BLOOD LACTATE REDUCTION AT THREE RECOVERY INTENSITIES FOLLOWING SEVERE ROWING EXERCISE

by

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Abstract

The purpose of this study was to observe the differences in rates of blood lactate reduction (BLR) at three recovery intensities (40% $V0_2max$, 60% $V0_2max$, and combined recovery) when subjects are highly trained and aerobically fit. Eight well-trained oarsmen (age = 23.2 yr, Ht = 189.6 cm, Wt = 85.3 kg, $V0_2max$ = 5.2 l / min or 61.6 ml / kg·min^{-1}) were tested in one pre-experimental procedure and three experimental treatments. The pre-experimental procedure involved the determination of $V0_2max$, and the loads at which 40 -, 50 -, and 60% $V0_2max$ occurred from a progressive load $V0_2max$. The three experimental treatments each involved three one minute maximal load intervals on the rowing ergometer to elevate blood lactate, followed by a 30 minute randomly assigned recovery on the rowing ergometer at either 40% $V0_2max$ (40R), 60% $V0_2max$ (60R), or combined recovery (CR). Blood samples, from an indwelling catheter placed in the cephalic vein, were taken at t=0, 1, 2, 3, 4, 5, 6, 9, 12, 15, 18, 21, 24, 27, and 30 min of recovery. Analysis of plasma samples revealed a mean resting blood lactate concentration ([Bla]) of 1.2 mM and a mean peak [Bla] following maximal exercise of 16.3 mM. ANOVA indicated that no significant differences occurred between the rates of lactate reduction for the three treatments ($p<.055$). With $p<.055$ and an effect size of eta=.31, further testing using a post-hoc multicomparison analysis revealed a significantly faster ($p<.05$) rate of BLR during the 60R treatment when compared to the rate of BLR for 40R. No further differences were revealed between any of the other comparisons (40R vs CR, or 60R vs CR). The significant differences between the rate of BLR for 60R compared to 40R may be due to the

subjects' high aerobic fitness, the specific nature of both their training and the recovery task, and physiological adaptations related to a high fitness level.

TABLE OF CONTENTS

Table of Contents cont.

LIST OF TABLES

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I Introduction

The accumulation of lactate in the body as a metabolite of muscular activity has been associated with both decreased intramuscular and blood pH (Fitts and Holloszy, 1976), resulting in a disruption of muscular and total body homeostasis (Graham, 1978). High levels of lactic acid and decreases in muscular pH eventually lead to muscular fatigue (Wenger and Reed, 1976). Resulting fatigue has been shown to be due to inhibition of glycolytic enzymes (Sutton et al., 1981) such as phosphofructokinase (Trivedi and Danforth, 1966) and lactate dehydrogenase (Karlsson et al., 1974) eventuating a shutdown of energy yeilding glycolysis. Lactate, at physiological concentrations, has also been shown to inhibit the release of free fatty acids (FFA) during exercise (Issekutz et al., 1975). "Consequently, it is of great importance to be able to remove the lactate and to restore the homeostasis of the body as soon as possible after exercise " (Hermansen et al., 1975, p. 101).

Early researchers (Jervell, 1928; Bang, 1936; and Newman et al., 1937) noted that during submaximal exercise, blood lactate concentration ([Bla]) consistently decreased towards the end of exercise. Further research has identified that, subsequent to heavy exercise, reduction of [Bla] is augmented by submaximal exercise during the recovery period (Gisolfi et al., 1966; Davies et al., 1970; and Hermansen and Stensvold, 1972). Although a number of studies have attempted to determine the optimal recovery intensity, there has been little agreement as to the most appropriate intensity. Agreement may only be with the observation that recovery exercise should not exceed the level of the anaerobic threshold (AnT) (Stamford et al., 1981; and McLellan and Skinner, 1982).

Comparisons of active recovery levels have, for the most part, attempted to analyze the differences among a series of single intensity work rates, although there seems to be evidence which points rather to the use of a combined intensity recovery to ultimately enhance the removal of lactate. Stamford et al. (1981) suggested a combined recovery protocol which would begin at an intensity above AnT and then drop well below AnT as the recovery continues. Testing this hypothesis, Dodd et al., (1984) observed that a high to low intensity, combined recovery protocol reduced [Bla] no differently than did that of a continuous low intensity recovery. As previously noted, recovery above AnT has been inappropriate for augmenting lactate reduction (Stamford et al., 1981; and McLellan and Skinner, 1982). Working above the AnT can only enhance lactate production not lactate utilization.

An alternate combined recovery may be proposed. Considering the elevated blood lactate concentration following severe exercise, the delivery rate of lactate to the active muscle tissue will be high, since Jorfeldt (1970) has shown that blood lactate concentration and lactate uptake by muscle is positively correlated. Therefore early in recovery a high intensity of exercise above AnT is not necessary. But as recovery continues and [Bla] decreases, increasing exercise intensity will proportionally increase the delivery rate of lactate to the active muscle (Guyton, 1982). Therefore, contrasting the combined recovery (CR) suggested by Stamford et al. (1981) and by staying below the AnT, blood lactate should be reduced at a higher rate. Whether this form of recovery is more appropriate than a continuous high intensity recovery is yet to be determined.

The inability to determine an optimal recovery intensity may be due to a myriad of factors. The amount of muscle mass (McGrail et al., 1978) and

the percentage of slow twitch fibres (Bonen et al., 1978 and 1979) involved in recovery execise, have to a certain degree, accounted for variations in rates of Bla reduction. It is also suggested that the level of fitness (Hubbard, 1973; and Weltman et al., 1977) and the training background (Evans and Cureton, 1983) of the individual may influence the appropriateness of a particular recovery intensity.

As mentioned above, a high intensity of exercise is shown to increase bloodflow to working muscles, while also increasing blood lactate delivery and uptake under conditions where the AnT is not exceeded (Stamford et al., 1981). Highly fit individuals when compared to less fit individuals are shown to have an AnT at a higher percentage of their $V0_2$max and as a result may be capable of recovering continuously at high percentage of their $V0_2$max while remaining fuel efficient, i.e., aerobic energy metabolism (McLellan and Skinner, 1982). By maintaining a high recovery intensity compared to a lower intensity, highly fit subjects will reduce [Bla] at a higher rate.

Therefore, the purpose of this investigation was to test two hypotheses with regards to a comparison of recovery intensities as they relate to the highly fit athlete. In training and competitive situations the achievement of an optimal rate of recovery from the fatigued state is of paramount importance to the highly fit athlete. More specifically the hypotheses of this investigation are:

1) That in highly fit individuals, a 60%$V0_2$ max recovery intensity will be significantly more effective in reducing [Bla] than a 40%$V0_2$ max recovery and a combined recovery, and

2) that in highly fit idividuals, a combined recovery will be more effective than a 40%$V0_2$ max recovery.

II Methods

Subjects

The reduction of lactate concentration in the venous blood, after maximal exercise, was determined during three recovery conditions: 40% $V0_2max$ (40R), 60% $V0_2max$ (60R) and combined recovery (CR). Ten male subjects, all highly trained oarsmen, began the experiment with the intent that each subject would complete one pre-experimental condition and three experimental treatments. The mean age, height and weight for these subjects were: 23.2 yrs, 189.6 cm and 85.3 kg respectively. The subjects were volunteers who responded to a request to the University of British Columbia Men's Varsity Rowing Crew for subjects. The final analysis includes the results obtained on eight of the 10 subjects. Two subjects were not included since one subject could not tolerate the duration of the treatment series, while analysis of the blood from a sample obtained from the second subject had revealed that during the 60R treatment, contamination of the blood had distorted the ability to measure accurately the concentration of lactate.

Permission to do this research was obtained from the Clinical Screening Committee for Research and Other Studies involving Human Subjects. Written informed consent was obtained from each subject prior to their participation in the study.

Pre-experimental Procedures

$V0_2max$, % $V0_2max$ (at various workloads), and heart rates were determined for each subject by means of their performance on a

continuous progressive rowing ergometer test. Workloads and heart rates corresponding to 40 -, 50 -, and 60 percent of the subjects maximal oxygen uptake (% $V0_2max$) determined from this procedure were used during the active recovery treatments of the experimental procedure.

The incremental maximal oxygen uptake ($V0_2max$) test was performed on a Gjessing rowing ergometer (Gjessing Ergorow, Bergen, Norway) which simulates the actual mechanics of rowing (Hagerman, 1984). The test involved a continuous work period with an incremental increase in workload. The initial workload began at 100 Watts (W) for the first two minute interval. The 100W workload was achieved by a one kilogram brakeload on the flywheel and 600 revolutions per minute (rpm) of the flywheel. Following the first interval and for each subsequent two minute interval, the workload was increased by 50W. These 50W increases in workload were brought about by an increase of 0.25 kg in load while still maintaining 600 rpm on the flywheel. Workloads were changed by sliding the weight on the lever bar to the next load level while the subject maintained their stroke rate at a level which would bring about 600 rpm of the flywheel. There was five equal increases in workload until the end of 10 minutes, at which point the subject was encouraged to increase stroke rate to a maximum, and the rpm of the flywheel as high as possible. In time with these increases, the load was increased to 3.25 kilogram meters (kgm) for one minute and then to either 3.50 kgm or 3.75 kgm for one minute or until volitional exhaustion or a plateau in $V0_2max$ was observed.

The volume of expired air (V_E), fraction of oxygen in expired air (F_EO_2), and the fraction of carbon dioxide in expired air (F_EC0_2) were measured every 15 s with an average calculated every minute. Heart

rate was measured at the end of every minute. Expired air was collected by means of a non-rebreathing mouthpiece, with gas samples passing through a low resistance pneumotachometer to measure ventilation. Samples from the expired air were analyzed by a Beckman 0_2 analyzer (OM - 14) and a Beckman Medical Gas LB -2 analyzer, measuring the 0_2 and $C0_2$ content of the expired air. These analyzers were interfaced with the Micro Nova System which ws programmed to display $V0_2$ (both in relative and absolute terms) every 15 s on a computer terminal in the testing laboratory. Known gas volumes and concentrations were used to calibrate all equipment prior to the $V0_2$ testing session.

Experimental Procedures

For each of the three experimental treatments, subjects completed three one minute maximal workloads on the rowing ergometer with one minute of passive rest after the first and second workload. These three minute workloads were selected, after consultation with a number of national team oarsmen and from personal experience while using the rowing ergometer, as a means to reach fatiguing levels quickly and also to elevate lactate levels. The maximal workloads were equivalent to the workload attained by each subject at $V0_2$max. These maximal efforts were used to elevate blood lactate levels.

Upon completing the third one minute interval the subjects performed a continuous active recovery for 30 minutes at one of either 40% $V0_2$max (40R), 60% $V0_2$max (60R) or combined recovery (CR) which included a continuous series of three 10 minute periods increasing in intensity from 40 to 50 to 60% $V0_2$max respectively until the end of the 30

minute recovery period. These recovery levels were selected as they represent the most common levels of recovery intensity which appear throughout the recovery literature (Davies et al., 1970; and Hermansen and Stensvold, 1972). Open circuit spirometry was used, as previously described, to monitor each subjects $V0_2$ throughout the recovery treatments. Every 15 s, $V0_2$ (both in l/min and ml/kg·min^{-1}) was displayed at a computer terminal in the testing laboratory. This direct feedback was used to aid in controlling the exercise intensity at the pre-assigned recovery $V0_2$. The sequence for the completion of the treatments by each subject was controlled by counterbalancing, which was randomly assigned prior to the experiment. Subjects were tested over a three week period with a minimum of three days and a maximum of four days separating each of the treatments.

Blood Sampling

Venous blood samples were drawn during each of the recovery treatments by means of either an 18 or 20 guage catheter inserted into the cephalic vein of the right arm. In one subject this procedure for catheterization was not used since vasospasm in the cephalic vein required the use of an alternate placement site into the radial vein. These sampling sites were used throughout the experiment. Both of these sampling sites provided an unrestricted flow of blood with minimal discomfort or impedence of the rowing motion at any intensity. A total of 16 samples of blood (1.5 ml/sample) were drawn for each treatment. Included in the 16 samples was a sample at rest, taken prior to exercise while the remaining 15 samples were taken during the

recovery period beginning at time 0 (end of the third one minute interval and the beginning of recovery) then at 1, 2, 3, 4, 5, 6, 9, 12, 15, 18, 21, 24, 27, and 30 minutes. Each sample of blood was drawn into a sterile syringe and was subsequently placed into a pre-marked and order sequenced vacutainer. These vacutainers contained sodium flouride which inhibits glycolytic intermediates (Lehninger, 1982) in the red blood cell (RBC). Infusion of normal saline was used to keep the cephalic or radial vein open and to clear the three foot intravenous extension tube of any blood from the previous sample. During the treatment period blood samples were stored on ice.

Plasma Analysis for Lactate Concentration

After all samples were collected they were centrifuged at 7,000 rpm for 10 minutes in a DAMON/IEC refridgerated centrifuge. At the end of 10 minutes the supernatant was separated from the RBC plug by micro-pipetting, and was then placed into a premarked storage tube. The RBC plug was discarded and the supernatant was then frozen at 20°C below zero. After all subjects had completed their three treatments, the samples were thawed and analyzed enzymatically in duplicate for lactate concentration according to the methods of Bergmeyer (1974) and McGrail et. al. (1978) with determinations made spectrophotometrically. Bergmeyer (1974) determined that there was a two percent reliability in repeated measures of lactate concentration from this method. During the analysis a standard lactate of a known concentration (5 mM) was included in the determinations, it was noted

that there was a three percent accuracy in this measurement. Sampling procedures were not measured for reliability.

Data Analysis

It is well established from lactate recovery research that the lactate recovery curve is a non-linear and exponential function (Hubbard, 1970; and Dodd et al., 1984). Therefore, this specifice exponential function:

$$y = ae^{-bt} + c$$

was chosen as its solution yields three meaningful parameters. In this equation *c* represents the resting baseline or asymptotic blood lactate concentration, *a* represents the lactate concentration above *c* at time zero, *e* is a constant, *b* represents the rate of blood lactate removal (BLR), and *y* represents the concentration of blood lactate at *t*, the time of each sample. This function was fit using a BMDP3R computer program for non-linear regression (Dixon, 1981), individually to the data collected from each of the subjects during all treatments. This procecdure resulted in three derived *b* values for each subject, one under each treatment condition. This data was then analyzed using a single factor anova with repeated measures; treatments was the independent variable while the rate (*b*) of lactate removal was the dependent variable. The single factor ANOVA with repeated measures was available from the BMDP2V computer program (Dixon, 1981). Statistical significance was accepted at the $p \leq 0.05$ level for all tests of significance. Effect size was determined to obtain a measure of the magnitude of the mean

difference among treatments, independent of the sample size. An effect size of 0.20 or greater is usually considered to exhibit a strong effect (Cohen, 1977).

III Results

Eight of the 10 subjects who were initially recruited, successfully completed this study. Table I contains the values for anthropometric (height, weight and age) and physiological ($V0_2max$, absolute and relative scores; and resting blood lactate concentration) measurements for each of the eight subjects. Results from two of the 10 participating subjects were excluded. In one case, the subject was unable to successfully complete all of the treatments and withdrew from the study. In the second case, the subject completed all treatments successfully, but enzymatic analysis of the plasma samples withdrawn during one treatment revealed contamination.

Observation of the $V0_2$ during each of the three recovery treatments indicated that the subjects performed close to their pre-selected level of intensity for the 40R, 60R, and CR treatments. The $\%V0_2max$ means of 41.5% ± 2.6 and 59.4% ± 4.5 were recorded for 40R and 60R respectively. The mean $\%V0_2max$'s achieved during the CR treatment were 44.3% ± 5.2, 49.2% ± 2.9, and 58.8% ± 3.0 respectively for each 10 minute period (40, 50, and $60\%V0_2max$).

Analysis of the plasma samples for resting blood lactate concentration revealed a mean resting concentration of 1.2 ± 0.5 mM. The mean resting [Bla] was considered to be within a normal range (.9 - 1.6 mM) for humans (Thews et al., 1985). Appendix C lists the blood lactate concentrations for each sample point during each treatment. Although there is consistency in the power output during the three, one minute workloads prior to each treatment (Appendix D), there are variations in the time to peak (Appendix C) and the measured level of

peak lactate concentration (Table II). Peaks in blood lactate concentration are shown to occur from (see Table II) zero to three

Table I Anthropometric and Physiological Data

Subject #	Height (cm)	Weight (kg)	Age (yr)	Maximal 0_2 uptake l/min	Maximal 0_2 uptake ml/kg.min^{-1}	[Bla] at Rest (mM)
S1	185.0	88.0	23	4.51	51.25	1.40
S2	186.6	86.6	24	4.61	53.22	0.77
S3	189.7	82.9	22	5.34	64.38	0.98
S4	199.9	96.5	20	5.30	54.92	1.20
S5	197.0	92.9	27	6.27	67.49	1.30
S6	184.0	78.0	23	5.98	76.67	1.40
S7	193.5	82.0	20	4.43	54.02	1.40
S8	179.4	75.9	27	5.40	71.15	0.94
Mean	189.6	85.3	23.25	5.23	61.64	1.20
± SD	± 6.9	± 7.0	± 2.7	± 0.68	± 9.6	± 0.25

minutes into recovery (mean peak blood lactate was 16.3 mM). Lactate concentration at the conclusion of the three one minute exercise bouts does not appear to be a function of the power output. The mean power outputs are consistent for each treatment as are the mean peak lactates for each treatment (Table II).

Table II Peak Blood Lactate [mM]

Subject	40R	60R	CR
S1	12.3	11.4	11.5
S2	14.8	15.0	16.6
S3	20.8	17.1	14.2
S4	14.1	17.0	19.5
S5	19.8	19.5	16.9
S6	20.9	22.4	18.5
S7	14.6	16.5	14.6
S8	12.7	16.5	14.6
Mean	16.3	16.9	15.8
±SD	±2.0	±3.2	±2.6

Initially all of the data were fit to curves, separately for each subject, using all concentration values beginning at time zero, the initiation of recovery. The goodness of fit (R^2) for the observed data was calculated for each set of data points. A number of low R^2 values (eg. $R^2 < 0.95$) calculated from the data indicated poor fits for subjects where peak values occurred after the initiation of recovery. Therefore the data were rerun using the peak values as the initial concentration point. The data prior to these peak values were ignored. The mean R^2 values for the peak value curves were 0.972 ± 0.02, 0.971 ± 0.01, and 0.971 ± 0.01 for 40R, 60R, and CR respectively, which was an improvement on the goodness of fit. An R^2 value of 0.97 or greater indicated that the data

points predicted by the computer program fit the observed data to a high degree. The rate of exponential decay of the curve represented by b, for blood lactate reduction for each treatment are found in Table III.

Table III Rates of Blood Lactate Reduction for Individual Treatments

Subject	40R	60R	CR
S1	-.017	-.051	-.019
S2	-.073	-.066	-.046
S3	-.040	-.074	-.039
S4	-.046	-.038	-.064
S5	-.015	-.074	-.066
S6	-.016	-.034	-.008
S7	-.051	-.054	-.037
S8	-.021	-.074	-.068
Mean	-.035	-.058	-.043
±SD	±0.021	±0.016	±0.022

The one-way ANOVA with repeated measures (Table IV) revealed no statistically significant differences between the rates of blood lactate reduction due to different recovery intensities ($F_{(2,4)} = 3.06$, $p<.055$). This value, although it is above the selected alpha level (0.05), clearly approaches significance. Determination of the effect size indicated that a large proportion of the total variability could be accounted for by the treatments (eta = .31), because of a large effect size and with $p < 0.055$, a

post-hoc multiple-comparison test using Newman-Keuls was implemented. The Newman-Keuls test revealed a significant difference between the 60R treatment and the 40R treatment ($p < .05$). No other comparison revealed a significant difference.

Table IV Source Table for one-way ANOVA with repeated measures

Source	Sum of Squares	df	Mean Square	F	Tail Probability
Subjects		7			
Treatments	0.00221	2	0.00111	3.60	0.055
Error	0.00431	14	0.00031		
Total	0.00652	23			

Further analysis of the data was considered through an attempt to determine the rate of BLR during both the initial and final 15 min of recovery. Comparisons were initiated with the intention of determining if there was a significant difference between each of the initial 15 min of all treatments and also if there was a significant difference between each of the final 15 min of all treatments. Although an attempt was initiated, there was not enough data points to achieve satisfactory curve fits.

Correlation Analyis

Correlation analysis was selected to examine the relationships among the dependent variable, the rate of blood lactate removal. The three r values for the correlations between 40R and 60R, 40R and CR, and 60R and CR, were r=0.06, r=0.16, and r=0.53 repectively. These

values were all very low and also non-significant. These findings may be due to the individual differences found in the rate of blood lactate reduction for these subjects and also possibly due to an error in blood sampling.

IV Discussion

Previous investigations (Gisolfi et al., 1966; Davies et al., 1970; Hermansen and Stensvold, 1972; and Belcastro and Bonen, 1975) have demonstrated that the rate of removal of lactate from the blood is enhanced with submaximal aerobic exercise, when compared to passively resting, following maximal exercise. This is due to the fact that active skeletal muscle (Jorfeldt, 1970; and Donovan and Brooks, 1983) is regarded as the major site of lactate removal. A number of other organs such as the heart (Griggs et al., 1966) and the splanchnic organs (Rowell et al., 1976) also metabolize lactate and are considered involved, to a lesser extent, in lactate reduction.

In the present study it was noted that, with continuous submaximal exercise, the lactate concentration in the blood steadily declined towards resting values. This is in accordance to observations of blood lactate concentration made by Jervell (1928) and Bang (1936). Lactate levels continued to decrease throughout the recovery period for all three treatments. A rest recovery was not compared to the three recovery treatments (40R, 60R, and CR), in this investigation, since it is well documented that all levels of exercise, which are below the anaerobic threshold, reduce blood lactate concentration at a higher rate than passive rest (Newman et al., 1937; Weltman et al., 1977; and Dodd et al., 1984).

Agreement upon the selection of a universally applicable recovery intensity appears to be non-existent (Gisolfi et al., 1966; Davies et al., 1970; Hermansen and Stensvold, 1972; Belcastro and Bonen, 1975; and Weltman et al., 1977). Although the ANOVA with repeated

measures for this study did not reveal a signifcantly different F ratio at the $p<.05$ level, the achieved p value, measured for the observed F ratio, was $p<.055$. This p value ($p<.055$) along with a relatively large effect size (eta=0.31) indicated that the treatments had affected the rate of reduction of blood lactate. Further testing under the above circumstances was considered justified. A Newman-Keuls post-hoc multicomparison test was implemented and indicated that there was a significant difference ($p<.05$) in the rate of [Bla] reduction between the 60R and 40R treatments.

This finding supports the stated hypothesis that in a group of highly fit individuals a 60% $V0_2max$ recovery intensity would be significantly more effective in reducing the lactate concentration in blood than would a 40% $V0_2max$ recovery intensity. These observations are in agreement with those of Hermansen and Stensvold (1972), in which a group of fit subjects ($V0_2max$ = 60 ml / kg·min^{-1}) attained the highest rate of lactate reduction during recovery at greater than 60% $V0_2max$. In contrast to these findings, Davies et al. (1970) and Belcastro and Bonen (1975) observed the highest rate of blood lactate reduction at intensities below 40% $V0_2$ max.

The post-hoc test did not reveal any significant differences between the other comparisons: 40R vs CR, and 60R vs CR. These results are in agreement with Dodd et al. (1984) who found no significant difference between a combined recovery and a continuous recovery. During the CR the initial 10 min averaged 44.3% $V0_2max$ rather than 40% $V0_2max$. The reason for the unexpectedly high percentage $V0_2max$ appeared to be due to the high intensity exercise which preceeded the initial 10 min of combined recovery. The small differences in $\%V0_2max$ (each 10 min period of the CR was 44.3-, 49.2-, and 58.8% VO_2max) can

be considered a limitation when attempting to sustain recovery at an accurate level. This alone could account for the CR being no more or less effective than either the 40R or 60R treatments. If 60R is only barely reaching significant difference from 40R then the CR (which tends to to be more like a 50% $V0_2$max recovery) has a rate of BLR which is between the 60R and 40R recovery.

A number of factors such as, the percentage of slow twitch fibres involved in recovery (Bonen and Belcastro, 1978; and 1979), the size of the muscle mass involved in recovery (McGrail et al., 1978), and the training and fitness status of the individual involved in recovery (Weltman et al., 1977; and Evans and Cureton, 1983), have all been suggested as possible reasons for these equivocal findings.

One of the major reasons the rate of lactate reduction was improved during the 60R treatment as compared to the 40R treatment, may be the aerobic fitness status of the group. Hubbard (1973) and Weltman et al. (1977) consider that the aerobic fitness status of an individual will reflect upon the appropriateness of a recovery intensity. In other words, an individual with a low aerobic capacity will recover most appropriately at a low percentage of their $V0_2$max, while an individual with high aerobic capacity will recover most appropriately at a higher percentage of their $V0_2$max. On closer inspection of recovery studies, an interesting trend appears, with regard to the relationship between a subject's aerobic power and their optimal intensity of recovery exercise. In studies where subjects are either on the low end of the aerobic power scale (< 50 ml / kg·min^{-1}) or where the subjects aerobic capacities vary over a wide range (≤ 50 ml / kg·min^{-1}) recovery exercise is found to occur at approximately 40% $V0_2$max (Gisolfi et al., 1966;

Davies et al., 1970; and Hubbard, 1973). On the other hand, with elevated aerobic power (> 55 ml / kg·min^{-1}) the optimal recovery intensity is observed at approximately 50 - 60% $V0_2$max for these subjects (Hermansen and Stensvold, 1972; and Hermansen et al., 1975).

The subjects who participated in the present study were a homogeneous group as indicated by the anthropometric and physiological data presented in Table I. The mean $V0_2$max values achieved (5.23 l / min) was representative of a highly fit group (Astrand and Rodahl, 1977), superior to observed $V0_2$max values of previous recovery investigations (Davies et al., 1970; Hubbard, 1973; and Belcastro and Bonen, 1975). Considering the athletic and training background of these competitive oarsmen, it was expected that they would achieve high aerobic capacities (Hagerman et al., 1979). The high $V0_2$max values further correspond to values observed in oarsmen of equivalent competitive ability (Bouckaert et al., 1983; Secher et al., 1983; and Hagerman, 1984).

The mean power output attained during the three one minute work periods was greater than the power outputs measured by Hagerman et al., (1979). This was expected since the subjects in the present study performed a shorter duration interval type exercise which quickly lead to fatigue and to an elevation in blood lactate concentration. The mean peak blood lactate concentration measured was 16.3 mM which is slightly higher than the mean peak measured in oarsmen by McKenzie and Rhodes (1982) but similar to those measured by Hagerman et al., (1979). This high blood lactate concentration is indicative of an intense work bout.

Lactate concentration at the conclusion of the three one minute workbouts did not appear to be a function of the power outputs, although the mean peak lactates (table II) and the mean power outputs (Appendix D) corresponded to one another. Individual differences in peak levels during similar power outputs may be due to dietary carbohydrate fluctuations and training fatigue. These subjects were not required to follow a set diet or cease their normal level of training during the period of the investigation. It has been shown that fluctuations in diet affect differnces in blood lactate concentration at similar power outputs (Rennie and Johnson, 1974; and Kelman et al., 1975). Randomization of treatments would bring about subjects experiencing training fatigue during different treatments and therefore individual differences in peak lactate would appear.

The high aerobic capacity, maintained by this subject group, is a reflection of the endurance training which takes up a large proportion of their training time (Hagerman, 1984). Mickelson and Hagerman (1984) determined that world-class oarsmen generate 72% of their total power output by utilizing 83% of their aerobic capacities. There can be no doubt that the group of subjects who participated in this study were highly trained, with a high aerobic power.

Evans and Cureton (1983) have shown that the training status of an individual affects recovery by allowing recovery to occur at a higher relative $V0_2$ than was possible prior to training. Training increases the oxidative capacity of the trained skeletal muscles and appears to increase the use of lactate as a preferred fuel for metabolism (Hochachka et al., 1985). The increased oxidative capacity and use of lactate as a fuel source would increase the rate of the uptake of lactate

and its subsequent disappearance from the blood. Tracer studies clearly demonstrate the uptake of labelled lactate by skeletal muscle and a concurrent evolution of $^{14}C0_2$, indicative of lactate oxidation (Jorfeldt, 1970; and Hubbard, 1973). Tracer studies comparing trained to untrained animals have shown that the evolution of $^{14}C0_2$ is greater at all levels of exercise for trained animals (Depocas et al., 1969; Freminet et al., 1975; Brooks and Divine-Spurgeon, 1982; and Donovan and Brooks, 1983).

The improved oxidative capacity of the skeletal muscle tissue due to training occurs at a number of levels. Enzymes which are important to oxidation are found at elevated concentrations in endurance trained individuals. Endurance training has been shown to increase the lactate specific heart isozyme of lactate dehydrogenase (LDH-H) in all trained skeletal muscle fibres and therefore not only improving the oxidative capacity of SO fibres, but also increasing that of the fast glycolytic fibres (FG) (Karlsson et al., 1974).

Determination of the anaerobic threshold on the subjects participating in this study was not performed. Oarsmen of equivalent training and competitive backgrounds have been shown to have high anaerobic thresholds occurring at approximately 83% of the $V0_2max$ while performing on a rowing ergometer (Mickelson and Hagerman, 1982). The level of the anaerobic threshold may determine the appropriateness of a recovery intensity (Mclellan and Skinner, 1979). In well-trained subjects their AnT may be much closer to their $V0_2max$ and would therefore be at a higher $\%V0_2max$ than an AnT of an untrained, less aerobically fit individual. The consequence of an AnT at a high percentage of an individual's $V0_2max$ would be a delay in the

onset of elevated blood lactate concentration as exercise intensity increases. This would allow individuals to remove lactate at a higher percentage of the $V0_2max$ without an elevation in the baseline concentrations of lactate in the blood (Stamford et al., 1981). Individuals who are capable of recovering at a higher percentage of $V0_2max$, increase bloodflow and lactate delivery to active skeletal muscle and thus improve lactate uptake while maintaining a lower production of lactate from working muscle tissue (Sahlin, 1982).

The performance of the recovery task involved the utilization of a rowing ergometer. The task itself involves a large percentage of the subject's muscle mass. The greater the extent of muscle participating in recovery, the greater the influence upon the rate of lactate reduction (McGrail et al., 1978; and Stamford et al., 1978). The muscle mass involved was, not only large (i.e., legs, arms, and back) but, because these muscles are involved in a task for which they are specifically trained, may have also increased the uptake of lactate and its subsequent reduction in the blood.

Summary

A reduction of lactate with submaximal exercise is paramount to the use of active recovery as a means to reduce lactate following exercise. It is suggested (Stamford et al., 1981) that as long as active recovery does not raise the baseline lactate levels substantially above the resting baseline levels, then there will be a reduction of lactate concentration in the blood. The appropriate intensity of submaximal exercise for recovery from prior severe exercise appears to be one in

which lactate production approaches but does not exceed lactate uptake (Hubbard, 1973; and Weltman et al., 1977).

This investigation studied the effects of three recovery intensities upon the rate of blood lactate reduction for eight well-trained oarsmen. It was determined that when these subjects recovered at 60% $V0_2max$ blood lactate was reduced at a significantly higher rate than 40% $V0_2max$, although it was not reduced any faster when compared to combined recovery. The significant difference between the rate of blood lactate reduction for 60R compared to 40R may be due to the subjects' high aerobic fitness, the specific nature of both their training and the recovery task, and the physiological adaptations related to a high fitness level. This investigation is only a first step, although these findings do suggest that the rate of lactate removal may be dependent upon the fitness level of the individual. Further research is needed to observe the differences among highly fit and lesser fit individuals and also to observe the role that a number of varied recovery tasks play in the enhancement of blood lactate reduction.

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APPENDIX A: REVIEW OF LITERATURE

Review of Literature

I Historical Background

The association of lactate accumulation and subsequent muscular fatigue was observed early in the 1900's when Fletcher and Hopkins (1907) attempted to classify the production of lactic acid as a metabolite of muscular activity. They found that lactic acid was spontaneously developed during anaerobic conditions in excised muscle, linking the lactate production to what they called "survival periods of subsisting irritability" (Fletcher and Hopkins, 1907, p. 301). It was felt that muscle had a chemical abiltiy to physiologically cope with lactate production, which was observed in the disappearance of lactate during recovery periods in an oxygen atmosphere (Fletcher and Hopkins, 1907).

Hill and co workers (1922 and 1923) were able to further elucidate the muscular recovery process with Meyerhof's (1920) discovery of glycolysis and glycogen as the ultimate precursor of lactate. These researchers observed that recovering muscle had an oxygen consumption and heat production far in excess of that in resting muscle. Both oxygen consumption and heat production remained elevated until the complete disappearance of lactate and restoration of muscle glycogen. It was thought that one-fifth to one-sixth of the lactate molecules were consumed (oxidized) so that the remaining fraction could be restored to glycogen (Hartree and Hill, 1922). The above findings were those which became the basis for the classical "0_2 debt" theory (Hill and Lupton, 1923).

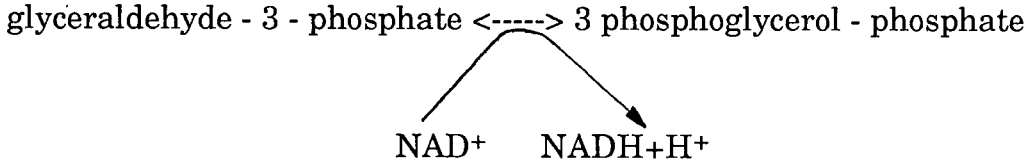
In an extensive series of experiments, Jervell (1928) concluded that the increase of lactate into the blood was "a sign of uneconomic

metabolism" which was produced to a large extent during periods of physical exertion and in certain disease states. Lactate was observed to have a fairly regular time course in blood following exercise, falling slowly to resting levels within a half an hour (Jervell, 1928). Further, Jervell (1928) noted that if submaximal exercise was performed during the recovery period, the lactic acid concentration would fall more rapidly than if the subject passively rested. These observations are somewhat contrary to the "0_2 debt" theory and were supported by another extensive lactic acid study carried out by Bang in 1936. Bang (1936) observed during a series of experiments that blood lactate concentration did not remain elevated throughout exercise. Lactate was consistently found to rise to a peak concentration at approximately 10 minutes of exercise and then steadily fall whether exercise was continued or not (Bang, 1936).

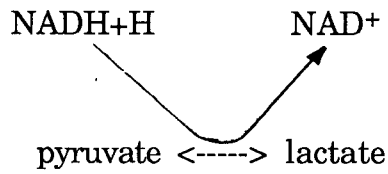
Newman et al., (1937) recorded findings which supported both the findings of Jervell (1928) and Bang (1936), demonstrating that with continuous exercise, blood lactate concentration did not keep rising but eventually declined to resting values. These papers (Jervell, 1928; Bang, 1936; and Newman et al., 1937) were contrary to the results observed by Hartree and Hill (1922), proposing that with submaximal to moderately severe exercise there is an enhanced removal of blood lactate and a possible reduction in lactate production.

II Lactic Acid And Muscular Performance

Under anaerobic conditions, there is a generation of nicotinamide adenine dinucleotide (NAD^+) in the reduced form ($NADH+H^+$) by and early step in glycolysis:



The reduction of pyruvate to lactate by LDH-M brings about an oxidation of $NADH+H^+$ to NAD^+:



Therefore the rapid reduction of pyruvate to lactate allows the muscle fibre and the organism to generate a temporary 0_2 debt in the form of lactate and by oxidizing $NADH+H^+$ to NAD^+ in the absence of 0_2 (Lehninger, 1982). The recyling of $NADH+H^+$ and NAD^+ solves the problem of reoxidizing $NADH+H^+$ when the 0_2 supply is insufficient, but the by-product, lactic acid is accumulated and this accumulation appears to be involved in fatigue (Wenger and Reed, 1976).

A number of investigations (Karlsson and Saltin, 1970; Klausen et al., 1972; Karlsson et al., 1974; Karlsson et al., 1975; and Weltman et al., 1977) have demonstrated that an elevated lactate concentration corresponds to a decrease in muscle performance. High concentration of lactic acid in the muscle appears to be responsble for exhaustion in very heavy exercise lasting two-and-a-half to six minutes in duration (Karlsson and Saltin, 1970). The association of an elevated lactate level and decreased performance time may be directly due to the muscles inability to produce energy due to the inhibitory effects associated with lactate on glycolysis (Klausen et al., 1972). Local muscle tissue does not

appear to be exclusively affected by excessive lactate concentration, there is evidence that lactate produced by one muscle group contributes to decreased performance in other muscle groups (Karlsson et al., 1974). It was further hypothesized that the size of the active muscle mass taking part in exhaustive exercise was an important factor with regards to the magnitude of the lactate change in other muscle groups. Therefore the larger the muscle mass involved in exhaustive exercise the greater the influence upon the biochemistry in other muscle groups (Karlsson et al., 1975).

With heavy exercise there is an increased production of lactate, a level beyond which can be removed at a lower energy expenditure (Jorfeldt et al., 1978). The increased muscle lactate is associated with elevated hydride ion concentration [H^+], decreased pH (Hermansen and Osnes, 1972), an inhibition of the glycolytic pathway and subsequently, inadequate supply of adenosine triphosphate for continuing muscle contraction (Tesch et al., 1978; and Sutton et al., 1981). Glycolytic intermediates such as phosphofructokinase (PFK) (Trivedi and Danforth, 1966) and lactate dehdrogenase (LDH) (Karlsson et al., 1974) are both inhibited at elevated lactate and [H^+] levels.

Increases in lactate and the concomitant elevation in [H^+] may also interfere with calcium ion (Ca^{++}) binding during muscle contraction. In amphibian muscle (Fitts and Holloszy, 1976) a strong inverse relationship (r= -.99, highly significant) was observed between an enhanced lactate concentration and a decrease in contraction force. During recovery in the same muscle, a strong inverse relationship (r= -.92, highly significant) described an increase in contraction force with a decrease in lactate concentration. Lactic acid was considered to

affect Ca^{++} binding in the muscle in two ways (Fitts and Holloszy, 1976). First, the elevated $[H^+]$ in the muscle may have lowered the apparent binding constant of Ca^{++} with troponin, reducing the number of available actomyosin cross bridges. Secondly, a decrease in pH may cause an increase in the amount of Ca^{++} bound to the sacroplasmic reticulum (SR) since the amount of Ca^{++} released from the SR is proportional to the pH in the range 6.45 - 7.9 (Nakumara and Schwartz, 1972; and Robertson and Kerrick, 1979).

These rather extensive perturbations on muscle tissue can be considered undesirable when continued performance, not fatigue is expected. Therefore the ability to return the muscle tissue to its resting level becomes of prime importance. Since lactate accumulation is implicated in the reduction of muscle performance, reducing its concentration in the blood then allows the body to return to its homeostatic level (Hermansen et al., 1975).

III The Role Of Exercise In Blood Lactate Reduction

IIIa Active Recovery

The importance of reducing elevated lactate levels and the evaluation of the role that particular recovery processes play, have clearly demonstrated that an active (submaximal) recovery is superior to a resting or passive recovery (PR) (Gisolfi et al., 1966; Davis et al., 1970; and Hermansen and Stensvold, 1972). However, efforts to categorize a specific optimal level of active recovery (AR) have yeilded conflicting results. Gisolfi et al., (1966) revealed that a relative $V0_2$ at approximately 50 percent of an individuals maximal oxygen uptake (% $V0_2max$) was most effective in reducing the concentration of lactate in

the blood. Unfortunately the choice of exercise intensity during AR seemed to be arbitrarily selected and compared only two levels of AR in two subjects. Davis et al., (1970) and Hermansen and Stensvold (1972), on the other hand, selected a series of AR levels which were performed following severe exercise. Davis et al., (1970) showed that for the group of subjects who performed in those recovery experiments, 40% $V0_2max$ appeared to be optimally suited for reducing blood lactate concentration. Results obtained by Hermansen and Stensvold (1972) pointed to a higher level of recovery, at 65% $V0_2max$ to produce the highest rate of blood lactate reduction.

The inability to find a singular recovery level remained the driving force in an effort to determine if an optimal level of AR existed. Belcastro and Bonen (1975) compared controlled and uncontrolled recovery exercise after exercise at 90% $V0_2max$. Uncontrolled AR at 51% and 56% $V0_2max$ and controlled AR at 30% and 45% were all shown to be more effective ($p<.01$) than PR, and than AR performed at approximately 60% and 80% $V0_2max$. In this case subjects may have exceeded their lactate threshold at 60% $V0_2max$ and above, where lactate production is in excess of removal. A linear equation fit to the data, predicted that AR at 32% $V0_2max$ would give an optimal rate of blood lactate reduction (Belcastro and Bonen, 1975). From a practical standpoint it appeared that subjects were capable of finding a level of recovery which best suited their needs as observed from the finding that uncontrolled recovery was as effective as a controlled recovery for reducing blood lactate concentration.

Bonen and Belcastro (1976), testing further uncontrolled recovery protocols, allowed subjects to self-select their own level of recovery

intensity. Subjects were required to complete three treatments: a constant jog, intermittent exercise, and rest, following a mile run. Continuous recovery involving aerobic exercise (free jog; 61.4 ± 9.2 % $V0_2max$) was found to significantly ($p<.001$) reduce blood lactate concentration when compared to free intermittent and resting recovery (Bonen and Belcastro, 1976). These researchers (Belcastro and Bonen, 1975; and Bonen and Belcastro, 1976) suggest that subjects were quite capable of intuitively determining their individual optimal intensity level for recovery exercise, with the single stipulation that recovery must involve a continuous level of aerobic exercise. Continuous exercise, compared to intermittent exercise, maintains bloodflow to those regions which remove lactate from the blood (Bonen and Belcastro, 1976) and therefore allows a continuous delivery of lactate to the working muscle.

IIIb Active Recovery Above The Anaerobic Threshold

Stamford et al., (1981), and McLellan and Skinner (1982) attempted to determine rates of lactate removal in relation to the anaerobic threshold (AnT). Although studies have implied that exceeding the AnT would effect lactate removal (Davis et al., 1970; and Hermansen et al., 1975), few efforts were made to test this hypothesis. Theoretically, if the AnT were surpassed by any level of active recovery, lactate production would encroach upon the rate of removal and therefore slow the recovery process (Stamford et al., 1981; and McLellan and Skinner, 1982).

Stamford et al., (1981) demonstrated that an active recovery level below 40% $V0_2max$ yielded a higher rate of lactate removal than active recovery above 70% $V0_2max$ (a level which exceeded the AnT) when a

resting baseline was used. When baselines relative to 40% $V0_2max$ and to 70% $V0_2max$ workloads were used, there was no difference in the rate of lactate removal for either active recovery. "Baseline refers to the concentration of blood lactate associated with a given intensity of exercise without preceding maximal exercise" (Stamford et al., 1981, p. 804). These results suggested to Stamford et al., (1981) that a combined active recovery which begins at, or slightly above AnT, then later drops well below AnT for the remainder of the recovery period would produce a higher rate of lactate removal then would a single level of AR.

Testing Stamford's hypothesis, Dodd et al., (1984) found that active recovery at 35% $V0_2max$ removed lactate at a higher rate than PR, and AR at 65% $V0_2max$ ($p<.05$). The 35% $V0_2max$ active recovery was not significantly different ($p>0.5$) than a combined recovery of 65% and 35% $V0_2max$. It was therefore concluded that a combined active recovery which exceeded the AnT was not superior to a continuous single level (35% $V0_2max$) active recovery.

Increments in recovery intensity were related to the AnT rather than to the $V0_2max$ in an experiment designed by McLellan and Skinner (1982). It was felt that by expressing the recovery intensities in terms of decrements from the AnT, the results would reveal a more homogeneous lactate removal pattern. Ten minutes of exercise at 90% $V0_2max$ prior to the recovery protocols was employed to elevate the lactate concentration of the blood. Following the initial exercise session a one minute rest period proceeded, either 20 minutes of further rest, or active recovery at AnT -30% (A-30), -20% (A-20), -10% (A-10), $\pm$10 (A+10). Subjects performed all treatments which were assigned in a randomized order. The dependent variable being the time required for

the blood lactate to achieve one-half the initial concentration ($t^1/_2$). The results revealed that all levels of active recovery brought about a significantly faster $t^1/_2$ than resting recovery. The $t^1/_2$ value was achieved for A-20, A-10, and A at a faster rate than A+10. A peak rate of lactate removal was determined, from regression analysis, to occur at 10 percent below AnT (A-10) which corresponded to 43% $V0_2max$, therefore showing that active recovery slightly below the AnT was best suited for peak lactate reduction for these subjects (McLellan and Skinner, 1982).

Evidence gathered from previous studies (Davis et al., 1970; and Hermansen et al., 1975) expresses the opinion that recovery in excess of the AnT would slow lactate removal from the blood. Although the research concerned with the selection of a single optimal recovery level provides a great deal of ambiguity (Hermansen and Stensvold, 1972; Bonen and Belcastro, 1976; Dodd et al., 1984), there seems to be no question that recovery exercise which occurs above the AnT does in fact slow the rate of lactate removal from the blood (Stamford et al., 1981; McLellan and Skinner, 1982; and Dodd et al., 1984).

IV Factors Influencing Lactate Reduction

IVa Fibre Type

Characteristically, researchers have been incapable of demarking a universally applicable intensity of active recovery which optimally removes lactate from the blood (Gisolfi et al., 1966; Davis et al., 1970; Hermansen and Stensvold, 1972; Belcastro and Bonen, 1975; Hermansen et al., 1975; and Bonen and Belcastro, 1976). Bonen et al., (1978, 1979) are

of the opinion that a number of factors could be responsible for these equivocal findings.

Muscle fibres are known to vary in their histochemical and biochemical characteristic (Gollnick and Armstrong, 1975) and from these differences there have been two main muscle fibre types identified. These are type I or slow oxidative (SO), which relies upon the oxidative pathway for energy production, and type II or fast glycolytic (FG), which relies upon anaerobic glycolysis for its principle energy source (Holloszy, 1967; and Saltin, 1973). A further identification of an intermediate muscle fibre, divides the type II fibres into IIa (fast oxidative glycolytic) and classifies the FG fibres as type IIb (Edgerton, 1976). Therefore it can be logically deduced that active muscle must in some way be responsible for the increased rate of lactate removal from the blood, in particular the red muscle fibres or slow oxidative fibres (SO) which are enzymatically suited for lactate uptake and oxidation (Gollnick et al., 1975). Enyzmatic characteristics are quite different in the two muscle fibre types (Lehninger, 1982). The enzyme lactate dehydrogenase (LDH) is found in a number of different isozymic forms. Two in particular, LDH-heart (H) form and LDH-muscle (M) form are found on opposite ends of the continuim. The FG fibres are found to contain predominantly the LDH-M isozyme which promotes the reduction of pyruvate to lactate under anaerobic condtions. The SO fibres contain predominantly the LDH-H isozyme which brings about the oxidation of lactate to pyruvate.

Bonen et al., (1978) observed a moderate correlation ($r=.544$) but statistically significant ($p<.05$) relationship between the percentage (%) of SO muscle fibres (vastas lateralis) and the rate of lactate removal from the blood. A moderate correlation points to other factors, in concert with

% SO fibres, influencing the optimal intensity of recovery. Using an additive, least squares, multiple regression model, Bonen et al., (1979) determined the influence of three factors, 1) the initial blood lactate concentration at the start of recovery, 2) the percentage of slow oxidative fibres in the vastus lateralis, and 3) the intensity of recovery exercise on the rate of lactate removal from the blood. Individually each factor showed only moderate correlations to the rate of lactate removal. Subsequently with the addition of each factor to the mathematical model, the multiple correlation coefficient (R) was increased to 0.91 ($p=0.012$).

IVb Muscle Mass

The size of the muscle mass involved in recovery exercise has also been implicated as a factor responsible for enhanced blood lactate reduction (McGrail et al., 1978; and Stamford et al., 1978). In a comparison of resting recovery (R), recovery using arm only exercise (AOE), recovery using leg only exercise (LOE), and recovery using a combination of leg and arm exercise (CE) McGrail et al. (1978) attempted to determine the relative effect of increasing muscle mass involvement on the rate of lactate reduction. The absolute 0_2 cost for each of the treatments were distinctly different; 0.73 ± 0.04, 1.04 ± 0.05, and 1.23 ± 0.10 l/min ($p<0.5$) for AOE, LOE and CE respectively. Although an effort was made to align the 0_2 cost of each recovery treatment these differences obscured the effects of increasing muscle mass on the rate of lactate reduction. Manipulation of the statistics identified a high positive correlation ($r=.92$) between the absolute recovery 0_2 and the rate of lactate reduction from the blood. The absolute 0_2 cost was considered to represent the active muscle mass involved in each of the three exercise

conditions. This strong relationship indicated that the mass of muscle involved in recovery influenced the rate of lactate removal from the blood (McGrail et al., 1978).

The effectiveness of increased muscle mass, "fatigued" muscle versus "non-fatigued" muscle, and elevated 0_2 availability on the rate of lactate reduction were observed by Stamford et al., (1978). Prior to recovery, pedalling exercise was performed by the right leg (one minute at 2.5 kg of resistance) to elevate the blood lactate concentration in the blood and to pre-fatigue the right leg. Subjects recovered by pedalling for 24 minutes with their 1) right leg (RL), 2) left leg (LL), 3) right leg as they breathed 100% 0_2 ($RL0_2$), and 4) with both legs (2L). Each of these treatments were performed at a workload of 50W. An additional two treatments were included; RL at 25W (RL25) and a non-exercising control (C). Significantly greater blood lactate removal occurred with 2L and RL25 ($p<.05$) than treatments C, RL, and LL. Disappearance of lactate from the blood was not impaired by a previously fatigued muscle group, since RL and LL were shown to bring about similar lactate reduction rates ($p>.05$). Breathing 100% oxygen during the recovery did little to improve lactate disappearance compared to any of the recovery treatments. Treatments involving RL25 and 2L recovery produced the smallest elevation in baseline lactate accumulation. A greater $V0_2$ and blood flow were associated with RL and LL treatments as was an elevated baseline blood lactate production when compared to RL25. The greatest influence to lactate disappearance revolves around maintaining the highest level of work output at the lowest possible elevation of the blood lactate baseline. Increasing the muscle mass involved in the recovery period at a relative workload meets this requirement since

production of lactate by the active muscle is kept to a minimum while local bloodflow is maintained. Increased bloodflow allows delivery of the lactate to active removal sites (Stamford et al., 1978). The RL25 treatment meets the above requirement and also retains an elevated bloodflow to the splanchnic region where lactate is also metabolized (Rowell et al., 1966).

IVc Training and Fitness Levels

Training and fitness appear to account for some of the disparity among the studies concerned with determining and optimal recovery intensity (Hubbard, 1973; and Weltman et al., 1977). Animal and human studies have shown to some degree, that the greater the aerobic fitness of an individual the higher the %$V0_2$max at which an optimal rate of lactate reduction occurs (Brooks and Divine-Spurgeon, 1982).

A paper by Evans and Cureton (1983) addressed the issue of training effect upon the rate of removal of lactate from the blood in humans. Two groups of subjects, both untrained, took part in this training study. The control group performed no exercise for the experimental period, while the training group trained four days a week for six weeks. The training involved six exercise bouts of four minutes duration each at a work capacity of 85% $V0_2$max (test $V0_2$max every week). In the pre-testing session both groups performed exercise at 110% $V0_2$max, to elevate blood lactate concentration, followed by either a resting recovery or an active recovery at 25% $V0_2$max. In the post-test both groups repeated the protocol from the pre-test (an absolute 25% $V0_2$max according to initial level of fitness), but the training group performed an additional active recovery protocol at a relative 25%

$V0_2$max level. Training increased $V0_2$max (l/min and ml/kg·min^{-1}) and ride time for the training group, 13.6%, 15.1% and 25.9% respectively, with only minor changes noted for the control group. These factors were significantly different between groups in the post-test session ($p<.05$). At the post-test, no differences were noted between the two groups with regards to absolute recovery intensity ($p>.05$). Significantly faster lactate reduction occurred during the relative recovery intensity between the two groups ($p<.05$). The observation that removal rate of lactate from the blood was enhanced with conditioning at relative $V0_2$ suggests that the increased rate was a function of the recovery work and $V0_2$. Whereas there was no difference in removal rate at an absolute recovery and the differences achieved due to the relative recovery intensity may only be due to the training groups ability to recover at a higher intensity (Evans and Cureton, 1983).

V The Metabolic Fate Of Lactate

Historically, researchers have considered that the metabolic fate of lactate, following exertion involved the oxidation of a small fraction ($^1/_5$ -$^1/_6$) of lactate to provide the energy to convert the remaining fraction to glucose in the process of gluconeogenesis (Hartree and Hill, 1922). Further, that the major site of lactate uptake and metabolism was that of the liver and that approximately 50% of all lactate was previously involved in glyconeogenesis (Rowell et al., 1966) in both of these studies (Hartree and Hill, 1922; and Rowell et al., 1966) indirect measurement of the lactate carbon fluxes were the basis for the above conclusions (Brooks and Fahey, 1984). Whereas, lactate tracer studies appear to provide a more direct method for evaluating the fate of lactate carbons during rest,

exercise, and recovery (Jorfeldt, 1970). Research using both radioactive (^{14}C) and non-radioactive (^{13}C) carbon isotopes are more extensive than reviewed here.

Va Human Studies

Working with evidence that muscle tissue could have a simultaneous release and uptake, Jorfeldt (1970) reached a number of conclusions with regards to the fate of lactate. Using ^{14}C-L(+) Lactate, rhythmic forearm exercise was performed (60 contractions/min at 10 kpm/min) for 60 min at normal and elevated blood lactate concentrations. During infusion of labelled lactate at normal concentrations, it was found that $^{14}CO_2$ was rapidly involved at the beginning of the steady infusion then reached a steady level after aproximately five minutes of infusion. By the end of 60 minutes of exercise, 80% of the ^{14}C was incorporated into expired $^{14}CO_2$. The net release of lactate was significantly greater ($p<.01$) at 10 min compared to 40 min with the mean net release decreasing during the 60 min exercise period. At the same time net uptake of lactate reached a constant level with no differences between 10 and 40 min of exercise (Jorfeldt, 1970).

To elevate levels of lactate beyond normal arterial concentrations a priming bolous of 15 mM of ^{14}C-L(+)- lactate was initially injected with a constant 3.5 mM per min infused until the end of exercise. After 10 min the net release and rate of net release was lower ($p<.05$) in the elevated lactate series than in the normal lactate series. Uptake of lactate reached a constant level after approximately four minutes with no differences observed between 10 and 40 minutes of exercise. Lactate uptake and rate of lactate uptake were positively correlated to blood

lactate concentration (r=0.65 and r=0.95 respectively). Following infusion, $^{14}C0_2$ in expired gas reached a constant level after about five minutes, 80% of ^{14}C was incorporated into $^{14}C0$ expired into $^{14}C0$ expired during exercise (Jorfeldt, 1970). The above evidence gives strong support to lactate being preferably oxidized rather than being incorporated in synthetic pathways such as glyconeogenisis. The skeletal muscle would appear to be an, if not the major site, of lactate uptake from the blood (Jorfeldt, 1970).

Hubbard (1973) compared the turnover of isotopically labelled lactate during rest and during 30 minutes of submaximal exercise (approximately 50% $V0_2max$) in human subjects. The labelled carbon (^{14}C) was measured in expired air, with 35 - 65% of the administered tracer carbon evolved after 30 min of exercise, while after 30 minutes of rest only 3 - 7% of $^{14}C0_2$ was observed. Similar findings by Mazzeo et al., (1983 and 1986) revealed cummulative $^{13}C0_2$ recovery in expired gas samples during exercise were much higher than during rest. Both studies (Mazzeo et al., 1983 and 1986) compared the effects of exercise, easy exercise (EE, 53.5% $V0_2max$) and heavy exercise (HE, 74% $V0_2max$). The rate of lactate oxidation was greatest in HE compared to EE and rest, with oxidation related to the intensity of exercise and lactate concentration (Mazzeo et al., 1983).

Lactate tracer studies (Jorfeldt, 1970; Hubbard, 1973; and Mazzeo et al., 1982 and 1986) involving humans at rest and during exercise have established that the primary metabolic fate of lactate may be that of oxidation. The enhanced rate of oxidation during exercise clearly identifies muscle tissue as the major source of lactate uptake and subsequent oxidation (Jorfeldt, 1970).

Vb Animal Studies

Oxidation of lactate as the major pathway involved in lactate reduction is supported by lactate tracer studies involving animals (Depocas et al., 1969; Donovan and Brooks, 1983; and Hochachka, 1985). These studies indicate that the turnover of lactate, which is the balance between lactate production and removal, may remain constant although the concentration is elevated in the blood. Lactate tracers are currently the best method to observe metabolic fluxes. Elevated blood lactate levels have been responsible for an increased lactate turnover rate.

Observing the replacement for lactate, Hochachka et al. (1985) found that lactate appeared to be the preferential energy source during light aerobic work. Lactate was shown to increase its metabolic turnover rate from rest to exercise by approximately 23.5 times (rest 12 umol / $kg \cdot min^{-1}$ to 282 umol / $kg \cdot min^{-1}$) in exercising Tammar Wallabies. Evidence that lactate is a potenially important aerobic fuel has been supported by other animal studies (Eldridge, 1975; Freminet et al., 1974; and Donovan and Brooks, 1983). It is known that lactate is not retained inside the body as a storage fuel, therefore two possible means by which lactate could contribute to metabolism may exist (Hochachka et al., 1985). The lactate may be formed endogenously or it may be formed in other tissues and then it is transported to sites of oxidation (ie. active skeletal muscle). The latter consideration appears to be the most likely, where lactate could be formed in FG fibres at about the same rate it is utilized by the more oxidative SO fibres (Hochachka et al., 1985).

Both animal and human studies have shown that lactate is readily taken up by aerobically working muscles. There appears to be a

greater turnover of lactate carbons by means of oxidation to $C0_2$ and H_20 as exercise increases up to a certain point. Trained muscle tissue appear to turnover lactate at a greater extent and at higher levels of exercise than do untrained individuals (Donovan and Brooks, 1983; Hochachka et al., 1985).

VI Summary

Historically lactate has been observed as a by-product of exercise metabolism, particularly of high intensity exercise which eventually causes fatigue. Disruption of the homeostatic balance maintained by the body appears to be a factor which, can to some degree, be associated with high concentrations of lactic acid. It has been shown that the concentration of lactate is reduced within blood during passive rest following severe exercise. The use of active recovery following exercise has revealed a much higher rate of [Bla] reduction than simply resting passively. Unfortunately there has been an inability to find an active recovery intensity which may be universally applicable. Reasons for these equivocal results appear to be due to several factors, such as: the size of the muscle mass involved in recovery; the percentage of slow twitch fibres involved in recovery; and the fitness and training level of the individuals involved in a recovery task.

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APPENDIX B: ABBREVIATIONS

Abbreviations

[ ]	concentration
[H^+]	Hydrogen ion concentration
FFA	Free fatty acid
PFK	Phosophofructokinase
LDH	Lactate dehydrogenase
LDH-H	Isozymic heart form of LDH
LDH-M	Isozymic muscle form of LDH
Ca^{++}	Calcium ion
^{14}C	Carbon fourteen (radioactive isotope of carbon)
^{12}C	Carbon twelve (most common isotope of carbon in nature)
HR	Heart rate
AnT	Anaerobic Threshold
$V0_2$	Volume of oxygen utilized
$V0_2$ max	Maximum volume of oxygen utilized
%$V0_2$ max	Percentage of the maximum volume of oxygen utilized
40R	40% $V0_2$max
60R	60% $V0_2$ max
CR	Combined recovery
V_E	Volume of expired air
F_EC0_2	Fraction of carbon dioxide in expired air
F_E0_2	Fraction of oxygen in expired air
l / min	Litres per min (absolute $V0_2$ max measure)
ml / kg·min^{-1}	Milliliters per kilogram per minute (relative $V0_2$ max measure)
min	Minutes
s	Seconds
h	Hours
yr	Year
W	Watts
rpm	Revolutions per minute
kg	Kilograms
kg·m	Kilogram meters
kg·m / min	Kilogram meter per minute
cm	Centimeters
mM	Millimolarity (equivalent to mmol·litres^{-1})
^oC	Degree celsius

abbreviations cont.

[Bla]	Blood lactate concentration
BLR	Blood lactate reduction
PR	Passive recovery
AR	Active recovery
± SD	Plus or minus the standard deviation
R^2	Goodness of fit
RBC	Red blood cells
SO	Slow oxidative muscle fibre (type I)
FOG	Fast oxidative muscle fibre (type IIa)
FG	Fast glycolytic muscle fibre (type IIb)

APPENDIX C: BLOOD LACTATE CONCENTRATIONS FOR ALL RECOVERY TREATMENTS

Appendix C: Blood Lactate Concentration For All Recovery Treatments

Blood Lactate Concentrations (mM)

Subject	Rec. Type	0	1	2	3	4	5	6	9	12	15	18	21	24	27	30
1	40R	14.8*	11.4	13.3	12.2	12.7	13.0	12.3	-	9.2	-	7.2	-	5.5	-	4.6
	60R	15.0*	13.7	13.2	11.7	9.8	10.8	10.3	9.7	8.3	6.6	4.9	3.4	4.2	3.5	2.3
	CR	16.6*	15.4	14.7	12.7	13.2	13.1	13.6	10.1	10.7	7.6	7.2	4.6	4.1	3.0	2.8
2	40R	9.0	-	8.6	9.9	7.8	6.6	8.0	6.0	6.0	4.2	3.2	3.2	2.5	2.2	2.9
	60R	11.4*	11.3	9.4	10.7	8.8	8.9	7.6	6.3	5.3	3.9	2.1	1.9	1.7	1.7	1.5
	CR	9.7	10.9	10.4	11.5*	10.7	7.2	7.8	8.4	5.9	5.0	3.9	2.7	1.6	1.5	0.90
3	40R	20.8*	20.4	19.9	17.1	17.2	16.3	15.7	12.6	10.4	7.6	6.4	5.1	3.3	2.3	1.2
	60R	17.1*	16.0	15.9	14.3	13.0	13.9	11.7	5.6	7.9	5.2	3.2	2.1	1.8	1.2	1.7
	CR	12.4	11.3	14.2*	13.5	13.7	11.4	11.2	9.7	8.9	5.2	4.5	2.6	1.7	1.1	1.2
4	40R	14.1*	12.1	11.2	11.2	10.4	9.2	10.0	6.3	5.0	1.2	2.4	3.2	1.0	1.0	1.3
	60R	16.0	17.0*	14.7	14.0	14.0	14.1	14.0	10.5	8.7	6.7	4.2	3.0	2.5	1.7	2.5
	CR	19.5*	19.3	15.2	13.7	15.1	13.7	12.6	10.3	9.6	7.3	5.8	3.9	3.3	3.1	2.3
5	40R	18.9	19.8*	16.4	-	17.2	16.4	-	14.3	12.8	8.6	8.6	6.4	4.9	4.2	3.0
	60R	19.5*	18.0	17.2	17.7	17.9	-	14.8	9.9	9.6	-	7.0	6.7	5.9	5.8	6.5
	CR	15.3	16.9*	14.9	14.3	12.2	12.7	12.6	11.7	9.5	9.6	6.9	7.8	6.7	5.6	5.6
6	40R	20.9*	18.5	16.8	15.4	15.4	15.7	15.8	13.3	12.0	11.2	7.8	6.3	5.1	3.4	3.0
	60R	22.4*	17.9	18.1	16.2	17.4	16.4	15.4	13.1	11.9	8.7	6.2	4.6	3.1	2.3	2.5
	CR	18.5*	14.5	13.9	14.9	14.8	13.7	14.9	13.5	10.1	8.9	7.7	5.6	4.9	3.5	3.0
7	40R	14.3	14.6*	14.0	14.1	11.7	11.5	10.9	9.0	7.6	6.1	5.4	3.6	3.1	2.4	1.6
	60R	16.5*	12.7	12.8	13.0	12.8	8.8	11.1	9.5	8.0	6.6	4.3	3.3	2.7	2.3	2.6
	CR	13.8*	13.7	13.4	10.0	13.2	10.3	11.1	9.5	7.6	6.0	4.5	3.4	3.3	2.2	2.1
8	40R	12.7*	9.0	9.7	10.7	11.2	8.8	8.1	8.9	6.3	4.9	3.9	2.7	2.0	1.3	1.2
	60R	12.1	13.3*	16.5	12.4	12.6	11.9	12.9	9.2	7.0	4.8	3.2	2.5	1.9	1.3	1.6
	CR	13.7	12.8	14.6*	13.3	11.6	11.4	10.1	8.2	7.2	4.3	2.8	1.6	0.90	0.70	0.60

APPENDIX D: POWER OUTPUTS

Appendix D: Power Outputs (Watts) for the three one minute work periods

		40	Mean	60	Mean	CR	Mean
S1	1	440	(3)	445	(2)	435	(1)
	2	429		440		435	
	3	435	435	435	440	435	435
S2	1	448	(1)	488	(3)	498	(2)
	2	461		461		456	
	3	445	465	451	467	424	460
S3	1	435	(1)	429	(3)	435	(2)
	2	445		445		440	
	3	429	437	429	435	429	435
S4	1	504	(1)	498	(3)	479	(2)
	2	456		456		461	
	3	456	472	429	461	440	460
S5	1	530	(2)	488	(1)	509	(3)
	2	461		488		480	
	3	445	479	488	488	461	486
S6	1	497	(3)	514	(2)	525	(1)
	2	497		480		480	
	3	445	480	457	484	474	493
S7	1	429	(1)	435	(3)	429	(2)
	2	424		429		416	
	3	408	421	416	429	419	422
S8	1	408	(3)	419	(2)	408	(1)
	2	408		419		414	
	3	408	408	408	415	398	406
		Mean	449		452		450
		± SD	±27		±26		±30

Brackets ( ) indicate the order in which the subject performed each treatment