

# Journal of Undergraduate Kinesiology Research

Official Research Journal of the Department of Kinesiology  
University of Wisconsin – Eau Claire

Volume 3 Number 2 May 2008

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## THE ROLE OF ACIDOSIS DURING MULTIPLE BOUTS OF HIGH-INTENSITY EXERCISE: SUBSEQUENT EFFECT OF RECOVERY IN ATTENUATING FATIGUE

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### ABSTRACT

**Chrismas, B.C.** The role of acidosis during multiple bouts of high-intensity exercise: subsequent effect of recovery in attenuating fatigue. *Journal of Undergraduate Kinesiology Research* 2008;3(2):1-18. **Purpose:** The etiology of fatigue following multiple bouts of high-intensity exercise and the subsequent limitations on exercise performance remains a highly contentious and controversial phenomenon. One important factor that could augment performance is the type of recovery. Therefore, the purpose of this study was to examine the effects of active recovery (*AR*) vs. passive recovery (*PR*) on blood acidosis during multiple bouts of forearm-wrist-flexion. **Methods:** Eight healthy, moderately active subjects, 3 male and 5 female (age,  $21.15 \pm 2.12$  y, height,  $168.13 \pm 7.38$  cm, weight,  $66.13 \pm 9.45$  kg) completed two multiple forearm-wrist-flexion trials in random order (*AR* and *PR*) separated by one week. All subjects completed three maximal exercise bouts with a 5 minute (min) *AR* or *PR*. **Results:** Following appropriate checks on underlying assumptions a two way repeated measures analysis of variance (*ANOVA*) was used to compare *AR* vs. *PR*. Statistical significance was set at  $p < 0.05$ . Lactate (*La*) removal was greater following *AR* ( $p = 0.001$ ). However, there was no significant difference between *pH* ( $p = 0.555$ ) or time to exhaustion (*TTE*) ( $p = 0.789$ ). **Conclusion:** Despite *AR* improving the removal rate of *La*, it did not improve *TTE*, therefore although acidosis may contribute to fatigue it appears there are multiple factors responsible for limiting exercise capacity. Due to the sporadic nature of current competitive sports it may be necessary to establish multiple recovery protocols reflective of the sport's individual characteristics.

**Key Words:** Lactate, *pH*, Fatigue, Recovery, Time To Exhaustion

## INTRODUCTION

The etiology of fatigue remains a highly contentious and controversial phenomenon (1,2). Multiple factors responsible for limiting exercise capacity can be found within the literature (3). However, identifying the appropriate mechanisms associated with fatigue has proved problematic (4). The relative contribution of such factors may depend on the mode, duration and intensity of the exercise protocol employed (5). Despite exercise physiologists endeavoring to establish the role of acidosis in limiting exercise capacity, this notion remains equivocal (6).

Central and peripheral causes of fatigue are at the forefront of current research (7). The majority of research indicates that peripheral changes (e.g. acidosis) are predominately responsible for limiting exercise capacity (8). Nevertheless, more recent research has suggested that the central nervous system (*CNS*) determines an individual's exercise capacity, regulating homeostasis and consequently determining fatigue (9). Contrastingly, multiple factors supporting the concept of a 'peripheral governor' as opposed to a 'central governor' are more prominent within the research. Numerous studies examining the effects of acidosis on exercise capacity have suggested that increased  $H^+$  concentration, due to decreased  $pH$  (3,12), increased  $La$  accumulation (15), increased  $P_i$  accumulation (13) and decreased muscle and blood buffering capacities (12,15) cause fatigue.

Recovery following multiple bouts of high-intensity exercise is fundamental to attenuate the effects of fatigue (e.g. acidosis) and therefore augment subsequent exercise performance (10). The effectiveness of the recovery in augmenting subsequent performance is dependent on a number of factors including the duration and intensity of the initial exercise and more importantly the mode, duration and intensity of recovery (5,10).

The concept that active recovery (*AR*) improves subsequent exercise performance compared to passive recovery (*PR*) remains equivocal (10). Predominately, research has focused on single bouts of exercise (11) with limited research examining recovery following multiple bouts of exercise (5,10). However, multiple, intermittent bouts of high-intensity exercise are more common in competitive sport (14) and therefore require further investigation. Limited research has investigated the effects of fatigue on localized muscle mass (16). Exercise physiologists may be unaware of the effects of fatigue and more importantly acidosis on small muscle mass. It is well documented that there exists differences between intra-muscular and whole blood values of  $pH$  (17). Therefore, it would be interesting to discover whether an individual is able to achieve acidosis and thus decrease  $pH$  to a level typically associated with whole body fatigue. Additionally, a comparison of recovery techniques (i.e. *AR* vs. *PR*) on small muscle mass could enable researchers to discover whether recovery following acidosis effects subsequent exercise performance.

Therefore, the aim of this study is to 1) examine the effects of *AR* v *PR* on acidosis, during multiple bouts of forearm wrist flexion, 2) establish whether *AR* improves subsequent exercise performance and 3) compare the differences if indeed there are any, between localized and whole body fatigue as determined by increased  $H^+$  concentration on subsequent recovery. The hypothesis to be tested is that there will be a significant difference between  $La$ ,  $pH$ , bicarbonate ( $HCO_3^-$ ) and base excess (*BE*) during *AR* and *PR*. Consequently, it is hypothesized that *AR* will augment subsequent exercise performance and therefore time to exhaustion (*TTE*) will be longer.

## METHODS

### Subjects

Eight healthy, moderately active subjects, 3 male and 5 female (age,  $21.15 \pm 2.12$  y, height,  $168.13 \pm 7.38$  cm weight,  $66.13 \pm 9.45$  kg) from the University of Hull completed a maximal (max) test followed by two exercise trials (AR and PR) in random order separated by one week. None of the subjects were trained specifically in sports involving extensive use of the forearm wrist flexion musculature. The study protocol was approved by the department's ethics committee. All testing was completed at the University of Hull physiology laboratory and conducted at sea level.

### Instrumentation



**Figure 1: Hand-made arm ergometer used for multiple forearm-wrist flexion.**

### Procedures

#### Initial consultation

Subject orientation began with an initial discussion of the study protocol and completion of the institutionally approved informed consent and medical questionnaire. All subjects were informed of the procedures and possible risks and side effects, and excluded on the basis of self-reported, pre-existing medical conditions contraindicative to the study's testing regimen. A standardized diet and exercise recording form was given to all subjects to record food and exercise undertaken 24 hours prior to testing. Following the first trial a copy of this form was handed to the subjects in order for them to duplicate, where possible, the same exercise and diet regime for the next trial. Furthermore, subjects were told to avoid spicy foods, caffeine, high-intensity exercise and alcohol 24 hours prior to the trials. All subjects were informed that they could withdraw from the study at any point without giving reasons. Anonymity and confidentiality was ensured.

### *Familiarization*

Prior to exercise testing the subject's height was measured using the Holtain Stadiometer (Holtain Ltd, Crymych, Dyfed) and mass was measured using the SECA balance scales (Vogel & Halke, Hamburg, Germany) which were calibrated prior to use following manufacturer's guidelines. In order to increase the validity and reliability of the study all subjects completed a familiarization trial, replicating the exercise protocol and blood sampling methods (as detailed below).

### *Maximal test*

Incremental,  $\max$  forearm-wrist-flexion was completed to exhaustion, at a rate of approximately 1 contraction every 2 seconds ( $s$ ). In order to measure forearm-wrist-flexion a hand-made arm ergometer was used (Figure 1). The arm ergometer was attached to a table and subjects exercised in the seated position. Subjects placed their exercising forearm into the ergometer and took hold of a bar. Once the subjects were sat in a comfortable position their arm was strapped into place in order to ensure the forearm musculature was responsible for the movement. The handle of the ergometer which the subjects were holding was connected by rope to a weight cradle ( $1\text{ kg}$ ). Subjects were instructed to touch both the downright and upright wooden blocks. A metronome was set to the exercising speed in order to ensure reliability. Incremental ( $0.5\text{ kg}$  every  $\text{min}$ ) forearm wrist flexion was completed until exhaustion. At the point of fatigue ( $TTE$ ) peak weight ( $PW$ ) was recorded.  $TTE$  was measured using a stop watch and the same tester did this throughout all trials to increase the reliability of results. If the subjects failed to hit the upright and downright bar of the ergometer consecutively in time with the metronome the test was terminated and it was assumed that the subjects had reached exhaustion. Otherwise subjects stopped the test themselves when they could no longer continue, thus defining volitional fatigue.

### *Exercise testing*

All trials were randomized and performed at the same time on the same day due to the effects of diurnal variation (18). Subjects completed three  $\max$  exercise bouts with a 5  $\text{min}$  recovery (active or passive) against the  $PW$  obtained from the max incremental exercise bout. Forearm wrist flexion was performed as detailed above. Studies using shorter recovery durations (i.e.  $< 3\text{ min}$ ) were unable to elicit differences in proton removal rate (17). Therefore, it was suggested that optimal recovery duration would be 4-5  $\text{min}$  (19).  $AR$  consisted of forearm-wrist-flexion against a  $0.5\text{ kg}$  load. During  $PR$  subjects rested in the exercising position.  $TTE$  was recorded for all trials.

### Blood sampling

Blood gas and blood  $La$  samples were collected during all trials. A pre-exercise resting sample and draws during the last  $\text{min}$  of the recovery periods were collected via a capillary finger prick and venous sample. Methodological discrepancies, including differences in values obtained due to sampling site, have invalidated research findings (17). Therefore, both capillary and venous samples were used in this study, to increase the reliability of the results.

### *Blood gases*

Capillary finger prick blood gas samples were collected in a  $200\ \mu\text{L}$  blood gas capillary tube (Roche, Mannheim, Germany) using the Accu-check softclix lansett device (Roche, Mannheim, Germany). Venous blood samples were drawn from a superficial vein in the antecubital space using a  $3\text{ mL}$  blood gas syringe with calcium balanced lithium heparin BD A-line (BD UK Ltd, Oxford) and a 26G Terumo Neolus (Terumo UK Ltd, Surrey) needle. These were sampled using the AVL blood gas analyzer (AVL Graz, Austria) which was calibrated according to manufacturer's guidelines.

### Blood lactate

The Accu-chek softclix lansett device (Roche, Mannheim, Germany) was used to collect a finger prick 100 microlitre blood  $La$  sample. A microcuvette cb 300 (Sarstedt, Numrbecht, Germany), containing heparin to stop the blood clotting and fluoride to stop glycolysis, was used to collect the subject's blood. Venous blood samples were taken as above and then transferred into a microcuvette cb 300 (Sarstedt, Numrbecht, Germany). All samples were placed in the YSI2300 STAT (YS1 Inc, Yellow Springs, OH) for blood  $La$  sampling.

### Statistical Analyses

Results were analyzed using Microsoft SPSS version 14.0 and graphs were drawn up using Microsoft Excel 2003. Prior to obtaining inferential statistics, descriptive statistics were obtained in order to check that the data did not violate any underlying assumptions. Furthermore, tests for normality were carried out. Additionally, as two sampling sites were used, a paired sample t-test was conducted to discover whether any significant difference existed between capillary and venous blood samples. There was a significant difference between capillary finger prick and venous blood samples (Table 2) and therefore only venous samples were used for inferential statistical analysis. A two-way repeated measures analysis of variance (*ANOVA*) was used to compare  $La$ ,  $pH$ ,  $HCO_3^-$ ,  $BE$  and  $TTE$  during  $AR$  and  $PR$  for all three exercise bouts. Statistical significance was set at  $p < 0.05$ .

## RESULTS

### Pre-exercise data

Mean  $\pm$  standard deviation (*stdv*) were obtained for resting venous and capillary blood  $La$  and blood gas data (Table 1). A paired samples t-test was conducted to compare resting venous and capillary blood samples for  $La$ ,  $pH$ ,  $BE$  and  $HCO_3^-$ . There was a significant difference between venous and capillary blood samples (Table 2) and therefore only venous blood samples were used in further statistical analysis. Furthermore, in order to obtain the effect size  $\eta^2$  was calculated. The results show that a large effect size ( $>0.14$ ) was observed for all results.

**Table 1: Mean  $\pm$  (stdv) for resting blood capillary and venous samples for AR and PR.**

	Mean ( $\pm$ stdv) AR		Mean ( $\pm$ stdv) PR	
	Capillary	Venous	Capillary	Venous
La (mmol/L)	1.40 $\pm$ 0.27	0.93 $\pm$ 0.19	1.08 $\pm$ 0.44	0.88 $\pm$ 0.23
Ph	7.40 $\pm$ 0.00	7.35 $\pm$ 0.03	7.39 $\pm$ 0.01	7.35 $\pm$ 0.03
BE(meq/L)	-0.03 $\pm$ 1.49	1.21 $\pm$ 1.45	-0.29 $\pm$ 1.28	0.93 $\pm$ 1.10
HCO <sub>3</sub> (mmol/L)	24.6 $\pm$ 1.72	27.53 $\pm$ 2.55	24.6 $\pm$ 1.63	27.45 $\pm$ 1.81

**Table 2: Paired samples t-test results for resting blood capillary and venous samples for AR and PR.**

	Paired t-test		eta <sup>2</sup>	
	AR	PR	AR	PR
La (mmol/L)	P < 0.01**	P = 0.10	1.31	1.01
Ph	P = 0.04*	P < 0.01**	1.67	1.52
BE(meq/L)	P < 0.01**	P = 0.01*	0.85	0.79
HCO <sub>3</sub> (mmol/L)	P = 0.01*	P = 0.01*	0.84	0.81

\*significant at the p &lt;0.05 level

\*\*significant at the p &lt;0.01

Prior to using the two-way repeated measures *ANOVA* a paired sample t-test was conducted to compare the difference between the resting venous blood samples for both *AR* and *PR*. Resting venous blood samples for *La*, *pH*, *BE* and *HCO<sub>3</sub><sup>-</sup>* for *AR* and *PR* were not significant (Table 3) and therefore the data did not violate any assumptions. In addition calculating *eta*<sup>2</sup> revealed a small effect size <0.06 for all results.

**Table 3: Paired samples t-test for resting venous blood La and blood gas samples between AR and PR.**

	Paired t-test	eta <sup>2</sup>
La (mmol/L)	P = 0.68	0.03
Ph	P = 0.86	0.00
BE(meq/L)	P = 0.49	0.05
HCO <sub>3</sub> (mmol/L)	P = 0.93	0.01

Subject descriptives

Mean descriptive data including age, height and mass were calculated for all subjects prior to testing in order to check that the data did not violate any underlying assumptions.

**Table 4: Mean ± (stdv) of subject's age, height and mass.**

Descriptives	Mean (± stdv)
Age (years)	21.15 ± 2.12
Height (cm)	168.13 ± 7.38
Mass (kg)	66.13 ± 9.45

Maximal testing

Preceding the exercise trials all subjects performed a max forearm-wrist-flexion test to obtain *PW* and *TTE*. Mean *PW* and *TTE* data was calculated from the data obtained.

**Table 5: Mean ± stdv for PW and TTE during max testing**

	Mean (± stdv)
Peak weight (kg)	5.56 ± 1.35
Max TTE (s)	593.38 ± 189.14

Recovery

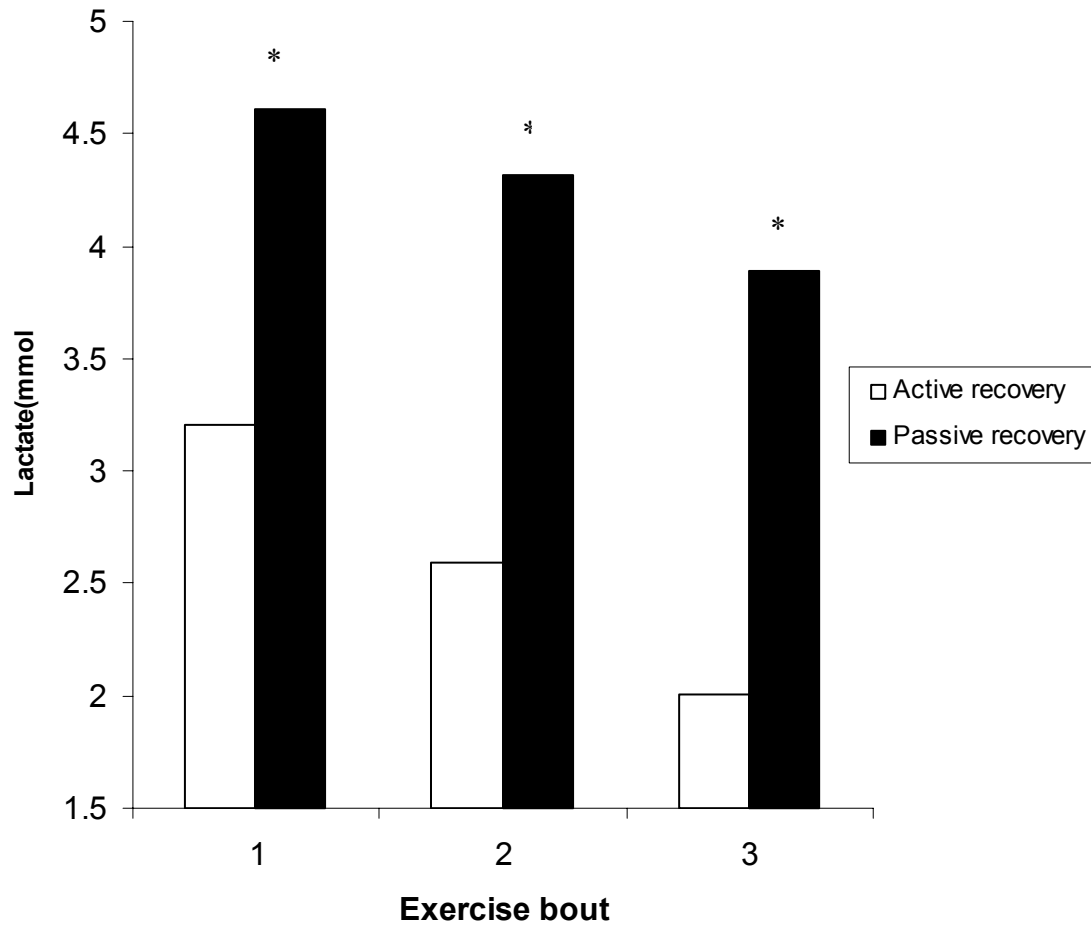
A two-way repeated measures ANOVA was conducted to compare  $La$ ,  $pH$ ,  $HCO_3^-$ , and  $BE$  during  $AR$  and  $PR$  for all three exercise bouts. There was a significant difference between  $La$ ,  $BE$  and  $HCO_3^-$  at the  $p < 0.01$  level. However, no significant difference was observed during recovery for  $pH$  (Table 6).

**Table 6: Two way repeated measures ANOVA results used to compare  $La$ ,  $pH$ ,  $HCO_3$  and  $BE$  during  $AR$  and  $PR$ .**

	p	f	eta <sup>2</sup>	Observed power
$La$ (mmol/L)	$P < 0.01^{**}$	15.12	0.69	0.87
Ph	$P = 0.56$	0.07	0.00	0.008
$BE$ (meq/L)	$P < 0.01^{**}$	14.39	0.67	0.9
$HCO_3$ (mmol/L)	$P < 0.01^{**}$	16.73	0.71	0.94

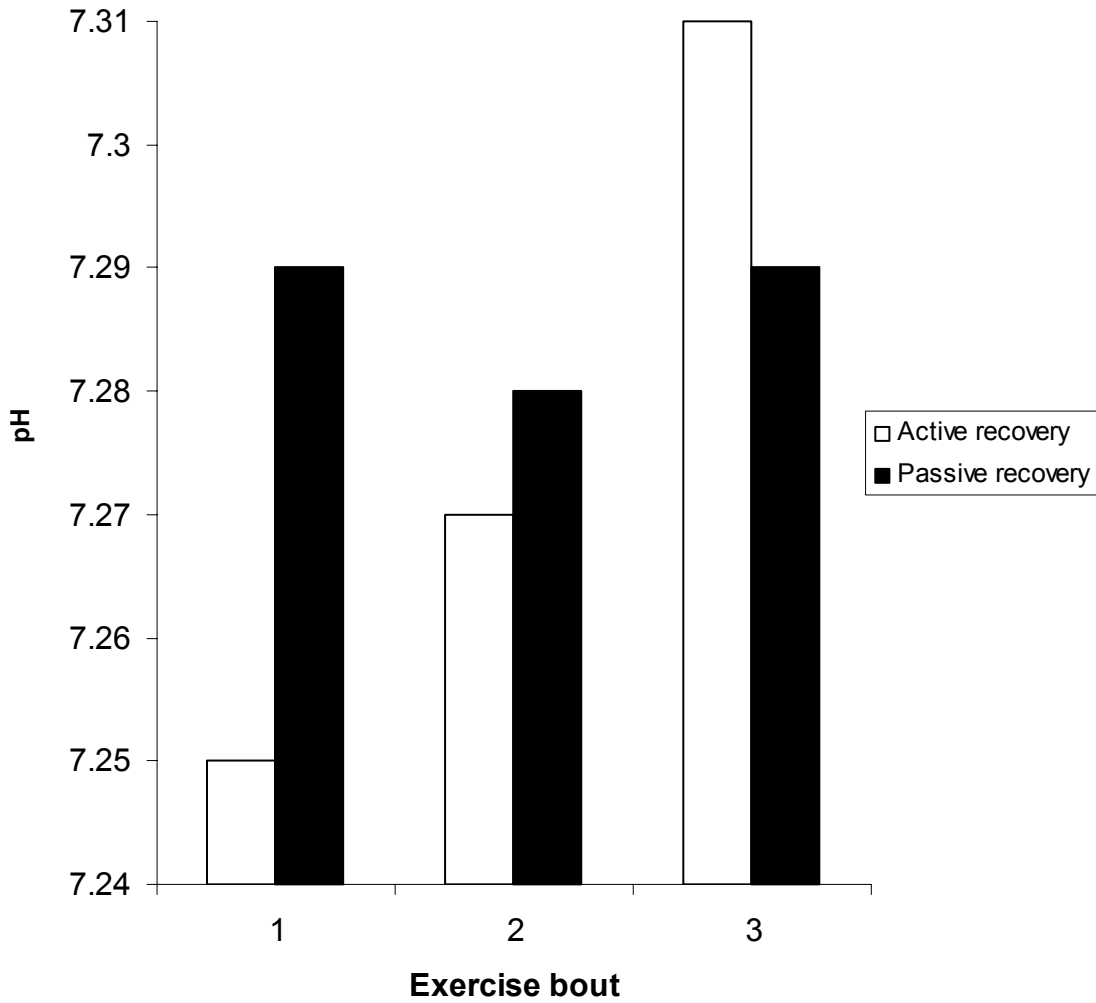
\*\*significant at the  $p < 0.01$  level

Graphs were drawn up for  $La$  and  $pH$  to compare the difference between all 3 exercise bouts during  $AR$  and  $PR$ .

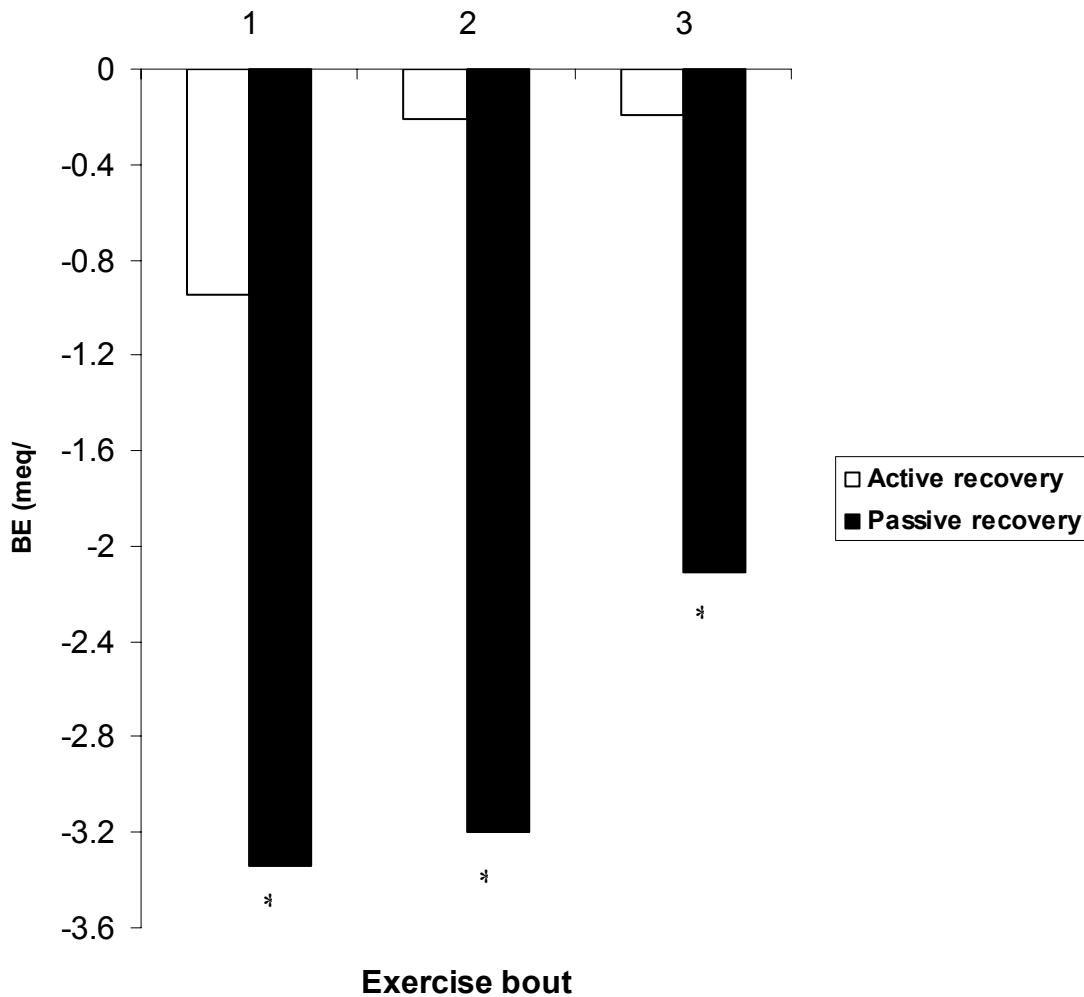


\* significant at the  $p < 0.05$  level

**Figure 2: Mean La concentration during the last minute of recovery for all three exercise bouts for AR and PR.**



**Figure 3: Mean Ph concentration during the last minute of recovery for all three exercise bouts during AR and PR.**

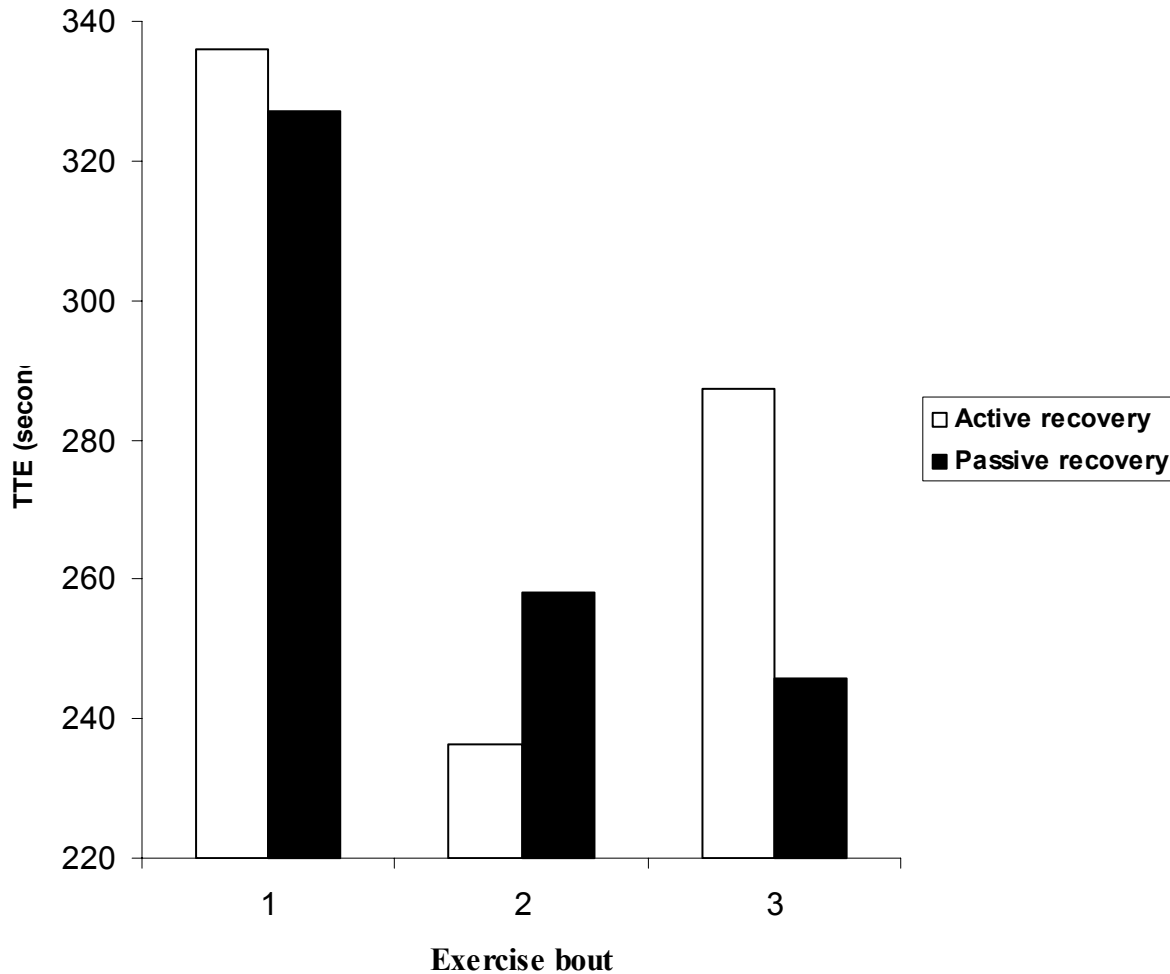


\* significant at the  $p < 0.05$  level

**Figure 4: Mean BE during the last minute of recovery for all three exercise bouts during AR and PR.**

Performance

A two-way repeated measures *ANOVA* was performed to compare *TTE* for all three exercise bouts during *AR* and *PR*. There was no significant difference in performance between recovery protocols ( $p = 0.789$ ,  $f = 0.08$ ,  $\eta^2 = 0.01$ , observed power 0.06). A graph was drawn up for *TTE* to compare the differences between all 3 exercise bouts during *AR* and *PR*.



**Figure 5: Mean TTE during AR and PR for all three exercise bouts**

## DISCUSSION

In support of the present study's hypothesis, there was a significant difference between  $L_a$  ( $p=0.009$ ),  $BE$  ( $p=0.007$ ) and  $HCO_3^-$  ( $p=0.005$ ) during  $AR$  and  $PR$ . However, in contrast to the hypothesis, despite a significant increased removal rate of  $L_a$  during  $AR$  this did not improve  $TTE$  ( $p=0.789$ ). Furthermore, there was no significant difference in  $pH$  ( $p=0.555$ ) between  $AR$  and  $PR$ . Therefore,  $AR$  did not appear to improve subsequent exercise performance.

### *The effects of $L_a$ during recovery from high-intensity exercise*

Figure 2 indicates that there was a significant decreased accumulation of  $L_a$  during  $AR$ . In related work, numerous studies support these findings and suggest that  $AR$  improves  $L_a$  removal (16,19,20,21,22,). Additionally, one study using an identical recovery period (5 min) to this study, found a decrease in  $L_a$  following  $AR$  (23). During  $AR$  there is an increased  $BF$  to the skeletal muscle increasing  $O_2$  delivery and enhancing proton removal via its transportation to slow twitch muscle fibers (24) decreasing metabolite accumulation and increasing the resynthesis of  $ATP$  (1). However, research remains equivocal as to whether this improved removal rate increases subsequent exercise performance (2).

In contrast to the findings of this study (Table 6), other research has suggested that *AR* does not improve the removal rate of *La* (16,25). It has been suggested that at least 10 min of *AR* is required in order to observe a decrease in *La* concentration (16, 25). However, these studies used shorter exercise and recovery periods < 4 min and 3 min and 15s and 3 min respectively. This suggests that exercise and recovery duration and intensity may affect the removal rate of *La*.

Blood *La* accumulation decreases between multiple bouts of high-intensity exercise (26). However, it is unclear as to whether this is a reflection of increased distribution volume of *La* (26) or an indication that acidosis has occurred (27). Nevertheless, Figure 2 highlights the significant reduction during *AR* and indicates that *La* concentration remained elevated during *PR*. This may suggest that during *AR* there was an increase in the uptake of *La*, as opposed to a reduction in the production (27). During *PR* the muscle may not be able to remove the accumulated *La* at a rate quicker than its production, potentially a result of a decrease in *BF* resulting in proton accumulation and the onset of acidosis. Contrastingly, the reduction in *La* concentration may simply be a reflection of increased volume of distributed *La*. If we assume that acidosis did occur, the results suggest that either *AR* does not attenuate the effects of acidosis due to the statistical insignificance of *TTE* ( $p=0.789$ ) (Figure 5), or that other factors alongside acidosis are responsible for limiting exercise capacity. Furthermore, peak *La* concentration can increase up to 25 mmol/L in plasma following exercise to fatigue (2). The highest *La* obtained in the present study was 7.24 mmol/L, with the majority of *La* values significantly lower (data not shown). Hence, this may suggest that subjects did not reach exhaustion in the present study and thus acidosis did not occur. In contrast, the inability to accumulate *La* may have been a reflection of the smaller muscle mass utilized. Plasma *La* values of 25 mmol/L are typical of whole body values (2) and therefore differences may have been expected.

Furthermore, *La* concentrations may vary depending on the sampling site. The differences in *La* concentrations referred to above could be due to discrepancies in sampling methods. Wide variations in the sampling methods used are evident within the literature (26). The results of the present study found a significant difference between capillary and venous blood *La* samples (Table 2). Similarly, decreased capillary blood *La* concentrations compared to femoral vein have been observed (26). In contrast a comparison between *NMR* and venous blood *La* measurements found no differences between conditions (28). Nevertheless, the authors suggested that inherent limitations in infrequent sampling associated with muscle biopsies could account for discrepancies in the results (28). In respect to the measurements used in the present study, it has been suggested that blood *La* measurements may not be reflective of intra-muscular activity. For example, a relatively small concentration of *La* is released into the blood (26). It is therefore suggested that exercise physiologists should not rely exclusively on blood *La* measurements as a direct indication of acidosis. The aforementioned inconsistencies and potential problems associated with the respective sampling measurements raise methodological issues in respect to inter-study comparison. Therefore, it is essential that exercise physiologists consider these differences in sampling methods when comparing and contrasting their research findings.

Regardless of these findings, the increased removal rate of *La* did not improve subsequent exercise performance in the present study (Figure 5) and therefore, the measurement of *La* as a performance indicator may be negligible (10). These results are either attributable to inconsistencies with sampling methods or they support the recent concept that *La* does not inhibit exercise performance (6). Subsequently, it has been suggested that *La* is actually beneficial as it may be utilized by other muscle fibers as a fuel for energy (2). These recent developments have led to the notion that acidosis may actually be ergogenic. Although this concept remains highly contentious, they appear to suggest that other metabolic factors could be responsible for limiting an individual's exercise capacity.  $H^+$

and  $P_i$  accumulation, resulting from an increase in  $ATP$  hydrolysis, may be responsible for inhibiting exercise capacity (4). Increased  $H^+$  concentration has been found to inhibit phosphofructokinase ( $PFK$ ) in particular in smaller muscle mass (2) and therefore this may have caused fatigue in the present study. The methodology of the present study prevented the measurement of intra-muscular metabolite accumulation and subsequent depletion of  $ATP$  and therefore the causes of fatigue can only be speculative. However, decreased  $pH$  causes a subsequent increase in accumulated  $H^+$  and  $P_i$  and therefore can, potentially, be used as an indirect indication of inhibited muscular contractile processes and increased  $ATP$  hydrolysis (28).

#### *The effect of AR vs. PR on pH following high-intensity exercise*

In contrast to the hypothesis, no significant difference in  $pH$  between  $AR$  and  $PR$  was found in the present study (Table 6). In light of these findings it has been suggested that fatigue is reached at varying  $pH$  levels, therefore indicating that  $pH$  does not directly contribute to fatigue (3,29). However, obtaining blood  $pH$  may not be a true reflection of intra-muscular  $pH$  (29). Intra-muscular  $pH$  concentrations are typically more acidic than blood  $pH$  (30) and hence a more true reflection of intra-muscular activity. Nevertheless, differences in  $pH$  could simply be attributed to differences in training status (14) and thus caution must be taken when interpreting findings.

Additionally, variation in the reported concentrations of  $pH$  within the literature may be due to differences in muscle fiber type (2). Type II fibers have a greater capacity to induce acidosis due to their inability to resynthesise  $ATP$  and thus they fatigue quicker. Forearm musculature contain a majority of Type II fibers and therefore should be sufficient to reduce  $pH$  to levels typically associated with acidosis (2). Reported whole body intra-muscular values of  $pH$  are  $\sim 6.2$  following fatigue (17), whereas reported blood  $pH$  values are  $\sim 7.1$  (29). The blood  $pH$  values observed in this study ranged from 7.16-7.38 during  $AR$  and 7.21-7.38 during  $PR$  (data not shown), suggesting that forearm musculature may not have been sufficient to decrease  $pH$  to levels typically associated with fatigue. Furthermore, irrespective of the decline in  $pH$  observed in the present study, this did not affect subsequent exercise performance (Figure 5). In support of these findings research has suggested that decreased  $pH$  is not a significant factor in fatigue (30).

Limited research has examined the effects of fatigue in localized muscle mass and therefore further research is required. The ability to decrease  $pH$  may simply be a reflection of training status and therefore may not accurately reflect the causes of fatigue. In addition, low statistical power could explain the statistical insignificance for  $pH$  (Table 6) in the present study. Furthermore,  $pH$  can be affected by  $PCO_2$  and sampling method and consequently may not be an accurate reflection of acidosis (2). Examining the effects of  $BE$  during recovery may be a more accurate measure of acidosis (30).

#### *BE as an indication of acidosis*

$BE$  has been classically considered as the 'gold standard' measure of acidosis (30). However, limited studies have examined the effects of  $BE$  with the majority of research using  $pH$  as an indication of acidosis. Figure 4 indicates that  $BE$  was significantly more acidic during  $PR$  suggesting that an increased amount of base was required in order to maintain a homeostatic  $pH$ . The addition of  $H^+$  into the bloodstream is accompanied by a drop in  $BE$  (31). Therefore, during  $PR$  it is suggested that there was an increase in accumulated  $H^+$ , demonstrating that  $PR$  is not as efficient as  $AR$  in

removing accumulated protons. However, this increase in  $H^+$  removal rate during *AR* measured indirectly via *BE* did not improve *TTE* in the present study (Figure 5) and may suggest that acidosis is not the main contributing factor of fatigue. Contrastingly, this could suggest that due to the smaller muscle mass utilized in this study the forearm wrist flexor musculature was able to cope with the exercise demand due to an enhanced buffering capacity (i.e.  $HCO_3^-$ ), maintaining a more homeostatic *pH* throughout the exercise bouts (Figure 3). However, these results are equivocal and further research is required in order to measure the effects of recovery following high-intensity exercise using *BE* as a measure of acid base balance.

#### Effects of recovery mode on subsequent exercise performance

In the present study, no significant difference was found for *TTE* between *AR* and *PR* (Figure 5). Therefore, the results of this study suggest that recovery mode does not effect subsequent exercise performance following multiple bouts of forearm wrist flexion to fatigue. Similarly, in light of these findings other research has suggested that *AR* does not improve exercise performance (10,11) and may actually decrease subsequent exercise performance (5,13,14,29). During *AR* mitochondrial *ATP* production is utilized to maintain muscle activity, therefore increasing the energy demand placed on the exercising muscle (32). *AR* may not be beneficial following high-intensity exercise as it inhibits the reoxygenation of myoglobin and *PCr* resynthesis (5). This could explain why no significant difference was observed in *TTE* between *AR* and *PR* (Figure 5). Too high an intensity during *AR* may result in further fatigue (33). Recovery intensity and duration were standardized in the present study for all subjects irrespective of *PW* and *TTE* obtained in the max test. Therefore, intensity of the *AR* for a number of subjects may have been too high, which could have accounted for the statistical insignificance of *TTE* ( $p=0.789$ ). Hence, research remains equivocal as to the effects of recovery following different duration and intensities of exercise. *AR* may increase performance following short-duration, high-intensity exercise (34) but not following exercise lasting  $> 5$  min. Nevertheless, differences in results may simply be a reflection of flawed and inconsistent methodologies.

Contrastingly, *AR* has improved subsequent exercise performance following multiple bouts of exercise lasting 6s combined with 30s of recovery (35). Subsequently, a 4 min *AR* improved exercise performance following 30s of high-intensity exercise compared to *PR* (34). This would appear to suggest that the longer the exercise duration the longer the recovery period required. This could explain why no difference in *TTE* was observed during the present study (Figure 5). The recovery period of 5 min used in the present study was based on previous research (19). A recovery period of 4-5 min has been suggested as an 'optimal' duration in order to improve performance. However, the exercise protocol used by Draper et al. may have been specific to climbing and therefore, in the present study, despite an improved removal rate of *La*, the 5 min recovery did not significantly improve *TTE* (Figure 5). *AR* lasting  $> 20$  min may be necessary to prevent decrease in subsequent performance (16).

Nevertheless, Figure 5 suggests that *TTE* was increased during the first and last exercise bout during *AR*. In contrast, during *PR*, *TTE* decreased throughout the three exercise bouts. The lack of statistical significance observed for *TTE* ( $p=0.789$ ) could therefore be due to reduced statistical power (observed power=0.06) and/or small sample size ( $n = 8$ ). Hence, it could be suggested that with a larger sample size *TTE* would be significantly longer during *AR*. However, the evidence for this is only circumstantial and requires further investigation. There is no physiological evidence to support this finding and this increase could have been due to methodological inconsistencies.

Although the majority of research advocates the use of *AR* following high-intensity exercise, it can be argued that *PR* may be more effective in attenuating the effects of fatigue and thus augmenting

subsequent performance. *PR* may improve performance following multiple bouts of high-intensity exercise (6). During high-intensity exercise *ATP* and *PCr* resynthesis are essential in order to continue to exercise (36). Failure of muscle recruitment and contraction processes due to decreased *ATP* and *PCr* resynthesis resulting in an increase in *ADP* may be a major factor limiting exercise performance (3). Therefore, if *PR* increases an individual's ability to generate *ATP* and *PCr* it may attenuate the effects of fatigue. However, the results of the present study do not appear to support this research (Figure 5). Studies incorporating short-duration, high-intensity exercise bouts (15.s) have found an improved performance following *PR* (5). Consequently, exercise duration could explain the inconsistencies in results. Hence care must be exercised when comparing results. Imposed experimental conditions utilized by Dupont, Blondel and Berthoin may have been insufficient to obtain an adequate decrease in *pH* to allow for acidosis to occur (2). Therefore, the shorter exercise period may have been the only reason that *PR* appeared to improve subsequent exercise performance. Additionally, intra-muscular measurements were not obtained in the present study and therefore caution must be taken when comparing results.

## CONCLUSIONS

The research examining acidosis and the subsequent effects of recovery on exercise performance remain controversial (1). However, the results of the present study, alongside previous research, have provided insight into the limitations of exercise capacity. Acidosis could indirectly contribute to inhibition of muscular contraction process nevertheless, this may only be one factor limiting exercise capacity (17). Fatigue may be specific to the research protocol employed. Subsequently, when investigating the causes of fatigue and effects of recovery on exercise performance it is essential to maintain a multi-dimensional approach (3). In addition, novel research criticizing the previous role of *La* appears to be supported by evidence in this study (6). However, wide variation in the study protocol employed alongside methodological inconsistencies in sampling methods could explain these findings.

Limited research has examined the effects of acidosis and recovery on smaller muscle mass. Statistical insignificance observed for *pH* and *TTE* could support the assumption that acidosis does not contribute directly to fatigue (28). However, the statistical insignificance could have been due to low statistical power and therefore the results of the present study remain inconclusive.

Nevertheless, the present study appears to support the assumptions that fatigue is caused by a multiplicity of factors that may be dependent on the type of exercise and recovery protocol employed (3). However, caution must be taken when interpreting research findings due to the inherent differences in study protocols employed (19).

## Limitations

The use of only in *vitro* measurements (i.e. blood sampling) as opposed to in *vivo* measures (i.e. *NMR*) may not have given an accurate reflection of acidosis (26). In addition, sampling time may have affected the results in the present study. Measurements were taken during the last minute of the recovery period and immediate changes in the concentration of the metabolites (e.g. *pH*) may have been missed. Measuring acid base kinetics (including  $Na^+$  and  $Cl^-$ ) may allow a more complete assessment of the effects of acidosis and allow exercise physiologists to gain further insight into the factors limiting subsequent performance following multiple bouts of high-intensity exercise (17).

In addition, the use of psychological measures alongside physiological measurements may have strengthened the results of the present study. Including psychological measures may have enabled a

more multi-dimensional analysis of fatigue in respect to the 'central governors' responsible for limiting exercise capacity (9).

Moreover, it could be suggested that the major limitation of the present study was the reduced statistical power due to a small sample size. Therefore, in order to increase the validity of future research it is important that exercise physiologists use a larger sample size, increasing statistical power.

#### Future research

Further research into the effects of fatigue following high-intensity exercise and the subsequent effect of recovery is clearly warranted. Based on the findings of the present study, supported by previous research, it is suggested that exercise physiologists need to maintain a multi-dimensional approach to analysis whilst reducing the number of confounding variables (4). Due to inherent differences in subject population demographics, sampling type and frequency, exercise mode, duration and intensity and subsequent duration, mode and intensity of recovery, inter-study comparison is increasingly problematic (14).

The etiology of fatigue in respect to 'central' and 'peripheral' governors remains a controversial phenomenon (9). Therefore, future research should focus on the various mechanisms associated with the above concepts. In order to accurately measure such mechanisms it is important that exercise physiologists use a variety of measurements (i.e. *NMR* and psychological measures). However, it is not expected that this phenomenon will be solved instantaneously. The mechanisms associated with fatigue and subsequent recovery are highly complex and it may take many more years and further advances in technological research to establish the major factors that limit an individual's exercise capacity.

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## ACKNOWLEDGEMENTS

I would like to thank my supervisor Dr Jason Siegler, for his expert guidance and knowledge relating to the writing of this manuscript. His assistance during the testing period is greatly appreciated as without his skills it would have not been possible to carry out the research.

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