Blood Lactate Clearance During Active Recovery After An Intense Running Bout Depends On The Intensity Of The Active Recovery

Running title: Lactate clearance during active recovery

Paul S Menzies*, Craig Menzies*, Laura McIntyre*, Paul Paterson, John Wilson, Ole J Kemi

Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow, United Kingdom

* The first 3 authors contributed equally and should be regarded as joint first authors.

Corresponding author:

Dr Ole J Kemi, Institute of Biomedical and Life Sciences, University of Glasgow, West Medical Building, Glasgow, G12 8QQ, United Kingdom.

Phone: +44 (0) 1413305962, Fax: +44(0) 1413305481, Email: o.kemi@bio.gla.ac.uk

Key Words: Active Recovery, Blood Lactate, Intensity, Exercise

ABSTRACT

High-intensity exercise training contributes to production and accumulation of blood lactate, which is cleared by active recovery. However, no commonly agreed intensity or mode for clearing accumulated blood lactate has yet evolved. We studied clearance of accumulated blood lactate during recovery at various exercise intensities at or below the lactate threshold (LT) after high-intensity interval runs that prompted lactate accumulation. Ten males repeated 5-minute running bouts at 90% of maximal oxygen uptake (VO_{2max}), which increased blood lactate from 1.0±0.1 mM to 3.9±0.3 mM. This was followed by recovery exercises ranging 0-100% of LT. Repeated blood lactate measurements showed faster clearance of lactate during active versus passive recovery, and that the decrease in lactate was more rapid during higher (60-100% of LT) versus lower (0-40% of LT) ($p<0.05$) intensities. The more detailed curve and rate analyses showed that active recovery at 80-100% of LT had the shortest time constants for 67% lactate clearance and highest peak clearance rates compared to 40% of LT or passive recovery ($p<0.05$). Finally, examination of self-regulated intensities showed enhanced lactate clearance during higher versus lower intensities, further validating the intensity-dependence of clearance of accumulated blood lactate. Therefore, active recovery after strenuous exercise clears accumulated blood lactate faster than passive recovery in an intensity-dependent manner. Maximum clearance occurred at active recovery close to the LT.

INTRODUCTION

Experimental (Haram et al., 2009; Kemi et al., 2005), clinical (Helgerud et al., 2007; Tjonna et al., 2008), and epidemiological (Lee, Sesso, Oguma & Paffenbarger, 2003; Moholdt, Wisloff, Nilsen & Slordahl, 2008) trials in both health and disease show that the beneficial effects of exercise training depend on the intensity at which the exercise training is performed, with high intensity superior to moderate-to-low intensities. Since the high-intensity exercise is performed above the lactate threshold (LT), i.e. the intensity at which lactate starts to accumulate in the skeletal muscle, the exercise is normally carried out in repeated bouts that are interspersed with recovery periods, as in an interval training regime. The reason for the lactate accumulation is that more of the pyruvate is converted to lactate by lactate dehydrogenase (LDH), primarily as a result of changes in the intramuscular redox state, and because oxidation of the excess lactate relies on redistribution by the blood flow to other muscles and the heart and liver (Gladden, 2004; Wasserman, Beaver & Whipp, 1986). Thus, muscle lactate is mirrored by blood lactate.

Since most of the lactate is oxidised by skeletal muscles working at a lower intensity, and since the lactate redistribution occurs via the blood flow (Gladden, 2004), active rather than passive recovery after lactate-accumulating exercise appears to be more effective at clearing accumulated lactate (Belcastro & Bonen, 1975; Boileau, Misner, Dykstra & Spitzer, 1983; Bonen, Campbell, Kirby & Belcastro, 1979; Hermansen & Stensvold, 1972). However, no commonly agreed strategies or optimal intensity of active recovery for clearing accumulated lactate have yet been identified. Previous studies have suggested active recovery intensities in the range 25-63% of the maximal oxygen uptake (VO_{2max}), (Boileau et al., 1983; Bonen & Belcastro, 1976; Dodd, Powers, Callender & Brooks, 1984; Hermansen & Stensvold, 1972) but these studies quantified the intensity of the active recovery to maximal aerobic capacity (VO_{2max}), where lactate production has a non-linear relationship to workload. Only recently have investigators related active recovery

intensities to LT (Baldari, Videira, Madeira, Sergio & Guidetti, 2004, 2005; Greenwood, Moses, Bernardino, Gaesser & Weltman, 2008), which may more directly link it to the workload at which production exceeds removal. However, these studies have not studied the intensity-dependence of active recovery in detail, or the temporal characteristics of lactate clearance. It also remains unclear whether active recovery should be enforced by a set exercise intensity, or whether voluntary control by the individual subject may be optimal (Bonen & Belcastro, 1976).

Here, after an interval run used in high-intensity interval training regimes, we determined the intensity at which blood lactate started to accumulate exponentially (LT), relating recovery intensities to this marker, and then studied the patterns of blood lactate clearance during passive and active recovery over a range of exercise intensities. We also studied the effect of self-regulated active recovery periods, by allowing the participant to control the active recovery exercise intensity.

METHODS

The study was approved by the Institutional Review Board, and all participants signed a consent form prior to inclusion in the study. Exclusion criteria were regular smoking, medication, and cardiovascular or metabolic disease or other dysfunction/disease that would impair exercise.

Participants

Ten moderately trained adult, healthy males volunteered for this study; their characteristics are presented in Table 1. Participants were asked to refrain from exhaustive exercise within 48 hours and avoid food and fluids except water within two hours of all laboratory visits.

Lactate threshold and maximal oxygen uptake

After a 10-minute warm-up by treadmill running (0% grade), below the LT at 8 km/h, the LT was assessed by an incremental ramp test protocol. While the treadmill remained at a 0% grade, velocity was increased by 0.5 km/h every 4 minutes until the intensity surpassed the LT, and blood lactate concentration [La^-] increased exponentially. LT was identified by the deflection point at which [La^-] started to increase, as observed by plotting [La^-] over intensity with algorithms developed for this purpose (Newell et al., 2007) and by visual curve inspection. Blood [La^-] was measured by analysing capillary blood samples taken from finger pricks after each increment (Analox GM7 Lactate Analyser, Analox, Hammersmith, UK). At each point, two samples were averaged. The lactate analyser was calibrated by standard solutions before and after each test. Oxygen uptake ($\dot{V}O_2$) (Servomex 4100 Gas Analyser, Servomex, Sussex, UK) and heart rate (Polar Heart Rate Monitor FS1, Kempele, Finland) were measured simultaneously throughout the protocol. On a subsequent day, $\dot{V}O_{2max}$ was assessed by an exhaustive treadmill running test protocol, in which intensity was increased every two minutes until voluntary exhaustion. $\dot{V}O_2$ and heart rate were measured throughout the test, and $\dot{V}O_{2max}$ was defined when at least 3 of the

following criteria were observed: (1) VO_2 plateaued despite increased intensity, (2) respiratory exchange ratio >1.15, (3) post-exercise [La^-] >8 mM, and (4) heart rate within 10 bpm from the age-predicted maximum, according to published guidelines (Duncan, Howley & Johnson, 1997).

Active and passive recovery trials

Each recovery trial started with a 10-minute warm-up followed by a 5-minute high-intensity run at 90% of VO_{2max}. Immediately thereafter, they continued with the recovery bouts, either at 100%, 80%, 60%, 40%, or 0% (complete rest for passive recovery) of the LT, by setting the 0% grade treadmill to the velocity that corresponded to the designated intensity in relation to the speed at which LT was defined. In addition, the participants also undertook a self-regulated active recovery session, in which the exercise intensity was controlled by the participant by selection of treadmill velocity, but no guidelines were provided as to exercise intensity, apart from the subjects being informed that they were to exercise at an intensity of their own choice. Each recovery trial was separated by at least 48 hours with the order of the trials being randomised. Each trial continued until [La^-] levels returned to resting levels. Capillary blood by finger pricks were sampled for analysis of [La^-] before and after the warm-up, at the end of the 5-minute high-intensity interval, and thereafter every 4 minutes during the active or passive recovery until return to baseline. Heart rate was also recorded at the same time-points as [La^-].

Computational and statistical analysis

Lactate recordings during the recovery trials were first normalised to a relative scale such that the resting and peak [La^-] were set to a values of 0 and 1, respectively; thus standardising the amplitude, whereupon the 1st derivative of the lactate clearance was computed to identify the maximal rate of clearance during each recovery session. Secondly, an exponential decay curve was

fitted for each individual trial to assess the time constant for 67% ($^2/_3$) clearance of the accumulated [La^-]. The fitted curves were compared to the raw curves by regression analysis.

Data are presented as means±standard error 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 active and passive recovery trials, whereas the one-way analysis of variance (ANOVA) with a Scheffe post-hoc test was used to assess the time constants and maximal rates of lactate clearance between the active and passive recovery trials. Statistical significance was reached at $p<0.05$.

RESULTS

Physical and physiological characteristics, including VO_{2max} and LT, are shown in Table 1. These measurements indicate a moderate level of fitness.

Table 1 near here.

To monitor the intensity of the recovery sessions, heart rates were recorded continuously. These recordings confirm that the participants exercised at the intended recovery intensity (Figure 1). For instance, the recorded heart rates during the 100% of LT active recovery trial are in close agreement with the heart rates at LT, as determined from the LT test (Table 1). A closer inspection of the recordings revealed that the individual variation was linked between the 100% LT recovery trial and the LT test. Thus, those that deviated from the mean value did so on both records. The self-regulated active recovery trials were on average performed at 79±5% of LT (range 55-102% of LT). This was confirmed by comparing the running velocities and heart rates to those observed during active recovery at set intensities (Figure 1).

Figure 1 near here.

The 5-minute run at 90% of VO_{2max} resulted in blood [La^-] rising from baseline levels at 1.0±0.1 mM to 3.9±0.3 mM (range 2.1-6.7 mM) measured immediately after the run (Figure 2A). Blood [La^-] returned to baseline levels within 32 minutes of active or passive recovery, but faster after active recovery compared to passive. Furthermore, exercise intensities approaching LT cleared accumulated blood lactate faster than low exercise intensities, as active recovery intensities at 100-60% of LT were more effective at clearing accumulated blood lactate than active recovery at 40% of LT ($p<0.05$) or passive recovery at 0% of LT ($p<0.01$). Active recovery at 40% was not

different from passive recovery (p>0.05). The measured blood [La^-] levels are shown in Figure 2A, and Figure 2B shows the normalised lactate.

Figure 2 near here.

Next, we computed the 1st derivative of each individual lactate clearance curve; see example traces in Figure 3A. This allowed us to analyse the peak rate of clearance at each of the active recovery intensities as well as the passive recovery. Active recovery at 80% and 100% of LT was equal, but occurred with a higher peak rate than during recovery intensities of 60% and 40% of LT, or passive recovery at 0% of LT. Moreover, active recovery at 60% and 40% of LT was also associated with a higher peak rate of lactate clearance than passive recovery. The self-regulated intensity, which occurred at 79±4% of LT, confirmed these results (Figure 3B).

Figure 3 near here.

We also fitted the exponential decay on the basis of each lactate clearance curve in order to analyse the time constant for lactate clearance; see example trace in Figure 4A. First, R^2 values ranging from 0.971 to 0.995 (p<0.01), and a coefficient of variation at 4% show a close relationship between the measured lactate clearance curves and the fitted exponential decay curves. This analysis showed that the time constants for 67% ($^2/_3$) lactate clearance were similar between active recovery intensities of 80% and 100% of LT, and that these were smaller (i.e. faster clearance of lactate) than active recovery at 60%, 40%, and passive (0%) recovery intensities. Furthermore, active recovery at 60% of LT cleared lactate faster than active recovery at 40% of LT or passive recovery, whereas there was no difference between 40% of LT and passive recovery. The self-

regulated active recovery intensity again confirmed these results (Figure 4B), and these results parallel those of the rate analysis by the 1st derivatives of the lactate clearance curves (Figure 3B).

Figure 4 near here.

Because the self-regulated active recovery trials contained a wide intensity range; 55-102% of LT, this allowed us to investigate further whether the intensity-dependence of lactate clearance during active recovery was true also under these conditions. As displayed in Figure 5, normalised lactate clearance during the individual self-regulated active recovery trials show a relationship that largely confirms the intensity-dependence of active recovery. The active recovery trials at low intensities cleared lactate slower than trials at higher intensities. It is also noteworthy that the two highest exercise intensities displayed relatively slow lactate clearance patterns, but both were above the LT (102% and 104% of LT), which suggests that the active recovery was performed under conditions in which excess lactate was still being produced by the exercising skeletal muscles, which may slow the clearance of accumulated blood lactate.

Figure 5 near here.

DISCUSSION

This study shows that the decrease in accumulated blood lactate after treadmill running at 90% of VO_{2max} is more effective when followed by active rather than passive recovery, and that active recovery at 80-100% of the individual LT, i.e. at or just below the LT, is more effective than active recovery at lower exercise intensities. Active recovery at 60% of LT was also more effective than 40% of LT. Thus, blood lactate clearance during active recovery displays a dose-response relationship between LT and passive recovery, and the active recovery intensities at or close to LT are preferable for blood lactate clearance. This confirms previous studies showing that active recovery clears blood lactate faster than passive recovery, though the intensity-dependence of the active recovery as a function of LT had not been established (Belcastro & Bonen, 1975; Boileau et al., 1979; Bonen & Belcastro, 1976; Bonen et al., 1979; Dodd et al., 1984; Gupta, Goswami, Sadkukhan & Mathur, 1996; Hermansen & Stensvold, 1972; Mondero & Donne, 2000). These studies have shown that active recovery for clearing lactate may be most effective in the range 25-63% of VO_{2max}, where the top end of the range approaches 80-100% of LT. Critically, in contrast to our study, the previous studies have quantified the intensity of the active recovery relative to VO_{2max}. This approach may confound the results because blood lactate accumulates non-linearly at intensities above the LT, and because the LT may vary widely between individuals with respect to its relationship to VO_{2max}. Only a few studies have related the active recovery intensity to the LT (Greenwood et al., 2008) or the ventilatory threshold (Baldari et al., 2004, 2005), which serves as a correlate to LT. However, although these studies also indicate that active recovery depends on the exercise intensity, they did not investigate the temporal or dose-response characteristics of the intensity-dependence of the active recovery.

This study also suggests that the exercise intensity to optimise lactate clearance during active recovery does not need to be fixed, but rather that the subject may in fact be able to control the

intensity such that it clears lactate with equal effectiveness as active recovery fixed at high intensities relative to LT. This was demonstrated by the use of self-regulated intensities, in which the subjects were given no instructions as to exercise intensity. Thus, the exercise intensity was measured, but not controlled by investigators. The reason for the equal effectiveness was that the subjects chose to run at an active recovery intensity close to 80% of the LT, and in fact, those who deviated from this intensity experienced slower lactate clearance. The biological feedback system that led the subjects to choose the most effective lactate recovery intensities remains unknown.

The importance of clearance of accumulated blood lactate is under continuing debate, but it has been recognized that elevated levels of skeletal muscle and blood lactate are associated with impaired muscle function and exercise performance (Andrews, Godt, & Nosek, 1996; Hogan, Gladden, Kurdak & Poole, 1995; Minshull, Gleeson, Walters-Edwards, Eston & Rees, 2007; Sahlin & Ren, 1989; Westerblad & Allen, 1992). Although the cause-effect relationship between lactate and fatigue remains unclear (Gladden, 2004), it is clear that accumulation of lactate may at least indirectly contribute to reduced performance, because conversion of lactic acid to lactate releases H^+ that leads to a metabolic acidosis with subsequent inhibition of glycolytic rate-limiting enzymes, lipolysis, and contractility of the skeletal muscles (Brooks, 2002; Gollnick, Bayly & Hodgson, 1986; Gladden, 2000; Mainwood & Renaud, 1985). Whether lactate production causes or reflects fatigue, it may be used as a measurable marker of it because of its correlation with muscle fatigue and performance. Thus, it becomes relevant to design strategies that clears blood lactate after high intensity exercise bouts, as this enables a faster recovery of the subject and may support subsequent high-intensity exercise, leading to greater overload and consequently enhanced training adaptation. This study contributes toward this end.

The mechanisms by which accumulated lactate is cleared include oxidation by working skeletal muscles. This mainly occurs by oxidative type I fibres, whereas the bulk production is confined to the glycolytic type II fibres, as well as myocardial oxidation and gluconeogenesis via the Cori cycle (Gladden, 2000, 2004). These are rate-limited by the monocarboxylate transporter-facilitated lactate shuttle to the blood and by the blood flow itself (Bonen et al., 2000). Thus, though measuring blood lactate only indirectly assesses the intramuscular environment, it opens a window of opportunity that can be repeatedly accessed with a high temporal resolution, which direct intramuscular measurements cannot match.

By definition, the LT occurs at the highest exercise intensity where lactate production and removal are balanced. However, our results suggest that the threshold intensity may increase when [La^-] increases to above resting levels. If it had remained constant, one would have expected the [La^-] to remain stable during the active recovery trials at 100% of LT and the self-regulated trials where the intensity exceeded the LT. This did not happen, despite careful determination of the LT that included re-testing where doubts occurred as to its accuracy. In fact, active recovery at 100% of LT very effectively cleared accumulated blood lactate. It is conceivable that under conditions of accumulated lactate, oxidation of lactate is increased and/or production is reduced.

The high intensity exercise bout at 90% of VO_{2max} inducing lactate accumulation was designed to reflect the high intensity bouts frequently utilized by aerobic interval training programmes (Haram et al., 2009; Helgerud et al., 2007; Kemi et al., 2005; Tjonna et al., 2008). These intervals are typically interspersed by 2-4 minute active recovery periods that are aimed at clearing lactate and reducing fatigue to enable more intervals. Our results suggest that 2-4 minutes may not be sufficient for this purpose, though this may be improved by active recovery at an exercise intensity close to the LT. This may therefore have implications for improving exercise training programmes

or performance during intermittent-type or repeated sports events. In swimming, this concept has already been demonstrated (Greenwood et al., 2008).

CONCLUSION

This study demonstrates that active recovery after strenuous aerobic exercise leads to a faster clearance of accumulated blood lactate than passive recovery, and that the rate of blood lactate clearance depends on the exercise intensity of the active recovery, with peak lactate clearance rates occurring at intensities close to LT. This was observed by measuring blood lactate clearance during active and passive recovery ranging 100-0% of the individual LT. Active recovery intensities at 100-80% of LT were more beneficial than exercise intensities at 60% of LT or below, and this dose-response relationship also existed during active recovery at 60%, 40% and 0% (passive recovery) of LT. Thus, after a strenuous high intensity aerobic exercise bout at an intensity close to VO_{2max}, the fastest lactate clearance is achieved by active recovery at an exercise intensity close to or just below the individual LT.

ACKNOWLEDGEMENTS:

The authors are indebted to the subjects for participating in this study. The study was supported by funding from the University of Glasgow.

CONFLICT OF INTEREST:

None

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Table 1. Participant characteristics.

	Mean	SEM	Range	
			Minimum	Maximum
Age (years)	21.1	0.4	21	23
Height (cm)	180.8	1.7	175.0	190.5
Body mass (kg)	75.3	2.8	65.0	90.8
VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	56.6	1.4	51.5	61.9
Maximal heart rate (bpm)	201.3	0.8	198	205
Velocity at LT (km/h)	10.0	0.3	8.6	11.2
VO_2 at LT ($mL \cdot kg^{-1} \cdot min^{-1}$)	36.8	1.4	31.0	45.6
%VO_{2max} at LT (%)	64.9	1.3	59.6	73.7
Heart rate at LT (bpm)	162.8	2.7	143.0	170.0

Participant characteristics at the inclusion to the study (n=10). VO_{2max}: maximal oxygen uptake; LT: lactate threshold; VO_2: oxygen uptake; SEM: standard error of the mean.

FIGURE LEGENDS

Figure 1. Heart rates during active and passive recovery sessions. *: different from other intensities, $p<0.01$; #: different from other intensities apart from self-regulated or 80% of LT intensities, respectively, $p<0.01$.

Figure 2. **A**: Blood lactate concentration ($[La^-]$) at baseline (warm-up), after a 5-minute run at 90% of maximal oxygen uptake (VO_{2max}; exercise, 0 min on X axis), 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 (79±5% of LT). **B**: Normalised blood lactate clearance during active and passive recovery. The baseline is marked with a dashed line. *: different from passive (0% of LT), $p<0.01$ and 40% of LT, $p<0.05$. Note that 40% of LT and passive recovery were not different from one another.

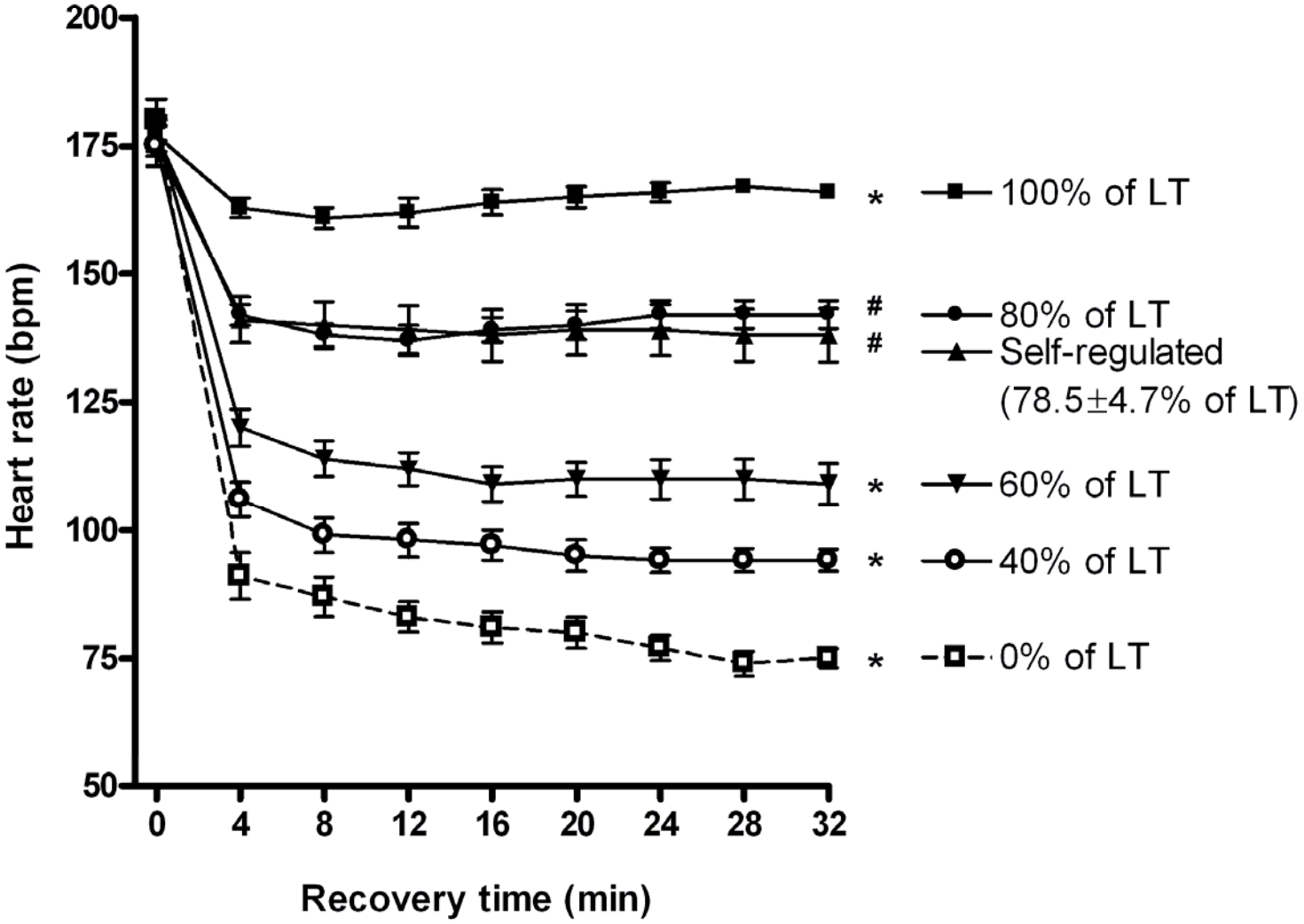
Figure 3. **A**: Example graph of lactate clearance during active recovery after a 5-minute bout of high-intensity exercise at 90% of maximal oxygen uptake; the active recovery intensity in this example is 60% of the lactate threshold (LT). The 1st derivative of the lactate clearance curve is plotted 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 60% and 40% of LT, $p<0.05$; *: different from passive recovery (0% of LT), $p<0.01$; ●: different from passive recovery (0% of LT), $p<0.05$.

Figure 4. **A**: Example graph of lactate clearance and its fitted exponential decay curve during active recovery after a 5-minute bout of high-intensity exercise at 90% of maximal oxygen uptake; the active recovery intensity in this example is 60% of the lactate threshold (LT). Inset is the correlation between the two curves and the 67% ($^2/_3$) time constant. **B**: 67% ($^2/_3$) time constants of

lactate clearance during each intensity of active or passive recovery. #: different from 60% of LT, $p<0.05$; *: different from passive recovery (0% of LT), $p<0.01$; ●: different from 40% of LT and passive recovery (0% of LT), $p<0.05$. Note that 40% of LT and passive recovery were not different from one another.

Figure 5. Normalised individual blood lactate clearance curves during active recovery at self-recovery exercise intensities. Individual curves are displayed, but labelled into 10-percentile blocks with respect to relative intensity of the lactate threshold (LT). This resulted in each block containing two trials. Note that the lowest exercise intensities resulted in slowest clearance of blood lactate, whereas the higher intensities tended to clearance blood lactate faster.

Figure 1


200
175
150
125
100
75
50
Heart rate (bpm)
0
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8
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16
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32
Recovery time (min)
*
#
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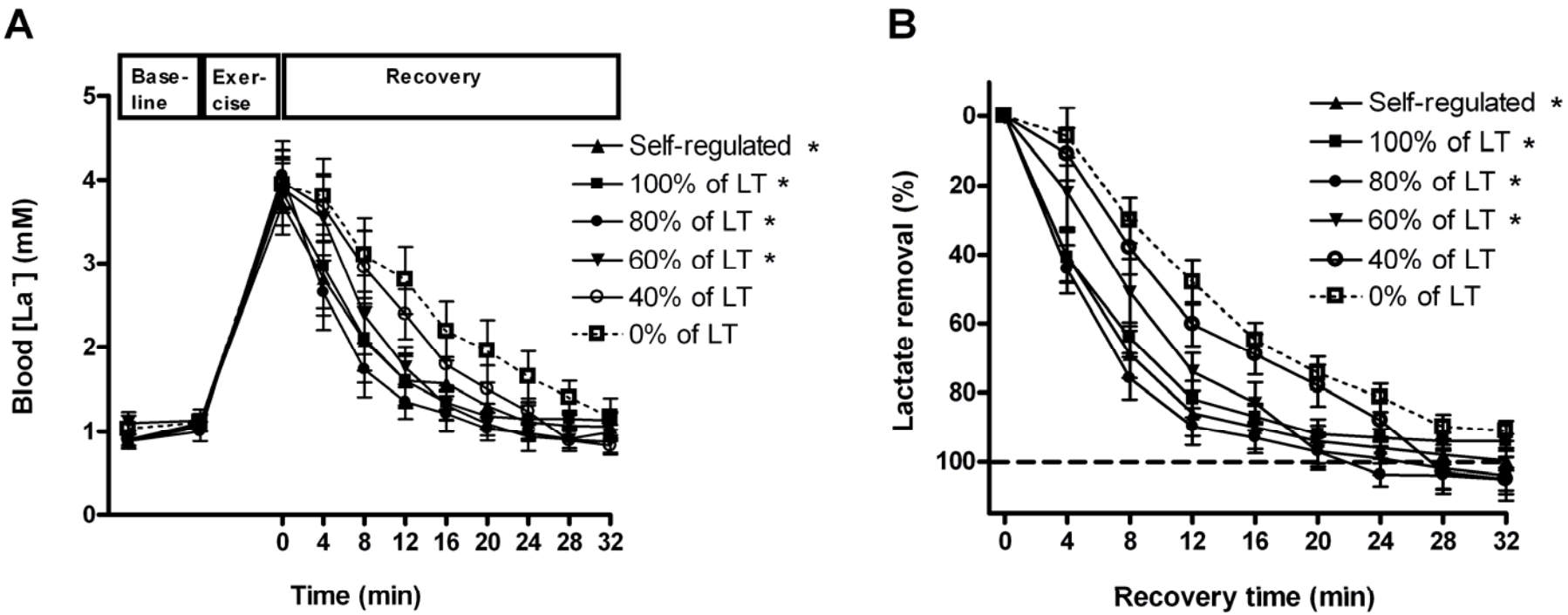
100% of LT
80% of LT
Self-regulated
(78.5±4.7% of LT)
60% of LT
40% of LT
0% of LT

Figure 2


A
Base-line
Exer-cise
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Blood [La⁻] (mM)
0
1
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Time (min)
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100% of LT *
80% of LT *
60% of LT *
40% of LT
0% of LT
B
Lactate removal (%)
0
20
40
60
80
100
0 4 8 12 16 20 24 28 32
Recovery time (min)
Self-regulated *
100% of LT *
80% of LT *
60% of LT *
40% of LT
0% of LT

Figure 3

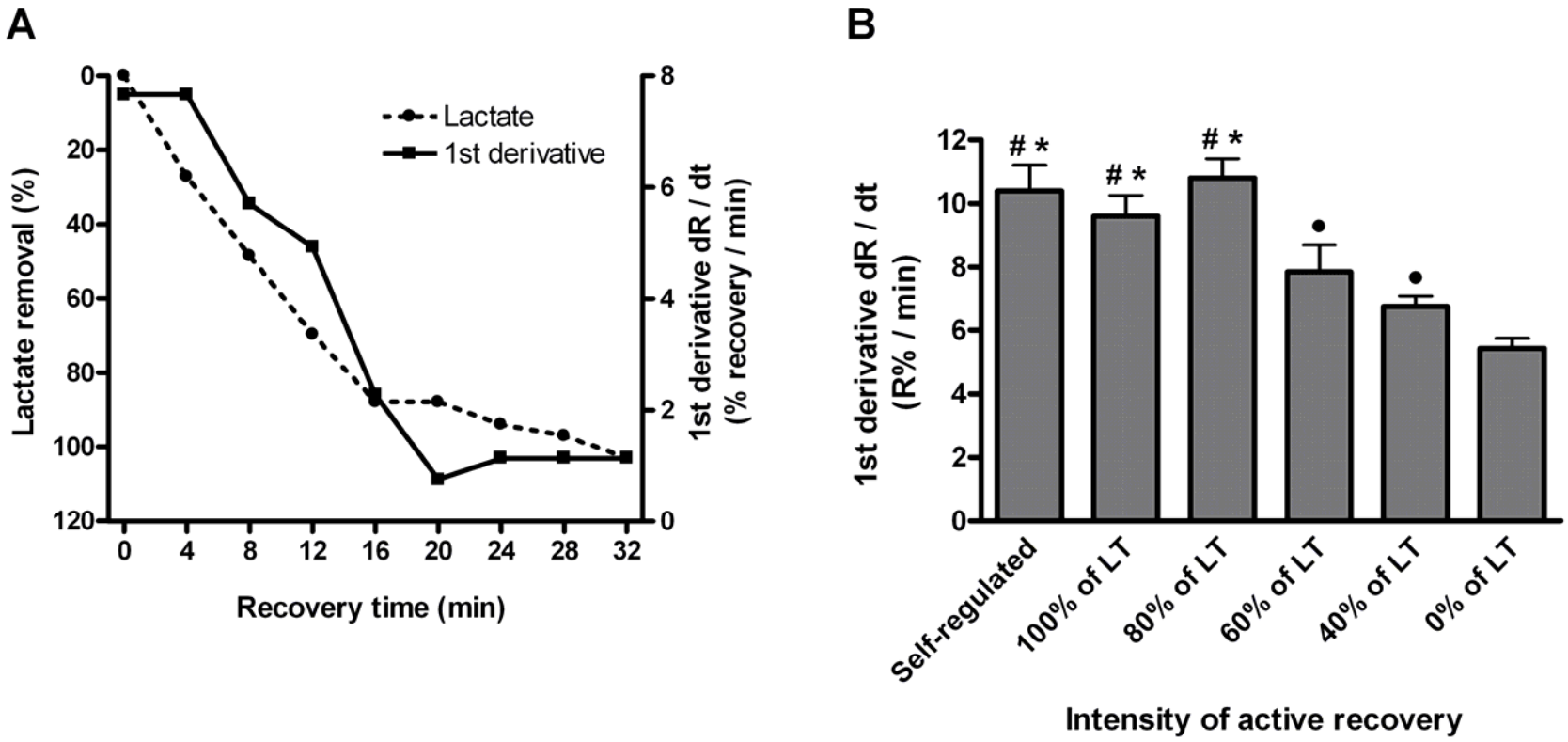

A
Lactate removal (%)
1st derivative dR / dt
(% recovery / min)
Lactate
1st derivative
0
20
40
60
80
100
120
8
6
4
2
0
0 4 8 12 16 20 24 28 32
Recovery time (min)
B
1st derivative dR / dt
(R% / min)
12
10
8
6
4
2
0
*
*
*
Self-regulated
100% of LT
80% of LT
60% of LT
40% of LT
0% of LT
Intensity of active recovery

Figure 4

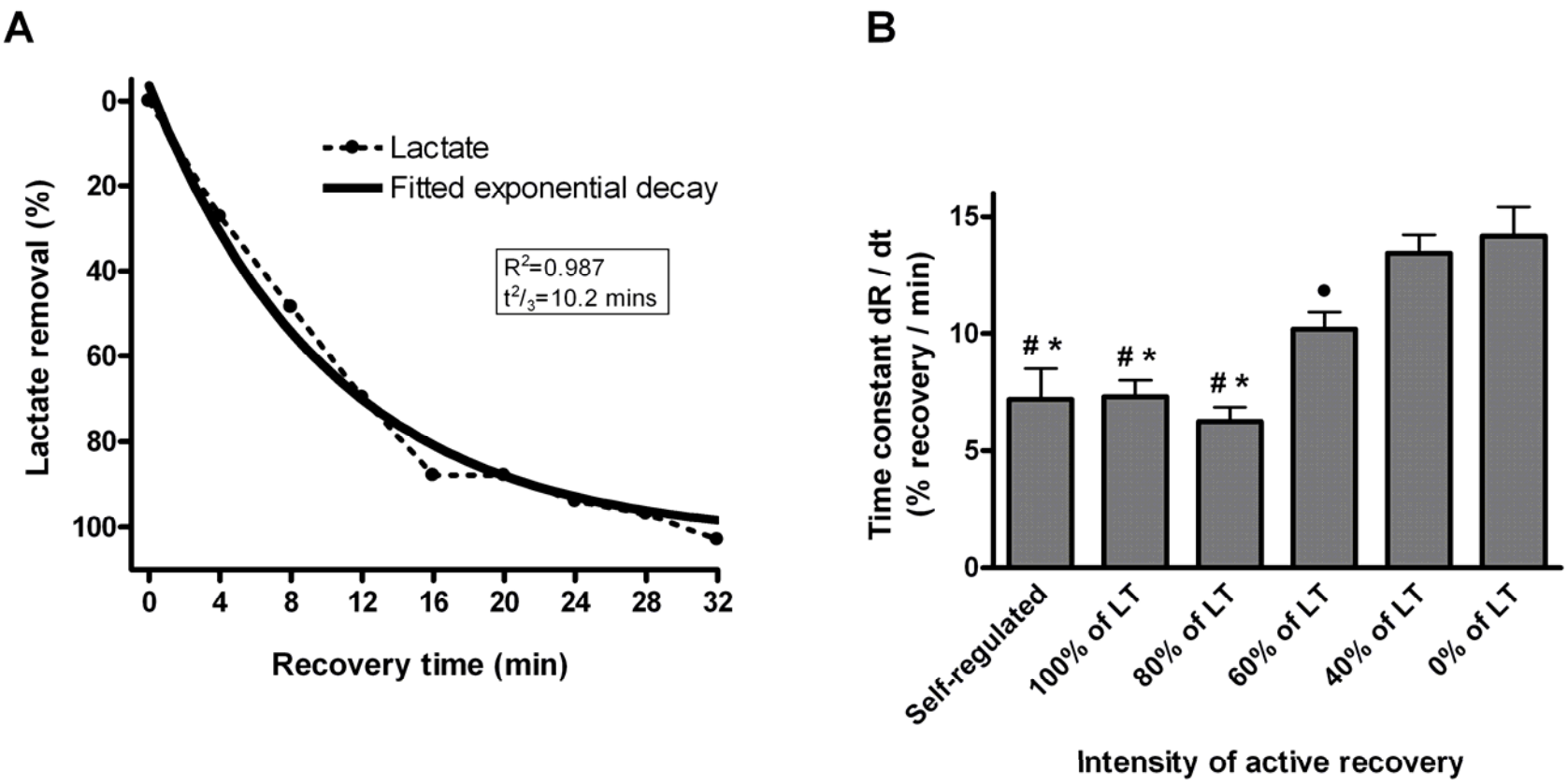
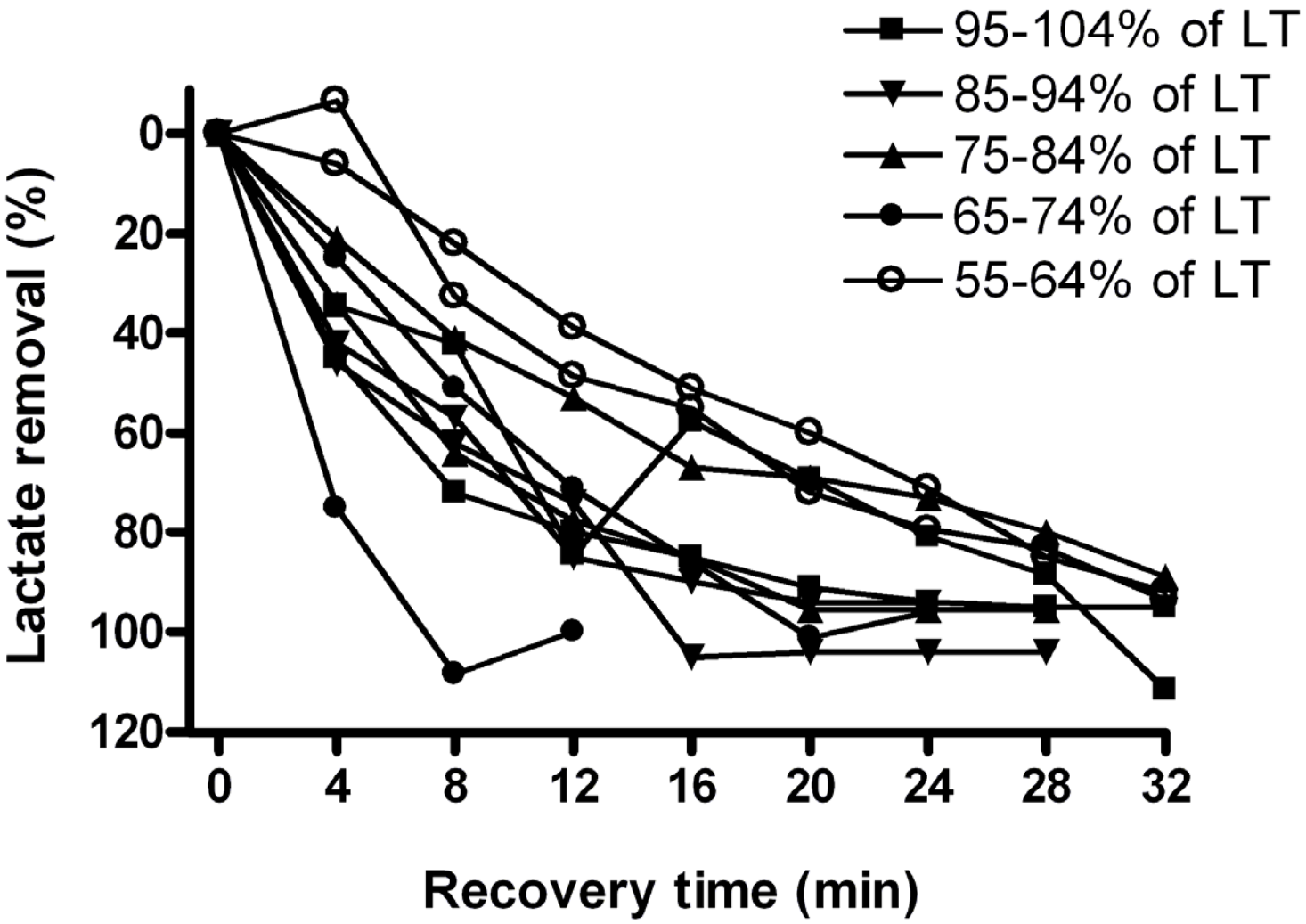

A
Lactate removal (%)
0
20
40
60
80
100
Lactate
Fitted exponential decay
R²=0.987
t²/₃=10.2 mins
0
4
8
12
16
20
24
28
32
Recovery time (min)
B
Time constant dR / dt
(% recovery / min)
15
10
5
0
*
*
*
Self-regulated
100% of LT
80% of LT
60% of LT
40% of LT
0% of LT
Intensity of active recovery

Figure 5


95-104% of LT
85-94% of LT
75-84% of LT
65-74% of LT
55-64% of LT
Lactate removal (%)
0
20
40
60
80
100
120
0
4
8
12
16
20
24
28
32
Recovery time (min)