Active vs. Passive Recovery
for 6 s Supramaximal Cycle Intervals

by

Tracey Doyle
B.Sc. Human Kinetics
University of Ottawa, 1995

A Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of

MASTER OF SCIENCE

in the School of Physical Education

We accept this thesis as conforming
to the required standard

[redacted]
Dr. David Docherty, Supervisor
School of Physical Education

[redacted]
Dr. Howard Wenger, Departmental Member
School of Physical Education

[redacted]
Dr. John Walsh, Outside Member
Department of Psychological Foundations

[redacted]
Dr. Patrick Neary, External Examiner
Department of Physical Education, Malaspina University College

Supervisor: Dr. David Docherty

ABSTRACT

The purpose of this study was to determine which recovery mode (active, passive, or active + passive) allows for greater maintenance of power output over ten 6 s supramaximal cycle intervals and whether recovery length and the number of intervals performed influences the superiority of one mode of recovery compared to another. Twenty-one female subjects participating in varsity level field hockey, rugby, soccer, or basketball performed 10 intervals of 6 s all-out cycle sprints at a load equal to 100 $g \cdot kg^{-1}$ on six different occasions, with a minimum of 48 hrs in between testing sessions. The recovery intervals varied in duration (30 or 60 s) and type (active, passive, or half active + half passive), for a total of 6 different modes which were presented randomly. It was found that passive recovery or active + passive recovery allows greater maintenance of peak power and mean power than active recovery whether recovery intervals are 30 or 60 s in duration and whether 5 or 10 intervals are performed.

[redacted]
Dr. D. Docherty, Supervisor (School of Physical Education)

[redacted]
Dr. H. Wenger, Departmental Member (School of Physical Education)

[redacted]
Dr. J. Walsh, Outside Member (Department of Psychological Foundations)

[redacted]
Dr. Patrick Neary, External Examiner (Department of Physical Education, Malaspina University-College)

Table of Contents

List of Tables

List of Figures

Acknowledgments

I would like to offer my sincerest thanks to the following people:

Dr. David Docherty: for three years of editorial, administrative, and academic guidance. Thank you for your patience and understanding in providing a significant portion of that guidance from a distance, and for taking me on in the first place!

Dr. Howard (Howie) Wenger: for your enthusiasm, ideas and encouragement.

Dr. John Walsh: for your expertise and support.

Dr. Patrick Neary: for agreeing to be a part of my defense "team".

Dona Tomlin and Darren Kruisselbrink: for our lengthy chats on various thesis-related issues and frustrations; these conversations were integral to the completion of my thesis with sanity intact!

Gladys and Norma: for the friendly smiles and invaluable assistance in jumping through all necessary hoops; Gladys you definitely made my life easier on more than one occasion!

Lindsey Close, Tracy Morgan, all my athletes, and everyone else who provided invaluable help in the lab… some of you as both subjects and testers… I really enjoyed meeting all of you; you made it fun to do research!

To all the friends I have made in Victoria who made it feel a little more like home, especially Michelle, Sarah, Megan, Tanya, and Michele.

An especially big hug, kiss, and thank you to my friends at home for being the best friends anyone could possibly ask for. Its you guys that make my life as great as it is. Thanks for being my family! To name but a few… Nat, Bill, Brad, Sheri, Will, Rob, Claire, Lori, Stina, and Jason… there are more, and I love you just as much!

Dedication

For my Mom

I love you

I miss you

Introduction

Research into recovery from exercise has used a range of intensities, durations, and exercise modes for both the work and recovery periods. One area that has remained relatively untested, however, is recovery from supramaximal work of less than 10 s in duration. Sports such as field hockey, ice hockey, rugby, and soccer are intermittent in nature and require quick bursts of high-intensity effort followed by periods of lower intensity work, or a rest period in which play has stopped. When training for such sports with short duration, high intensity, intermittent demands, knowing which form of recovery allows for the greatest maintenance of power output would be useful in obtaining optimal performance.

Margaria, Oliva, Di Prampero, and Cerretelli (1969) concluded that in supramaximal exercise, energy is not derived from glycolytic metabolism until phosphagen stores have been depleted or have reached a critical level. It had previously been determined that it takes 10-15 s for this to occur (Margaria, Cerretelli, Di Prampero, Massari, & Torelli, 1963). Therefore, lactic acid is not produced in this time frame. It was also concluded that a short recovery period would be necessary to replenish phosphagen stores and enable a subject to repeat the previous exercise using the same energy source. However, Jacobs, Tesch, Bar-Or, Karlsson, and Dotan (1983) found that there was pronounced accumulation of lactate in muscle after cycling exercise of only 10 s in duration, indicating that glycolysis does take place in this brief time frame, and that perhaps the energy systems should be viewed as a continuum as opposed to distinct and separate components.

Lactate accumulation is often discussed in relation to fatigue (Tesch, Sjödin, Thorstensson, & Karlsson, 1978), and lactate clearance times are frequently the dependent measure in studies examining recovery (Ainsworth, Serfass, & Leon, 1993; Gisolfi, Robinson, & Turrell, 1966; Hermansen & Stensvold, 1972; Stamford, Moffatt, Weltman, Maldonado, & Curtis, 1978; Weltman, Stamford, Moffatt, & Katch, 1977). Lactate accumulation is the most general and frequently cited cause of muscular fatigue (Fitts & Holloszy, 1976), and seems to be the basis for the rationale of how active recovery could be more beneficial than passive recovery (Belcastro & Bonen, 1975).

High muscle and blood lactate levels and the consequent increase in $[H^+]$ and decrease in pH are inversely related to the capacity to do physical work (Hermansen & Osnes, 1972). This acidosis can inhibit glycolytic enzyme activity (Trivedi & Danforth, 1966) as well as decrease muscle contractility (Fitts & Holloszy, 1976). As a decrease in pH impairs the release of Ca^{++} from the sarcoplasmic reticulum (Nakamaru & Schwartz, 1972), and H^+ competes for binding sites on troponin C, the formation and rate of cycling of cross bridges can be compromised (McCartney et al., 1986).

By engaging in active recovery, muscle blood flow is maintained to allow the clearance of lactate from the muscle fiber and its circulation to regions such as the liver, other muscles, and the heart. Lactate can also be utilized as a substrate by other fibers in the same muscle where it was formed (Hildebrandt, Schütze, & Stegemann, 1992). There is some controversy regarding whether or not lactate, and its effects on muscle metabolism and function, is the true cause of fatigue (Bangsbo, Johansen, & Saltin, 1993). For example, Sjøgaard, Adams, and Saltin (1985) suggested that muscle fatigue could be related to changes in the K^+ gradient across the muscle cell membrane. A loss of K^+ from

the muscle cell causes a decrease in the speed of propagation as well as the amplitude of the action potential (Juel, Bangsbo, Graham, & Saltin, 1990).

Active recovery has been repeatedly shown to improve lactate clearance and, presumably, enhance recovery (Belcastro & Bonen, 1975; Dodd, Powers, Callender, & Brooks, 1984; Gisolfi et al., 1966; Stamford, Weltman, Moffatt, & Sady, 1981). In studies that measured recovery based on subsequent performance, active recovery again has shown to be superior to passive recovery (Ainsworth et al., 1993; Bangsbo et al., 1993; Kaczynski, Montgomery, Koziris, Travlos, & Turcotte, 1988; Signorile, Ingalls, & Tremblay, 1993; Weltman et al., 1977). Based on such results, it is apparent that, even if lactate accumulation is not the major cause of fatigue, active recovery provides other benefits that facilitate subsequent performance. Weltman et al. (1977) felt that there must be other critical factors and suggested that, in addition to the oxidation of lactic acid, the increased muscle blood flow in active recovery aids such processes as temperature regulation within the muscle and the resynthesis of glycogen and replenishment of phosphagen stores in low-oxidative fibers.

The only studies to have examined active versus passive recovery for repeat performances of a supramaximal activity of less than 10 s in duration are Signorile et al. (1993) and Ahmaidi et al. (1996). Signorile et al. examined whether active or passive recovery would allow for the best maintenance of power output over successive intervals. It was suggested that if phosphagen stores are nearly depleted after this type of exercise, as suggested by Margaria et al. (1969), then active recovery would be detrimental in that it would further reduce phosphagen stores. It was hypothesized, however, that active recovery would prove to be more beneficial, due to the current evidence that the anaerobic alactic energy system does not work independently of the lactic and aerobic systems in the

first 10 s of activity. The protocol used was eight 6 s supramaximal work intervals on a cycle ergometer, separated by 30 s active or passive recovery intervals. The results were as hypothesized: active recovery allowed for a better maintenance of power output on successive intervals. Ahmaidi et al. (1996) also used 6 s supramaximal cycle intervals, but used an increasing load with every trial, and 5 minutes of active or passive recovery in between. At higher work intensities, peak power output and lactate clearance were enhanced by active recovery. The protocols of these two studies were quite different. However, both demonstrated that active recovery was superior to passive recovery for very short-term supramaximal performance.

Due to the lack of information concerning recovery of supramaximal intensity exercise lasting less than 10 s, practitioners writing anaerobic alactic training programs have been required to make 'educated guesses' about what will prove to be most effective. One approach has been to use a combination of active and passive recovery during the recovery interval. The rationale is that the benefits provided by active recovery, such as increased blood flow, lactate removal and temperature regulation (Weltman et al., 1977), can occur during the first half of the allotted time, and passive rest in the second half will allow greater phosphagen repletion. Another strategy has been to use passive recovery when the number of intervals is low (≤ 5) and active recovery when using a higher number of repeats. The assumption would be that over the course of 5 'alactic' intervals, very little lactic acid would be produced. However, as the number of intervals increases and phosphagen stores are further depleted, lactate accumulation could become more of a factor in fatigue. The half recovery time of muscle ATP and CP stores is commonly stated to be between 30 and 60 s (Tesch, Thorsson, & Fujitsuka, 1989). Active recovery has been found to be superior to passive recovery with 30 s recovery intervals (Signorile et al.,

1993), but no studies have examined longer intervals. Since a 60 s recovery interval for 6 s work intervals represents a work to rest (W:R) ratio of 1:10, and the W:R ratio for alactic training is normally 1:5 or 1:6 (Coaching Association of Canada, 1981), it is unlikely that this form of training would ever be done with recovery longer than 60 s. To date, these strategies have not been tested for effectiveness.

The purpose of this study was to determine which recovery mode (active, passive, or active + passive) allows for greater maintenance of power output over ten 6 s supramaximal cycle intervals and whether recovery length and the number of intervals performed influences the superiority of one mode of recovery compared to another.

Research Questions

1. Which recovery mode (active, passive, or active + passive) allows for the greatest maintenance of power output (as expressed by peak power output, mean power output, and fatigue index) over ten 6 s supramaximal cycle intervals?

2. Does the superiority of one recovery mode over another (as expressed by peak power output, mean power output, and fatigue index) change when the length of recovery is changed from 30 s to 60 s?

3. Is the superiority of one recovery mode over another (as expressed by peak power output, mean power output, and fatigue index) related to the number of intervals performed?

Operational Definitions

Peak Power Output (PPO): the highest power output in Watts (W) in 1 s of a 6 s work interval; expressed relative to body weight ($W \cdot kg^{-1}$).

Mean Power Output (MPO): the average power output in Watts (W) over the entire 6 s work interval; expressed relative to body weight ($W \cdot kg^{-1}$).

Fatigue Index (FI): the difference between peak power and the lowest power output in 1 s of a 6 s work interval, divided by peak power and expressed as a percentage.

Difference Score: the absolute difference between the average of the first three and the average of the last three sprint intervals; calculated for PPO, MPO and FI.

Active Recovery (A): cycling at a self-selected intensity

Passive Recovery (P): sitting on the cycle ergometer

Active + Passive Recovery (AP): cycling at a self-selected intensity for the first half of the recovery interval, sitting on the cycle ergometer for the second half.

Trained State: subjects were currently training for varsity level field hockey, rugby, soccer, or basketball, such that they were training a minimum of 4 times per week.

Assumptions

1. The load at which the subjects were required to cycle was sufficient to cause a drop-off in power output over 10 intervals.
2. Subjects made a maximal effort during each trial on every day of testing.
3. Subjects were capable of making a consistent effort for all testing.
4. Workloads were applied consistently for every trial under every condition.

Delimitations/Limitations

1. 21 subjects were used.
2. The subjects were from various sporting backgrounds.
3. The subjects were in a trained state.
4. It was up to the subjects to make a maximal effort for every trial under every condition.
5. The workload was applied by hand, requiring precision in both timing and load placement.

Methods

Subjects

Following approval by the University of Victoria Human Research Ethics Committee, 31 female subjects were recruited from the university athletic community. All were actively involved in either field hockey, rugby, soccer, or basketball and therefore were accustomed to training at maximal intensities. All testing procedures were thoroughly explained, including any potential risks and the level of commitment required. Informed consent was obtained, and confidentiality of results assured.

Three subjects withdrew prior to commencing the study due to injuries or the inability to commit to the time required. Throughout the course of the study, 7 more subjects also withdrew due to injury and other priorities as well as due to illness. Twenty-one subjects completed the study, however, two did not perform a $\dot{V}O_2$max test due to illness.

Testing Procedures

Eight testing days were required to perform a Wingate test, six interval protocols, and a $\dot{V}O_2$max test, in that order. There was a minimum of 48 hrs in between tests. All of the intermittent tests were performed at the same time of day to control for any circadian effects that may have been present. Prior to each testing session, the subjects were required to abstain from alcohol, caffeine, and vigorous exercise for at least 6 hrs, abstain from smoking for at least 2 hrs, and avoid having a heavy meal in the 3 to 4 hrs before testing.

There was at least one Professional Fitness and Lifestyle Consultant present at all testing sessions, and all other lab personnel were trained and experienced in all testing procedures.

All sprint cycling protocols were conducted on the same modified Monarch friction braked cycle ergometer with toe clips. The seat height was adjusted for each subject such that their leg was almost fully extended when the pedal was in the lowest position. The height was recorded and kept constant for subsequent sessions. The anaerobic tests (with the exception of the Wingate test) were set with a resistance that equaled 100 $g \cdot kg^{-1}$ of body mass for each subject. A resistance of 90 $g \cdot kg^{-1}$ was used in a pilot project, as this is the resistance recommended for all subjects for a 30 s Wingate test (Canadian Society for Exercise Physiology, 1993). In some cases, this resistance was found to be insufficient to elicit a large decrease in power output. In further pilot work, resistances of 90, 100 and 110 $g \cdot kg^{-1}$ were tested and it was found that a resistance of 100 $g \cdot kg^{-1}$ elicited higher peak and mean power outputs while demonstrating a consistent drop in performance over 10 intervals.

The intensity for the active recovery was self-selected by each of the subjects. All subjects pedaled at a rate of 75 rpm and selected the specific resistance (within 0.25 kp) after the first 6 s sprint was performed. The same intensity was then used for all intervals and recovery conditions. Self-selection for recovery was used by Belcastro and Bonen (1975) as part of a study to determine optimal recovery intensity from cycling exercise. Using blood lactate removal rates as a dependent variable, they predicted that the optimum intensity for recovery was 32% of $\dot{V}O_2max$. However, they found that the blood lactate removal rates were similar across a wide range of recovery intensities (27.7-45.3% of $\dot{V}O_2max$), as well as during the self-selected recovery intensities. This wide range of

effective recovery intensities makes self-selection of recovery intensity a viable option that is also practical for application in sport and training situations.

As all active testing procedures were maximal, high levels of verbal encouragement were provided at all times and were consistent for subjects across all tests. The calibration for the cycle ergometer used for all sprint testing was checked before the days testing and again if the ergometer was moved or otherwise modified. The calibration held day-to-day.

Wingate Test. This test was done for the purpose of describing the population being studied. Subjects warmed-up for 5 min on the cycle ergometer at an intensity that elicited a HR of about 150 bpm, followed by a 5 min rest and stretching. They then performed a 30 s Wingate test with a resistance of 90 $g \cdot kg^{-1}$, followed by a 5 min cool-down at a self-selected cycling intensity. Peak power, mean power, and fatigue index were calculated.

Once recovered, the subjects practiced the 6 s protocol 3 times with 60 s of passive rest between intervals 1 and 2, and 30 s of active rest between intervals 2 and 3. This was done in order to familiarize the subjects with the test protocol.

Interval Testing. With the exception of the duration and mode of recovery in between intervals, the protocols for these 6 days were the same. The test began with a 5 min warm-up for which the intensity was self-selected on the first day, and equivalent to the chosen recovery intensity on each subsequent day. Subjects were then given 5 min to rest and stretch their quadriceps, hamstrings, and calves (30 s each side), as well as to do any upper body stretching that they felt was necessary. After cycling for 30 s at the warm-up intensity, ten 6 s supramaximal work intervals were performed in which the subjects

were encouraged to pedal as fast and as hard as they could. The work intervals began from a pedaling start. Subjects were instructed to pedal fast and hard 2 s before the application of the load; the clock started as soon as the load was applied. The same cues were given for every trial, with the subject hearing "pick it up - pick it up - ready - and - go". The load was applied starting with "ready - and" and was fully applied by "go". Flywheel revolutions were counted using a photocell attached to the frame of the cycle ergometer and connected to an electronic counter.

Each interval was separated by one of 6 conditions:

1. 30 s active (self-selected intensity)
2. 30 s passive (sitting on cycle ergometer)
3. 60 s active
4. 60 s passive
5. 15 s active followed by 15 s passive
6. 30 s active followed by 30 s passive

In order to control for changes in fitness level that may have occurred during the course of the study, the order in which these conditions were presented was randomized. After the last interval, the subjects cycled at a self-selected intensity for at least 5 min. Peak power output, mean power output, and fatigue index were calculated for every 6 s interval for every subject.

In order to verify the reliability of the sprint testing, an intraclass correlation was carried out between the total number of flywheel revolutions counted for the first interval on the first day of sprint testing and the first interval on the second day for every subject. A correlation of .92 was obtained. In addition, an intraclass correlation was done using the

number of flywheel revolutions in the first second of the 6 s test. The resulting correlation coefficient was .88.

$\dot{V}O_2$max. Subjects were instructed to do their own stretching before the cycling test began. The test was continuous and incremental with a built-in warm up consisting of an initial 4 min stage at the same intensity that was used for recovery from the sprint intervals. After the warm-up stage, the workload was increased every 2 min based on the subjects body size and their response to the previous workload. As the subjects began to have difficulty with the workloads and the respiratory exchange ratio reached 1.0, the workload was increased every minute. Oxygen consumption was measured using a Sensormedics Vmax system, and was expressed relative to body mass. $\dot{V}O_2$max was considered to have been achieved when 2 or more of the following occurred: (a) oxygen consumption failed to increase (by more than 2%), or decreased, when the load was increased; (b) the respiratory exchange ratio exceeded 1.15; (c) predicted heart rate (HR) maximum was achieved; (d) volitional fatigue.

The average test length was 14 min 40 s including the 4 min warm-up interval. After the test, subjects were encouraged to continue cycling at an easy, self-selected intensity for several minutes.

Body Composition. Height, weight, and skinfold measurements were taken. Using Harpenden skinfold calipers, the 8 skinfold sites were triceps, biceps, subscapular, suprailiac, supraspinale, abdominal, front thigh, and medial calf. All measurements were taken on the right side of the body, except for the abdominal site, which was taken 5 cm to

the left of the umbilicus (Ross & Marfell-Jones, 1991). All skinfold measurements were taken by the same tester.

Data Analysis

A one-way repeated measures analysis of variance was conducted for each of the three dependent variables of peak power output (PPO), mean power output (MPO), and fatigue index (FI) using the mean for each variable over 10 intervals, the mean for the first five intervals only, and the difference scores over 10 intervals. Fisher's LSD was used for all post hoc tests. An alpha level of .05 was used for all statistical tests.

Results

Subject Characteristics

Of the 21 females who completed the study, nine played rugby, eight played field hockey, and two subjects were involved in soccer and basketball. Table 1 summarizes the age, height, weight, $\dot{V}O_2max$, and Wingate for all subjects, showing the mean, range, and standard deviation. Means, ranges, and standard deviations for skinfold measurements are shown in Table 2.

Table 1

Mean, Range, and Standard Deviation for Age, Height, Weight, $\dot{V}O_2max$, and Wingate Results of all Subjects (N=21 for all measures except $\dot{V}O_2max$ where N=19)

	Age	Height	Weight	$\dot{V}O_2max$	Wingate ($W \cdot kg^{-1}$)		
	(yrs.)	(cm)	(kg)	($mL \cdot kg^{-1} \cdot min^{-1}$)	PPO	MPO	FI
Mean	20.3	168.7	66.9	54.4	9.4	7.7	34.2
Range	18 - 24	162 - 177	56.7 - 79.6	51.0 - 59.7	7.8 - 10.9	6.5 - 8.9	16.5 – 45.8
SD	2.1	4.1	6.8	2.6	0.9	0.8	6.6

Table 2

Mean, Range, and Standard Deviation for all Skinfold Measurements (N=21)

(mm)	tricep	bicep	subscapular	iliac crest	supraspinale	abdominal	front thigh	med. calf
Mean	7.9	15.5	11.6	16.5	12.1	16.7	24.1	13.3
Range	3.4-17.6	8.8-27.5	7.7-20.9	7.6-25.2	6.0-20.5	6.3-29.2	11.8-31.7	7.3-21.6
SD	3.2	4.2	3.2	5.8	4.3	6.1	5.3	3.4

Recovery Intensity

The recovery intensity chosen by the subjects averaged 37.5% of $\dot{V}O_2max$, as measured during the warm-up phase of the $\dot{V}O_2max$ test performed at the same load and rpm as the recovery intervals. The range was from 30 to 47.8%, with a standard deviation of 5.57%.

Differences Among Recovery Conditions for 10 Intervals

Prior to performing the repeated measures ANOVAs, all interval data was examined for univariate normality and multivariate normality using Mauchly's test of sphericity. All assumptions were met ($p > .05$). Fisher's LSD was used for post hoc comparisons, as suggested by Howell (1995). Post hoc tests were done between all three conditions for 30 s recovery and 60 s recovery, resulting in six comparisons; three for the 30 s recovery conditions (A30-P30, A30-AP30, P30-AP30), and three for the 60 s recovery conditions (A60-P60, A60-AP60, P60-AP60). No comparisons were made between 30 s and 60 s conditions.

Peak Power Output. A repeated measures ANOVA indicated differences between means of the recovery conditions for PPO ($F_{5,100} = 28.377$; $p < .0001$). Subsequent Fisher's LSD post hoc tests showed that significant differences existed between A30-P30, A30-AP30, A60-P60, A60-AP60, and P60-AP60 ($\alpha = .05$). Figure 1 illustrates the differences between all six recovery modes. The difference between P30-AP30 was not statistically significant.

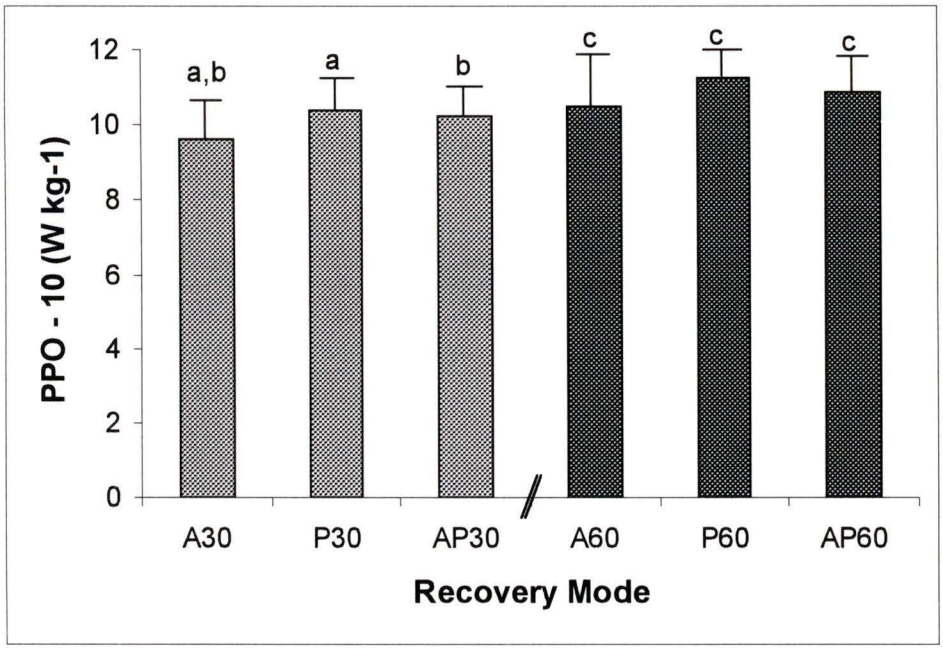


Figure 1. Mean PPO (SD) across 10 intervals for each recovery mode. Corresponding letters denote significant differences between conditions ($\alpha = .05$).

For 30 s of recovery, P and AP modes allowed for superior performance across 10 intervals, while with 60 s of recovery all three conditions were statistically different, with P having the highest mean, followed by AP and A.

Mean Power Output. A repeated measures ANOVA for MPO also indicated differences between recovery conditions ($F_{5,100} = 52.768$; $p < .0001$). Similar to PPO, post hoc LSD tests showed differences between A30-P30, A30-AP30, A60-P60, and A60-AP60 ($\alpha = .05$). P and AP were not significantly different for either 30 or 60 s of recovery. As seen in figure 2, the pattern between the recovery conditions is the same for both 30 and 60 s of recovery, with P and AP both being superior to A.

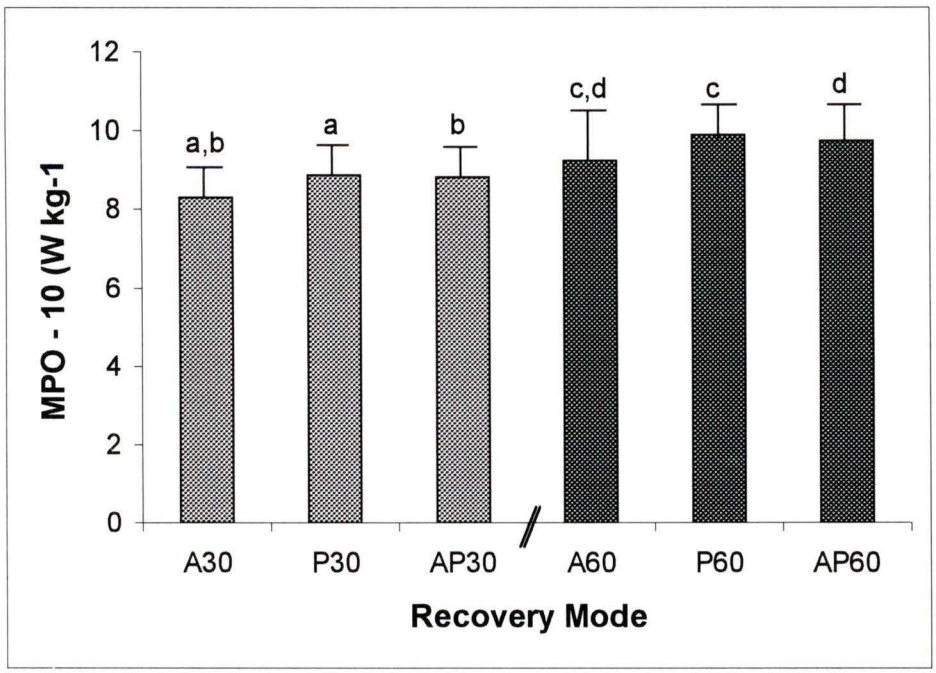


Figure 2. Mean MPO (SD) across 10 intervals for each recovery mode. Corresponding letters denote significant differences between conditions ($\alpha = .05$).

Fatigue Index. The repeated measures ANOVA for FI produced an $F_{(5,100)}$ of 6.578 ($p < .0001$). The post hoc tests showed that there were no statistically significant differences between any of the recovery conditions for 30 s, but that AP60 was significantly different from both A60 and P60. As can be seen from Figure 3, the AP60 recovery condition resulted in a lower fatigue index, indicating that it allowed for superior recovery with respect to the ability to maintain power for 6 s within each interval.

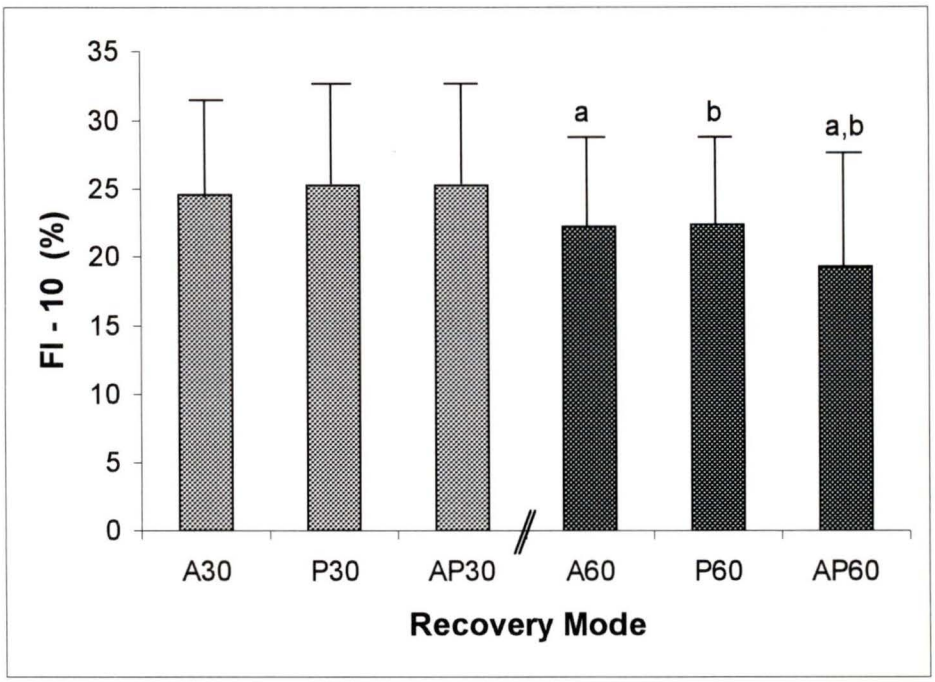


Figure 3. Mean fatigue index (SD) across 10 intervals for each recovery mode. Corresponding letters denote significant differences between conditions ($\alpha = .05$).

Differences Among Recovery Conditions for 5 Intervals

The results obtained using the means after only 5 intervals are identical to those found after 10 intervals with the exception that there were no significant differences between any of the modes or durations for FI. The $F_{5,100}$ values for ANOVAs on PPO, MPO, and FI were 12.945 ($p < .0001$), 24.319 ($p < .001$), and 3.913 ($p < .01$) respectively.

Differences Among Recovery Conditions Using Difference Scores

A repeated measures ANOVA of the difference scores over 10 intervals for PPO indicated significant differences ($F_{5,100} = 11.446$; $p < .0001$). Post hoc tests showed, however, that there were no significant differences between the conditions of interest. The same was seen for MPO ($F_{5,100} = 15.935$; $p < .0001$). The ANOVA for the difference scores of FI indicated that there were no significant differences between any conditions ($F_{5,100} = 1.429$; $p = .220$).

Discussion

The main finding from this study was that for 6 s supramaximal cycle intervals with 30 or 60 s second recovery intervals, either passive recovery or a combination of active recovery followed by passive recovery is superior to active recovery alone. This allows for better maintenance of both peak power and mean power over 5 or 10 intervals, but does not influence the fatigue index within each interval. Without additional measures, such as muscle biopsies, conclusions cannot be drawn about the physiological reasons for these findings. It may be assumed that the passive recovery allows for a more complete repletion of ATP and CP stores, whereas active recovery is considered to facilitate removal of metabolites likely to accumulate from repeated high intensity efforts. However, the need to maximally replenish phosphagen stores for such short-term high intensity work may outweigh the other benefits provided by active recovery.

There are two exceptions to the general statement made above, both with respect to 60 s recovery intervals. With 60 s of recovery, passive recovery is superior to the combination recovery which is in turn superior to active recovery for peak power output. Perhaps with the longer recovery time the additional ATP-CP repletion made possible with passive recovery is enough to produce a statistically significant difference compared to the combination recovery, whereas with only 30 s of recovery the mean peak power output for passive recovery is greater than for the combination, however the differences are not statistically significant. In addition, the fatigue index for 60 s of combination recovery is significantly lower than for both active recovery and passive recovery. The mean fatigue index is the average of the fatigue indexes for each 6 s sprint interval, and it is possible for this measure to stay low as intervals progress if the absolute power output

drops while the relative difference between high and low power over 6 s stays the same. The mean fatigue index was not influenced by recovery mode at all over 5 intervals, nor with 30 s of recovery over 10 intervals. Perhaps with 30 s of active recovery followed by 30 s of passive recovery the active portion provides an advantage with respect to maintaining power output over 6 s that is not seen with the measures of mean power due to decreasing peak power.

The only study directly comparable to the current one was conducted by Signorile et al. (1993). Contrary to the current findings, these authors found that active recovery was superior to passive recovery for eight 6 s sprint intervals. There are several differences between the two studies, the most relevant being the use by Signorile et al. of 6 male athletes involved in power lifting, sprinting, soccer, and triathlons, compared to 21 females involved in sports that require high intensity, short duration, intermittent efforts. In addition, Signorile et al. used a load of 140 $g \bullet kg^{-1}$ for the work interval intensity, whereas the current study used 100 $g \bullet kg^{-1}$. While it has been found that significant differences exist between genders with respect to performance in anaerobic power tests (Mayhew & Salm, 1990), whether these differences would extend into mechanisms of fatigue and recovery is unclear. It is possible that the differences in athletic background could impact the results, as different types of training could result in different adaptations at the muscular level. Perhaps the concentrations of certain substrates and enzymes could influence the effectiveness of different recovery modes. Hamilton, Nevill, Brooks, and Williams (1991) found that as compared to endurance-trained runners, athletes involved in multiple sprint-type sports had enhanced maximal performance on ten 6 s all-out sprints on a treadmill followed by 30 s of passive recovery. While the subjects used by Signorile et al. were not all endurance athletes, the Hamilton et al. study serves to demonstrate that

athletes with obviously different training backgrounds can respond differently to the same stimulus. The load used could also be a factor, as a load of 140 $g \cdot kg^{-1}$ is very high, and would likely result in more pronounced fatigue over 10 intervals than a load of 100 $g \cdot kg^{-1}$. It is possible that with such a high load factors such as metabolite clearance, tissue temperature regulation, and glycogen resynthesis, which are all aided by active recovery (Weltman et al., 1977), play a greater role in permitting the maintenance of power output than they do when a lower load is used. More work would need to be done in this area to determine if all of these factors truly have a significant impact on what kind of recovery is most effective.

While other studies comparing active recovery to passive recovery for high-intensity sprint intervals of less than 10 s are lacking, there have been several studies in the last few years that have examined other aspects of this type of activity. The following discussion will focus on those studies that are directly relevant to the protocol used for this study, which consisted of 6 s cycle sprints.

Gaitanos, Williams, Boobis, and Brooks (1993) performed muscle biopsies of the vastus lateralis before and after the first and last sprints of a 10 interval protocol. Muscle ATP dropped to 87 % of resting values after the first sprint, and was 68 % both before and after the tenth sprint. Muscle CP dropped to 43 % after the first sprint and values for before and after the tenth sprint were 49 % and 16 %, respectively. This would seem to suggest that ATP dropped to 68% and remained at that level while CP continued to go through cycles of being depleted and replenished, each time falling to a lower percentage of its resting levels.

Through additional measures of muscle glucose, glycogen, lactate, and pyruvate, Gaitanos et al. (1993) calculated that for the first sprint, 44 % of the anaerobic ATP

production was from glycolysis, with the remainder being derived primarily from CP degradation. During the last sprint, it was calculated that 80 % was supplied from CP and only 20 % from glycolysis. The authors noted that in four of seven subjects the estimated ATP production from glycolysis in the last sprint was zero. It was suggested that acidosis caused by maximal anaerobic glycogenolysis could have been responsible for the decreased glycogenolytic and glycolytic rates observed in the last interval. The possibility of an aerobic contribution to energy production in the last sprint was also discussed, based on the fact that MPO was still 73 % of the initial sprint, despite a substantial drop in anaerobic glycogenolysis.

These results would seem to provide some support for how passive recovery could be superior to active for this type of activity. If CP degradation plays a progressively more important role in ATP production as the intervals progress, then getting maximal repletion of this substrate is key to the ability to sustain power. The fact that the current study found that either passive recovery or a combination recovery was superior to active recovery even when only five intervals are performed indicates that the reliance on CP degradation likely starts very quickly.

In an investigation of the effects of creatine supplementation on the performance of repeated cycle sprints, Balsom, Ekblom, Söderlund, Sjödin, and Hultman (1993) found that the concentration of blood lactate decreased with supplementation, even when the amount of work being performed had increased. It was postulated that the mechanism for both the increased performance and decreased blood lactate concentration was higher resting values of CP as well as an increased rate of CP resynthesis during recovery. Balsom, Söderlund, Sjödin, and Ekblom (1995) conducted a similar study and took biopsies from the vastus lateralis. After 5 sprints CP levels decreased to 35 % of resting

values without creatine supplementation and to 46 % after creatine supplementation. Muscle lactate values were also lower after the creatine supplementation.

These studies demonstrate that higher initial CP levels will enhance this type of performance. If, as previously discussed, acidosis from anaerobic glycolysis is responsible for decreased glycolytic rates and enhanced CP levels allow for less reliance on anaerobic glycolysis, the resulting lower lactate levels could help to maintain the contribution of ATP from anaerobic glycolysis. The relevant point to the current study is that the maximum replenishment of CP stores during recovery periods will allow for the best maintenance of power.

The results obtained in the current study using "difference scores" did not support those found using the means, however, this can likely be explained by the general trend seen in the power scores throughout the 10 intervals. Maximal power output is generally not seen in the first interval, likely due to a warm-up effect, both physically and mentally. Making a maximal effort against high resistance for every interval involves a significant psychological component in addition to the physical effort. Despite efforts made to prevent the subjects from pacing themselves, in many cases power output would increase slightly for the last one or two intervals. This resulted in an initial increase in power output, followed by a decrease and then another increase towards the end of the intervals. The means of the first and last three scores were used to calculate the difference score in order to minimize the effects of extreme high and low scores. However, due to this non-linear trend, this may have had the effect of minimizing the differences seen within each condition as well as across conditions.

There is also some debate as to the reliability of difference scores in general. In a discussion on this topic, Allen and Yen (1979) state that a final difference score tends to

be more unreliable than the two scores used in its calculation. The error variance of a difference score will be as large or larger than the error variance of the two original scores. Due to the nature of this type of testing and the question surrounding the reliability of difference scores, it suggested that this may not be an appropriate method for trying to quantify fatigue for interval testing.

The mean self-selected recovery intensity in the current study was 37.5 % of $\dot{V}O_2max$, with a range of 30 to 47.8 %. This is within the optimal range for active recovery stated by Belcastro and Bonen (1975) and Davies, Knibbs, and Musgrove (1970). However, since the recovery intervals are preceded by supramaximal workloads, these percentages do not reflect the actual oxygen consumption during recovery, which was not measured. CP resynthesis has been shown to be dependent on oxygen delivery to the muscle (Sahlin, Harris, & Hultman, 1979), therefore the actual oxygen consumption during recovery could be a variable of interest. Balsom et al. (1993) did measure oxygen uptake during the 6 s sprint as well as the subsequent 30 s passive recovery interval for sprints seven, eight, and nine of a 10 interval protocol and found that the actual oxygen consumption was 71 % of $\dot{V}O_2max$. Balsom, Gaitanos, Ekblom, and Sjödin (1994) also found an oxygen consumption of 72 % for sprints six, seven, eight, and nine of a 10 interval protocol with passive recovery. Unfortunately, there are no measures for active recovery periods with this type of protocol for comparison.

This study has demonstrated that passive recovery or a combination of active and passive recovery is superior to active recovery alone for the maintenance of power output over repeated intervals of 6 s cycle sprints. This was true whether the duration of the recovery interval was 30 or 60 s, and for both 5 and 10 intervals. It is assumed that this

pattern exists because passive recovery allows for a more complete repletion of ATP and CP stores in the working muscle.

Conclusions

- Passive recovery or a combination of half-active, half-passive recovery is superior to active recovery for repeated 6 s sprint intervals

- This pattern exists whether the recovery intervals are 30 or 60 s in duration, with one exception; with 60 s recovery intervals, passive recovery is superior to the combination recovery which is in turn superior to active recovery for the maintenance of peak power output.

- The pattern is also identical with respect to peak power output and mean power output whether 5 or 10 intervals are performed

- The fatigue index for each 6 s interval is not influenced by recovery mode with one exception; with 60 s of recovery over 10 intervals the combination recovery mode produced a lower fatigue index than both active and passive recovery

Future Research Questions

1. What are the differences in the depletion and repletion of ATP and CP for active versus passive recovery for 6 s cycle sprint intervals?

2. What are the differences in levels of muscle lactate, glucose, and glycogen for active versus passive recovery for 6 s cycle sprint intervals?

3. Does the proportion of fast twitch fibers to slow twitch fibers influence the superiority of one recovery mode over another?

4. Are there differences in recovery patterns between males and females?

5. Does the load used for the work interval influence the recovery pattern?

6. What influence does training background have on recovery?

7. Are there differences in recovery patterns between adults and children?

References

Ahmaidi, S. , Granier, P. , Taoutaou, Z. , Mercier, J. , Dubouchaud, H. , & Prefaut, C. (1996). Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. Medicine and Science in Sports and Exercise, 28, 450-456.

Ainsworth, B. E. , Serfass, R. C. , & Leon, A. S. (1993). Effects of recovery duration and blood lactate level on power output during cycling. Canadian Journal of Applied Physiology, 18, 19-30.

Allen, M. J. & Yen, W. M. (1979). Introduction to measurement theory. Monterey, CA: Brooks/Cole Publishing Company.

Balsom, P. D. , Ekblom, B. , Söderlund, K. , Sjödin, B. , & Hultman, E. (1993). Creatine supplementation and dynamic high-intensity intermittent exercise. Scandinavian Journal of Medicine & Science in Sports, 3, 143-149.

Balsom, P. D. , Gaitanos, G. C. , Ekblom, B. , & Sjödin, B. (1994). Reduced oxygen availability during high intensity intermittent exercise impairs performance. Acta Physiologica Scandinavica, 152, 279-285.

Balsom, P. D. , Söderlund, K. , Sjödin, B. , & Ekblom, B. (1995). Skeletal muscle metabolism during short duration high intensity exercise: Influence of creatine supplementation. Acta Physiologica Scandinavica, 154, 303-310.

Bangsbo, J. , Johansen, L. , & Saltin, B. (1993). The effect of severe exercise on fatigue and anaerobic energy production during subsequent intense exercise – the importance of active recovery. In T. Reilly, J. Clarys, & A. Stibbe. (Eds.), Science and Football II (pp. 107-113). London: E&FN SPON.

Belcastro, A. N. & Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 39, 932-936.

Canadian Society for Exercise Physiology. (1993). Certified Fitness Appraiser resource manual. Gloucester, Canada: Author.

Coaching Association of Canada. (1981). National Coaching Certification Program Coaching Theory: Level 3. Ottawa, Canada: Author.

Davies, C. T. M. , Knibbs, A. V. , & Musgrove, J. (1970). The rate of lactic acid removal in relation to different baselines of recovery exercise. Internationale Zeitschrift fuer Angewandte Physiologie Einschleisslich Arbeits physiologie, 28, 155-161.

Dodd, S. , Powers, S. K. , Callender, T. , & Brooks, E. (1984). Blood lactate disappearance at various intensities of recovery exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 57, 1462-1465.

Fitts, R. H. & Holloszy, J. O. (1976). Lactate and contractile force in frog muscle during development of fatigue and recovery. American Journal of Physiology, 231, 430-433.

Gaitanos, G. C. , Williams, C. , Boobis, L. H. , & Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. Journal of Applied Physiology, 75, 712-719.

Gisolfi, C. , Robinson, S. , & Turrell, S. (1966). Effects of aerobic work performed during recovery from exhausting work. Journal of Applied Physiology, 21, 1767-1772.

Hamilton, A. L. , Nevill, M. E. , Brooks, S. , & Williams, C. (1991). Physiological responses to maximal intermittent exercise: Differences between endurance-trained runners and games players. Journal of Sports Sciences, 9, 371-382.

Hermansen, L. & Osnes, J. -B. (1972). Blood and muscle pH after maximal exercise in man. Journal of Applied Physiology, 32, 304-308.

Hermansen, L. & Stensvold, I. (1972). Production and removal of lactate during exercise in man. Acta Physiologica Scandinavica, 86, 191-201.

Hildebrandt, W. , Schütze, H. , & Stegemann, J. (1992). Cardiovascular limitations of active recovery from strenuous exercise. European Journal of Applied Physiology and Occupational Physiology, 64, 250-257.

Howell, D. C. (1995). Fundamental statistics for the behavioural sciences (3rd ed.). Belmont, CA: Duxbury Press.

Jacobs, I. , Tesch, P. A. , Bar-Or, O. , Karlsson, J. , & Dotan, R. (1983). Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 55, 365-367.

Juel, C. , Bangsbo, J. , Graham, T. , & Saltin, B. (1990). Lactate and potassium fluxes from human skeletal muscle during and after intense, dynamic, knee extensor exercise. Acta Physiologica Scandinavica, 140, 147-159.

Kaczynski, M. , Montgomery, D. L. , Koziris, P. , Travlos, A. K. , & Turcotte, R. A. (1988). The effects of active and passive recovery on blood lactate concentration and performance in a simulated ice hockey task (Abstract). Canadian Journal of Sport Sciences, 13, 61P-62P.

Margaria, R. , Cerretelli, P. , Di Prampero, C. , Massari, C. , & Torelli, G. (1963). Kinetics and mechanism of oxygen debt contraction in man. Journal of Applied Physiology, 18, 371-377.

Margaria, R. , Oliva, R. D. , Di Prampero, P. E. , & Cerretelli, P. (1969). Energy utilization in intermittent exercise of supramaximal intensity. Journal of Applied Physiology, 26, 752-756.

Mayhew, J. L. & Salm, P. C. (1990). Gender differences in anaerobic power tests. European Journal of Applied Physiology and Occupational Physiology, 60, 133-138.

McCartney, N. , Spriet, L. L. , Heigenhauser, G. J. F. , Kowalchuk, J. M. , Sutton, J. R. , & Jones, N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. Journal of Applied Physiology, 60, 1164-1169.

Nakamaru, Y. & Schwartz, A. (1972). The influence of hydrogen ion concentration on calcium binding and release by skeletal muscle sarcoplasmic reticulum. Journal of General Physiology, 59, 22-32.

Ross, W. D. & Marfell-Jones, M. J. (1991). Kinanthropometry. In J. D. MacDougall, H. A. Wenger, & H. J. Green (Eds.), Physiological testing of the high-performance athlete (2nd ed., pp. 223-308). Champaign, IL: Human Kinetics Books.

Sahlin, K. , Harris, R. C. , & Hultman, E. (1979). Resynthesis of creatine phosphate in human muscle after exercise in relation to intramuscular pH and availability of oxygen. Scandinavian Journal of Clinical and Laboratory Investigation, 39, 551-557.

Signorile, J. F. , Ingalls, C. , & Tremblay, L. M. (1993). The effects of active and passive recovery on short-term, high intensity power output. Canadian Journal of Applied Physiology, 18, 31-42.

Sjøgaard, G. , Adams, R. P. , & Saltin, B. (1985). Water and ion shifts in skeletal muscle of humans with intense dynamic knee extension. American Journal of Physiology, 248, R190-R196.

Stamford, B. A. , Moffatt, R. J. , Weltman, A. , Maldonado, C. , & Curtis, M. (1978). Blood lactate disappearance after supramaximal one-legged exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 45, 244-248.

Stamford, B. A. , Weltman, A. , Moffatt, R. , & Sady, S. (1981). Exercise recovery above and below anaerobic threshold following maximal work. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 51, 840-844.

Tesch, P. , Sjödin, B. , Thorstensson, A. , & Karlsson, J. (1978). Muscle fatigue and its relation to lactate accumulation and LDH activity in man. Acta Physiologica Scandinavica, 103, 413-420.

Tesch, P. A. , Thorsson, A. , & Fujitsuka, N. (1989). Creatine phosphate in fiber types of skeletal muscle before and after exhaustive exercise. Journal of Applied Physiology, 66, 1756-1759.

Trivedi, B. & Danforth, W.H. (1966). Effect of pH on the kinetics of frog muscle phosphofructokinase. Journal of Biological Chemistry, 241, 4110-4114.

Weltman, A. , Stamford, B. A. , Moffatt, R. J. , & Katch, V. L. (1977). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. Research Quarterly, 48, 786-796.

Appendix A

Review of Literature

Introduction

This review of literature will address four main topics: the ATP-CP system, the causes of muscular fatigue, studies which have compared active and passive recovery modes, and the issue of recovery from supramaximal efforts of less than 10 s.

The ATP-CP System

ATP-CP Depletion

The adenosine triphosphate-creatine phosphate (ATP-CP) system is the immediate energy source in skeletal muscle. Both ATP and CP will decrease upon activation of a muscle fiber, but not to the same degree. At very heavy loads, CP can fall to as low as 0, but a large store of ATP will remain (Hultman, Bergström, & McLennan Anderson, 1967). Hultman et al. as well as Karlsson, Diamant, and Saltin (1971) reported that ATP levels decrease when CP levels are low, Karlsson et al. stating a threshold level of 60 to 80% of the initial resting CP values.

Hultman et al. (1967) states that ATP usually does not decrease below about 60% of resting initial values. After examining the data published in 14 different studies that measured ATP and CP depletion through muscle biopsy (Balsom, Söderlund, Sjödin, & Ekblom, 1995; Bangsbo, Johansen, & Saltin, 1993; Cheetham, Boobis, Brooks, & Williams, 1986; Gaitanos, Williams, Boobis, & Brooks, 1993; Hirvonen, Rehunen, Rusko, & Härkönen, 1987; Hultman et al., 1967; Karlsson et al., 1971; Karlsson, Nordesjö, Jorfeldt, & Saltin, 1972; Kim, Kim, Lee, Kim, & Son, 1993; Knuttgen & Saltin, 1972; Jacobs et al., 1982; McCartney et al., 1986; Tesch & Karlsson, 1984; Tesch, Thorsson, &

Fujitsuka, 1989), it seems clear that this is most likely the case, since the lowest value found for ATP was 53% of the initial resting level (Hultman et al., 1967). This was found in an individual subject during cycle exercise that lead to exhaustion in 1 to 2 minutes. The average ATP level at exhaustion was 62%. In this same protocol, the CP level for one subject was reported as 0, and the group average was 10% of initial resting level. It should be noted that in studies where data for individual subjects was reported (McCartney et al., 1986; Knuttgen & Saltin, 1972; Hultman et al., 1967), there appears to be considerable variability in depletion between subjects.

ATP-CP Repletion

It is commonly stated that the half-time for the replenishment of CP is from 30 to 60 s (Tesch et al., 1989; Petersen & Cooke, 1994). Petersen and Cooke reported a value of 26 s through the use of phosphorus nuclear magnetic resonance spectroscopy (^{31}P-NMR), but did not provide any actual CP values making further comment difficult. Tesch et al. stated that their results agree with other studies obtaining values of 30 to 60 s, but for the mean values of CP that they provide, a half recovery of CP would be equal to 70% of the initial resting value, and the actual level after 60 s is only 55%.

Similarly, McCartney et al. (1986), stated that although the high energy phosphates were almost replenished at the end of a 4 minute recovery period, subsequent power output was substantially diminished. The validity of concluding that ATP and CP were replenished is questionable, however, because recovery was only measured in two subjects and their individual results were not the same. Hultman et al. (1967) also measured ATP and CP recovery. Again there was considerable variability between subjects and only a small amount of data for most measures, but after 5 minutes, the ATP

of two out of five subjects was at least 100% replenished, and three out of five were back to at least 100% CP. At 10 minutes recovery two out of two subjects were more than 100% recovered in both ATP and CP.

Contrary to this, Bangsbo et al. (1993) found that after 10 minutes of recovery, ATP and CP replenishment were still incomplete. The protocols and intensities in these two studies are quite different, however if the half-recovery time of CP from 0 is even as long as 60 s, recovery should be 99.9% complete in 10 minutes. Similarly, Knuttgen and Saltin (1972) stated that after 6 minutes of recovery, ATP and CP stores were not completely recovered.

Changes to ATP-CP Levels With Training

Karlsson et al. (1971) found that a group of subjects with higher $\dot{V}O_2max$ values (average 61 $ml \cdot kg^{-1} \cdot min^{-1}$) and presumably an overall higher fitness level than the lower $\dot{V}O_2max$ group, had higher resting ATP levels. In testing a group of military conscripts before and after training, Karlsson et al. (1972) found that their resting concentration of ATP increased. Contrary to these findings, Linossier, Denis, Dormois, Geyssant, and Lacour (1993) found no change in resting mean ATP and CP with sprint training. While not necessarily related to training, Hirvonen et al. (1987) found that better sprinters used more CP and used it faster than their slower (yet still highly trained) counterparts. It is possible that this is due to genetic factors.

Causes of Muscular Fatigue

Muscle and Blood Lactate

Muscle and blood lactate are commonly measured in studies on muscular fatigue. Cause and effect conclusions cannot be drawn from these measures, because both muscle and blood lactate values are a reflection of the efflux and uptake by both mediums (Jorfeldt, 1970). In each case, an elevated level of lactate is generally assumed to be detrimental to physical performance and it is for this reason that muscle and blood lactate clearance are often studied. In general, blood lactate is considered to be a reflection of lactic acid produced in the muscle (Weltman, Stamford, & Fulco, 1979). In this review, muscle and blood lactate will be considered equally in this respect unless the purpose of the study in question warrants a different interpretation.

Effects of Lactate

In the athletic community, lactate accumulation in working muscle is accepted as the cause of perceived fatigue during high intensity exercise (Bangsbo et al., 1993). Based on the number of studies using lactate clearance as the measure of recovery (for example Dodd, Powers, Callender, & Brooks, 1984; Hermansen & Stensvold, 1972; Stamford, Moffatt, Weltman, Maldonado, & Curtis, 1978), the role of lactate in fatigue is also perceived to be important by the scientific community.

Fitts and Holloszy (1976) found that an increase in muscle lactate concentration was inversely proportional to a decrease in contractile force in isolated frog sartorius muscles. Karlsson, Bonde-Petersen, Henriksson, and Knuttgen (1975) suggested that elevated muscle lactate concentrations, or related factors such as an elevated H^+

concentration served as limiting factors in muscular performance. Acidosis resulting from lactic acid accumulation has also been negatively correlated with sprint performance (Jones, Sutton, Taylor, & Toews, 1977). These are only three examples of observations of diminished performance that coincide with elevated levels of muscle lactate, but none can state conclusively that lactate was the only cause of fatigue or explain exactly how lactate causes this phenomenon.

Within the process that leads from the derivation of energy from a substrate to the actual contraction of a muscle fiber, there are several areas that have been proposed to be influenced by elevated muscle lactate. During exercise, pH is maintained through the binding of H^+ to hemoglobin and bicarbonate anions in plasma. When the rate of lactic acid production and the release of H^+ to plasma exceeds buffer and metabolite removal capacities, acidosis results (Ainsworth, Serfass, & Leon,1993) which decreases the affinity of phosphofructokinase (PFK) for fructose-6-phosphate, perhaps stopping lactic acid production before pH falls low enough to cause damage to the cell (Trivedi & Danforth, 1966). In addition, Karlsson, Hultén, and Sjödin (1974), found significant product inhibition of lactate dehydrogenase (LDH) at low concentrations of lactate and pyruvate, causing a decrease in rate of reaction.

An increase in $[H^+]$ also interferes with Ca^{++} binding to troponin (Fuchs, Reddy, & Briggs, 1970) and causes an increase in the Ca^{++} binding capacity of the sarcoplasmic reticulum (Nakamaru & Schwartz, 1972). These mechanisms could both serve to decrease the number of calcium ions bound to troponin during excitation-contraction coupling, therefore diminishing the number of active interactions between actin and myosin, and decreasing the contractile force of the muscle (Fitts & Holloszy, 1976).

The Role of Lactate Questioned

Despite these findings, there is evidence that lactate accumulation through such mechanisms may not be the main cause of muscular fatigue. Bangsbo et al. (1993) found that the effects of increased lactate and pH on exhaustive leg extension exercise were not very dramatic. The muscle concentration of lactate and muscle pH varied widely between subjects, and muscle pH increased after a second exercise bout, despite having been previously lowered. It was concluded that other factors must cause fatigue and impaired performance.

In research on frogs, Fitts and Holloszy (1976) reached a similar conclusion as the return of contractile force lagged behind the removal of lactate. If the development of fatigue was largely due to lactate accumulation, then there must have been a secondary effect, caused by the elevated lactate, that persisted after lactate removal. In contrast to this, Hermansen and Osnes (1972) found in humans that at the start of a fifth exercise bout, blood pH was much lower than at the start of the first or second bouts, but subjects could still maintain the same exercise intensity. They concluded that blood pH was not a limiting factor in such circumstances.

A lack of association between blood lactate and performance was also found by Weltman et al. (1979), as a significantly elevated blood lactate concentration prior to a repetition of 5 minutes of maximal exercise produced no difference in work output. It was concluded that elevated blood lactate was not detrimental to maximal effort performances. Weltman, Stamford, Moffatt, and Katch (1977) also found that lactate removal does not need to be complete in order to attain initial performance values for high intensity, short duration anaerobic exercise.

In addition, Juel, Bangsbo, Graham, and Saltin (1990) suggested that large individual variations in pH at exhaustion argue against a pH effect on Ca^{++} affinity for tropomyosin, or an inhibition of PFK as the only critical factors in fatigue. While some findings may contradict each other, the fact that the mechanisms of muscular fatigue are poorly understood is clear. A reduction in lactate concentration does not appear to be a reliable indicator of recovery from fatigue (Hildebrandt, Schütz, & Stegemann, 1992).

Other studies (Hermansen & Osnes, 1972; Meyer et al., 1991; Weltman & Regan, 1983) have also found that increased blood or muscle lactate does not correspond to diminished performance. These findings strongly suggest that lactic acid production alone is likely not a major contributor to muscular fatigue.

Other Potential Sources of Muscular Fatigue

In a study of one-legged knee extension, Sjøgaard, Adams, and Saltin (1985) proposed an alternative source of muscle fatigue. During dynamic exercise with a limited muscle mass, arterial lactate and electrolyte concentrations were slightly elevated, with more marked elevations in corresponding venous concentrations. It was felt that this, combined with increased blood flow through the exercising muscle, prevented lactate accumulation and a concomitant decrease in pH, but caused a significant loss of K^+ from the muscle. They suggested that fatigue may be related to changes in the K^+ gradient across the muscle cell membrane as opposed to acidosis in the cell. In a similar discussion, Juel et al. (1990) stated that a loss of K^+ from the muscle cell causes the speed of propagation of the action potential to be decreased, as well as a decrease in its amplitude. Only 70% of the gross K^+ efflux is pumped back into the cell by the Na-K pump. The other 30% is moved into other compartments and taken up by other tissues.

As another possibility, differences observed in the activity of LDH and LDH isoenzymes between fiber types may influence the production of lactic acid as well as its transport between fiber types, indirectly affecting contractibility and fatigue (Tesch, Sjödin, Thorstensson, & Karlsson, 1978). Also, McCartney et al. (1986) felt that it was possible that the fatigue experienced by their subjects in repetitions of 30 s supramaximal cycling could have been centrally mediated.

Active Recovery From High-Intensity Exercise

Evidence that Active Recovery is Superior to Passive

Single bout exercise. The studies of Belcastro and Bonen (1975), Gisolfi, Robinson, and Turrell (1966), and McGrail, Bonen, and Belcastro (1978) all had subjects perform high intensity exercise sessions of 3 to 6 min in duration, on either a cycle ergometer or on a treadmill, and then monitored their blood lactate during recovery. In each case, active recovery was seen to provide superior lactate removal.

Dodd et al. (1984), Stamford et al. (1978), and Stamford, Weltman, Moffatt, and Sady (1981) all examined supramaximal cycle exercise lasting from 40 to 60 s, and also found that lactate clearance was enhanced with active recovery.

Exercise repeated after a recovery period. Having subjects repeat a test that causes fatigue can provide more information than a single test, as lactate may not be the most important factor involved in fatigue, as previously discussed. Bangsbo et al. (1993) had subjects perform a single repeat of one-limb knee extension to exhaustion separated by 10 minutes of active or passive recovery, seven 15 s intervals with 15 s recovery, and another 2.5 min recovery period. They found more lactate removal and a longer

performance time before exhaustion with active recovery. Weltman et al. (1979) had subjects cycle at an intensity greater than $\dot{V}O_2max$ for 5 minutes, rest (either actively or passively) for 20 minutes, and then repeat the high-intensity exercise. They also found that the active recovery periods allowed for better maintenance of intensity during the repeated test. Weltman et al. (1977) had subjects perform 2 one minute all-out cycle ergometer tests, separated by either 10 or 20 minutes of active or passive recovery. Active recovery resulted in higher post-recovery pedal revolutions.

Multiple repetitions of exercise. Hermansen and Stensvold (1972) had subjects do supramaximal runs on a treadmill for 60 s, followed by 4 min of rest. This was repeated 3 times and followed by 30 minutes of active or passive recovery at different intensities. Lactate removal rates were measured, and the rate during active recovery was faster.

In examining active versus passive recovery with hockey players, Kaczynski, Montgomery, Koziris, Travlos, and Turcotte (1988) required players to do 4 repeats of a repeated sprint skate (each of which takes about 15 s), then recover for 6 min. This was repeated 6 times. The active recovery modes produced lower blood lactates and better repeat performance.

Signorile, Ingalls, and Tremblay (1993) had subjects perform 8 repeats of a 6 s supramaximal cycling effort, separated by 30 s of active or passive recovery. The active recovery allowed for the best maintenance of power output across the trials. Recently, Ahmaidi et al. (1996) also used 6 s sprints as a work interval, but in this case the intensity of the work was increased with every trial and the recovery was 5 minutes of active or passive cycling. In this case active recovery was also found to be superior to passive at the higher work intensities producing higher peak power output and greater lactate clearance. It should be noted that the resistance setting used by Signorile et al. was 140 $g \cdot kg^{-1}$, and

the resistance at which active recovery was found to be beneficial by Ahmaidi et al. was approximately 84 $g \cdot kg^{-1}$. The same relationship was found at the approximate intensities of 113 and 127 $g \cdot kg^{-1}$.

Benefits of Active Recovery

According to Weltman et al. (1977), the benefits of active recovery include lactate removal, tissue temperature regulation, glycogen resynthesis, and phosphate resynthesis. Lactate removal during passive recovery may be ineffective due to low blood flow rates. Skeletal muscle blood flow increases with increasing exercise intensity, therefore facilitating the transport of lactate to removal sites (Belcastro & Bonen, 1975; Gisolfi et al., 1966). The 'muscle pump' mechanism and oxidation by neighboring muscle fibers is also enhanced with active recovery (Signorile, et al., 1993).

Because the role of lactate clearance in repeat performance has been brought into question, it seems that there are other benefits to active recovery that have either not been examined or clearly elucidated. It is clear, however, that for the types of activity and recovery that have thus far been examined, active recovery, through whatever mechanisms, is superior to passive.

Intensity of Active Recovery

Many different intensities have been used in studies of active recovery. Dodd et al. (1984), Belcastro and Bonen (1975), Weltman et al. (1979), Stamford et al. (1978), Stamford et al. (1981), Kaczynski et al. (1988), and Davies, Knibbs, and Musgrove (1970), all incorporated recovery intensities ranging from 15 to 80.8% of $\dot{V}O_2max$ into their studies. In all cases, an intensity between 30 and 45% of $\dot{V}O_2max$ was determined to

be optimum. In all of these studies, recovery was carried out on a cycle ergometer. Belcastro and Bonen (1975) determined that self-selected intensities for recovery on the cycle ergometer were within an optimal range for recovery. Kaczynski et al. (1988) also incorporated a self-selected recovery mode, while skating, and the intensities selected by the subjects did not produce as favorable results as cycling at 40% $\dot{V}O_2$max. The skating was, however, still superior to passive recovery.

It was suggested by Stamford et al. (1981) that a progressive decrease in exercise intensity from above the anaerobic threshold (AT) to below it could be more effective than a single exercise intensity of either above or below AT. Dodd et al. (1984) tested this hypothesis, and found that a recovery intensity of 35% $\dot{V}O_2$max was superior to such a high-to-low combination. The combination recovery was, however, superior to an intensity of 65% $\dot{V}O_2$max and to passive recovery.

It should be noted that the optimal intensity for recovery can be difficult to determine, due to individual subject differences (Dodd et al., 1984). Also, it is possible that the optimal intensity for treadmill exercise could be higher than for cycling exercise, based on data from Hermansen and Stensvold (1972), where the highest lactate removal rate for treadmill running was seen at 63% $\dot{V}O_2$max. It is also possible that these subjects were more highly trained than those in the cycling studies (Belcastro & Bonen, 1975).

Recovery from Supramaximal Exercise of less than 10 s

According to Margaria, Oliva, Di Prampero, & Cerretelli (1969), lactic acid production began only after 10 to 15 s, after adenosine triphosphate (ATP) and creatine phosphate (CP) stores had been depleted, or had reached a critical level of depletion.

Therefore, after a short recovery period the phosphagen stores were replenished, and the performance could be repeated using the same energy source. No appreciable lactic acid was formed and accumulated in blood, therefore the exercise could be repeated indefinitely.

If energy pathways are sequentially linked as stated above, it is possible that active recovery between supramaximal efforts of less than 10 s duration would only serve to further deplete the compromised phosphagen stores. Without sufficient recovery time, the phosphagen stores would be further depleted with every repetition and the working musculature would become increasingly reliant on anaerobic glycolysis (Signorile et al., 1993).

Since the time of the study by Margaria et al. (1969), it has been shown that there is pronounced muscle lactate accumulation within 10 s of the initiation of supramaximal cycling, indicating that anaerobic glycolysis has been extensively activated within this time (Jacobs, Tesch, Bar-Or, Karlsson, & Dotan, 1983; McCartney et al., 1986). According to Koziris and Montgomery (1991), anaerobic glycolytic metabolism reaches a maximal level within the first 15 s of all-out cycling.

In such short duration efforts, not only is significant lactate produced, but the replenishment of phosphagen stores does not guarantee a return to the initial level of performance. In the study of McCartney et al. (1986), it was stated that the resynthesis of both ATP and CP was almost complete during the recovery periods, but the power output on repeated trials was substantially lower than for the initial test. This was perhaps an over-generalization, however, as it was based on the data of two subjects, taken after 4 minutes of rest after the second and fourth exercise periods of 30 s of maximal cycling. One of the subjects demonstrated ATP recovery above resting values and CP recovery to

only 78 and 84% of resting. The other subject's ATP recovered to 83 and 76% and CP to 72 and 75%. Not only are the results quite different between subjects for ATP replenishment, but it is questionable whether CP values between 72 and 84% of resting can be considered to be almost complete recovery.

There is an equilibrium between CP and C + P_i that is affected by changes in pH. An increase in acidity can result in a decrease in the concentration of CP (Karlsson et al., 1975). If lactate is produced in significant quantities during this type of exercise, then perhaps active recovery would be beneficial. In fact it has been shown to be superior to passive recovery (Signorile et al., 1993; Ahmaidi et al., 1996). These studies are the only ones to date to examine active versus passive recovery for supramaximal intermittent exercise of such a brief duration. In the Signorile et al. study, the work time was 6 s, with 30 s recovery intervals. Based on the work of Margaria et al. (1969), it is possible that longer recovery intervals would permit improved maintenance of power output with passive recovery, and conceivable that active recovery could impair repeat performance. However, as with other types of exercise, the benefits of active recovery may not be related primarily to lactate removal. This is demonstrated by the Ahmaidi et al. study which also used 6 s supramaximal work intervals, but had 5 minutes of active or passive recovery in between. The protocols of these two studies were quite different, however both serve to begin to demonstrate that active recovery is superior to passive recovery for very short-term supramaximal performance.

Many questions remain as to the true time-course of phosphagen repletion, the role of lactate in muscular fatigue, and the many other potential causes of fatigue. What does seem clear is that individuals can respond very differently to the same stimulus and that there are likely several mechanisms responsible for fatigue, working independently and/or

as a chain of events. Active recovery has repeatedly been shown to enhance repeat performance that is considered to be anaerobic lactic or aerobic in nature, however more work needs to be done to confirm that this phenomenon applies to anaerobic alactic intervals.

References

Ahmaidi, S. , Granier, P. , Taoutaou, Z. , Mercier, J. , Dubouchaud, H. , & Prefaut, C. (1996). Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. Medicine and Science in Sports and Exercise, 28, 450-456.

Ainsworth, B. E. , Serfass, R. C. , & Leon, A. S. (1993). Effects of recovery duration and blood lactate level on power output during cycling. Canadian Journal of Applied Physiology, 18, 19-30.

Balsom, P. D. , Söderlund, K. , Sjödin, B. , & Ekblom, B. (1995). Skeletal muscle metabolism during short duration high intensity exercise: Influence of creatine supplementation. Acta Physiologica Scandinavica, 154, 303-310.

Bangsbo, J. , Johansen, L. , & Saltin, B. (1993). The effect of severe exercise on fatigue and anaerobic energy production during subsequent intense exercise - the importance of active recovery. In T. Reilly, J. Clarys, & A. Stibbe. (Eds.), Science and Football II (pp. 107-113). London: E&FN SPON.

Belcastro, A. N. & Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 39, 932-936.

Cheetham, M. E. , Boobis, L. H. , Brooks, S. , & Williams, C. (1986). Human muscle metabolism during sprint running. Journal of Applied Physiology, 61, 54-60.

Davies, C. T. M. , Knibbs, A. V. , & Musgrove, J. (1970). The rate of lactic acid removal in relation to different baselines of recovery exercise. Internationale Zeitschrift fuer Angewandte Physiologie Einschleisslich Arbeits physiologie, 28, 155-161.

Dodd, S. , Powers, S. K. , Callender, T. , & Brooks, E. (1984). Blood lactate disappearance at various intensities of recovery exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 57, 1462-1465.

Fitts, R. H. & Holloszy, J. O. (1976). Lactate and contractile force in frog muscle during development of fatigue and recovery. American Journal of Physiology, 231, 430-433.

Fuchs, F. , Reddy, V. , & Briggs, F. N. (1970). The interaction of cations with the calcium-binding site of troponin. Biochemical and Biophysical Acta, 221, 407-409.

Gaitanos, G. C. , Williams, C. , Boobis, L. H. , & Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. Journal of Applied Physiology, 75, 712-719.

Gisolfi, C. , Robinson, S. , & Turrell, S. (1966). Effects of aerobic work performed during recovery from exhausting work. Journal of Applied Physiology, 21, 1767-1772.

Hermansen, L. & Osnes, J. -B. (1972). Blood and muscle pH after maximal exercise in man. Journal of Applied Physiology, 32, 304-308.

Hermansen, L. & Stensvold, I. (1972). Production and removal of lactate during exercise in man. Acta Physiologica Scandinavica, 86, 191-201.

Hildebrandt, W. , Schütz, H. , & Stegemann, J. (1992). Cardiovascular limitations of active recovery from strenuous exercise. European Journal of Applied Physiology and Occupational Physiology, 64, 250-257.

Hirvonen, J. , Rehunen, S. , Rusko, H. , & Härkönen, M. (1987). Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. European Journal of Applied Physiology and Occupational Physiology, 56, 253-259.

Hultman, E. , Bergström, J. , & McLennan Anderson, N. (1967). Breakdown and resynthesis of phosphorylcreatine and adenosine triphosphate in connection with muscular work in man. Scandinavian Journal of Clinical and Laboratory Investigation, 19, 56-66.

Jacobs, I. , Bar-Or, O. , Karlsson, J. , Dotan, R. , Tesch, P. , Kaiser, P. , & Inbar, O. (1982). Changes in muscle metabolites in females with 30-s exhaustive exercise. Medicine and Science in Sports and Exercise, 14, 457-460.

Jacobs, I. , Tesch, P. A. , Bar-Or, O. , Karlsson, J. , & Dotan, R. (1983). Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology, 55, 365-367.

Jones, N. L. , Sutton, J. R. , Taylor, R. , & Toews, C. J. (1977). Effect of pH on cardiorespiratory and metabolic responses to exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology,43, 959-964.

Jorfeldt, L. (1970). Metabolism of L(+)-lactate in human skeletal muscle during exercise. Acta Physiologica Scandinavica, (Suppl. 338).

Juel, C. , Bangsbo, J. , Graham, T. , & Saltin, B. (1990). Lactate and potassium fluxes from human skeletal muscle during and after intense, dynamic, knee extensor exercise. Acta Physiologica Scandinavica, 140, 147-159.

Kaczynski, M. , Montgomery, D. L. , Koziris, P. , Travlos, A. K. , & Turcotte, R. A. (1988). The effects of active and passive recovery on blood lactate concentration and performance in a simulated ice hockey task (Abstract). Canadian Journal of Sport Sciences, 13, 61P-62P.

Karlsson, J. , Bonde-Petersen, F. , Henriksson, J. , & Knuttgen, H. G. (1975). Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. Journal of Applied Physiology, 38, 763-767.

Karlsson, J. , Diamant, B. , & Saltin, B. (1971). Muscle metabolites during submaximal and maximal exercise in man. Scandinavian Journal of Clinical and Laboratory Investigation, 26, 385-394.

Karlsson, J. , Hultén, B. , & Sjödin, B. (1974). Substrate activation and product inhibition of LDH activity in human skeletal muscle. Acta Physiologica Scandinavica, 92, 21-26.

Karlsson, J. , Nordesjö, L. -O. , Jorfeldt, L. , & Saltin, B. (1972). Muscle lactate, ATP, and CP levels during exercise after physical training in man. Journal of Applied Physiology, 33, 199-203.

Kim, K. -J. , Kim, Y. -S. , Lee, D. -K. , Kim, C. -K. , & Son, N. -W. (1993). Measurement of anaerobic performance on the basis of the differences between ATP-PC and lactate system. Korean Journal of Sport Science, 5, 82-110.

Knuttgen, H. G. & Saltin, B. (1972). Muscle metabolites and oxygen uptake in short-term submaximal exercise in man. Journal of Applied Physiology, 32, 690-694.

Koziris, L. P. & Montgomery, D. L. (1991). Blood lactate concentration following intermittent and continuous cycling tests of anaerobic capacity. European Journal of Applied Physiology and Occupational Physiology, 63, 273-277.

Linossier, M. -T. , Denis, C. , Dormois, D. , Geyssant, A. , & Lacour, J. R. (1993). Ergometric and metabolic adaptation to a 5-s sprint training programme. European Journal of Applied Physiology and Occupational Physiology, 67, 408-414.

Margaria, R. , Oliva, R. D. , Di Prampero, P. E. , & Cerretelli, P. (1969). Energy utilization in intermittent exercise of supramaximal intensity. Journal of Applied Physiology, 26, 752-756.

McCartney, N. , Spriet, L. L. , Heigenhauser, G. J. F. , Kowalchuk, J. M. , Sutton, J. R. , & Jones, N. L. (1986). Muscle power and metabolism in maximal intermittent exercise. Journal of Applied Physiology, 60, 1164-1169.

McGrail, J. C. , Bonen, A. , & Belcastro, A. N. (1978). Dependence of Lactate Removal on muscle metabolism in man. European Journal of Applied Physiology, 39, 89-97.

Meyer, R. A. , Adams, G. R. , Fisher, M. J. , Dillon, P. F. , Krisanda, J. M. , Brown, T. R. , & Kushmerick, M. J. (1991). Effect of decreased pH on force and phosphocreatine in mammalian skeletal muscle. Canadian Journal of Physiology and Pharmacology, 69, 305-310.

Nakamaru, Y. & Schwartz, A. (1972). The influence of hydrogen ion concentration on calcium binding and release by skeletal muscle sarcoplasmic reticulum. Journal of General Physiology, 59, 22-32.

Petersen, S. R. & Cooke, S. R. (1994). Effects of endurance training on recovery from high-intensity exercise. In F. I. Bell & G. H. VanGyn (Eds.), Access to Active Living: Proceedings of the 10th Commonwealth & International Scientific Congress (pp.227-231). Victoria, Canada: University of Victoria.

Signorile, J. F. , Ingalls, C. , & Tremblay, L. M. (1993). The effects of active and passive recovery on short-term, high intensity power output. Canadian Journal of Applied Physiology, 18, 31-42.

Sjøgaard, G. , Adams, R. P. , & Saltin, B. (1985). Water and ion shifts in skeletal muscle of humans with intense dynamic knee extension. American Journal of Physiology, 248, R190-R196.

Stamford, B. A. , Moffatt, R. J. , Weltman, A. , Maldonado, C. , & Curtis, M. (1978). Blood lactate disappearance after supramaximal one-legged exercise. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology,45, 244-248.

Stamford, B. A. , Weltman, A. , Moffatt, R. , & Sady, S. (1981). Exercise recovery above and below anaerobic threshold following maximal work. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 51, 840-844.

Tesch, P. A. & Karlsson, J. (1984). Muscle metabolite accumulation following maximal exercise: A comparison between short-term and prolonged kayak performance. European Journal of Applied Physiology and Occupational Physiology, 52, 243-246.

Tesch, P. , Sjödin, B. , Thorstensson, A. , & Karlsson, J. (1978). Muscle fatigue and its relation to lactate accumulation and LDH activity in man. Acta Physiologica Scandinavica, 103, 413-420.

Tesch, P. A. , Thorsson, A. , & Fujitsuka, N. (1989). Creatine phosphate in fiber types of skeletal muscle before and after exhaustive exercise. Journal of Applied Physiology, 66, 1756-1759.

Trivedi, B. & Danforth, W. H. (1966). Effect of pH on the kinetics of frog muscle phosphofructokinase. Journal of Biological Chemistry, 241, 4110-4114.

Weltman, A. & Regan, J. D. (1983). Prior exhaustive exercise and subsequent, maximal constant load exercise performance. International Journal of Sports Medicine, 4(3), 184-189.

Weltman, A. , Stamford, B. A. , & Fulco, C. (1979). Recovery from maximal effort exercise: Lactate disappearance and subsequent performance. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 47, 677-682.

Weltman, A. , Stamford, B. A. , Moffatt, R. J. , & Katch, V. L. (1977). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. Research Quarterly, 48, 786-796.

Appendix B

Means and Standard Deviations

Table 3

Means and Standard Deviations for 10 Intervals (N=21)

Measure	Recovery Condition	Mean (W•kg-1)	SD
PPO	A30	9.6	1.1
	P30	10.4	0.8
	AP30	10.2	0.8
	A60	10.5	1.3
	P60	11.2	0.9
	AP60	10.9	1.0
MPO	A30	8.3	0.9
	P30	8.9	0.8
	AP30	8.8	0.9
	A60	9.2	1.2
	P60	9.9	0.7
	AP60	9.7	1.0
FI	A30	24.6	6.9
	P30	25.4	6.7
	AP30	25.3	7.1
	A60	22.2	6.6
	P60	22.4	7.0
	AP60	19.3	7.4

Table 4

Means and Standard Deviations for 5 Intervals (N=21)

Measure	Recovery Condition	Mean (W•kg-1)	SD
PPO	A30	10.1	1.2
	P30	10.8	0.9
	AP30	10.7	0.9
	A60	10.7	1.3
	P60	11.4	1.0
	AP60	11.0	1.0
MPO	A30	8.9	1.0
	P30	9.5	0.9
	AP30	9.4	1.0
	A60	9.5	1.3
	P60	10.2	0.8
	AP60	9.9	0.9
FI	A30	22.2	6.8
	P30	22.8	6.1
	AP30	23.2	7.8
	A60	20.1	6.2
	P60	19.9	6.9
	AP60	18.3	7.2

Table 5

Means and Standard Deviations for Difference Scores (N=21)

Measure	Recovery Condition	Mean (W•kg-1)	SD
PPO	A30	1.5	1.3
	P30	1.1	1.2
	AP30	1.3	0.8
	A60	0.4	0.7
	P60	0.3	0.8
	AP60	0.4	0.7
MPO	A30	1.7	1.1
	P30	1.5	1.0
	AP30	1.5	0.9
	A60	0.7	0.8
	P60	0.7	0.7
	AP60	0.6	0.7
FI	A30	6.8	7.8
	P30	8.4	7.3
	AP30	6.0	6.0
	A60	6.1	6.6
	P60	7.3	6.8
	AP60	4.1	3.9

VITA

Surname: Doyle **Given Names:** Tracey Elizabeth

Place of Birth: Ottawa, Ontario, Canada

Educational Institutions Attended:

University of Victoria	1995 to 1998
University of Ottawa	1991 to 1995

Degrees Awarded:

B.Sc. Human Kinetics	University of Ottawa	1995

Honours and Awards:

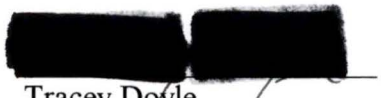
University of Victoria Fellowship	1995-98
University of Ottawa Silver Medal	1995
Canadian Society for Exercise Physiology Award	1995
University of Ottawa Merit Scholarships	1992, 1994

PARTIAL COPYRIGHT LICENSE

I hereby grant the right to lend my thesis to users of the University of Victoria Library, and to make single copies only for such users or in response to a request from the Library of any other university, or similar institution, on its behalf or for one of its users. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by me or a member of the University designated by me. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Title of Thesis:

Active vs. Passive Recovery for 6 s Supramaximal Cycle Intervals

Author 

Tracey Doyle
September 3,1998