Blood Lactate Clearance After Maximal Exercise Depends On Active Recovery Intensity

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ABSTRACT

Aim. High-intensity exercise is time-limited by onset of fatigue, marked by accumulation of blood lactate. This is accentuated at maximal, all-out exercise that rapidly accumulates high blood lactate. The optimal active recovery intensity for clearing lactate after such maximal, all-out exercise remains unknown. Thus, we studied the intensity-dependence of lactate clearance during active recovery after maximal exercise.

Methods. We constructed a standardized maximal, all-out treadmill exercise protocol that predictably lead to voluntary exhaustion and blood lactate concentration >10mM. Next, subjects ran series of all-out bouts that increased blood lactate concentration to 11.5±0.2mM, followed by recovery exercises ranging 0% (passive)-100% of the lactate threshold.

Results. Repeated measurements showed faster lactate clearance during active versus passive recovery ($p<0.01$), and that active recovery at 60-100% of lactate threshold was more efficient for lactate clearance than lower intensity recovery ($p<0.05$). Active recovery at 80% of lactate threshold had the highest rate of and shortest time constant for lactate clearance ($p<0.05$), whereas the response during the other intensities was graded (100%=60%>40%>passive recovery, $p<0.05$).

Conclusion. Active recovery after maximal all-out exercise clears accumulated blood lactate faster than passive recovery in an intensity-dependent manner, with maximum clearance occurring at active recovery of 80% of lactate threshold.

Key words: Active recovery, Blood lactate, Exercise intensity, Maximal exercise.

INTRODUCTION

Evidence is mounting that the magnitude of improvements in athletic performance and other exercise and health parameters are linked to the intensity of the exercise; high-intensity exercise producing greater effects than lower intensities.[1,2] Such high-intensity exercise also leads to fatigue, which has been targeted by introducing active recovery. Attention has turned to designing exercise training programs that sustain and accumulate the highest possible exercise intensity for as long as possible, facilitated by efficient active recovery that replenishes energy stores and reduces symptoms of fatigue. To this end, active recovery is preferable to passive recovery,[3-7] and the effect depends on the intensity of the active recovery.[8-10] However, this has not yet been studied after maximal, all-out exercise, which is employed during competitive sports or very high-intense exercise training sessions. This will be addressed in the current study.

In exercising humans, measuring the degree and mechanisms of fatigue remains difficult. A repeatable proxy measure is that of blood lactate concentration ([La^-]). Blood lactate accumulates during high-intensity exercise because changes in the redox state of the working muscles accentuate conversion of pyruvate to lactate, of which the bulk oxidation occurs peripheral to the production.[11-12] This results in lactate diffusing with relative ease to the blood.[13] Thus, because the onset and development of fatigue correlates well with accumulation of lactate,[14-18] blood [La^-] levels effectively provide an accessible measure of muscle lactate and muscle fatigue that may be repeated serially during and after exercise.

Recently, it was demonstrated that blood lactate clearance during active recovery depends on the work intensity of the active recovery, with the rate of blood lactate clearance peaking at active recovery of 80-100% of the individual lactate threshold.[10] However, this was assessed under conditions where lactate accumulation and fatigue was limited to a moderate level (blood [La^-] of

4 mM), and hence was likely not substantially impeding exercise. In the present study, we designed controlled protocols where exercise could only be sustained for 30 seconds, and used two repeated 30-second exercise bouts to raise blood [La^-] to ~12 mM, after which we aimed to study the intensity-dependence of lactate clearance during active recovery. The hypothesis was (i) that lactate clearance also under these conditions would be faster during active rather than passive recovery, and (ii) that the effect of active recovery would be exercise intensity-dependent.

MATERIALS AND METHODS

Study Design

We initially established maximal, all-out exercise procedures that reproducibly increased blood [La^-] by ~10mM. To study the effect of passive versus active recovery and the exercise intensity-dependence of active recovery for lactate clearance after high initial lactate loading, we measured blood [La^-] at baseline (resting), immediately after maximal, all-out exercise, and continuously during passive or active recovery sessions until baseline levels were re-established. Active recovery was performed at a range of exercise intensities from low to that corresponding to the individual's lactate threshold; the highest exercise intensity at which no more excess lactate is produced. Each test session including initial tests of maximal oxygen uptake (VO_{2max}) and lactate threshold were separated by at least 48 hours.

Subjects

12 adult, healthy males participated voluntarily in this study. The subjects were physically active on a recreational, but not professional, basis (typically running- and cycling-based activity), with an exercise training/physically active hours/week ranging 5-15 of varying intensity; characteristics are presented in Table 1. Subjects were asked to avoid exhaustive exercise within 48 hours and food and fluids except water within 2 hours of all laboratory visits. The study conformed to the Declaration of Helsinki (2000) and was approved by the Institutional Research Board (IRB), and all subjects signed an informed consent form prior to inclusion. Exclusion criteria were regular smoking, medication, and cardiovascular or metabolic disease or other dysfunction/disease that impaired exercise.

Table 1

Procedures

VO_{2max}: After warm-up consisting of a 10-minute run on a flat treadmill at 8 km/h, VO_{2max} was assessed by increasing the treadmill gradient by 2% every 2 minutes until exhaustion. Oxygen uptake (VO_2) by analyzing continuous 1-minute Douglas bag-collected expired air samples (Servomex 4100 Gas Analyser, Servomex, Sussex, UK) and heart rate (Polar Heart Rate Monitor FS1, Kempele, Finland) were measured throughout the test, and VO_{2max} was considered achieved when at least three of the following criteria were met: (i) VO_2 plateaued despite increased exercise intensity, (ii) respiratory exchange ratio >1.15, (iii) post-exercise [La^-] >8 mM, and (iv) heart rate within 10 bpm of the age-predicted maximum.[19]

Lactate Threshold: After a warm-up as described above, lactate threshold was assessed by increasing the treadmill velocity by 0.5 km/h every 4 minutes while the gradient remained 0%, until the intensity surpassed the lactate threshold and blood [La^-] increased exponentially. The lactate threshold was identified as the deflection point at which [La^-] started to increase after plotting blood [La^-] over intensity, by visual curve inspection and using algorithms developed for this purpose.[20] Blood [La^-] was analyzed from finger prick capillary blood samples (Analox GM7 Lactate Analyser, Analox, Hammersmith, UK) taken at each intensity. 2 samples were averaged at each point, and the lactate analyzer was calibrated by standard solutions before and after each test. Also, VO_2 and heart rate were measured throughout the protocol.

Maximal, All-Out Exercise: Maximal, all-out exercise was used to induce fatigue and raise blood lactate to unsustainable levels. Since this is a non-standard test on a treadmill, a series of trials were undertaken to identify a treadmill protocol in which standardized exercise i) could be sustained at the highest intensity for 2 successive 30-second running bouts with a 1-minute recovery period in between, and ii) yielded a reproducible increase in blood [La^-] of ~10 mM. For

this purpose, treadmill gradient was manipulated 15-20% and velocity 10-16 km/h during randomized trials; each trial separated by at least 48 hours. These trials demonstrated that a standard treadmill protocol of 2 30-second runs at 15 km/h and a 20% gradient with a 1-minute recovery period in between yielded the desired effect (Table 2). As such, this protocol was applied for the subsequent experiments to induce fatigue and increase blood [La^-] >10 mM.

Table 2

Active and Passive Recovery After Maximal Exercise: Next, after a 10-minute warm-up as described above, each subject ran 2 30-second maximal, all-out runs on a 20% graded treadmill at 15 km/h, interspersed by a 1-minute resting period that consisted of recovery by free walking outwith the treadmill. This exercise increased blood [La^-] by >10 mM (see results). To help achieve this, subject were motivated verbally. Immediately after the maximal exercise, the subjects continued with recovery bouts, each randomized to either of 100%, 80%, 60%, 40%, or 0% (complete rest for passive recovery) of the individual lactate threshold, or a self-selected intensity, in which the exercise intensity was controlled by the subjects with no prior instructions; each trial was separated by at least 48 hours. Capillary blood was sampled by finger pricks for analysis of [La^-] before and after the warm-up, at the end of the maximal exercise, every minute for the first 5 minutes of the recovery, and thereafter every 4 minutes from the eight minute onward during the active or passive recovery bouts until [La^-] returned to baseline. 2 samples were averaged at each point, and the lactate analyzer was calibrated as described above. Heart rate was also recorded at the same sample times as [La^-].

Computational Analysis: Lactate recordings were normalized such that lactate clearance during active or passive recovery could be quantified on a relative scale. From each individual trial, an

exponential decay curve was fitted to assess the time constant ($^2/_3$) for clearance of the accumulated [La^-] (see Figure 3a for a typical example illustrating the analysis), and the 1st derivative of the lactate clearance was computed to identify the maximal rate of clearance (see Figure 4a for a typical example illustrating the analysis). The fitted curves were compared to the raw curves by regression analysis.

Statistical Analysis

Data are presented as means±standard errors of the mean (SEM). A repeated measures general linear model with the Scheffe post-hoc test was used to assess differences in the repeated measurements between the trials, whereas a one-way analysis of variance (ANOVA) with a Scheffe post-hoc test was used to assess differences in blood lactate responses between trials. Statistical significance was set at $p<0.05$. Post-analysis power calculations for n (12) showed that the observed differences in lactate clearance between recovery trials rejected the null hypothesis with a probability (power) ranging 0.87-0.97, whereas the intraclass correlation coefficient assessed the reliability of duplicate measurements (0.97, $p<0.05$).

RESULTS

Physical and physiological characteristics of the subjects are shown in Table 1, indicating a moderate level of fitness.

The first set of experiments identified a protocol consisting of 2 consecutive runs at 15 km/h on a 20% graded treadmill to result in blood [La^-] increasing from 0.9±0.1 mM to 12.1±0.7 mM (Table 2) and voluntary exhaustion at the end of the trial; thus, this was used as the protocol for maximal, all-out exercise (see Material and Methods for more detail). In contrast, running at 16 km/h and 20% gradient could only infrequently be sustained for 2x30 seconds.

Thus, the next set of experiments included intensity-dependent active or passive recovery sessions immediately following the maximal, all-out exercise. Heart rates were recorded to monitor the intensity of the recovery sessions (Figure 1). This confirmed that the subjects exercised at the intended recovery intensities. These recordings together with those of running velocity also suggested that the self-regulated active recovery was performed on average at 82±2% of the lactate threshold.

Figure 1

Figure 2 shows the measured blood [La^-] levels (2a) and the normalized lactate clearance curves (2b) during active and passive recovery following the maximal exercise. The 2 30-second exhaustive running bouts increased blood [La^-] from baseline levels at 0.9±0.1 mM to 11.5±0.3 mM (range 10.6-12.3 mM), with the peak occurring ~4 minutes after the end of the maximal run. Blood [La^-] returned to baseline levels within 80 minutes of passive recovery, but considerably faster after active recovery; at the latest after 60 minutes during active recovery at 40% of lactate

threshold. However, active recovery at higher intensities (60-100% of lactate threshold) cleared accumulated blood lactate faster than active recovery at 40% of lactate threshold ($p<0.05$) or passive recovery ($p<0.01$). The repeated measures analysis of the time course of lactate clearance did not identify a difference between active recovery intensities of 60%, 80%, or 100% of lactate threshold.

Figure 2

Based on the individual lactate clearance curves, we fitted exponential decay curves in order to analyze the time constant of lactate clearance (Figure 3). The comparison between the measured and the fitted exponential decay curves of lactate clearance showed R^2 values ranging 0.85 to 0.99 ($p<0.01$) and a coefficient of variation at 5%; therefore indicating close fitting. This analysis showed that the fastest time constant for lactate clearance occurred during recovery at 80% of lactate threshold ($p<0.05$). This was also confirmed by the self-regulated active recovery, performed at an intensity of 82±2% of lactate threshold. Moreover, passive recovery (0% of lactate threshold) presented with the slowest time constant for lactate clearance ($p<0.05$), whereas no differences occurred between active recovery at 100%, 60%, and 40% of lactate threshold.

Figure 3

Finally, we computed the 1st derivative of each individual lactate clearance curve in order to analyze the peak rate of lactate clearance (Figure 4). Active recovery at 80% of lactate threshold resulted in the fastest peak rate of lactate clearance ($p<0.05$), which was also confirmed by the self-regulated active recovery at 82±2% of lactate threshold. Passive recovery had the slowest peak rate of lactate clearance ($p<0.05$), whereas active recovery at 40% of lactate threshold had a slower

peak rate of lactate clearance than active recovery at higher intensities ($p<0.05$). No differences occurred between active recovery at 100% and 60% of lactate threshold.

Figure 4

DISCUSSION

This study shows that active recovery is superior to passive recovery for clearing accumulated blood lactate after short bouts of maximal, all-out exercise that substantially raise blood [La^-] (>10 mM), and that lactate clearance depends on the intensity of the active recovery to the point where it peaks at an active recovery intensity of 80% of lactate threshold. This is in line with previous results indicating intensity-dependence of lactate clearance during active recovery after a bout of exercise that raised blood [La^-] to ~4 mM.[10] Thus, lactate clearance during active recovery is intensity-dependent after both low and high accumulation of lactate. The finding that active recovery is preferable to passive recovery is not new,[5-8,21,22] but the intensity-dependent dose-response relationship of the active recovery is only now becoming apparent. As such, this informs strategies to facilitate recovery after both aerobic sub-$\dot{V}O_{2max}$ and anaerobic supra-$\dot{V}O_{2max}$ exercise bouts. This may therefore improve the outcome of exercise training programs or even exercise performance.[9,23,24]

Importantly, active recovery in this study was set relative to the lactate threshold of the individual, i.e., to the exercise intensity where lactate accumulation starts, whereas previously, active recovery has been set relative to $\dot{V}O_{2max}$, which may have confounded the results because blood lactate accumulation is not fixed to $\dot{V}O_{2max}$. Also, the few recent studies that have related active recovery to the onset of blood lactate accumulation did not investigate the spatiotemporal characteristics of lactate clearance,[3,4,9] or did so only after a modest initial increase in blood lactate.[10]

Although evidence linking lactate to muscle fatigue exists,[14,16] the nature of the relationship remains controversial.[25,26] Whether lactate causes or reflects fatigue, accumulation of lactate contributes to intramuscular acidosis by release of H^+, which reduces muscle performance via inhibition of metabolic pathways and contractility.[11,13,15] Thus, it becomes important to clear the

accumulated lactate; chiefly achieved by oxidation. Active recovery facilitates this by reducing muscle work to a level where no further excess production occurs and where oxidation is maximized by maintenance of both a higher blood flow that redistributes accumulated lactate and a higher metabolic activity in lactate-consuming oxidative type I muscle fibers and the myocardium. Our results indicate which active recovery intensity range best achieves this, but also suggest that even under optimal active recovery conditions, this process requires ~30 minutes. Compared to clearance after a modest loading of blood lactate,[20] this is substantially prolonged. In fact, since increasing blood [La^-] by a factor of 2.5 increases the time required to reestablish resting values by the same order of magnitude, it suggests that blood lactate clearance is not driven by its concentration gradient, but rather by capacity for oxidation, which is driven by metabolic rate.

Interestingly, during active recovery at 100% of lactate threshold, blood [La^-] did not remain constant, but rapidly decreased. This suggests that the theoretical framework of the lactate threshold demarcating between lactate production and elimination may be invalid at high blood [La^-] or may have shifted to a higher intensity than the lactate threshold identified from the point of exponential increase in blood [La^-]. If the lactate threshold had remained static, active recovery at this intensity should have sustained and not reduced blood [La^-].

CONCLUSIONS

Following maximal, all-out exercise that rapidly increases blood [La^-] to >10 mM, active recovery is beneficial for blood lactate clearance. Furthermore, the intensity-dependent dose-response relationship as a function of the individual lactate threshold dictates that active recovery at or close to 80% of lactate threshold yields the fastest rate and shortest time constant for clearance of accumulated lactate. This may inform strategies for rapid recovery and elimination of excess lactate after strenuous and maximal intensity exercise bouts, though it should be pointed out that our hypothesis was tested in a homogenous group of active and healthy young men, whereas different groups of subjects or fitness levels may present with different intensity-dependent active recovery kinetics.

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Table 1. Physical and physiological characteristics of the subjects.

Table 2. Blood lactate concentration after 2x30 second treadmill running bouts with interspersing 1 minute active recovery.

Figure 1. Heart rates during active and passive recovery sessions. Different from other intensities; **: $p<0.01$, except 80% of lactate threshold (LT) and self-regulated intensities that were different from other intensities, but not each other.

Figure 2. a: Blood lactate concentration ($[La^-]$) at baseline (warm-up), after maximal exercise (time 0 min), and during active or passive recovery at exercise intensities ranging 100-0% of the individual lactate threshold (LT) and a self-regulated active recovery exercise intensity (82±2% of LT). **b**: Normalized blood lactate clearance during active and passive recovery. The baseline is marked with a dashed line. Different from other intensities; **: $p<0.01$, *: $p<0.05$.

Figure 3. a: Example graph of a single-subject lactate clearance and its fitted exponential decay curve during active recovery after maximal exercise; the active recovery intensity in this example is 80% of the lactate threshold (LT). Inset is the correlation between the two curves and the $^2/_3$ time constant. **b**: Time constants for $^2/_3$ lactate clearance during each intensity of active or passive recovery. Different from other intensities; *: $p<0.05$ (80% of LT and self-regulated active recovery intensities were not different from each other); different from all other intensities; #: $p<0.05$.

Figure 4. a: Example graph of a single-subject lactate clearance during active recovery after maximal exercise; the active recovery intensity in this example is 80% of the lactate threshold (LT). The 1st derivative of the lactate clearance curve is plotted on the same graph on the Y_2 axis,

illustrating the peak rate of clearance during the recovery. **b**: Peak rates of lactate clearance during each intensity of active or passive recovery. Different from other intensities; *: $p<0.05$ (80% of LT and self-regulated active recovery intensities were not different from each other); different from all other intensities; #: $p<0.05$.