

**Title:** Active recovery strategy and lactate clearance in elite swimmers

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## ABSTRACT

**Background:** Swimming requires sustained high performance, with limited recovery between heats, recovery strategies are essential to performance but are often self-regulated and sub-optimal. Accordingly, we investigated a physiologically determined recovery protocol.

**Methods:** Fifteen (m=9, f=6) international junior age group swimmers participated in this study. The average age of the participants was  $15.8 \pm 1.5$  years. All participants completed a lactate elevation protocol (8 x 50 m sprints), followed by one of three recovery strategies, 1) velocity at lactate threshold ( $V_{LT}$ ), 2) coach prescribed protocol (COA) and 3) national governing body recommendations (NGB) and thereafter a 200-m time trial.

**Results:**  $[\text{lac}^-]_B$  was similar between trials at baseline (pooled data:  $1.3 \pm 0.4 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P>0.05$ ) but increased following 8x50 m sprints (pooled data  $9.5 \pm 3.5 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ ) and reduced in all conditions (mean reduction  $6.4 \pm 1.7 \text{ mmol}\cdot\text{l}^{-1}$ ).  $[\text{lac}^-]_B$  remained elevated following NGB ( $5.6 \pm 0.8 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ ) compared with COA ( $2.3 \pm 1.7 \text{ mmol}\cdot\text{l}^{-1}$ ) and  $V_{LT}$  ( $1.7 \pm 1.2 \text{ mmol}\cdot\text{l}^{-1}$ ) but was blunted during the 200-m time trial in  $V_{LT}$  ( $6.4 \pm 1.7 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ ). Time trial performance was similar between trials;  $V_{LT}$  ( $2.24 \pm 0.12 \text{ min}$ ), COA ( $2.23 \pm 0.14 \text{ min}$ ) and NGB ( $2.22 \pm 0.13 \text{ min}$ ,  $P>0.05$ ).

**Conclusions:** Despite similar performance, individually prescribed recovery strategy with a physiological basis will preserve repeated exercise performance performed on the same day.

**Key words:** Lactate threshold, recovery, swimming, performance, athletes, endurance

## INTRODUCTION

Performance conducted in aquatic mediums poses additional physiological challenges when compared with land-based events, primarily caused by the resistance and drag of the fluid medium. Despite the well documented anthropometric markers that determine successful performance in swimmers (e.g. arm span and hand surface area (13), sprint swimmers must also possess high levels of power, speed, and endurance, supported via metabolism of fat and carbohydrates that is relative to the exercise intensity (17). Short duration, high intensity swimming events such as the 200-m require a large contribution from the muscles high-energy phosphate stores and glycolysis (up to 67%; (19) which are replenished via non-mitochondrial processes (29). The high anaerobic demand and repeated performance therefore presents a large metabolic challenge which is exacerbated in subsequent bouts of exercise and commonly in the absence of sufficient time for recovery (2). The large anaerobic component coupled with swim competition schedules, typically incorporate a series of preliminary heats and then a final, often on the same day and with limited time for recovery (17).

Lactate accumulation prior to exercise has been shown to reduce swimming performance (5,6,9). A rise in intramuscular  $[\text{lac}^-]_{\text{B}}$  increases the strong ion difference and elevates  $[\text{H}^+]$ , a natural physiological process which occurs to preserve intramuscular electroneutrality (4). However the accumulation of  $\text{H}^+$  (acidosis) contributes to the development of muscle fatigue, impairing intramuscular contractile properties (7,12) and key metabolic enzymes.(1). The accumulation of  $\text{H}^+$  is also associated with heightened perceptual responses (23) which are reversed by favorable changes in acid-base balance (24).

Although the case for the accumulation of lactate ions and muscular fatigue is not entirely clear, due to the importance of other biochemical markers in this process (30), the use of lactate as a marker of exercise intensity is commonplace within applied settings. Reductions in lactate concentrations during exercise is therefore considered advantageous for skeletal

muscle metabolic activity, force production and most importantly performance. In recognition of the implications for exercise performance, previous research from our group (15) and others (8,17) has designed and investigated the efficacy of different recovery protocols to accelerate lactate clearance which, has been shown to sustain performance in repeated bouts. However, this research to date research has focused on passive (e.g. standing/seated rest) or active (e.g. low absolute exercise intensity) recovery strategies with the latter being superior to the former. However, uncertainty remains surrounding the optimal intensity of the active recovery phase (9,26). To coincide with this, recovery protocols and subsequent intensities are largely uncontrolled and determined by athletes, coaches and in some instances governing bodies. We suggest that the use of an individual's physiological basis to determine if an optimal intensity for recovery strategies exists which can accelerate lactate clearance and positively influence performance. Accordingly, the aim of this study was to evaluate different models of active recovery used within elite swimming upon lactate clearance, the lactate response to, and performance of, 200-m time-trial.

## MATERIALS AND METHODS

### *Participants*

Following ethics approval from the host University 15 (male: n=6; female: n=9) elite international age group swimmers (within 2% of National Qualifying Time) provided written informed parental consent to participate in the study (see Table 1). Participants trained 9 times per week for 1 to 3 hrs per session (land: n=1; water: n=8) and weekly training distance was between 40 and 50 km. Participants completed a 24h diet record prior to their first exercise trial, which was then replicated prior to all subsequent trials. Participants abstained from caffeine in the 24h prior to testing and arrived at the swimming pool 2h post-prandial. Participants initially attended a briefing session where the experimental design was explained in full. Following this, participants completed three preliminary trials and three experimental trials outlined below.

**\*\* TABLE 1 AROUND HERE \*\***

### *Preliminary Trials*

During the first preliminary trial participants completed a full body composition assessment using dual energy x-ray absorptiometry (Lunar iDXA, GE Healthcare, Hertfordshire, UK). On a separate day, participants performed a maximal 200-m front crawl time trial (pooled time  $2:21 \pm 0.14$  min). Finally, participants performed a lactate profiling protocol for the determination of the individual velocity commensurate with the lactate threshold ( $V_{LT}$ ) according to the method of Pyne, Lee, & Swanwick (21) and defined as the first rise above resting levels by  $1 \text{ mmol}^{-1}$ . This intensity optimises lactate clearance through aerobic metabolic pathways (18). All swimmers performed seven incremental 200-m front crawl swims from a push start with the first 30 s slower than the maximal 200-m time trial time recorded in the second preliminary trial with the remaining swim efforts 5 s faster than the effort preceding it. Each increment was 5 min in duration allowing the swimmer to recover in

the pool prior to the next bout. Prior to the first and immediately following each increment  $[\text{lac}^-]_{\text{B}}$  was measured from an arterialised fingertip (Accu-Check, Safe T-Pro, Birmingham, UK). The analyzer was checked for accuracy prior to each test using standards provided by the manufacturer and precision from previous literature within the physiological range of  $1.0 \pm 18.0 \text{ mmol}\cdot\text{l}^{-1}$ (20). Subsequently a  $[\text{lac}^-]_{\text{B}}$ -velocity curve was produced for each athlete to determine  $V_{\text{LT}}$ .

### *Experimental trials*

Following the completion of an individual competition warm-up, participants completed 8 x 50-m maximal sprints (duration of sprint plus recovery was 1 min 30 s) from a competition start which was designed to maximise the individual's metabolic disturbance and create a worse case metabolic scenario for the swimmer. Subsequently swimmers performed one of three recovery strategies followed immediately by a 200-m front crawl time trial. The order of the experimental trials was randomised for each participant using a latin square and separated by a minimum of one week. Recovery strategy one was prescribed by the team coach based upon individual experience (hereon referred to as COA) consisting of 6 x 100-m front crawl efforts with a start interval of 1 min 40 s, 200-m kicking interspersed with a 10-m sprint at 50-m intervals. Second, participants completed 20 min front crawl swimming at the velocity of lactate threshold ( $V_{\text{LT}}$ ). Third, 20 min variable speed swimming comprising 200-m front crawl, 4 x 100-m freestyle, 4 x 100-m backstroke, 4 x 100-m kicking and finally 200-m individual choice swimming which is formed by generic guidance for regional high-performance age group centres issued by the national governing body (here on referred to as NGB). Both COA and NGB were self-paced protocols and in-line with recommendations from previous literature. At baseline, prior to and following 8 x 50-m sprints, following the recovery swim and following the subsequent 200-m time trial  $[\text{lac}^-]_{\text{B}}$  and ratings of whole body

perceived exertion (6 to 20 scale) were recorded. Heart rate was measured continuously throughout all trials using short range telemetry (Polar Team<sup>2</sup>, Kempele, Finland) and then averaged over 10 seconds for analysis. Time to complete each 25-m of the 200-m swim was recorded using a hand-held stopwatch by experienced timekeepers. Stroke rate throughout each 25-m was used to calculate mean stroke rate (SR) and mean stroke length (SL) for the 200-m effort as reported previously (28) where  $SR \text{ (cycles}\cdot\text{min}^{-1}) = (\text{stroke cycles to complete distance}/\text{time to complete distance}) \times 60$  and  $SL \text{ (m}\cdot\text{cycle}^{-1}) = \text{distance to swim}/\text{cycles to complete distance}$ .

### *Statistical analyses*

Changes in dependent variables over time throughout the experimental trials or between trials at specific time points were assessed using one-way repeated measures ANOVA with Bonferroni post-hoc analysis. A factorial repeated measures ANOVA was used to test for between trial differences in 200-m time trial performance. A priori  $\alpha$  was set at 0.05 and all results are presented as mean  $\pm$  SD. Effect size was calculated using Cohen's  $d$  ( $d = (x1 - x2)/\text{pooled } \sigma$ ). Statistical analysis was performed using SPSS for Windows (SPSS, Chicago, IL, USA).

## RESULTS

### *Physiological measures*

Baseline  $[\text{lac}^-]_{\text{B}}$  was not different between trials (pooled data  $1.3 \pm 0.4 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P>0.05$ ) and was unchanged following warm up in all trials ( $1.4 \pm 0.5 \text{ mmol}\cdot\text{l}^{-1}$ ). After the 8 x 50-m sprints,  $[\text{lac}^-]_{\text{B}}$  increased to  $9.5 \pm 3.5 \text{ mmol}\cdot\text{l}^{-1}$  (pooled data,  $P<0.05$ ) which was not different between trials ( $P>0.05$ , Figure 1). Following  $V_{\text{LT}}$ ,  $[\text{lac}^-]_{\text{B}}$  decreased to  $1.7 \pm 1.2 \text{ mmol}\cdot\text{l}^{-1}$  (mean reduction  $7.4 \pm 2.9 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ , effect size  $d:1.66$ ) and  $2.3 \pm 1.7 \text{ mmol}\cdot\text{l}^{-1}$  in COA (mean reduction  $7.5 \pm 3.0 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ , effect size  $d:0.77$ ), but remained elevated following NGB ( $5.6 \pm 0.8 \text{ mmol}\cdot\text{l}^{-1}$ ; mean reduction  $4.8 \pm 3.3 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P>0.05$ ; effect size  $d: -1.22$ ). Relative to baseline,  $[\text{lac}^-]_{\text{B}}$  was not different in  $V_{\text{LT}}$  (mean difference  $0.6 \pm 1.1 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P>0.05$ ) and COA (mean difference  $0.9 \pm 1.5 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P>0.05$ ) but greater for NGB (mean difference  $3.5 \pm 1.2 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ ). Post-recovery  $[\text{lac}^-]_{\text{B}}$  was not different between COA and  $V_{\text{LT}}$  ( $P>0.05$ , Figure 1). Post 200-m time trial  $[\text{lac}^-]_{\text{B}}$  increased in all trials (pooled data  $7.3 \pm 0.9 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ , Figure 1). The rise in  $[\text{lac}^-]_{\text{B}}$  in  $V_{\text{LT}}$  ( $6.4 \pm 1.7 \text{ mmol}\cdot\text{l}^{-1}$ ) was lower than both COA ( $8.2 \pm 2.6 \text{ mmol}\cdot\text{l}^{-1}$ , effect size  $d: 0.79$ ) and similar to NGB ( $7.2 \pm 1.5 \text{ mmol}\cdot\text{l}^{-1}$ ,  $P<0.05$ , effect size  $d: 0.49$ ). Heart rate and perceived exertion showed a main effect for time ( $P<0.05$ ) with no interaction effect in all trials (Table 2).

**\*\* FIGURE 1 AROUND HERE \*\***

### *Time Trial Performance*

Time trial performance following  $V_{\text{LT}}$  was  $2.24 \pm 0.12 \text{ min}$  which was similar to COA ( $2.23 \pm 0.14 \text{ min}$ ,  $P<0.05$ ; effect size  $d: 0.16$ ) and NGB ( $2.22 \pm 0.13 \text{ min}$ ,  $P<0.05$ ; effect size  $d: 0.13$ ). This was also similar to the baseline preliminary time trial performed ( $P>0.05$ ).

Accordingly, there was no difference in either stroke rate or stroke length between trials (Table 2,  $P > 0.05$ ).

**\*\* TABLE 2 AROUND HERE \*\***

## DISCUSSION

The key findings of this study demonstrate that active recovery strategies that are prescribed relative to the velocity corresponding to the lactate threshold is most effective at reducing  $[\text{lac}^-]_{\text{B}}$  concentrations following successive bouts of maximal swimming exercise. Although this does not affect performance time during a 200-m swimming performance in a subsequent exercise trial, the blunted post exercise lactate response could have important implications during competition settings where athletes are required to perform repeated high intensity exercise bouts. This is especially true within competitive swimming, where the use of preliminary rounds that precede a final is common within competition settings.

Metabolic provision is proportional to exercise intensity and short duration events such as the 200-m swimming time trial demand energy provision via anaerobic pathways and specifically via the use of anaerobic glycolysis, elevating lactate concentrations (11) which, during sustained exercise at high intensities results in the accumulation of  $\text{H}^+$  (14). The preservation of cellular homeostasis is important to performance and buffering mechanisms exist and operate continuously to limit excessive metabolic perturbations (22). However, high lactate concentrations are associated with reduced swimming performance (9,17) and a typical competition consists of repeated bouts, often with limited time for recovery (~30 min; 17). As a result, research has previously investigated the efficacy of active recovery strategies to optimise the removal of  $[\text{lac}^-]_{\text{B}}$  and enhance performance (28). To date, active recovery strategies are preferred to passive protocols and the use of fixed pace strategies are preferred to self-paced strategies (9). To our knowledge though, this is the first study to compare active recovery strategies that are determined using physiological profiling and compare these to guidelines issued by a governing body and the experience-derived protocol of the swimming squad head coach.

Both NGB and COA strategies were self-paced and intermittent in intensity ( $65 \pm 6\%$  and  $63 \pm 6\%$  max HR respectively), characteristics which are recognised as being sub-optimal in relation to exercise preparation and performance (10). Our data is line with previous findings which suggest that when swimmers are free to self-select their swimming pace they will adopt a pace that is equivalent to 60-70% of their maximum (16,27). Importantly, the use of a recovery strategy prescribed at the swimmer's individual  $V_{LT}$  which was higher in intensity ( $75 \pm 8\% HR_{max}$ ), helped to achieve the desired pre-competition baseline values ( $<2.0 \text{ mmol}\cdot\text{l}^{-1}$ ) following a short ( $\sim 20$  min) cool down period. Although this did not impact performance when quantified by subsequent swim time trial performance, it did lead to a blunted  $[\text{lac}^-]_B$  response.

The need to conduct cool down protocols within a pool environment is preferred over land-based protocols due to the beneficial effects of hydrostatic compression when submerged. The fluid shifts and subsequent changes in blood flow will enhance the capacity to remove blood lactate, (3,5) which is a by-product of high intensity exercise and is associated with decreased exercise performance (25). The importance of exercise intensity in promoting optimal lactate clearance, therefore, should not be overlooked. Protocols that are self-paced, intermittent or characterised by sub-optimal intensities ( $\sim 60\%$  maximum swim pace) may result in reduced blood flow to the working muscles, resulting in lower metabolic demand and/or removal of metabolic waste products. It therefore seems reasonable to suggest that a protocol designed to take place in the water, which is continuous and prescribed relative to an individual's  $V_{LT}$  will result in greater lactate clearance (3). However future research may wish to investigate the efficacy of the reported intervention in a setting that mirrors a traditional competition format that is matched for the number of repeated bouts and time between each repeated effort.

The use of an active recovery strategy that is continuous in nature and prescribed relative to the individuals  $V_{LT}$  provides comparable reductions in  $[\text{lac}^-]_B$  but importantly, also blunts the accumulation of lactate during subsequent 200-m race pace swimming. Although performance on a 200-m time-trial was similar, this could result in increased performance on subsequent and repeated trials where elevated  $[\text{lac}^-]_B$  are known to reduce swimming performance since elevated  $[\text{lac}^-]_B$  have been shown to impair subsequent swimming performance (5,6,9). Accordingly, we recommend that the generic guidance provided to athletes is revised. To make the transition to the applied practitioner, the authors acknowledge a need to repeat this process in line with the athletes training cycle, adopting this approach will account for changes in the  $V_{LT}$  throughout a periodised training programme and ensure that the recovery strategies are specific and relative to an individual's current training status.

## **CONCLUSION**

Findings here provide support for the use of active recovery strategies that are conducted at an individual velocity corresponding to the lactate threshold. Although this approach appears not to be associated with immediate performance improvements, the data suggests that lactate concentration was reduced to a larger extent during subsequent exercise compared with protocols conducted at lower intensities. This could have important implications within a competition setting where multiple bouts of intense exercise are required with short recovery periods.

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## Integrity of Research and Reporting

### Ethical Standards

All experimental procedures and methods of assessment used in this study were ethically approved by the host universities ethics committee and conform to the laws of the United Kingdom.

### Conflicts of interest:

No conflicts of interest for each of the authors.

### Funding:

None

## TABLES

Table 1: Physical characteristics of the elite age group swimmers (N=15).

	Males (N=6)	Females (N=9)
Age (years)	16.7 ± 1.9	15.2 ± 1.0
Height (m)	1.80 ± 0.12	1.68 ± 0.07
Body mass (kg)	70.7 ± 10.8	61.6 ± 6.8
Body fat (%)	10.6 ± 2.0	17.3 ± 2.8
200 m PB (mm:ss)	02:28 ± 00:30	02:40 ± 00:20
Weekly Training Volume (m)	42,000 - 56,000	

Table 2: Physiological responses recorded during each recovery 200-m trial.

	Recovery Trial		
	V <sub>LT</sub>	COA	NGB
Time-trial (min <sup>-1</sup> )	2.24 ± 0.12	2.23 ± 0.14	2.22 ± 0.13
Stroke Rate (cycles·min <sup>-1</sup> )	47 ± 17	48 ± 18	48 ± 17
Stroke Length (m·cycle <sup>-1</sup> )	2.01 ± 0.71	2.00 ± 0.69	2.02 ± 0.70
HRmax (beats·min <sup>-1</sup> )	180 ± 17	177 ± 14	185 ± 9
Mean HR (beats·min <sup>-1</sup> )	135 ± 13	124 ± 17	126 ± 13
Max RPE	18 ± 2	18 ± 2	18 ± 2
Mean RPE (AU)	13 ± 5	13 ± 5	12 ± 5

V<sub>LT</sub> Cool down recommended relative to the velocity at lactate threshold, COA cool down prescribed by the coach of the group, NGB cool down prescribed by the National Governing Body, <sup>A</sup> Different to COA, <sup>B</sup> Different to NGB.

## FIGURE CAPTIONS

**Figure 1** Mean change in  $[\text{lac}^-]_{\text{B}}$  concentrations ( $\text{mmol}\cdot\text{l}^{-1}$ ) during each recovery protocol. A different from baseline, B different from COA, C different from  $V_{\text{LT}}$ , D different from NGB, \* less than peak lactate concentration.