). Active recovery trials at low intensities cleared lactate slower than trials at higher intensities up to 100% of lactate threshold (Menzies et al., 2010). Nevertheless, lactate clearance during active recovery in these studies was still smaller than the observed decrease of 1 $mmol \cdot L^{-1} \cdot min^{-1}$ in in the present study (Spierer et al., 2004). These decreases of 1 $mmol \cdot L^{-1}$ in 1 minute, are unlikely to be explained by elimination of lactate only.

However, lactate metabolism in other tissues like heart, brain and skeletal muscles strongly depends on the availability of lactate (Gladden, 2000). The considerations about metabolic effects of vascular occlusion during TUT, correspond with data of VO_2 during strength training. These show lower values during exercise sets and higher values during rest intervals, although both are elevated during sets and rest intervals over the course of training (Farinatti and Castinheiras Neto, 2011). Thus, higher aerobic capacity was associated with lactate metabolism over the course of training.

Concerning the different pattern of [La] development for arm and leg exercise, a greater decrease of [La] during exercise is observed at higher [La] values in exercise progression at the LE2 exercise mode. In accordance with these findings, Richter et al. (1988) observed that lactate uptake in active legs raises when a higher muscle mass is activated. Another study suggested that lactate

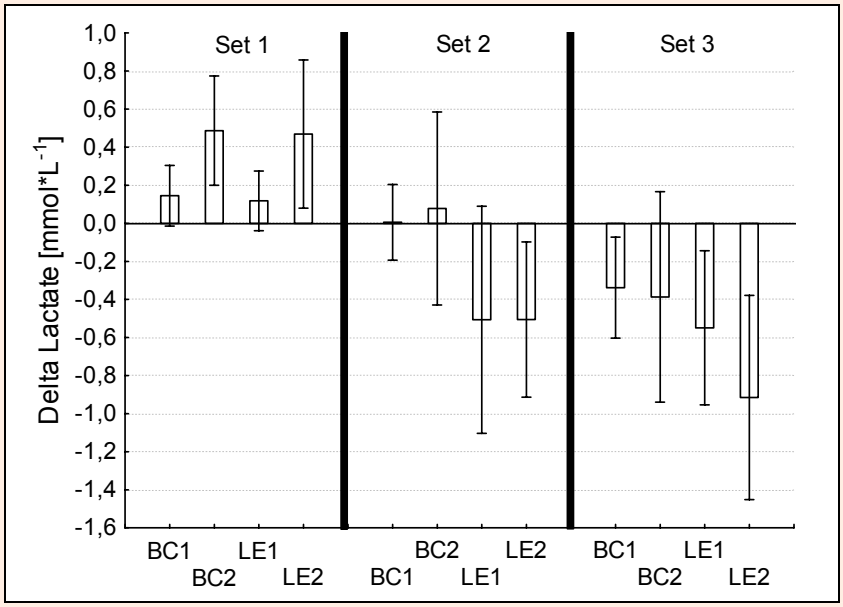

Figure 2. Δ blood lactate concentration (post-pre) within set 1, 2 and 3 for each intervention (one legged: LE1; two legged: LE2; one armed: AC1; two armed: AC2) (mean ± SD).

uptake and subsequent oxidation are also dependent on an elevated metabolic rate (Van Hall et al., 2003), which might increase over time with exercise progression. Further reasons for different rates of lactate disposal can be seen in various muscle fiber distributions. All fiber types switch from net production at low lactate concentrations to net consumption at higher concentrations (Donovan & Pagliassotti, 2000) although this transition occurred at lower lactate concentrations for Type I and IIa fibers, when compared with IIx fibers.

Conclusion

The main finding in this investigation was a marked drop in [La] during intensive strength exercise and an increase only occurring during the recovery phases. The decrease became larger as the exercise protocol progressed. However, due to methodological limitations, we cannot refer to changes in tissue metabolism, which might explain the observed decreases. Future studies should investigate the possible physiological reasons for the decrease in [La] during exercise. A detailed analysis of [La] during multiple set circuit training of the same or different muscle groups could extend the knowledge about lactate distribution and metabolism during strength training. We suggest that the decrease in [La] is independent of the absolute amount of [La], and rather relates to the preloading of the same muscle group.

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Key points

- Blood lactate concentration [La] decreases during the 2nd and 3rd set of a resistance exercise program of the leg extensor muscles.
- [La] decreases during the 3rd set of a resistance exercise program of the arm flexor muscles.
- A significant increase of [La] only appears during the first set, during rest periods and after the last set.
- The decline of [La] during sets becomes larger over the course of exercise.

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