

Effect of Recovery Interventions on Lactate Removal and Subsequent Performance

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The recovery process in sport plays an essential role in determining subsequent athletic performance. This study investigated the effectiveness of different recovery interventions after maximal exercise. Eighteen trained male cyclists initially undertook an incremental test to determine maximal oxygen consumption. The four recovery interventions tested were: passive, active (50% maximal oxygen uptake), massage, and combined (involving active and massage components). All test sessions were separated by 2 to 3 days. During intervention trials subjects performed two simulated 5 km maximal effort cycling tests (T_1 and T_2) separated by a 20 min recovery. Performance time for the tests (t_1 , t_2); blood lactate (BLa) during T_1 , T_2 , and every 3 min during recovery; and heart rate (HR) during the recovery intervention and T_2 were recorded. Combined recovery was found to be better than passive ($P < 0.01$) and either active or massage ($P < 0.05$) in maintenance of performance time during T_2 . Active recovery was the most effective intervention for removing BLa at minutes 9 and 12, BLa removal during combined recovery was significantly better than passive at minute 3, and significantly better than passive, active, and massage at minute 15. In conclusion, combined recovery was the most efficient intervention for maintaining maximal performance time during T_2 , and active recovery was the best intervention for removing BLa.

■ Key words: Fatigue, blood lactate, massage.

Introduction

The recovery process is of particular importance in events where an athlete may have to compete on more than one occasion during a competition in a single day such as track, swimming, cycling, and rowing.

It is generally accepted [2,12] that intense physical activity results in the production of lactate, and the accumulation of lactate in exercising muscle is thought to be a major determinant of fatigue [10,14]. However, a definitive relationship has not yet been established. Furthermore, several studies have concluded that the accumulation of muscle lactate does not exert a significant negative effect on subsequent performance, therefore the role of lactate in fatigue is questionable [4,15,16,17].

General agreement exists over the fact that active recovery (sustained exercise at a sub-maximal intensity) is more efficient than passive recovery (rest) in improving the recovery process [1,4,6–9,14–16]. Another modality commonly used on the sports field to facilitate the recovery process is sports massage. However, scientific data concluding that massage results in positive physiological changes from the recovery process viewpoint are limited, and no studies to our knowledge have examined a combined active/massage regime.

The purpose of the present study was to compare the effects of four different recovery interventions following maximal cycling exercise (laboratory simulated 5 km time trial), on the rate of blood lactate removal and subsequent maximal cycling performance capacity.

Methods

Subjects

The subjects were 18 healthy trained male cyclists. All subjects were tested either pre-season or at the beginning of the competitive season. Written informed consent was obtained from each individual prior to enrolment, and all procedures were approved by the Human Ethics Committee of the University of Dublin.

Testing protocol

All subjects reported for testing on 6 separate occasions over a 3 week time frame, all testing sessions and recovery interventions were carried out in a temperature controlled environmental chamber ($18 \pm 1^\circ\text{C}$).

Maximal O_2 uptake test

The initial test session involved a medical examination and a continuous maximal incremental cycling test using a Kingcycle Trainer/Tester unit (EDS Portapromt Ltd., England) and the cyclist's own bicycle to determine individual $\dot{V}\text{O}_2\text{max}$ and lactate (BLa) profile, followed by an 10 min active recovery period at a self-selected intensity. Metabolic parameters were recorded using a Metamax cardiorespiratory unit (Cortex Ltd., Leipzig, Germany), heart rate (HR) by radiotelemetry (Polar Electro, Kempere, Finland), and blood lactate (BLa) from finger tip capillary samples using a YSI 1500 Sport Lactate Analyser (Yellow Spring Instruments, Ohio, USA). Prior to each test the Metamax analyser was volumetrically calibrated using a 3 L Hans Rudolph syringe, the O_2 and CO_2 analysers were calibrated using an alpha standard gas (17.8% O_2 , 5.1% CO_2) and room air.

Following an initial data collection period at rest (3 min), a progressive incremental step protocol was used. Initial load was 120 W, duration 3 min, increment 40 W, and ramp rate $8\text{ W} \times \text{s}^{-1}$. All cyclists were instructed to cycle to exhaustion at a self-selected cadence and gear ratio. Two 30 cm fans (Everal Ltd., England) were used to cool the cyclist.

5 km maximal effort test

The second test session consisted of a 5 min warm-up at 150 W, a 5 km maximal effort test, 10 min cycling at a self-selected intensity, followed by 20 min seated recovery and subsequent performance of a second 5 km maximal effort test, following a further 2 min warm-up at 150 W. This test session served to familiarise the subjects with the distance and test protocol and to investigate replication of the performance times for the first 5 km test, since reproduction of maximal performance on subsequent test days was an essential protocol requirement.

In the 3rd to 6th test sessions, following Kingcycle calibration and a 2 min pre-test warm-up at 150 W, each subject undertook two 5 km maximal effort tests (T_1 and T_2) separated by 20 min. Following the first 5 km test, the subjects remained stationary on the bicycle for one min, over the following 15 min the different recovery interventions were performed, recalibration of the Kingcycle took 2 min, and during the final 2 min the subjects undertook the standard pre-test warm-up (150 W) before undertaking the second 5 km test (see Fig. 1). Performance time for the 5 km tests (t_1 and t_2), blood lactate (BLa) during T_1 , T_2 , and at 3 min intervals during the recovery period, and HR during the recovery intervention, and T_2 were recorded.

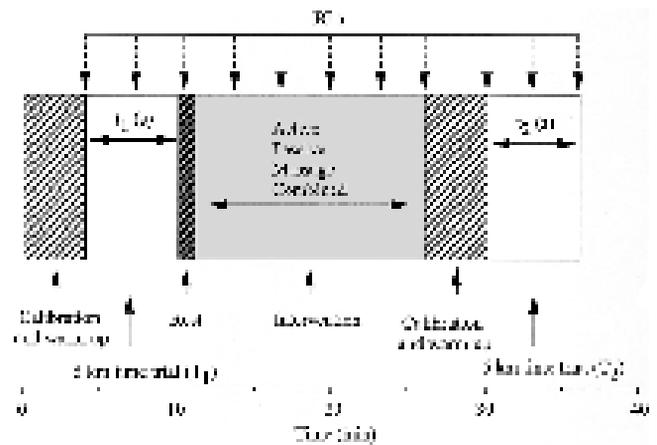


Fig. 1 Schematic illustrating event sequence for randomised intervention trials, blood lactate (BLa) were collected at the time points indicated.

Recovery interventions

The four recovery interventions investigated were passive, active, massage, and combined recovery; intervention sequences were randomised across cyclists to eliminate bias. During passive recovery, following the initial 5 km time trial, the cyclist dismounted the bicycle and remained seated at rest on a chair for 15 min. Active recovery consisted of sub-maximal cycling at a load equivalent to 50% of individual $\dot{V}\text{O}_2\text{max}$. Massage consisted of three basic manipulations (effleurage, stroking and taponement) applied to the posterior part of the lower extremities in the supine position, the same certified masseur applied massage to all cyclists. Combined recovery consisted of pedalling at a sub-maximal load equivalent to 50% of $\dot{V}\text{O}_2\text{max}$ for the initial 3.75 min of the intervention, followed by the massage application in the supine position for 7.5 min (3.75 min per leg), and finally cycling for the final 3.75 min at the same equivalent load.

Each recovery intervention lasted for 15 min, no stretching was undertaken during the recovery interventions, but the cyclists were allowed to rehydrate freely, provided they always consumed an equal volume of similar drinks on the different test days.

Statistical analysis

ANOVA for repeated measures was used to detect significant differences across the four different recovery interventions, an α level of 0.05 was selected to infer statistical significance. Post-hoc analysis of significant differences were investigated using the Scheffe F test. Group results are presented as mean and standard error of the mean (SEM).

Results

The physical characteristics of the cyclist ($n=18$) who took part in this study were age 25 ± 0.9 yr, body mass 72 ± 1.6 kg, $\dot{V}\text{O}_2\text{max}$ $68 \pm 1.7\text{ ml} \times \text{kg}^{-1} \times \text{min}^{-1}$, maximum power output 364 ± 9 W, power at T_{Lac} 283 ± 10 W, cycling experience 5 ± 0.3 yr, mean \pm SEM.

No statistically significant differences were recorded in performance time for the first 5 km maximal effort test (t_1) or in BLA data before or following the first 5 km maximal effort test across the different test sessions (see Table 1).

Performance capacities (s) across recovery interventions were compared by computing the time difference $t_2 - t_1$. The mean increase in time trial time (2.9 ± 1.5 s) was significantly lower following combined recovery compared with the other interventions tested, see Table 2. The rate of lactate removal in $\text{mM} \times \text{min}^{-1}$ was calculated over each successive 3 min interval during the different recovery interventions by calculating Δ BLA over each interval and dividing by time (see Fig. 2), both the active and combined interventions were more efficient than either the passive or massage interventions for removal of BLA (see Fig. 2). Over the entire intervention phase the mean \pm SEM rates of BLA removal were 0.37 ± 0.03 , 0.38 ± 0.04 , 0.21 ± 0.04 , and $0.16 \pm 0.06 \text{ mM} \times \text{min}^{-1}$ for active, combined, massage, and passive interventions, respectively. Commencing the 2nd time trial, mean BLA were significantly higher ($P < 0.01$) following passive and massage interventions, however, following the 2nd time trial no statistically significant differences in mean BLA across interventions were noted.

The lowest HR data were recorded during the passive and massage interventions, as well as during the massage portion of combined recovery. The highest HR data were recorded during active recovery and the active portions of the combined recovery. Fig. 3 shows mean heart rate during the four different recovery interventions.

Discussion

The principal finding of this study was that the combined recovery intervention was significantly better than passive, active, or massage recovery interventions in terms of maintenance of performance (see Table 2) during subsequent maximal effort. In addition we have shown that both the active and combined interventions were more efficient ($P < 0.05$) than the passive or massage interventions for removal of BLA following exercise. However, during the combined recovery intervention the highest rate of BLA removal occurred during the active phases (see Fig. 2, min 0–3 and min 12–15).

The exact mechanisms by which combined recovery induced the best maintenance of performance capacity (minimum difference between 2nd and 1st time trial time) are unclear, and more research is clearly warranted in this area. One possible reason was the higher rates of BLA removal recorded (see Fig. 2) during the active portions of the combined recovery intervention (initial and final elements). Although similar rates of BLA removal occurred during active recovery, and over the entire recovery period no significant difference was noted (active $0.37 \pm 0.03 \text{ mM} \times \text{min}^{-1}$ versus combined $0.38 \pm 0.04 \text{ mM} \times \text{min}^{-1}$, $P > 0.05$), performance capacity in the 2nd maximal effort test was significantly less than that recorded following combined recovery (6.9 ± 1.3 versus 2.9 ± 1.5 s, $P < 0.05$). This difference was noted despite similar pre-intervention BLA (see Table 1), implying that other factors must be crucial for maintaining performance during subsequent exercises. At a muscular level, factors effecting maximal exercise performance include lactate concentration, glycogen stores, pH and ATP/ADP ratio. One possible factor effecting subsequent performance

Table 1 Mean blood lactate concentration before and after the initial 5 km maximal effort test (T_1) and performance time for the initial 5 km maximal effort test (t_1) preceding the different interventions, $n = 18$, data are mean \pm SEM

Intervention	BLA (mM)		Performance time
	Pre T_1	Post T_1	t_1 (s)
Passive	1.8 ± 0.1	8.9 ± 0.5	379 ± 4.5
Active	1.9 ± 0.1	8.5 ± 0.5	379 ± 4.2
Massage	2.0 ± 0.1	9.5 ± 0.6	379 ± 4.2
Combined	2.0 ± 0.0	9.2 ± 0.6	382 ± 6.3

Table 2 Mean increase in 5 km trial time (s) \pm SEM across intervention, $n = 18$, difference calculated as time for 2nd trial minus time for 1st trial ($t_2 - t_1$)

Intervention	Mean increase (s)	SEM
Passive	9.9^{**}	1.6
Active	6.9^*	1.3
Massage	7.7^*	1.5
Combined	2.9	1.5

** implies combined recovery significantly less at $P < 0.01$, * implies combined recovery significantly less at $P < 0.05$

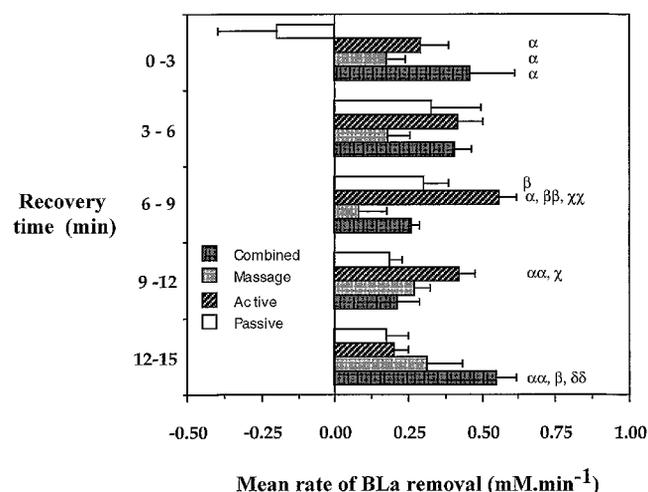


Fig. 2 Bar graph of mean rate of BLA removal ($\text{mM} \times \text{min}^{-1}$) over successive 3 minute intervals during the different recovery interventions, $n = 18$, bars denote SEM. Symbol codes for statistical significance; α and $\alpha\alpha$ indicates greater than passive at $P < 0.05$ and $P < 0.01$, respectively, β and $\beta\beta$ indicates greater than massage at $P < 0.05$ and $P < 0.01$, respectively, γ and $\gamma\gamma$ indicates greater than combined at $P < 0.05$ and $P < 0.01$, respectively, and $\delta\delta$ indicates greater than active at $P < 0.01$.

could be the restoration of muscle glycogen during the recovery period. While this variable was beyond the measurement scope of the present study, previous investigations have examined the efficiency of passive and active recovery on the repletion of intramuscular glycogen stores. Some researchers refute the hypothesis that active recovery impedes muscle glycogen

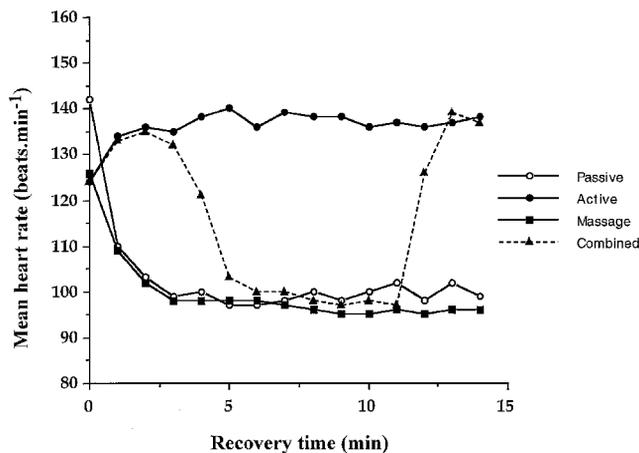


Fig. 3 Graph of mean heart rate (beats \times min⁻¹) versus time (min) across the different recovery interventions, n = 18.

resynthesis following intense exercise [14] while others support it [5]. In addition there are numerous studies demonstrating an increase in muscle glycogen during passive recovery following intense exercise [3,5,13]. Peters-Futre et al. [13] reported no significant differences in rates of muscle glycogen resynthesis following intense exercise between active and passive recovery. Although non-significant, glycogen resynthesis tended to be greater during passive recovery (37.2 vs. 24.0 mM \times kg⁻¹ww in 90 min). In their study high-intensity intermittent bicycle exercise was used to deplete muscle glycogen levels by 70% and elevate blood lactate levels to > 13 mM.

Choi et al. [5] designed a study to compare the effect of both passive and active recovery on the resynthesis of muscle glycogen during a 60 min recovery period. Six untrained subjects performed three 1 min exercise bouts at approximately 130% $\dot{V}O_2$ max with 4 min rest periods. The recovery interventions used after exercise were either active (30 min at 40–50% $\dot{V}O_2$ max, 30 min seated rest) or passive (60 min seated rest). They reported that mean muscle glycogen content after 60 min of passive recovery had increased by 15.0 ± 4.9 mM \times kg⁻¹ww, in contrast to a decrease of 6.3 ± 3.7 mM \times kg⁻¹ww following 60 min active recovery ($P < 0.05$).

The intervention phase in the present study was considerably shorter (15 min compared to 60 min), therefore a direct comparison between studies is not possible since biopsy samples for glycogen resynthesis rates were only reported at the end of the recovery period by Choi et al. [5]. Similar rates of glycogen repletion could have occurred during passive, massage, or the massage portions of the combined recovery interventions, and based on the data of Choi et al. [5] this would imply higher glycogen stores at the start of the 2nd time trial compared to active recovery. While we have no data supporting this speculation, more detailed research is warranted in this area.

Results obtained in this study show that exercise at an intensity below T_{lac} (active recovery and the active phases of combined recovery, intensity 50% $\dot{V}O_2$ max) induced a greater rate of BLA removal than the other recovery interventions, see Fig. 2. These findings are in agreement with results from previous studies [6,9,17]. The mechanisms by which active recovery

induces a greater rate of BLA removal are not completely understood. BLA concentration during recovery from maximal exercise is the product of a complex interplay of multiple factors. These include: the bicarbonate buffer system, efflux of lactate from the muscle into the blood, local blood flow, and fractional removal or uptake by the liver, skeletal muscles, and the heart. Active recovery was the most effective strategy for increased blood flow to the working muscles, as indicated by the increased heart rate, compared to the other recovery interventions. Massage also produces an increase in tissue blood flow through increased arterial inflow and increased venous compliance. However, during the massage elements lactate clearance was lower (see Fig. 2), and during the massage intervention the mean rate of lactate removal was similar to passive and significantly lower ($P < 0.01$) than either active or combined interventions. Other factors are also involved in the uptake of lactate by the organs/tissues. Jorfeldt et al. [11] reported that translocation of lactate from the muscle to the blood showed a linear relationship with intracellular lactate concentrations up to approximately 4 mM \times kg⁻¹ww, after which there was a constant rate of lactate efflux. If lactate transport across the cell membrane is carrier mediated, then a high blood flow would have little effect on accelerating the rate of lactate efflux from intracellular to the extracellular compartments.

In conclusion, combined recovery resulted in the best maintenance of performance capacity during the second 5 km maximal effort test. This finding could be explained by a high rate of BLA removal during the active portions of this intervention, coupled with a speculated higher rate of intramuscular glycogen restoration during the massage portion, compared to active recovery. Finally, of the interventions tested, active and combined recovery were the most efficient for the removal of blood lactate following maximal cycling exercise.

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