The Aerobic Response to Intense Intermittent Exercise

by

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We accept this thesis as conforming to the required standard

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ABSTRACT

This study examined the relationships between the aerobic response to intense intermittent exercise, the ability to resist fatigue over repeated bouts and $\dot{V}O_2max$. On the basis of cycle $\dot{V}O_2max$ scores, 13 female recreational soccer players were assigned to either a low aerobic power group (LAP, n = 6, mean (SD) $\dot{V}O_2max$ = 34.4 (2.4) $mL \cdot kg^{-1} \cdot min^{-1}$) or to a high aerobic power group (HAP, n = 7, $\dot{V}O_2max$ = 47.6 (3.8) $mL \cdot kg^{-1} \cdot min^{-1}$). Within 2 weeks of the $\dot{V}O_2max$ test, subjects performed 10 6-s sprints on a cycle ergometer, loaded at .075 $kp \cdot kg^{-1}$ of body weight with 30-s of passive recovery between sprints. Following the sprints were 5 minutes of active recovery at 35% $\dot{V}O_2max$, then 10 minutes of passive recovery.

LAP and HAP subjects generated similar peak 6-s power (7.8 (1.2) vs 8.1 (0.8) $W \cdot kg^{-1}$, respectively, p =.58) but HAP had a smaller decrement in power (% DO) over the 10 sprints (LAP vs HAP: 18.0 (7.6) vs 8.8 (3.7) % DO, p =.02). The HAP group also consumed significantly more oxygen than LAP in 9 of the 10 sprint-recovery cycles (p <.05), averaging 25.9 (4.2) $mL \cdot kg^{-1} \cdot min^{-1}$ above pre-exercise levels versus 21.3 (1.7) $mL \cdot kg^{-1} \cdot min^{-1}$ in the LAP (p =.02). Modest relationships existed between the average increase in $\dot{V}O_2$ above pre-exercise values during the sprint, recovery or sprint-recovery cycles and % DO (r = -.40 to -.52, p =.07 to .17) as well as between % DO and $\dot{V}O_2max$ (r = -.65, p =.02) with a stronger relationship seen between $\dot{V}O_2max$ and the aerobic response to the sprint-recovery series (r = .76 to .78, p =.002). $\dot{V}O_2$ was

similar between groups for 14 of the 15 minutes of recovery ($p = .45$ to $.90$), with HAP consuming more oxygen than LAP during the first minute of passive recovery ($p = .04$).

Thus, the enhanced ability of the HAP group to resist fatigue during intense intermittent exercise may be related to both $\dot{V}O_2max$ and an increased aerobic contribution to sprint and recovery bouts.

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Acknowledgments

Without the support of my family, friends, job-share partner Holly, and my supervisor Howie, this thesis would never have been completed. Thank you all for your assistance and encouragement.

Dedication

I dedicate this thesis to my husband John, who folded copious quantities of laundry as I puzzled over spreadsheets and typed (with both fingers) for so many evenings and to my children, Sam, Henry, Bennett and Paige who will happily no longer have to ask "Are you STILL working on your Masters?"

Introduction

Though generally associated with exercise of long duration and low intensity, the aerobic metabolic system is stimulated even in high intensity exercise lasting a few minutes (Gaitanos, Nevill, Brooks and Williams, 1991). For example, the majority of energy required for a single bout of brief (<10 seconds) dynamic maximal exercise is provided through phosphocreatine (PCr) breakdown and glycogenolysis leading to lactate formation (Jacobs, Tesch, Bar-Or, Karlsson and Dotan, 1983), however aerobic metabolism may supply as much as 20% of the energy (Bogdanis, Nevill, Lakomy, Graham and Louis, 1996b).

Following exercise, oxygen consumption ($\dot{V}O_2$) remains elevated for some time (Berg, 1991; Gaesser and Brooks, 1984). At least part of the oxygen consumed immediately post-exercise is used to restore PCr in muscle and oxygen stores in tissue and blood (diPrampero, Peeters and Margaria, 1973). $\dot{V}O_2$ also remains elevated as a result of increased core temperature (Brooks, Hittleman, Faulkner and Beyer, 1971), increased circulating levels of epinephrine and norepinephrine (Gladden, Stainsby and McIntosh, 1982), elevated cardiac and respiratory function (Bahr and Sejersted, 1991), increased lactate oxidation and glycogen repletion (Gaesser and Brooks, 1984) during the post-exercise period. In intermittent exercise, if the recovery period is insufficient to allow complete recovery of the muscle, performance on subsequent exercise bouts may be compromised. Since many team sports such as soccer and rugby require intermittent bursts of all-out effort lasting a few seconds, interspersed with periods of rest or lower intensity activity (Mayhew and Wenger, 1985; McLean,

1992), it is surprising that few studies have investigated the aerobic response to brief intermittent repeats (Buono and Roby, 1982; Chamari, Ahmaidi, Fabre, Ramonatxo and Préfaut, 1995; Gaitanos et al., 1991; Hamilton, Nevill, Brooks and Williams, 1991; Weltman, Stamford and Fulco, 1979).

When performing brief (6-s) all-out sprints, oxygen consumption increases rapidly at the onset of sprinting (Chamari et al., 1995) and increases with subsequent sprints (Chamari et al., 1995; Gaitanos et al., 1991; Hamilton et al., 1991), attaining levels as high as 70 or more per cent of $\dot{V}O_2$max prior to reaching a plateau or decreasing (Hamilton et al., 1991). During intermittent exercise, any increase in $\dot{V}O_2$ during the sprints should increase the energy available for muscle contraction by supplementing energy provided anaerobically. The increased energy should translate into more work during the sprints. Further, since aerobically supplied energy does not result in hydrogen ion accumulation, which is implicated in fatigue (Wenger and Reed, 1976), this should further contribute to the maintenance of power over repeated bouts. In support of this, Hamilton et al. (1991) found a moderate relationship between the aerobic response during repeated sprint-recovery intervals and fatigue in peak power ($r = -.60$). However, since Hamilton et al. (1991) pooled sprint and recovery oxygen consumption for each sprint-recovery interval, it is unclear whether the ability to resist fatigue over repeated sprints is related to increased utilization of oxygen during the sprint, the recovery intervals or both. Furthermore, factors affecting resting oxygen consumption did not appear to be adequately controlled (Bahr, 1992) in the study by Hamilton et al. (1991), which could have had an impact on exercise $\dot{V}O_2$.

$\dot{V}O_2$ remains elevated between repeated bouts of 6-second exercise (Chamari et al., 1995) and following repeated sprints (Hamilton et al., 1991), presumably contributing to the energy needed for recovery from the preceding sprint. Increased $\dot{V}O_2$ in the recovery interval may be associated with enhanced PCr restoration (Bogdanis, Nevill, Boobis and Lakomy, 1996a), which should result in better performance on subsequent exercise bouts. Adaptations due to aerobic training, such as increased number and size of mitochondria, enhanced capillary density and increased aerobic enzyme activity (Anderson and Hendriksson, 1977; Holloszy and Coyle, 1984), and a faster adjustment of oxygen uptake at the onset of exercise (Hickson, Bomze and Holloszy, 1978) may result in increased oxygen consumption during both the sprint and recovery intervals for endurance trained athletes. Hamilton et al. (1991) found a strong relationship between the aerobic response during the sprint-recovery interval and maximal oxygen consumption ($\dot{V}O_2max$; r = .83). However, due to the pooling of sprint and recovery oxygen consumption, it is yet to be determined if the relationship to $\dot{V}O_2max$ is with the sprint $\dot{V}O_2$, recovery $\dot{V}O_2$ or both. It is therefore the intent of this study to determine:

a) if the ability to resist fatigue over repeated sprints is related to the oxygen consumed during sprints, in the brief recovery interval between sprints, and/or over the entire sprint recovery cycle, and;

b) if the ability to resist fatigue over repeated sprints is related to oxygen consumption, is oxygen consumption during sprints, in the recovery interval or the sprint recovery cycle related to aerobic fitness ($\dot{V}O_2max$), and;

c) if the ability to resist fatigue over repeated sprints is related to oxygen consumption, is oxygen consumption during sprints, in the recovery interval or the sprint recovery cycle related to a faster adjustment of oxygen uptake at the onset of exercise.

Methodology

Subjects

Nineteen female soccer players aged 19-42 years were recruited for the study (Table 1). All subjects were members of organized soccer teams and played or practiced soccer at least twice per week. The nature and the purpose of the investigation was explained to each participant before she signed an informed consent (Appendix 1), in accordance with the Human Ethics Committee of the University of Victoria. Subjects with $\dot{V}O_2max$ scores > 43 $mL \cdot kg^{-1} \cdot min^{-1}$ were assigned to a high aerobic power group (HAP, n = 7) whereas subjects with $\dot{V}O_2max$ scores < 38 $mL \cdot kg^{-1} \cdot min^{-1}$ were assigned to the low aerobic power group (LAP, n = 6), with the intent of forming two distinct groups. The data for the 6 subjects with moderate aerobic power were not considered in comparisons between HAP and LAP groups.

Instrumentation

All exercise tests were carried out on a friction-loaded cycle ergometer (model 818, Monark) interfaced with an electronic revolution counter (Micro Projects) which enabled power output to be monitored and recorded. The product of flywheel revolutions and the load were used to determine work and power throughout each exercise period. The best average 6-s power over a sprint will be referred to as peak power (PP). The percent drop-off from highest average 6-s power occurring in one of the first 3 6-s sprints to the lowest average 6-s power in one of the final 3 6-s sprints will be referred to as percent drop-off (% DO).

Expired gases were collected using breath by breath mode and analyzed prior to, during and post-exercise using a Vmax 229 Metabolic Measurement Cart (Sensormedics) for determination of oxygen consumption ($\dot{V}O_2$), ventilation ($\dot{V}_E$), volume of carbon dioxide produced ($\dot{V}CO_2$), respiratory exchange ratio (RER) and $\dot{V}O_2$max. Subjects breathed through a low-resistance valve (Rudolph 2700). The gas analyzers were calibrated using primary standard reference gases before and after each test.

Table 1

Physical Characteristics of Low Aerobic Power (LAP) and High Aerobic Power (HAP) Groups

Group		Age (year)	Height (cm)	Weight (kg)	Sum of Skinfolds (mm)
LAP	Mean	34	167.7	68.6	87.0
	(SD)	(7)	(7.6)	(7.4)	(22.3)
HAP	Mean	30	167.3	60.4	54.3
	(SD)	(8)	(4.2)	(4.8)	(9.3)
	p	.39	.94	.03	.005

Design

Subjects participated in two laboratory sessions. In the first laboratory session, anthropometric data was collected, then the subjects performed a $\dot{V}O_2$max test and a

1-minute oxygen kinetics test, followed by familiarization to the sprint test protocol. In the second session, baseline heart rate and resting $\dot{V}O_2$ were established, then the sprint test series was performed. To control for potential effects on resting $\dot{V}O_2$, this session took place either early in the morning after an overnight fast or 4 hours postprandial with subjects having engaged in minimal activity during the day. Subjects were asked to travel to the laboratory by car, motorcycle or bus to eliminate unnecessary activity (Gore and Withers, 1990). Subjects also abstained from caffeine, alcohol, tobacco and drugs for the previous 4 hours and refrained from intense physical exercise for 24 hours prior to this session (Short and Sedlock, 1997). Intense physical exercise included any exercise session longer than 30 minutes that was perceived as intense by the subjects. The two laboratory sessions took place within a 2-week period to minimize effects of changes in training.

Procedures

$\dot{V}O_2$max test. Height, weight and sum of skinfolds (triceps, biceps, subscapular, iliac crest and medial calf) were measured, then $\dot{V}O_2$max was determined using a maximal oxygen uptake protocol on a cycle ergometer as described by Thoden (1991). Heart rate response was monitored using a Polar Heart Rate Monitor and recorded every minute throughout the test. All subjects met at least 2 of the following criteria of $\dot{V}O_2$max: a leveling or decrease in $\dot{V}O_2$ with increasing workload, RER in excess of 1.10, reaching the age-predicted maximum HR, or volitional exhaustion. After 5 minutes of recovery subjects resumed cycling at the load that elicited $\dot{V}O_2$max for one minute to estimate the rate of adjustment of oxygen

consumption at the onset of exercise. During this oxygen kinetics test subjects synchronized their pedaling rate with the sound of a metronome and the appropriate resistance on the flywheel was obtained within 5 seconds of the start of the test. After a further 10-15 minutes of recovery the subject practiced 6-second sprints on a cycle ergometer until comfortable with the protocol and confident of producing an all-out effort from a stationary start.

Resting $\dot{V}O_2$ and the sprint test series. Upon arrival at the laboratory, the subject rested for 30 minutes with the average $\dot{V}O_2$ over the last 10 minutes taken as resting $\dot{V}O_2$ (Short and Sedlock, 1997). Next, subjects performed a low-intensity warm-up consisting of stretching and submaximal cycling for 5 minutes at 50-60 rpm against a resistance of 0.5 kp followed by a moderate intensity warm-up consisting of two 10-second sprints at 85 and 115 rpm against a resistance of 1.0 kp, separated by 60 seconds of recovery. Finally, a five-minute stretching period completed the warm-up period. A similar warm up has previously been shown to result in only minor metabolic disturbances (Gaitanos, Williams, Boobis and Brooks, 1993).

The sprint test series consisted of 10 all-out 6-second sprints interspersed with 30 seconds of rest-recovery. The cycle ergometer was loaded with .075 $kp \cdot kg^{-1}$ body weight for all sprints. To standardize the measure, each sprint was initiated from a stationary start and subjects remained seated during all sprints, with feet secured to the pedals by toe clips. Subjects were encouraged to produce an all-out effort on each sprint and to avoid breath holding during exercise, as this can affect the ventilatory response (Fujihara, Hildebrandt and Hildebrandt, 1973). The rest periods between

sprints consisted of rest-recovery, with the subject seated quietly on the cycle ergometer. For the first 5 minutes following the sprint tests, the subjects performed an active recovery at a power output corresponding to 35% $\dot{V}O_2$max (Belcastro and Bonen, 1975) followed by a passive recovery for 10 minutes. During the sprint test series and for 15 minutes post-exercise, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$ and RER were monitored continuously utilizing the breath by breath mode of the MMC.

Statistical procedures

Relationships between variables were assessed using Pearson product-moment correlation coefficients. A two-way ANOVA with repeated measures was used to compare LAP versus HAP groups on performance variables (6-s work and $\dot{V}O_2$ during exercise, recovery and per sprint-recovery period) during the 10 repeats of exercise, then appropriate post hoc analysis was used to follow up significant F values. Student t-tests were used to discern differences between HAP and LAP in physical characteristics, PP, TW, % DO and $\dot{V}O_2$max. Statistical significance was accepted at the .05 level. All results are presented as means (standard deviation).

Results

While LAP and HAP differed significantly in $\dot{V}O_2max$ when expressed either in absolute (mean (SD) LAP versus HAP: 2.36 (.32) vs 2.86 (.22) $L \cdot min^{-1}$; p =.006) or relative terms (LAP versus HAP: 34.4 (2.4) vs 47.6 (3.8) $mL \cdot kg^{-1} \cdot min^{-1}$, p =.00001), there were no significant differences between groups in total work (TW) or peak 6-s power (PP) (Table 2).

As seen in Figure 1 while both groups generated similar 6-s power during the first 6 sprints (p =.13 to .69), the HAP group maintained higher power output in each of the final 4 sprints (p =.010 to .048), which resulted in the HAP group demonstrating a smaller decrement in % DO over the sprint test series (LAP vs HAP: 18.0 (7.6) vs 8.8 (3.7) %, p =.02) (Table 2).

The HAP group utilized more oxygen during 9 out of the 10 sprint-recovery cycles (p =.02 to .049, Figure 2) and tended to use more oxygen during both sprinting and recovery periods (Figure 3), though statistical significance was not demonstrated in all intervals.

No significant difference between LAP and HAP were seen for resting oxygen consumption or for the first 20 seconds of the 1-minute oxygen kinetics test (Figure 4) however, oxygen consumption was significantly higher in HAP during all subsequent 10 second periods (p =.001 to .048).

As seen in Table 3, no significant relationships were found between % DO and the increase in $\dot{V}O_2$ above pre-exercise levels during the 6-s sprints, 30-s recovery periods or the sprint-recovery cycles (r = -.40 to -.52, p =.07 to .17), however, a moderately high relationship was found between % DO and $\dot{V}O_2max$ (r = -.65, p =.02).

Finally, stronger relationships between $\dot{V}O_2$max and the aerobic response to the 6-s sprints, the 30-s recovery periods and the sprint-recovery cycles were found ($r = .76$ to $.78$, $p = .002$) (Table 3).

During active recovery, when the difference in the oxygen cost of the work was eliminated, there was no difference between HAP and LAP in $\dot{V}O_2$ ($p = .45$ to $.90$). The first minute of passive recovery was the only period during the 15-minutes of recovery where a significant difference in $\dot{V}O_2$ was seen, with HAP consuming significantly more oxygen than LAP ($p = .04$).

Table 2

Comparison Between Low Aerobic Power (LAP, n = 6) and High Aerobic Power (HAP, n = 7) Subjects' $\dot{V}O_2max$, Total Work Over 10 6-s Sprints (TW), Best Average Power Over a 6-s Sprint (PP) and Percentage Drop-off in Power Over the 10 Sprints (% DO)

Group		$\dot{V}O_2max$		TW		PP		% DO
		($L \cdot min^{-1}$)	($mL \cdot kg^{-1} \cdot min^{-1}$)	(J)	($J \cdot kg^{-1}$)	(W)	($W \cdot kg^{-1}$)	(%)
LAP	Mean	2.36	34.4	29244	424	543	7.8	18.0
	(SD)	(.32)	(2.4)	(5652)	(49)	(118)	(1.2)	(7.6)
HAP	Mean	2.86	47.6	27924	463	490	8.1	8.8
	(SD)	(.22)	(3.8)	(2918)	(37)	(48)	(0.8)	(3.7)
	p	.006	.00001	.60	.14	.30	.58	.02

Table 3

Pearson Product-moment Correlations Between the Percent Drop-off in Power (% DO), $\dot{V}O_2max$ and the Aerobic Response to the Sprint-test Series for LAP and HAP Subjects

	Average $\dot{V}O_2$ per 36-s sprint-recovery cycle ($mL \cdot kg^{-1} \cdot min^{-1}$)	Average $\dot{V}O_2$ per 6-s sprint ($mL \cdot kg^{-1} \cdot min^{-1}$)	Average $\dot{V}O_2$ per 30-s recovery ($mL \cdot kg^{-1} \cdot min^{-1}$)	$\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$)
% DO	- .41 (p =.16)	- .52 (p =.07)	- .40 (p =.17)	- .65 (p =.02)
$\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	.78 (p =.002)	.76 (p =.002)	.77 (p =.002)	

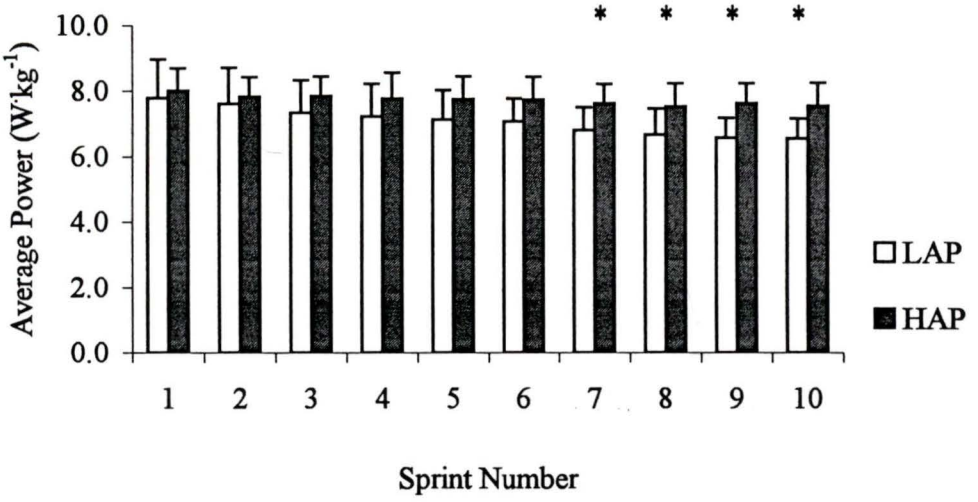

Figure 1. Average power per sprint (mean (SD)) over 10 6-s sprints for high aerobic power (HAP) and low aerobic power (LAP) groups (* p <.05. ** p <.01).

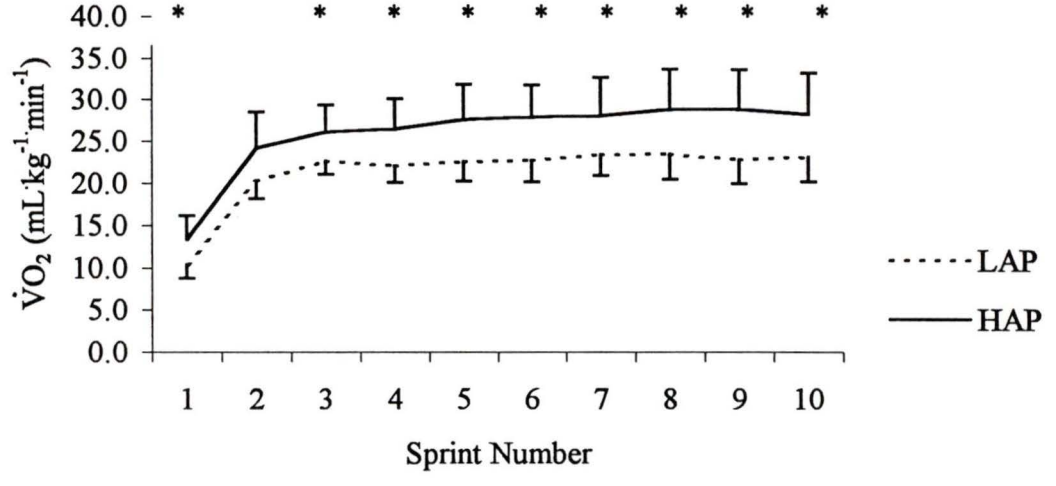

Figure 2. Mean (SD) oxygen consumption above pre-exercise levels per sprint-recovery cycle for low aerobic power (LAP) and high aerobic power (HAP) groups (* p <.05. ** p <.01).

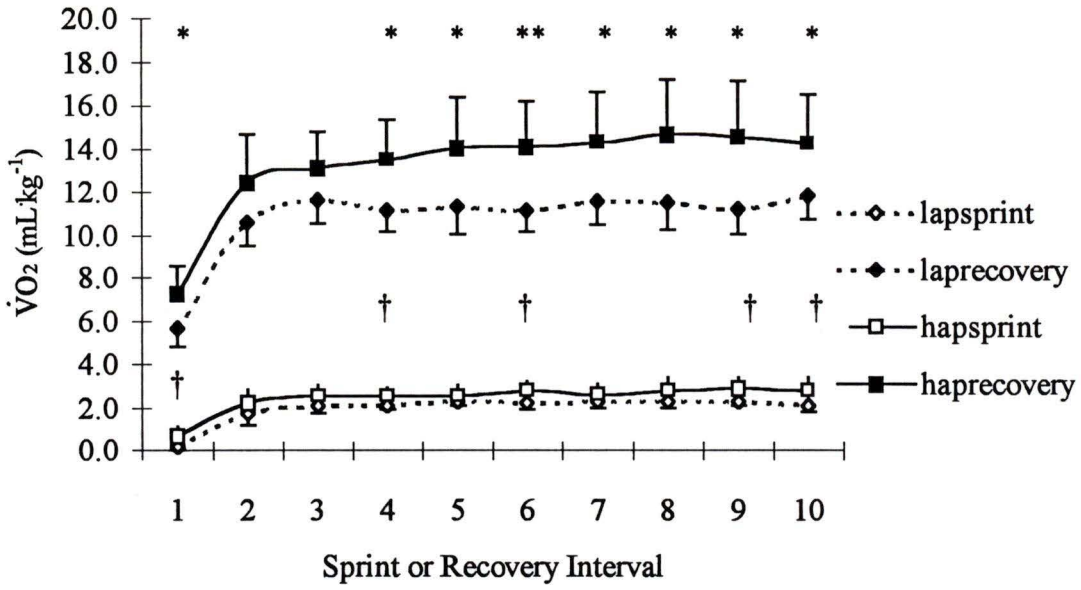

Figure 3. Mean (SD) oxygen consumption above pre-exercise levels for sprint and recovery intervals for LAP and HAP groups (* $p < .05$. ** $p < .01$ for recovery; † $p < .05$. †† $p < .01$ for sprint).

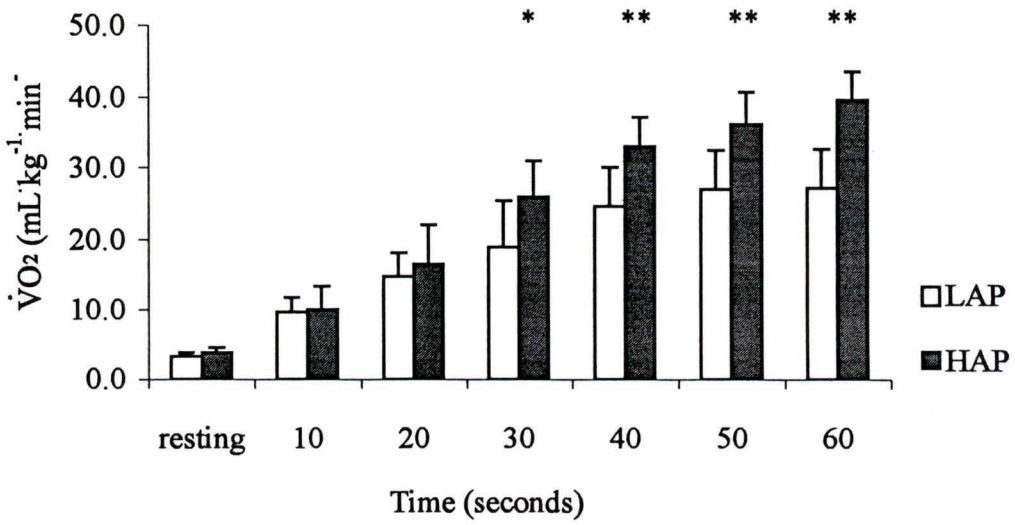

Figure 4. Mean (SD) oxygen consumption at rest and for a 1-minute oxygen kinetics test performed at 100% $\dot{V}O_2max$ (* $p < .05$. ** $p < .01$).

Discussion

As seen in Table 1, the LAP and HAP groups were similar in age (mean (SD) LAP vs HAP: 34 (7) vs 30 (8) years, p =.39) and height (LAP vs HAP: 167.7 (7.6) vs 167.3 (4.2) cm, p =.94) but the LAP group was significantly heavier (LAP vs HAP: 68.6 (7.4) vs 60.4 (4.8) kg, p =.03), which could be attributed, at least in part to their higher skinfolds (LAP vs HAP: 87.0 (22.3) vs 54.3 (9.3) mm, p =.005). As seen in Table 2, both absolute and relative maximal aerobic power were significantly higher in the HAP group (2.36 (.32) versus 2.86 (.22) $L \cdot min^{-1}$, p =.006 and 34.4 (2.4) vs 47.6 (3.8) $mL \cdot kg^{-1} \cdot min^{-1}$, p =.00001, for LAP and HAP, respectively). Compared to a population of normally active females, this range of $\dot{V}O_2max$ scores is considered good to superior (Cooper, 1977). However, Colquhoun and Chad (1986) found somewhat higher $\dot{V}O_2max$ values for state and international level female soccer players, who averaged 47.9 $mL \cdot kg^{-1} \cdot min^{-1}$ on the treadmill. Much of this difference can be explained by the difference in testing mode since treadmill $\dot{V}O_2max$ values have been shown to be 7-19% higher than cycle $\dot{V}O_2max$ scores (Léger, 1996).

It is also apparent from Table 2 that over the sprint test series no significant differences were seen between LAP and HAP groups for total work or peak power. Peak power averaged 543 (118) versus 490 (48) W (p =.30), or 7.8 (1.2) versus 8.1 (0.8) $W \cdot kg^{-1}$ (p =.58) for LAP and HAP, respectively. Few comparisons for females performing repeated 6-second exercise could be found, however Simoneau, Lortie, Boulay, Marcotte, Thibault and Bouchard (1986) found that sedentary females averaged only 5.8 $W \cdot kg^{-1}$ while 74 active females tested by Maude and Shultz (1989)

averaged 7.6 $W \cdot kg^{-1}$ for the first 5-s of a 30-second Wingate test, suggesting that, as expected, the subjects in the present study generated higher power outputs than sedentary subjects and were similar to other active females.

To minimize the effect of the difference in weight between groups ($p = .03$) results are expressed per unit of body weight. As for the 10 sprints within the sprint test series, the average power generated in each of the first 6 sprints was similar between LAP and HAP ($p = .13$ to $.69$), yet the HAP group maintained higher power in each of the final 4 sprints ($p = .01$ to $.048$) (Figure 1), resulting in the HAP group experiencing a smaller decrement in power over the 10 sprints than the LAP group (HAP vs LAP: 8.8 (3.7) vs 18.0 (7.6) % DO, $p = .02$), as seen in Table 2. It thus appears that while the LAP and HAP groups generated similar peak power, the HAP group was more successful at maintaining power and at resisting fatigue over the repeated sprints. Similar results were seen in the study by Hamilton et al. (1991) where mean power fatigue of male games players (GP) and endurance trained (ET) athletes over 10 treadmill sprints was evaluated. While assignment to GP and ET groups was on the basis of the individuals' chosen sport rather than $\dot{V}O_2max$, $\dot{V}O_2max$ was considerably higher in the ET group ($p < .01$), allowing for comparisons on the basis of aerobic power. In support of the present investigation, according to their data both ET and GP groups achieved similar peak (mean 6-s) power (NS) yet the ET demonstrated a significantly smaller decrement in power over the 10 sprints ($p < .05$). In the interpretation of their results, however, Hamilton et al. (1991) suggests that the GP tended to produce higher peak power, despite the fact that the difference between ET and GP was not statistically significant. They attributed the apparent increased peak

power in the GP to an adaptive response of sprint training, however, this likely only partially explains their results.

A moderate relationship of r = -.65 (p =.02) was seen between $\dot{V}O_2$max and the percent decrement in mean power over the 10 sprints (Table 3). Likewise, Dawson, Fitzsimons and Ward (1993) and McMahon (in press) found similar relationships between relative $\dot{V}O_2$max and power decrement during 6 6-s cycle sprints (r = - .56, p <.05) and 6 15-s cycle sprints (r = - .63, p =.004), respectively. These results appear to support that enhanced maximal aerobic power may contribute to improving power recovery over repeated intervals.

The improved power recovery seen in the HAP group may be the result of a larger aerobic contribution to the sprinting activity or an improved ability to recover between sprints. When exercising at the same submaximal percent of $\dot{V}O_2$max, aerobically trained individuals consume more oxygen than untrained due to their higher $\dot{V}O_2$max. As seen in Figures 2 and 3, the HAP group consumed significantly more oxygen than LAP throughout most of the 10 recovery and sprint-recovery bouts but only 5 of the 10 sprints. $\dot{V}O_2$ in Figure 2 is expressed as a rate ($mL \cdot kg^{-1} \cdot min^{-1}$) to facilitate comparison to the literature, whereas $\dot{V}O_2$ in Figure 3 is expressed as volume ($mL \cdot kg^{-1}$) to more easily make relative oxygen cost comparisons.

It may be that the 6-s interval is too short to reliably measure oxygen consumption since the inclusion or exclusion of a single breath can have a significant impact on average $\dot{V}O_2$ when only 2 or 3 breaths make up the averaging interval, as is the case in a 6-s interval. Additionally, breathholding, though strongly discouraged, did occur during the sprints and this has been shown to have an impact on the aerobic

response (Fujihara et al., 1973). With breath holding, oxygen consumption may be unequally distributed between the sprint and recovery intervals, which could affect the averaged scores of each, having a larger impact on the brief 6-s intervals. In pilot work performed in this laboratory, the day to day variation in $\dot{V}O_2$ for 10 6-s all-out sprints interspersed with 30-s passive recovery was investigated (n = 13). Reliability for oxygen consumption during the sprint-recovery cycle and the 30-s recovery were high (Safrit, 1990; R = .93 and .91, respectively) with the reliability for $\dot{V}O_2$ during the 6-s sprinting interval lower (R = .81). Furthermore, considerable fluctuation in $\dot{V}O_2$ for the 6-s sprint occurred between individual sprints from day 1 to day 2, ranging from R = .42 (sprint 2) to R = .86 (sprint 3). Some of the variation may be explained by the variation in day to day performance (R = .97), which could affect all subsequent $\dot{V}O_2$ values.

In both LAP and HAP, oxygen consumption increased rapidly after the onset of the first sprint, increasing further with each sprint-recovery period (Figure 2). In examining the aerobic response to constant intensity intermittent exercise (Buono and Roby, 1982; Weltman et al., 1979) or to increasing intensity intermittent exercise (Chamari et al., 1995) or to maximal effort intermittent bouts (Bogdanis et al., 1996a; Hamilton et al., 1991), others have also reported $\dot{V}O_2$ increases with subsequent repetitions. Furthermore, with the aid of muscle biopsy analysis, Bogdanis et al. (1996a) found that a mismatch between anaerobic energy release and power output during a second sprint was partially compensated for by an increased contribution of aerobic metabolism, as reflected in increased oxygen consumption in sprint 2. The peak oxygen

consumption (averaged over 30-s) achieved over the sprint test series was 78.6 (11.3) % of $\dot{V}O_2max$ in the LAP group and 75.8 (12.5) % of $\dot{V}O_2max$ in the HAP group ($p = .68$). Hamilton et al. (1991) reported similar peak $\dot{V}O_2max$ percentages in GP (70.7%) and ET (72.7%) (NS), however the higher maximal oxygen uptake of the ET (60.8 $mL \cdot kg^{-1} \cdot min^{-1}$) and GP subjects (52.5 $mL \cdot kg^{-1} \cdot min^{-1}$) may account for some of the difference, as well as the different mode of exercise.

The delay in fully activating aerobic metabolism at the onset of exercise seen in both HAP and LAP (Figures 2 and 3) is well established (Hughson and Morissey, 1982; Powers, Dodd and Beadle, 1985) and has been attributed to the time required for an increase in cellular ADP levels to trigger an increase in oxidative phosphorylation, an increase in blood flow to the working muscle and desaturation of oxymyoglobin (Astrand, Astrand, Christensen and Hedman, 1960; Hickson et al., 1978; Jobis and Duffield, 1967). As seen in Figure 2, the HAP group appears to utilize more oxygen sooner than the LAP group, likely as the result of their higher maximal aerobic power. Improvement in maximal aerobic power may enhance oxygen kinetics through more rapid oxygen transport and increased utilization of oxygen at the muscle. Aerobic training results in increased muscle blood flow, which is accomplished through elevated cardiac output (Ekblom and Hermansen, 1968), increased muscle capillarization (Anderson and Hendriksson, 1977) and an improved ability to vasodilate (Sinoway, Musch, Minotti and Zelis, 1986). Oxygen transport in the endurance trained is further improved by increases in blood volume and total hemoglobin (Kjellberg, Rudhe and Sjostrand, 1949). Increased oxygen extraction in the aerobically trained has been attributed to increased concentrations of aerobic enzymes, increased mitochondrial

number, size and surface area (Holloszy and Coyle, 1984) and increased myoglobin (Saltin and Rowell, 1980). Aerobic training has also been linked to a faster adaptation to gas exchange (Hagberg, Nagle and Carlson, 1978; Hickson et al., 1978). Powers et al. (1985) found that within a group of long distance runners who had similar training habits, those with higher $\dot{V}O_2$max scores achieved a more rapid $\dot{V}O_2$ adjustment at the onset of exercise.

The results of the 1-minute oxygen kinetics test performed by subjects in the present investigation appear to support the notion of faster oxygen kinetics in the aerobically trained state. As seen in Figure 4, oxygen consumption increases at a much faster rate in the HAP group, such that after 20-s at a power output corresponding to $\dot{V}O_2$max, the HAP group was utilizing significantly more oxygen than the LAP group ($p < .05$). As this represented a similar percentage of $\dot{V}O_2$max (LAP vs HAP: 54.1% vs 54.4%, $p = .94$), the benefit of a higher $\dot{V}O_2$max can be seen as enabling the subjects to consume more oxygen sooner. It appears that after the first 30 seconds of increasing $\dot{V}O_2$, LAP $\dot{V}O_2$ begins to plateau, while HAP $\dot{V}O_2$ continues to increase throughout the 60 seconds, with both groups attaining similar peak percentages of $\dot{V}O_2$max in the 1-minute period (HAP vs. LAP: 83.9 (8.5) versus 81.1 (11.0) % $\dot{V}O_2$max, $p = .62$). The superior oxygen kinetics seen in the HAP group translates into a faster onset of aerobic metabolism (Hickson et al., 1978), which may result in decreased reliance on anaerobic lactic energy sources.

Even a single 6-s bout of high intensity exercise is enough to significantly lower ATP/PCr stores, elevate blood and muscle lactates and depress pH (Gaitanos et al., 1993). As pH decreases through increases in anaerobic lactate metabolism, key

enzymes such as phosphofructokinase (PFK) and lactate dehydrogenase (LDH) are inhibited (Page, 1981). Since PFK is the rate-limiting enzyme of glycolysis (Page, 1981), the inhibition may impair both the aerobic and anaerobic degradation of carbohydrate to energy. Also, inhibition of LDH, especially the H isozyme, will hinder the oxidation of lactate to pyruvate in the recovery period (Page, 1981). Furthermore, the depressed pH will disrupt the excitation-coupling process by decreasing the amount of Ca++ released from the sarcoplasmic reticulum (Fuchs, Reddy and Briggs, 1970) and interfere with Ca++-troponin binding (Nakamura and Schwartz, 1972). Restoration of PCr levels during recovery is critical for maintenance of power during repeated 6-s sprints (Gaitanos et al., 1993) as it helps to minimize the contribution of anaerobic lactic energy. Bogdanis, Nevill, Boobis, Lakomy and Nevill (1995) found that power recovery on repeated 30-s cycle sprints and resynthesis of PCr proceeded in parallel, confirming the relevance of PCr availability for force recovery. Complete repletion of ATP/PCr stores may require 3-5 minutes (Hultman, Bergstrom and McLenan-Anderson, 1967) or even longer (Harris, Edwards, Hultman, Nordesjo, Nylind and Sahlin, 1976), so with only 30 seconds of recovery between exercise bouts, ATP/PCr stores will only be partially restored. Furthermore, with subsequent high intensity work bouts, as in the present study, ATP/PCr will be progressively depleted (Gaitanos et al., 1993; Yoshida and Watari, 1993) and there will be increased reliance on anaerobic glycolysis (Wooton and Williams, 1983) which may adversely affect performance (Sahlin, 1992).

PCr recovery appears to be dependent on oxygen supply to the muscle (Harris et al., 1976) and has been coupled to oxygen consumption in the immediate post-

exercise period (Hultman et al., 1967; Piiper and Spiller, 1970) so the superior levels of $\dot{V}O_2$ seen in the HAP group during the 30-s recovery periods (Figure 3) may contribute to PCr recovery. If more PCr is replenished by HAP participants in the 30-s recovery bouts, as suggested by the $\dot{V}O_2$ data (Figure 3), the result could be a diminished reliance by HAP on anaerobic glycolysis for subsequent sprints, which may at least partially explain why the HAP group were more successful at maintaining power throughout the 10 sprint repeats. In support of this, Balsom, Ekblom and Sjodin (1994) found that by inducing increases in hemoglobin, which also increased $\dot{V}O_2$max, subjects performing 15 6-s treadmill sprints could do so with lower post-exercise blood lactates, which may endorse the notion of increased reliance on alactic and/or aerobic energy sources with improved oxygen delivery.

After the initial increase in oxygen consumption, the increase in $\dot{V}O_2$ is not as great, with LAP $\dot{V}O_2$ for sprint-recovery and recovery intervals appearing to plateau sooner than HAP $\dot{V}O_2$ (Figures 2 and 3). If LAP participants are relying more on anaerobic glycolysis, due either to an inability to rapidly initiate aerobic metabolism or because of an inability to restore PCr in the brief recovery periods, the concomitant increase in H+ may be slowing aerobic metabolism by mechanisms previously discussed. Since LAP and HAP groups performed similar amounts of work on each of the first 6 sprints (Figure 1) and the HAP group consumed significantly more oxygen during 5 out of the initial 6 sprint-recovery bouts (Figure 2), the LAP group must have been augmenting aerobic metabolism with more alactic and lactic energy than the HAP group. In support of this, the HAP group tended to utilize more oxygen per 100 joules of work performed during the 10 sprints (LAP vs HAP: 30.4 (2.7) vs 33.5 (3.1)

$mL \cdot 100\ J^{-1}$, p =.08). While these differences were not statistically significant, closer inspection of the raw data for subject 12 (Appendix 2) reveals that her aerobic response was similar to other LAP participants yet her ability to generate power was lower, resulting in a high $\dot{V}O_2$ to work ratio, similar to that seen in the HAP group. With only 6 subjects in the LAP group, one unusual score will have a large impact on a t-test (Safrit, 1990). If anaerobic lactic metabolism was higher in the LAP participants, the resulting acidosis may have expressed itself in the LAP group as an inability to maintain power, as seen beyond the sixth sprint (Figure 2). Endurance training results in lower blood and muscle lactates for the same absolute submaximal workload (Karlsson, 1971) due to decreased production of lactate as a result of increased reliance on other energy systems (Holloszy and Coyle, 1984) and/or increased lactate clearance (Brooks and Donovan, 1983). Neither blood or muscle lactates were measured in the present investigation, however, using a similar protocol Hamilton et al. (1991) found that following multiple treadmill sprints the ET, who had similar peak power to the GP (NS), had a smaller performance decrement ($p < .05$), consumed more oxygen ($p < .05$) and had lower blood lactate levels ($p < .05$) than the GP, suggesting smaller contribution from anaerobic glycolytic energy production or enhanced lactate removal in the ET.

Some authors have reported enhanced lactate removal in endurance trained individuals (Freund, Lonsdorfer, Oyono-Enguelle, Lonsdorfer and Bogui, 1992; Gisolfi, Robinson and Turrell, 1966; Oyono-Enguelle, Marbach, Heitz, Ott, Gartner, Pape, Vollmer and Freund, 1990) while others have failed to find a relationship between lactate removal and aerobic fitness (Evans and Cureton, 1983). Changes associated with enhanced $\dot{V}O_2max$ that may lead to improved lactate removal include both

increased buffering capacity and increased blood flow, which results from blood volume, capillary density and cardiac output enhancements. Increased capillary density, as seen in endurance trained individuals, provides a decreased diffusion distance between capillaries and muscle cells, enhancing the movement of oxygen and nutrients to and the removal of H+ and lactate from muscle (Anderson and Hendriksson, 1977). Tesch and Wright (1983) reported a strong relationship between capillary density and blood lactate concentrations.

Moderately high correlations (Safrit, 1990) of r = .76 to .78 (p =.002) are seen between the aerobic response to the sprint, recovery and the sprint-recovery cycle versus $\dot{V}O_2$max (Table 3), suggesting that the aerobic response to repeated bouts of brief high intensity effort is related to $\dot{V}O_2$max. Hamilton et al. (1991) found that ET athletes consumed more oxygen over 10 sprint-recovery cycles than GP ($p < .05$) and reported a correlation between $\dot{V}O_2$max and the aerobic response to the sprint recovery interval similar to that found in the present investigation ($r = .83$; $p < .01$). It would seem that the higher the maximal oxygen uptake the greater the likelihood of the athlete being able to use more oxygen during the sprint, recovery or entire sprint-recovery interval. Even if an athlete with a $\dot{V}O_2$max of 35 $mL \cdot kg^{-1} \cdot min^{-1}$ could use 100% of her maximal oxygen uptake during intense intermittent exercise, an athlete with a $\dot{V}O_2$max of 50 $mL \cdot kg^{-1} \cdot min^{-1}$ who achieved only 75 - 80% of her $\dot{V}O_2$max would utilize more oxygen.

The Pearson product moment correlation coefficients between % DO and net $\dot{V}O_2$ for the sprint, recovery and sprint-recovery intervals were moderate (Safrit, 1990), ranging from r = -.41 - -.50 (Table 3), failing to meet statistical significance. It may be

that the limited range of sprint, recovery and sprint-recovery cycle $\dot{V}O_2$ values across subjects (< 15 $mL \cdot kg^{-1} \cdot min^{-1}$) and the relatively homogeneous % DO values (72-95%) made a large correlation coefficient difficult to attain. The more homogeneous the group is with respect to the trait being measured, the lower the resulting correlation coefficient will be (Baumgartner, 1991). Furthermore, with only 13 subjects, even 1 or 2 sets of scores that do not comply with the general trend can have a sizable negative impact on the correlation coefficient.

While moderately high correlations are seen between % DO and $\dot{V}O_2max$ ($r = -.65$) and between $\dot{V}O_2max$ and net $\dot{V}O_2$ during sprint and/or recovery periods ($r = .76$ to $.78$), the correlation between % DO and net $\dot{V}O_2$ during sprint and/or recovery periods ($r = -.40$ to $-.52$) is weaker (Table 3). Perhaps genetic factors affecting $\dot{V}O_2max$ impact somewhat differently on the % DO than the aerobic response to exercise. It may be that rate of recovery is determined more by genetic factors associated with $\dot{V}O_2max$, such as the percentage of slow twitch fibres, whereas the aerobic response to exercise is influenced by both central and peripheral factors which are sensitive to training. In support of this, Colliander, Dudley and Tesch (1988) demonstrated that individuals in a "low fast twitch" group were superior to a "high fast twitch" group in restoring force between sets of concentric contractions. Furthermore, the metabolic profile of muscles fibres can be altered with endurance training through transformation of fast glycolytic fibers to the more oxidative fast oxidative glycolytic fibers (Jansson and Kaijser, 1977), thereby enhancing the oxidative capabilities of the muscle, but the enhancements do not always translate into improvements in $\dot{V}O_2max$ (Holloszy and Coyle, 1984).

When the difference in the oxygen cost of the work was eliminated, no significant differences in oxygen consumption between HAP and LAP groups were seen during the 5-minute active recovery period, though the HAP utilized significantly more oxygen during the first minute of passive recovery ($p = .04$). In the period immediately following exercise, a higher $\dot{V}O_2$ for the HAP group was expected since when compared to untrained individuals, fast EPOC following submaximal exercise of the same relative intensity is demonstrably higher in endurance trained individuals (Frey, Byrnes and Mazzeo, 1993; Hagberg et al., 1980; Sedlock, 1994; Short and Sedlock, 1997). Unfortunately the 5 minute active recovery period may have masked differences in EPOC, with everyone working at the same relative percentage of $\dot{V}O_2max$. Additionally, subjects did not always maintain the exact prescribed cadence. As expected (Short and Sedlock, 1997), since HAP and LAP were both working at 35% $\dot{V}O_2max$ during the active recovery period, for the first minute of the passive recovery phase $\dot{V}O_2$ was significantly higher in the HAP group ($p = .04$), but as recovery progressed no significant difference in the magnitude of EPOC was seen.

In summary, it appears that a superior level of maximal aerobic power may be beneficial as it appears to be associated with improved power maintenance during repeated supramaximal sprints. Those with a higher $\dot{V}O_2max$ tend to consume more oxygen in both the sprint and recovery intervals during repeated high intensity exercise bouts, which can be ascribed, at least in part, to their improved oxygen kinetics. The enhanced aerobic response during the recovery intervals may be responsible for improved PCr resynthesis, making more PCr available for subsequent sprints. The resulting higher levels of PCr at the end of each recovery period and increased aerobic

metabolism during sprinting will thus minimize anaerobic lactate contribution to the sprints. Since increased reliance on anaerobic glycolytic energy results in increased H+ accumulations, which have been associated with fatigue (Sahlin, 1992), increased use of the other two energy sources should enhance power recovery, as seen with HAP athletes in the present investigation. These findings support the practice of enhancing $\dot{V}O_2max$ through training so as to improve performance on repeated bouts of high intensity activity.

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APPENDIX ONE

Letter of Consent

THE AEROBIC RESPONSE TO INTENSE INTERMITTENT EXERCISE CONSENT FORM

The purpose of this research project is to investigate the relationship between oxygen consumption and fatigue during 10 repeated bursts of 6 second cycling. Information gathered in this study may be helpful in better understanding the physiological requirements of intense intermittent sport and improving training techniques. You will participate in 2 laboratory sessions as outlined below, however, your participation is completely voluntary and you can withdraw from the study at any time, without explanation.

You will participate in 2 laboratory sessions within a 2 week period: a maximal oxygen consumption test and a sprint test. The maximal exercise loads imposed during these tests will not exceed those which might be expected during competitive sports performance.

1) Today, height, weight and 5 skinfolds will be measured, then **maximal oxygen consumption** will be determined. During the maximal test, heart rate and oxygen consumption will be measured while you ride a cycle at loads that increase every 1-2 minutes until exhaustion. Once you have recovered from the maximal test, you will practice the sprint test protocol so that you feel comfortable with it for the sprint test day. This laboratory session should take approximately 1 hour.

2) Prior to the **sprint test session** you will refrain from intense physical exercise for 24 hours and abstain from alcohol, caffeine, tobacco and drugs for 2 hours. We also ask that you travel to the laboratory by car, motorcycle or bus for this session, in order to eliminate unnecessary activity. This session will take place either early in the morning after you have completed an overnight fast or at least 4 (but no more than 5) hours after your last meal. Upon arrival at the sprint test session baseline oxygen consumption and heart rate will be established while you sit quietly for 30 minutes. During this time you are free to read, write and/or listen to music but must remain seated as we will be measuring heart rate and oxygen consumption. Following baseline measures and a standard warm-up, you will perform 10 6-second all-out cycle sprints with 30 seconds of rest between each sprint. During the sprint test series and for 15 minutes following it, your heart rate and oxygen consumption will be measured. Also, core temperature will be measured during resting, before and after the first sprint, following the fifth and final sprints, then 3, 5 and 10 minutes post-exercise. Core temperature will be monitored by gently inserting a tympanic thermometer in the ear canal. This laboratory session should take approximately 1 hour.

Due to the intense nature of the exercise performed in this study, some subjects may experience symptoms such as nausea or dizziness during either of the testing sessions. If you experience any of these symptoms, please inform the investigator

testing sessions. If you experience any of these symptoms, please inform the investigator immediately and the test will be terminated. You can either reschedule another appointment to complete the test or withdraw from the study.

Following any of these testing sessions, you may experience some minor muscle soreness, similar to what you may encounter after a soccer match. Gentle stretching of the affected muscles should alleviate the stiffness, however if it persists for more than 48 hours or you would describe it as more than minor stiffness, please contact the researcher (477-8832).

Any data collected in the study will remain confidential; physiological data will be kept in a filing cabinet in a locked room. A code number will be the only identifying information for you on all data sheets associated with the study. The key to the coded numbers will be kept in a location separate from the data sheets and known only to the primary researcher, Dona Tomlin. All original data forms and the key to coded numbers will be destroyed within one year of the study, however, a computer data base of results using only code numbers to identify subjects will be archived and possibly used for writing further scientific papers. Furthermore, your name will not be attached to any published results, and your anonymity will be protected by describing all results in terms of the group response. If you withdraw from the study prior to the completion of all 3 sessions, your results collected to that point will not be used without your written approval.

Whether you participate, choose not to participate or withdraw from the study will have no bearing on your university grades, academic standing or your status as a soccer player with Castaways Soccer or any other soccer organization.

Having read the above, I agree to participate in the study entitled *The aerobic response to intense intermittent exercise*.

Signature of participant: _____ Date: _____

Name of participant (please print): _____

Researcher: Dona Tomlin, graduate student Phone: (250) 721-8388
Faculty Supervisor: Dr. H. A. Wenger Phone: (250) 721-8386

APPENDIX TWO

Raw Data

Table A-1

Average Power ($W \cdot kg^{-1}$) for 10 6-s Sprint (SP) for Low Aerobic Power (LAP) and High Aerobic Power (HAP) Subjects

Subject	SP 1	SP 2	SP 3	SP 4	SP 5	SP 6	SP 7	SP 8	SP 9	SP 10	Peak Power	Mean Power
LAP												
1	7.4	7.6	7.1	7.2	7.0	7.0	7.0	6.5	6.3	6.6	7.6	7.0
2	7.5	6.9	6.4	6.4	6.8	6.7	6.1	5.8	6.0	6.1	7.5	6.5
4	8.6	8.5	8.4	8.2	8.1	7.8	7.6	7.2	7.1	7.2	8.6	7.9
5	8.5	8.1	7.7	7.3	7.0	7.0	6.7	6.9	6.6	6.1	8.5	7.2
11	8.9	8.8	8.5	8.4	8.1	7.9	7.5	7.7	7.4	7.3	8.9	8.0
12	5.8	5.8	5.9	5.7	5.8	5.9	6.0	5.8	5.9	5.8	6.0	5.8
Mean	7.8	7.6	7.3	7.2	7.1	7.1	6.8	6.7	6.6	6.5	7.8	7.1
(SD)	(1.2)	(1.1)	(1.0)	(1.0)	(0.9)	(0.7)	(0.7)	(0.8)	(0.6)	(0.6)	(1.1)	(0.8)
HAP												
7	8.4	7.8	7.6	7.5	7.3	7.5	7.3	7.2	7.3	7.1	8.4	7.5
8	7.7	7.7	7.8	7.4	7.4	7.7	7.4	7.4	7.7	7.4	7.8	7.6
16	7.6	7.4	7.6	7.6	8.0	7.9	7.9	7.9	8.0	8.0	8.0	7.8
17	7.4	7.4	7.3	7.1	7.2	7.3	7.2	7.0	7.0	7.0	7.4	7.2
18	9.2	9.1	9.1	9.4	9.2	9.1	8.9	8.8	8.7	8.8	9.4	9.0
19	8.5	8.2	8.1	8.1	7.9	7.7	7.5	7.4	7.6	7.4	8.5	7.8
20	7.2	7.3	7.3	7.3	7.2	6.9	7.0	6.8	6.9	6.9	7.3	7.1
Mean	8.0	7.8	7.8	7.8	7.8	7.7	7.6	7.5	7.6	7.5	8.1	7.7
(SD)	(0.7)	(0.6)	(0.6)	(0.8)	(0.7)	(0.7)	(0.6)	(0.7)	(0.6)	(0.7)	(0.7)	(0.6)

Table A-2

Oxygen Consumption ($mL \cdot kg^{-1} \cdot min^{-1}$) above Pre-exercise Levels for Each 36-s Sprint-Recovery Cycle (SRC) for Low Aerobic Power (LAP) and High Aerobic Power (HAP) Subjects

Subject	SRC 1	SRC 2	SRC 3	SRC 4	SRC 5	SRC 6	SRC 7	SRC 8	SRC 9	SRC 10	Average
LAP											
1	9.5	21.2	22.1	21.1	20.5	20.4	22.2	22.1	19.8	22.4	20.1
2	8.6	17.9	22.4	21.1	23.4	23.1	23.2	21.9	21.4	23.3	20.6
4	13.1	21.6	21.7	21.1	21.9	23.5	23.7	22.9	23.0	22.3	21.5
5	9.5	18.0	23.7	21.8	22.4	20.5	21.0	22.5	22.2	22.2	20.4
11	11.0	23.2	25.2	26.0	26.8	27.4	28.0	29.5	28.3	25.8	25.1
12	10.6	19.3	20.5	21.2	20.3	21.5	22.3	21.7	22.1	22.8	20.2
Mean	10.4	20.2	22.6	22.0	22.6	22.7	23.4	23.4	22.8	23.1	21.3
(SD)	(1.6)	(2.1)	(1.6)	(2.0)	(2.4)	(2.6)	(2.5)	(3.0)	(2.9)	(1.4)	(1.9)
HAP											
7	9.2	21.0	27.0	24.7	26.6	24.7	27.6	27.4	27.2	25.1	24.1
8	12.5	19.1	22.1	21.8	22.5	25.2	23.0	23.0	22.0	21.1	21.2
16	12.8	24.0	24.9	25.6	26.1	27.9	26.6	27.9	28.8	29.5	25.4
17	12.1	22.9	24.6	25.9	26.3	27.9	26.6	27.6	28.9	26.8	25.0
18	17.3	30.8	32.8	33.6	36.4	36.0	37.9	39.1	38.1	36.3	33.8
19	16.8	29.2	25.1	26.8	26.3	24.9	26.8	28.6	29.3	31.2	26.5
20	12.9	22.3	25.7	26.9	28.9	28.3	27.9	28.2	27.5	27.5	25.6
Mean	13.4	24.2	26.0	26.5	27.6	27.9	28.1	28.8	28.8	28.2	25.9
(SD)	(2.8)	(4.3)	(3.3)	(3.6)	(4.3)	(3.9)	(4.6)	(4.9)	(4.8)	(4.8)	(3.9)

Table A-3

Oxygen Consumption ($mL \cdot kg^{-1}$) Above Pre-exercise Levels for Each 6-s Sprint Interval (S) for Low Aerobic Power (LAP) and High Aerobic Power (HAP) Groups

Subject	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10
LAP										
1	0.4	1.9	2.1	2.1	2.2	2.0	2.1	2.2	2.1	2.0
2	0.1	1.4	2.5	2.1	2.5	2.5	2.8	2.6	2.5	2.6
4	0.2	2.0	1.9	2.1	2.1	2.2	2.3	2.2	2.3	1.9
5	0.0	0.9	1.5	1.8	2.2	1.8	1.8	2.1	1.9	1.7
11	0.4	2.0	2.1	2.1	2.3	2.5	2.3	2.3	2.4	2.3
12	0.3	1.8	2.7	2.4	2.3	2.4	2.4	2.7	2.6	2.4
Mean	0.2	1.7	2.1	2.1	2.3	2.2	2.3	2.3	2.3	2.1
(SD)	(0.2)	(0.5)	(0.4)	(0.2)	(0.2)	(0.3)	(0.3)	(0.3)	(0.2)	(0.3)
HAP										
7	0.3	1.5	2.8	2.0	2.7	2.5	2.5	2.3	2.6	2.2
8	1.1	1.6	1.9	2.3	1.9	3.1	2.3	2.3	2.2	2.2
16	0.4	2.5	2.7	2.7	2.6	2.9	2.6	2.9	3.0	3.1
17	0.2	2.2	1.9	2.7	2.1	2.2	2.2	2.5	3.3	2.4
18	0.6	2.9	3.2	3.2	3.3	3.3	3.7	3.9	3.6	3.9
19	1.3	3.0	2.9	2.2	2.5	2.5	2.5	3.0	2.8	2.9
20	1.1	2.0	2.2	2.7	2.5	3.1	2.8	2.7	2.7	2.8
Mean	0.7	2.2	2.5	2.5	2.5	2.8	2.6	2.8	2.9	2.8
(SD)	(0.4)	(0.6)	(0.5)	(0.4)	(0.5)	(0.4)	(0.5)	(0.5)	(0.5)	(0.6)

Table A-4

Oxygen Consumption ($mL \cdot kg^{-1}$) Above Pre-exercise Levels for Each 30-s Recovery Interval (R) for Low Aerobic Power (LAP) and High Aerobic Power (HAP) Groups

Subject	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10
LAP										
1	5.0	10.6	11.2	10.6	10.3	10.3	11.2	11.1	9.8	11.5
2	4.8	9.4	11.6	10.8	12.3	11.6	11.0	10.6	10.5	11.4
4	7.4	10.8	11.1	10.6	10.9	11.7	11.8	11.3	11.5	11.4
5	5.2	10.3	12.1	10.9	11.4	10.3	10.5	11.3	11.3	11.6
11	5.5	9.8	10.4	10.9	9.9	10.4	11.3	10.7	10.8	11.2
12	6.0	12.5	13.5	13.1	13.3	12.6	13.5	14.0	13.4	14.0
Mean	5.7	10.6	11.7	11.2	11.3	11.1	11.6	11.5	11.2	11.8
(SD)	(0.9)	(1.1)	(1.1)	(1.0)	(1.3)	(1.0)	(1.0)	(1.3)	(1.2)	(1.1)
HAP										
7	5.7	11.4	13.5	12.8	13.3	12.4	14.3	13.8	13.6	12.8
8	6.5	10.1	11.3	10.9	11.3	13.1	11.6	11.6	10.9	10.7
16	6.6	11.8	12.3	13.0	13.1	13.9	13.7	14.0	14.3	14.6
17	6.7	11.7	12.2	13.5	13.4	14.2	13.7	14.6	14.7	13.7
18	9.5	16.0	16.6	16.9	18.6	18.5	19.1	19.7	19.5	17.9
19	8.6	14.9	12.5	14.0	13.8	12.7	13.5	14.5	15.0	15.8
20	7.0	11.2	13.2	13.4	14.8	13.8	14.1	14.4	13.8	13.7
Mean	7.2	12.5	13.1	13.5	14.1	14.1	14.3	14.6	14.5	14.2
(SD)	(1.3)	(2.1)	(1.7)	(1.8)	(2.3)	(2.1)	(2.3)	(2.5)	(2.6)	(2.3)

Table A-5

Oxygen Consumption ($mL \cdot kg \cdot min^{-1}$) at Rest and for Each 10-s of a 1- minute Oxygen Kinetics Test (O_2K) Performed at 100% VO_2max

Subject	Resting VO_2	O_2K 10-s	O_2K 20-s	O_2K 30-s	O_2K 40-s	O_2K 50-s	O_2K 60-s
LAP							
1	2.5	11.0	14.2	17.0	21.9	25.7	22.5
2	3.9	6.9	11.8	8.6	16.4	18.9	22.1
4	3.2	12.4	19.6	25.4	31.6	33.2	33.4
5	3.8	7.4	10.5	14.4	22.3	23.4	22.8
11	3.2	9.6	17.2	25.2	28.7	31.6	31.8
12	2.9	9.9	15.1	21.8	26.9	29.6	31.3
Mean	3.3	9.5	14.7	18.7	24.6	27.1	27.3
(SD)	(0.5)	(2.1)	(3.4)	(6.6)	(5.5)	(5.4)	(5.4)
HAP							
7	4.4	9.6	14.5	25.7	31.5	35.3	40.4
8	3.6	10.8	18.5	26.6	32.8	32.4	37.0
16	2.5	4.0	6.5	21.7	32.3	39.9	38.5
17	3.9	11.7	16.8	26.8	32.8	32.4	38.7
18	3.8	15.4	23.7	35.1	39.7	41.7	46.4
19	4.2	7.7	13.7	18.8	26.6	30.5	33.8
20	4.5	10.0	20.7	27.2	36.0	41.1	42.3
Mean	3.8	9.9	16.3	26.0	33.1	36.2	39.6
(SD)	(0.7)	(3.5)	(5.6)	(5.1)	(4.0)	(4.7)	(4.0)

APPENDIX THREE

Review of Literature

The aerobic response to single bouts of submaximal and maximal exercise has been studied extensively (Ceretelli, Pendergast, Pagnelli and Rennie, 1979; Davis, Whipp and Wasserman, 1979; di Prampero, Boutellier and Pietsch, 1983; Fox, Robinson and Weigman, 1969; Girondola and Katch, 1973; Hagberg, Hickson, Ehsani and Holloszy, 1980; Hickson, Bomze, Holloszy, 1978) but few have investigated the aerobic response to intense intermittent exercise (Bogdanis, Nevill, Boobis and Lakomy, 1996; Buono, and Roby, 1982; Chamari, Ahmaidi, Fabre, Ramonatxo and Prefaut, 1995; Hamilton, Nevill, Brooks and Williams, 1991; Weltman, Stamford and Fulco, 1979). The aerobic response to intense intermittent exercise is likley related to aerobic fitness (Hamilton et al., 1991) and may have an impact on the aerobic energy contribution during sprinting and in the brief recovery period (Bogdanis et al.,1996). This, in turn, may enhance performance on repeated bouts of high intensity exercise. Some researchers have suggested that the adaptations associated with endurance training should enhance recovery from high intensity intermittent exercise (Rhodes and Twist, 1990; Thoden, 1991) and since the theoretical basis is so compelling, many coaches and athletes have accept this conjecture as truth. However, as will be seen, the evidence supporting aerobic fitness as a means of improving performance of intense intermittent exercise is not yet conclusive.

Energy Demands for High Intensity Exercise

In activities requiring brief periods of high intensity effort, most of the energy for exercise is provided through anaerobic processes (Jacobs, Tesch, Bar-Or, Karlsson and Dotan, 1983). Since only limited amounts of adenosine triphosphate (ATP) are stored in muscle, immediate energy for short-term high intensity exercise is provided by the removal of a phosphate from phosphocreatine (PCr) to create energy. Once ADP levels rise, additional ATP can be produced through transphosphorylation, where

ATP is produced from 2 ADP (Brooks and Fahey, 1984). The high rate of energy production associated with the alactic system will be limited if the stores of high energy compounds are depleted, if enzymes such as creatine kinase, which generates ATP through PCr, or myokinase, the transphosphorylation enzyme, are inhibited, or if the creatine kinase equilibrium is shifted (Sahlin, 1992). Exclusive use of ATP and PCr is only possible in high intensity activities lasting less than about 2-3 s (Balsom, Seger, Sjodin and Ekblom, 1992), so for high intensity exercise of more than a few seconds both anaerobic alactic and lactic processes are required.

Anaerobic glycolysis involves the breakdown of carbohydrate (glycogen or glucose) and is of primary importance in supplying energy for high-intensity activities lasting longer than 10 seconds up to approximately 2 minutes. A 6-s bout of exercise requires nearly equal contribution from alactic and glycolytic energy sources (Gaitanos, Williams, Boobis and Brooks, 1993). In the process of anaerobic glycolysis, Pi, ADP and lactic acid, which dissociates into lactate and hydrogen ion (H+), are produced (Sahlin, 1992). Hydrolysis of ATP under aerobic or anaerobic conditions results in the release of Pi, ADP or H+, all potentially fatiguing agents (Sahlin, 1992). The focus of this discussion will be the H+ since it's accumulation depresses pH and is thereby implicated in fatigue in high intensity exercise (Sahlin, 1992). Potential mechanisms whereby excess H+ may induce fatigue include impairment of membrane permeability to Na+ and K+ which results in membrane hyperpolarization (Sjogaard, 1986), increased Ca++ requirement for any given tension (Robertson and Kerrick, 1976), decreased calcium release from the sarcoplasmic reticulum (Nakamura and Schwartz, 1970), competition with Ca++ at the troponin binding site (Bonen and Belcastro, 1976), and the inhibition of key enzymes such as phosphofructose kinase (PFK), lactate dehydrogenase (LDH) (Bonen and Belcastro, 1976), myokinase and creatine kinase (Sahlin, 1992). Inhibition of PFK, the rate limiting enzyme of glycolysis, or LDH, the enzyme responsible for catalyzing the conversion of pyruvate

to lactate, will impede glycolysis whereas inhibition of creatine kinase or myokinase will hinder the rate of alactic energy production. Regardless of the exact mechanisms whereby pH decrements cause fatigue, reductions in muscle pH are associated with decreased tension development (Fabiato and Fabiato, 1978; McCartney, Heigenhauser and Jones, 1983).

Though generally associated with exercise of long duration and low intensity, the aerobic metabolic system is stimulated even in high intensity exercise lasting a few seconds (Gaitanos et al., 1993). Furthermore, oxygen consumption appears to increase with subsequent high intensity bouts, regardless of whether the succeeding exercise bouts are constant intensity (Buono and Roby, 1982; Weltman et al., 1979), increasing intensity (Chamari et al., 1995) or an all-out effort (Hamilton et al., 1991). Bogdanis et al. (1996) suggest that aerobic metabolism may contribute as much as 40% of the energy for a second 30-s sprint during intermittent all-out sprints. Furthermore, it appears that oxidative metabolism is essential in the recovery process following exercise (Harris, Edwards, Hultman, Nordesjo, Nylind and Sahlin, 1976), including recovery between repeated bouts (Bogdanis et al., 1996).

Recovery from High Intensity Exercise

The return of the muscle to it's pre-exercise state following exercise is a process known as recovery. The recovery process is biphasic with the initial rapid phase of recovery lasting 10 seconds to 5 minutes followed by a slower second recovery phase lasting anywhere from a few minutes to a number of hours (Gaesser and Brooks, 1984). During recovery, oxygen consumption is elevated to help restore metabolic processes to pre-exercise conditions. The post-exercise oxygen uptake beyond that required at rest has been termed excess post-exercise oxygen consumption (EPOC) (Gaesser and Brooks, 1984).

The fast phase of recovery is marked by rapidly declining oxygen consumption and heart rate. It is during this period that tissue stores of oxygen are quickly replenished (Gaesser and Brooks, 1984) and most of the ATP and PCr depleted in the muscle are restored, with 70% of the phosphagens restored within 30 seconds and 100% restored within 3 to 5 minutes (Hultman, Bergstrom and McLenan-Anderson, 1967). Once depleted, PCr is not restored until after the demands of high intensity exercise have ceased (diPrampero et al., 1983). Additionally, Harris et al. (1976) found that no replenishment of PCr occurred when the circulation was occluded, suggesting that oxygen is required for the process.

The increased metabolism marking the slow recovery period has been associated with the removal of lactate and H+ (Gaesser and Brooks, 1984; Sahlin, 1992), elevated body temperature (Brooks, Hittelman, Faulkner and Beyer, 1971), the cost of increased respiratory and cardiac functions (Gaesser and Brooks, 1984), the effect of catecholamines (Gladden, Stainsby and McIntosh, 1982) and the cost of glycogen resynthesis (Gaesser and Brooks, 1984). Recovery is not complete until all of these factors have returned to control levels.

Factors Infuencing Recovery from Intense Intermittent Exercise

The ability to recover from high intensity intermittent exercise is influenced by the nature of both the exercise and the recovery periods. The aim of the recovery interval is to return the muscle to it's pre-exercise condition by restoring ATP/PCr and tissue oxygen stores, ridding the muscle of excess H+ and converting lactate into usable substances, such as pyruvate or glycogen, which can then be degraded to produce energy. Generally, the more that exercise disrupts homeostasis, the greater the effect on recovery metabolism (Brehm and Gutin, 1986). The more complete these

restorative processes the greater the ability to generate force on subsequent work intervals.

High intensity exercise of short duration (< 5 s) results in decreased ATP/PCr stores, which upon completion of exercise, can be completely restored in a few minutes (Hultman et al., 1967). However, if the subsequent recovery interval is less than a few minutes long, as in many team sports (Mayhew and Wenger, 1985; McLean, 1992), the ATP/PCr stores may be only partially restored before demands of the next exercise bout strike, resulting in compromised performance on subsequent bouts. Moreover, as ATP/PCr stores are progressively depleted with subsequent high intensity work bouts (Gaitanos et al., 1993; Yoshida and Watari, 1993), there will be increased reliance on anaerobic glycolysis (Wooton and Williams, 1983). The metabolic consequence of increased anaerobic glycolysis is an increase in H+ concentration and a depressed pH, which may adversely affect performance by disrupting contractile processes (Sahlin, 1992).

When the duration of high intensity exercise exceeds more than a few seconds, ATP/PCr stores will be rapidly depleted and anaerobic glycolysis, with the concomitant increase in H+, will be required to provide energy at an increased rate. As evidence, muscle and blood lactate levels are significantly increased following 6 (Gaitanos et al., 1993) and 10 (Jacobs et al., 1983) seconds of all-out effort. It is not surprising that following exercise which results in depletion of ATP/PCr stores and increased lactate and H+ accumulation, it will require longer to return to the pre-exercise state. Once H+ have accumulated, existing transport and metabolic pathways are less efficient, slowing the rate of recovery from exercise.

The length of the recovery interval between repeats of high intensity bouts of exercise will also affect recovery. Wooton and Williams (1983) found that, while power output decreased over repeated 6-s all-out sprints with either 30 s or 60 s of recovery between sprints, power output declined less when 60 s of recovery was

allowed. A longer recovery interval ensures more complete recovery, however, in sports requiring intermittent bursts of all-out effort the recovery periods may last only a few seconds so performance on subsequent bouts may suffer.

During brief intervals of recovery, at least some of the ATP, PCr and oxymyoglobin is restored. While restoration of the oxygen-myoglobin stores can take 10-80 seconds (Chance, Dait, Zhang, Hamoaka and Hagerman, 1992) complete phosphagen recovery may require 3-5 (Hultman et al., 1967) or even 8 minutes (Harris et al., 1976). The rate of post-exercise PCr resynthesis is controlled by the rate of oxidative metabolism within the muscle (Taylor, Bore, Styles, Gadian and Radda, 1983), and in the absence of circulation, little PCr is regenerated (Colliander, Dudley and Tesch, 1988; Harris et al., 1976; Yoshida and Watari, 1997).

The ability to recover from exercise resulting in lactate production depends on the capacity to tolerate, buffer and/or rapidly remove H+ from working muscle (Sahlin and Henriksson, 1984). Important buffers within muscle include PCr, inorganic phosphate, protein-bound histidine residues and carnosine (Parkhouse and McKenzie, 1984). Once in the blood, lactic acid is effectively buffered by sodium bicarbonate. Approximately 65% of the lactate is converted to pyruvate by LDH, then undergoes aerobic degradation via the Krebs Cycle and electron transport system, with the remaining 35% converted to glucose and/or glycogen, secreted in urine and sweat or converted to protein (Parkhouse and McKenzie, 1984). Most of lactate oxidation occurs in skeletal muscle, particularly the slow twitch fibres (Bonen and Belcastro, 1976). The restoration of muscle pH is critical for optimal force production on subsequent exercise since the rate of PCr resynthesis is influenced by the metabolic environment of muscle, especially the concentration of H+ (Arnold, Matthews and Radda, 1983), but also the ATP concentration and the rate of oxidative phosphorylation within working muscle (Jansson, Dudley, Norman and Tesch, 1990; Tesch and Wright, 1983). Arnold et al. (1983) reported that the rate of PCr

resynthesis was slower following heavy versus light work, which may be related to the effect pH has on creatine kinase (Sahlin, Harris and Hultman, 1975).

Fox, Robinson and Weigman (1969) have suggested that since passive recovery does not result in further breakdown of ATP/PCr, it is optimal when complete ATP/PCr restoration is necessary. Active recovery is recommended following exercise that is of an intensity and duration that will result in lactic acid accumulation as it increases the rate of lactate removal (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976) likely through increased blood flow which should increase oxygen delivery to the muscle and promote lactate efflux from the muscle (Bonen and Belcastro, 1976; Gollnick, Bayly and Hodgson, 1986). Furthermore, low intensity exercise recruits slow twitch fibers and these fibers are better equipped to oxidize lactate (Tesch and Wright, 1983).

Possible Role of Aerobic Fitness in Enhancing Recovery from Intense Exercise

The most widely accepted measure of aerobic fitness, maximal oxygen consumption ($\dot{V}O_2max$) represents the maximum rate at which aerobic metabolism can supply energy (Thoden, 1991). Another widely used index of aerobic fitness, aerobic capacity, identifies by blood lactate or ventilatory parameters the maximal steady state exercise intensity that can be sustained. Increases in $\dot{V}O_2max$ and aerobic capacity result from endurance training (Ekblom, Astrand, Saltin, Stenberg and Wallstrom, 1968). Aerobic capacity measures have proven useful in predicting success in distance running events (Costill, Thomason and Roberts, 1973; Tanaka and Matsuura, 1984) with some scientists (Boulay, Hamel, Simoneau, Lortie, Prud'homme and Bouchard, 1984) regarding it as a superior measure of endurance fitness in comparison to $\dot{V}O_2max$.

Thoden (1991) proposes that aerobic training may enhance the ability of the muscle to recover following anaerobic exercise. He acknowledges that while it is unlikely that the recovery processes require the high levels of $\dot{V}O_2$max associated with distance running, an athlete with higher aerobic fitness will tax nonoxidative sources less and thereby recover at a more rapid rate from exercise. Theoretically, an increase in aerobic fitness could enhance recovery from anaerobic performance both by supplementing anaerobic energy during the exercise and by providing aerobically derived energy at a faster rate during the recovery period. Additionally, any improvements that aid in transport to or from the muscle, such as increased blood flow, could augment heat dissipation and removal of lactate and H+.

Individuals with high maximal aerobic power exhibit increased concentrations of aerobic enzymes (Holloszy and Coyle, 1984), increased mitochondrial number, size and surface area (Holloszy and Coyle, 1984) and increased myoglobin (Saltin and Rowell, 1980), all contributing to improved oxygen extraction by muscle. Aerobic training also results in increased muscle blood flow, which is accomplished through elevated cardiac output (Ekblom and Hermansen, 1968), increased capillarization of muscle tissue (Anderson and Hendriksson, 1977; Saltin and Rowell, 1980; Tesch and Wright, 1983) and an improved ability to vasodilate (Sinoway, Musch, Minotti and Zelis, 1986). Oxygen delivery in the endurance trained is further improved by increases in blood volume and total hemoglobin (Kjellberg, Rudhe and Sjostrand, 1949). Together, these enhancements result in an increased rate of oxygen consumption during maximal exercise (Ekblom et al., 1968) and decreased time to reach peak $\dot{V}O_2$ during exercise (Hagberg, Hickson, Ehsani and Holloszy, 1980), which may result in less lactic acid accumulation (Ceretelli, Pendergast, Paganelli and Rennie, 1979). In conjunction with enhanced ATP/PCr stores (Park, Brown, Park, Cohn and Chance, 1988) and elevated myokinase and creatine kinase concentrations (Thorstensson, Sjodin and Karlsson, 1975) seen in trained athletes, these adaptations should result in

an ability to supply more energy through the phosphagen and aerobic systems, even for high intensity exercise, thus decreasing the reliance on anaerobic glycolysis and thereby stemming the rise in H+ during brief intermittent work. Endurance training results in lower blood and muscle lactates for the same absolute submaximal workload (Karlsson, 1971) due to decreased production of lactate as a result of increased reliance on other energy systems (Holloszy and Coyle, 1984) and/or increased lactate clearance (Brooks and Donovan, 1983). With reduced anaerobic glycolysis during exercise, less energy is required during the recovery period to rid the muscle of H+ and lactate, thereby hastening the recovery process.

Lactate removal from muscle is enhanced by increased buffering capacity and increased blood flow, which results from blood volume, capillary density and cardiac output enhancements. Increased capillary density as seen in endurance trained individuals, provides a decreased diffusion distance between capillaries and muscle fibres, enhancing movement of oxygen and nutrients to and the removal of H+ and lactate from the muscle (Holloszy and Coyle, 1984). Enhanced oxygen delivery to muscles post-exercise potentially accelerates the rate of PCr resynthesis, an oxygen dependent process (Colliander et al., 1988; Harris et al., 1976). Tesch and Wright (1983) found a significant correlation between capillary density and blood lactate concentration, suggesting an improved efflux of lactate as a result of increased capillary density.

Other training effects seen in aerobically trained individuals that may hasten recovery are improved temperature regulation during and after exercise (Baum, Bruck and Scwennicke, 1976), better mobilization and utilization of fuel substrates (Bloom, Johnson, Park, Rennie and Sulaiman, 1975) and increased hypertrophy of and selective recruitment of slow-twitch and fast twitch type a muscle fibres (Gollnick, Armstrong, Saubert, Piehl and Saltin, 1972). The increased activity of the H form of LDH associated with endurance training (Sjodin, 1976) should also facilitate recovery by

favouring the oxidation of lactate to pyruvate. This adaptation provides ready fuel, in the form of pyruvate, for aerobic metabolism and helps normalize pH by consuming H+ (Gladden and Yates, 1993). Thus it appears that the metabolic and circulatory adaptations associated with high levels of aerobic power may facilitate faster recovery from high intensity exercise.

Research on the Relationship between Aerobic Fitness and Recovery from Intense Exercise

Numerous indicators have been used to assess recovery from exercise including force or power recovery, oxygen consumption, muscle and blood lactates, muscle pH and muscle PCr. When appropriate, research on single bouts of submaximal and maximal exercise will be presented, but it is the intention of this review to focus predominantly on the relationship between aerobic fitness and recovery from high intensity intermittent exercise.

Hamilton et al. (1991) compared the aerobic response of endurance trained (ET) runners ($\dot{V}O_2max = 60.8 \pm 4.1$ mL·kg^{-1}·min^{-1}) and games players (GP) ($\dot{V}O_2max = 52.5 \pm 4.9$ mL·kg^{-1}·min^{-1}) during and following repeated all-out 6-s treadmill sprints. While assignment to GP and ET groups was on the basis of the individuals' chosen sport rather than $\dot{V}O_2max$, maximal aerobic power was considerably different ($p < .01$), allowing for comparisons on the basis of aerobic power. Although endurance training does not result in conversion of fast twitch to slow twitch fibers, endurance trained athletes tend to have more slow twitch fibers (Gollnick and Saltin, 1983), enhanced oxidative capacity of all 3 fiber types (Pette, 1984), increased anaerobic threshold (Davis, Frank, Whipp and Wasserman, 1979), with most, but not all, achieving high rates of aerobic power (Ekblom et al., 1968). Not surprisingly, Hamilton et al. (1991) found that ET athletes consumed significantly

more oxygen during each sprint-recovery cycle than GPs ($p < .05$) since when exercising at the same percentage of $\dot{V}O_2max$, trained individuals will consume more oxygen than untrained due to their higher $\dot{V}O_2max$. They also found a strong correlation ($r = .83$) between the average increase in oxygen consumption above pre-exercise levels per treadmill sprint and $\dot{V}O_2max$. Similarly, in subjects who completed 2 30-s all-out cycle sprints, Bogdanis et al. (1996) found a high correlation between $\dot{V}O_2max$ and the percent of energy contributed by aerobic metabolism on sprint 1 ($r = 0.79$) and sprint 2 ($r = 0.87$). $\dot{V}O_2max$ thus appears to determine the magnitude of the aerobic response to repeated sprints.

Consuming more oxygen during sprinting may result in less reliance on anaerobic glycolysis and thus less lactic acid production, which could manifest itself in less lactic acid and H+ accumulation. Since the accumulation of H+ is implicated in fatigue, decreased accumulations create a more favourable contractile environment (Sahlin, 1992). If, as well, lactate is removed from muscle faster by an efficient aerobic system, as hypothesized to happen in individuals with higher aerobic power, even less lactate will accumulate, resulting in less disruption of homeostatic pH levels. Unfortunately, most studies examining lactate changes as a result of aerobic fitness rely on blood lactate measures which only reflect muscle lactate, providing indirect evidence about lactate production and removal in the muscle from lactate accumulation in blood. Some authors have reported enhanced lactate removal in endurance trained athletes (Freund, Lonsdorfer, Oyono-Enguelle, Lonsdorfer and Bogui, 1992; Gisolfi, Robinson and Turrell, 1966; Oyono-Enguelle, Marbach, Heitz, Ott, Gartner, Pape, Vollmer and Freund, 1990) while others have failed to find a relationship between lactate removal and $\dot{V}O_2max$ (Evans and Cureton, 1983; Schreiner, 1988). With the exception of Schreiner (1988), who examined blood and muscle lactates over 4 repeated bouts of high intensity exercise, all of these investigations measured blood lacates after a single bout of high intensity exercise.

Gisolfi et al. (1966) reported higher rates of blood lactate disappearance following a bout of exhaustive treadmill running in a distance runner versus a sprinter, however, with only 2 subjects this study needs to be replicated with a larger subject pool. Freund et al. (1992) found that highly trained athletes could clear lactate from working muscles faster than sedentary controls and, based on venous blood lactates, Oyono-Enguelle et al. (1990) suggest that aerobic training may enhance lactate removal following anaerobic exercise. By comparing blood lactate disappearance rates of trained versus less trained subjects from different studies, Bonen and Belcastro (1976) also concluded that trained subjects have faster lactate disappearance rates. The results of Tesch and Wright (1983) strongly support the claim that aerobic fitness is related to lactate clearance as they found a significant correlation between capillary density, which is known to increase with endurance training (Saltin and Rowell, 1980) and blood lactate concentrations. Furthermore, Tesch and Wright (1983) found that capillary density was related to the rate of force recovery over repeated sprints.

Evans and Cureton (1983) failed to find differences in blood lactate disappearance between control subjects and subjects who underwent a 6 week aerobic conditioning program which succeeded in elevating $\dot{V}O_2max$ by 15%. Apparently, the $\dot{V}O_2max$ of the control group did not change over the 6 weeks, however, as no values were reported, it is difficult to assess if, perhaps the control group's $\dot{V}O_2max$ was of sufficient magnitude to result in enhanced lactate removal, even at the start of the study. Schreiner (1988) failed to find differences in blood or muscle lactate removal over 4 30-s cycle sprints, between high ($\dot{V}O_2max > 54.6$ $mL \cdot kg^{-1} \cdot min^{-1}$) and low ($\dot{V}O_2max < 50.9$ $mL \cdot kg^{-1} \cdot min^{-1}$) aerobic power groups. It may be that $\dot{V}O_2max$ was too similar between the low and high aerobic power groups to clearly show differences or that the passive recovery between bouts suppressed the expression of the aerobic enhancements. Having relatively more females in the low aerobic power group may also have obscured the results. Since female-male differences in a 30-s test cannot be

explained by gender differences in body composition, aerobic power, strength or neuromuscular factors (Nindl, Mahar, Harman and Patton 1995; Mayhew and Salm, 1990), the possibility of biological differences in anaerobic exercise capacity between the sexes exists, and this may have been reflected in lactate removal rates documented by Schreiner (1988). Furthermore, the high aerobic power group tended to produce higher power outputs throughout all 4 trials (trials 1 through 3, $p > .05$; trial 4, $p < .05$), which may partially be explained by gender differences in strength (Mayhew and Salm, 1990). Caution must be exercised when comparing individuals on relative force recovery such as percentage of decrement (% decrement) over trials, as is conventionally done, especially when peak force varies considerably between athletes. Since subjects who produce higher peak force on initial trials have the potential to display greater absolute force decrement and therefore greater % decrement, this force recovery index may misrepresent the data. For example, greater % decrement trends has been associated with more powerful initial efforts (Dawson, Fitzsimmons and Ward, 1993; Hamilton et al., 1991). It may be that % decrement is more appropriate when comparing athletes that can be matched on initial peak force, however, this is not always possible.

Hakkinen and Myllyla (1990) found that, whereas power and strength athletes generated higher peak force, endurance athletes were able to maintain a 60% isometric contraction for much longer ($p < .001$) and following a 3 minute rest period, endurance athletes displayed the best relative force recovery. Neither $\dot{V}O_2max$ or fiber type was measured but the results might suggest that differences in force recovery are at least partially the result of differences in aerobic fitness, as aerobic fitness is generally superior in endurance trained athletes (Saltin and Astrand, 1967). Likely, differences in force recovery also reflect peak power differences resulting from the different training regimens of strength, power and endurance athletes (Hakkinen, Mero and Kauhanan, 1989). Gaiga and Docherty (1995) found that subjects participating in 9 weeks of

interval training that successfully increased $\dot{V}O_2$max by 6-7% displayed increases in peak power and mean power in all 4 repeated 30 s maximal cycling sprints, with slightly greater improvements seen in the final 2 sprints. However, from the results it is difficult to establish if recovery improved or if the interval training merely enhanced the ability to generate peak power since total work and peak power improved in all 4 repeats, with little change in the absolute power decrement from sprint 1 to sprint 4.

Both McMahon (in press) and Dawson et al. (1993) investigated the relationship between $\dot{V}O_2$max and power recovery for repeated cycle ergometer sprints. Whereas Dawson et al. (1993) found a moderate relationship between relative $\dot{V}O_2$max and power decrement during 6 6-s sprints ($r = - 0.56$; $p < .05$), McMahon (in press) failed to confirm a like relationship for 10 6-s sprints ($r = - 0.2$; $p = .4$) yet verified a significant $\dot{V}O_2$max-power decrement relationship over 6 15-s sprints ($r = - 0.63$; $p = .004$). Protocols differed somewhat in that Dawson et al. (1993) utilized a passive recovery and a 24-s recovery interval while McMahon (in press) employed active recovery for 90-s between 15-s sprints and 30-s between 6-s sprints. Perhaps the 30-s active recovery employed by McMahon (in press) between 6-s sprints utilized energy that would otherwise be available for PCr restoration, since even with passive recovery 30-s of recovery is likely inadequate for complete replenishment of PCr stores (Wooton and Williams, 1983). With inadequate time to fully replenish ATP/PCr stores (Hultman et al., 1967), each subsequent bout of exercise results in reduced PCr levels (Gaitanos et al., 1993), regardless of fitness level. It thus appears that, regardless of fitness level, the effect of an active recovery at eliminating exercise-induced acidosis (Hermansen ad Stensvold, 1972) and removing lactate (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976) is outweighed by the inability to regenerate PCr during a 30-s recovery interval. The 90-s recovery between the 15-s sprints was likely long enough to derive the benefits of active recovery, thus providing a means for expressing differences in aerobic conditioning. Dawson et al. (1993) had another

group of subjects perform 6 all-out 40 m treadmill sprints, departing every 30 seconds and found that a lower % decrement score was associated with a higher $\dot{V}O_2max$ value ($r = - 0.62$; $p < .001$). In summary, the relationships presented here seems to suggest that aerobic metabolism may contribute to improving force recovery over repeated intervals.

Although no comparisons were made to aerobic fitness level, Bogdanis, Nevill, Boobis, Lakomy and Nevill (1995) found that power recovery on repeated 30 s cycle sprints and resynthesis of PCr proceeds in parallel, confirming the relevance of PCr availability for force recovery. In a subsequent study, Bogdanis et al. (1996) analyzed pre and post exercise muscle biopsies from subjects who performed 2 30-s sprints separated by 4 minutes of passive recovery. From these results they demonstrated strong relationships between power recovery in the first 10 seconds of the second sprint and the resynthesis of PCr ($r = 0.84$) and between power recovery and endurance fitness ($r = 0.94$), as represented by the percentage of $\dot{V}O_2max$ corresponding to a blood lactate concentration of 4 mmol/L. Effectively these results link PCr resynthesis to both power recovery and endurance fitness. Interestingly, no relationship, significant or otherwise, was reported between $\dot{V}O_2max$ and power recovery or PCr restoration, so one must assume that no significant relationships were found. It may be that aerobic capacity, as characterized by 4 mmol/L blood lactate (Tanaka and Matsuura, 1984), is a better index of recovery than $\dot{V}O_2max$. Improvements in aerobic capacity are associated with improvements in oxidative capacity (Kuno and Itai, 1992) and lactate clearance (Fukuba, Walsh, Cameron, Morton, Kenny and Banister, 1992).

Only one other study compared differences in recovery from high intensity intermittent exercise and aerobic capacity (Bell, Snydmiller, Davies and Quinney, 1997). Highly trained endurance athletes performed 3 1-minute sprints at 125% $\dot{V}O_2max$, after which EPOC was measured. Utilizing ventilatory threshold as the

measure of aerobic capacity, the authors failed to find a significant relationship between aerobic capacity and recovery. While ventilatory threshold and 4 mmol/L lactate concentration both purport to represent aerobic capacity, slightly different factors affect each (Brooks, 1985), so a comparison is not always possible.

In failing to find any significant relationships between aerobic fitness ($\dot{V}O_2max$ and aerobic capacity) and EPOC (the rate of EPOC recovery and the magnitude of EPOC) following the sprints, Bell et al. (1997) concluded that there does not appear to be a relationship between aerobic fitness and recovery from repeated bouts of high intensity exercise in highly trained endurance runners. It must however be stressed, as conceded by the authors, that the population studied were highly trained (mean $\dot{V}O_2max$ = 63.1 $mL \cdot kg^{-1} \cdot min^{-1}$; range = 54.4 - 70.3 $mL \cdot kg^{-1} \cdot min^{-1}$), so even subjects with "low" aerobic power scores were quite fit and may have had an aerobic fitness level sufficient to result in enhanced recovery. The possibility of an aerobic fitness threshold exists, beyond which recovery is enhanced, however, this hypothesis remains to be tested. Unfortunately, the interpretation of the EPOC results may have been hampered by an apparent lack of pre-exercise controls in terms of circadian effects, previous exercise or the thermic effect of food, all which affect resting oxygen consumption (Poehlman, 1989) and therefore EPOC. Moreover, conclusions on the rate of recovery were based on the 1/2 time recovery of EPOC, which may be inappropriate. While it has been shown that the 1/2 time EPOC recovery for submaximal exercise improves with training (Henry and Berg, 1950; Hagberg et al., 1980; Girondola and Katch, 1973), this index may be inappropriate for cross-sectional studies. It may be that an individual's rate of recovery is determined more by genetic factors, such as the percentage of slow oxidative fibres, in that while rate of recovery can be improved, $\dot{V}O_2max$ is only one of the contributing factors. For example, Colliander et al. (1988) demonstrated that individuals in a "low fast twitch" group were superior to a "high fast twitch" group in restoring force between sets of

concentric contractions. Also, the metabolic profile of muscle fibers can be altered with endurance training through transformation of fast glycolytic fibers to fast oxidative glycolytic fibers (Jansson and Kaijser, 1977), thereby enhancing the oxidative capacity of the muscle, but the enhancements do not always translate into improvements in $\dot{V}O_2$max (Holloszy and Coyle, 1984). Furthermore, improvements in 1/2 time recovery may indicate better recovery, however similar 1/2 time recovery scores do not necessarily suggest that recovery rate was the same for different individuals. If 1/2 time EPOC recovery is the same for 2 individuals who differ in aerobic power, clearly the one with higher $\dot{V}O_2$ at the end of exercise, which is likely the one with superior aerobic power (Bogdanis et al., 1996; Hamilton et al., 1991), will utilize more oxygen in the same period of time. Therefore, use of the 1/2 time recovery rate may not accurately reflect differences in the rate of recovery.

When exercising at the same percentage of $\dot{V}O_2$max, trained individuals will consume more oxygen than untrained due to their higher $\dot{V}O_2$max. Therefore, at the start of the recovery period oxygen consumption is elevated, resulting in a greater potential for fast EPOC magnitude. As well, ATP/PCr stores in trained individuals tend to be higher (Park et al., 1988) and since PCr replenishment has been coupled to fast EPOC (Hultman et al., 1967; Piiper and Spiller, 1970), it is not surprising that fast EPOC following submaximal exercise of the same relative intensity is demonstrably higher in endurance trained individuals (Frey, Byrnes and Mazzeo, 1993; Hagberg et al., 1980; Sedlock, 1994; Short and Sedlock, 1997). Given the same percentage of $\dot{V}O_2$max, trained individuals have a larger magnitude of fast EPOC whereas total EPOC tends to be about the same and total recovery time shorter (Frey et al., 1993; Hagberg et al., 1980; Sedlock, 1994; Short and Sedlock, 1997). Hamilton et al. (1991) found that immediately following 10 repeated all-out treadmill sprints where endurance athletes (ET) consumed significantly more oxygen than games players (GP) during each sprint-recovery cycle, ET athletes tended to consume more oxygen, whereas GPs

consumed more oxygen during the remaining 14 minutes of recovery, resulting in a similar net EPOC, supporting similar patterns of EPOC seen in submaximal studies (Frey et al., 1993; Hagberg et al., 1980; Sedlock, 1994; Short and Sedlock, 1997). With more oxygen consumed sooner, the fit individual should be able to restore more ATP/PCr. The high post-exercise $\dot{V}O_2$ associated with higher aerobic power may be advantageous in priming the aerobic system to consume more oxygen immediately post-exercise which, if used to replenish ATP/PCr stores, should be advantageous for repeated exercise, especially when subsequent exercise is primarily dependent on PCr breakdown. This may at least partially explain why the ET athletes were more successful in maintaining initial power output throughout the 10 repeats. Whereas the ET and GP averaged similar power over the 10 sprints (612 versus 603 watts, respectively), and the GP tended to generate higher power outputs on the initial 4 sprints, the ET power output on the final 6 sprints tended to exceed that of the GP.

Few studies have investigated the relationship between EPOC following high intensity exercise (>$\dot{V}O_2$max) and aerobic fitness (Bell et al., 1997; Gitto, 1995; Hamilton et al., 1991) and only Gitto (1995) considered both fast and slow EPOC magnitude. Gitto (1995) examined the EPOC-aerobic fitness relationship following single bouts of 3 different supramaximal treadmill sprints lasting 1-2 minutes. Unfortunately, 6 of the 12 subjects had $\dot{V}O_2$max scores between 53.6 and 55.8 $mL \cdot kg^{-1} \cdot min^{-1}$, likely too similar for comparison on the basis of $\dot{V}O_2$max, as even day to day variations can be 2 $mL \cdot kg^{-1} \cdot min^{-1}$ (Thoden, 1991). She found a significant relationship between relative $\dot{V}O_2$max and the magnitude of fast EPOC following the 1-minute sprint at 8 mph/15% grade ($r = .63$), however failed to find significant relationships between $\dot{V}O_2$max and the rate or magnitude of EPOC for either of the other sprint protocols. She proposed the significant relationship to be spurious however, methodological differences must also be considered as the other 2 sprints were based on volitional exhaustion rather than a timed endpoint, and thereby resulted in an

exhaustive effort and higher blood lactates. Whereas no differences were seen in the magnitude of fast EPOC for the 3 sprints, total and slow EPOC were significantly higher in the volitional fatigue sprints compared to the 1-minute sprint, suggesting different metabolic demands. Finally, Gitto (1995) concluded that the rate of recovery was not related to $\dot{V}O_2max$ differences, however this may be due to it's characterization based on the 1/2 time recovery, as already discussed.

Direct evidence from ^{31}P-MRS studies appear to support the relationship seen between aerobic fitness, submaximal exercise and EPOC. Increased rates of PCr resynthesis following submaximal exercise have been documented in endurance trained athletes versus control subjects (McCully, Boden, Tuchler, Fountain and Chance, 1989) and in endurance trained athletes versus sprinters, middle-distance runners and control subjects (McCully, Vanderborne, De Meirleir, Posner and Leigh, 1992) where PCr resynthesis rate was measured in units of $mmol \cdot min^{-1} \cdot kg^{-1}$ of muscle mass. Furthermore, McCully and Posner (1992) demonstrated enhanced PCr resynthesis following only 2 weeks of muscle specific aerobic training. Unfortunately, $\dot{V}O_2max$ was not measured in any of these studies, so extrapolation of the findings to $\dot{V}O_2max$ is somewhat limited. Even though most endurance trained athletes possess high levels of aerobic power, all of the factors that determine $\dot{V}O_2max$ have yet to be determined (Holloszy and Coyle, 1984).

Few studies have investigated the relationship between aerobic fitness and PCr resynthesis following a single bout of high intensity exercise (Cooke, 1993; Petersen and Cooke, 1994; Takahashi, Inaki, Fujimoto, Katsuta, Anno, Niitsu and Itai, 1995) or high intensity intermittent exercise (Yoshida and Watari, 1993). Cooke (1993) and Petersen and Cooke (1994) utilized ^{31}P-MRS to investigate differences in pH recovery and PCr resynthesis between high aerobic power (HAP) and low aerobic power (LAP) groups following a single 2-minute bout of high-intensity exercise. Cooke (1993) found no differences in pH or PCr recovery between the groups, based

on the 1/2 time recovery of pH or PCr and non-linear regression model results, so concluded that $\dot{V}O_2max$ was a poor predictor of PCr recovery. It may be more appropriate to interpret the lack of difference in the PCr half-time recovery as a better recovery rate in HAP as, due to their higher initial PCr ($p < .05$), they actually replenished more PCr in the same time.

Petersen and Cooke (1994) found no significant differences between HAP and LAP groups in % PCr resynthesis following 2 minutes of high intensity exercise, however, they found that while pH for HAP and LAP was similar during rest and exercise, the HAP group had significantly faster pH recovery in the post-exercise period ($p < .05$). The lack of difference in % PCr resynthesis between groups does not necessarily suggest equal rates of PCr resynthesis. If the HAP group had larger resting PCr stores, as suggested by the results of Cooke (1993) and Park et al., (1988), for any given % PCr resynthesized the HAP group would replenish more PCr. In the post-exercise period, pH for both HAP and LAP decreased further from end-exercise, however the LAP pH tended to decrease more (n.s.). It may be that the HAP group was able to perform more of the exercise aerobically as a result of their enhanced oxidative capacity (Holloszy and Coyle, 1984) thus accumulating less H+, or perhaps they were able to clear lactate and H+ from the muscle more rapidly because of aerobic-induced improvements in local circulation (Anderson and Hendriksson, 1977).

Nonconformation of results from high intensity exercise PCr resynthesis rates (Cooke, 1993; Petersen and Cooke, 1994) to PCr resynthesis rates following submaximal exercise (McCully et al., 1992; McCully et al., 1989) are suggested by Cooke (1993) to be due to the high intensity nature of the exercise performed in her study which significantly altered pH. However, in another investigation $\dot{V}O_2max$ was found to be significantly correlated to the rate of PCr recovery in exhaustive exercise which resulted in similar pH decrements (Takahashi et al., 1995). Rather, it may be

that the lack of significant differences between HAP and LAP group in PCr resynthesis are more related to differences in methods for assessing PCr recovery.

It may also be that the impact of aerobic power is not as obvious with single bouts of exercise but becomes more pronounced with subsequent bouts of high intensity exercise. Yoshida and Watari (1993) used MRS to examine PCr resynthesis following 4 repeats of 2 minutes of moderately high intensity exercise, and in spite of using % PCr resynthesis to compare endurance trained and control subjects, they clearly demonstrated that endurance trained subjects ($\dot{V}O_2max$ = 73.6 $mL \cdot kg^{-1} \cdot min^{-1}$) had significantly faster PCr recovery than control subjects ($\dot{V}O_2max$ = 46.6 $mL \cdot kg^{-1} \cdot min^{-1}$), which became increasingly apparent after the first bout of exercise. It may be that the 2 groups of subjects used by Yoshida and Watari (1993) had such vastly different levels of aerobic fitness that even differences in % PCr resynthesis were apparent.

Summary and Conclusions

A strong relationship between aerobic fitness and the aerobic response to repeated cycle (Bogdanis et al., 1996) and treadmill (Hamilton et al., 1991) sprints has been established, suggesting that for high intensity intermittent exercise, aerobic fitness is important in determining the magnitude of the oxidative response. The elevation of exercise $\dot{V}O_2$ is at least partially responsible for the larger fast component of EPOC seen in endurance trained athletes following single bouts of submaximal exercise (Frey et al., 1993; Hagberg et al., 1980; Sedlock, 1994; Short and Sedlock, 1997) and, possibly, intense intermittent exercise (Hamilton et al., 1991). Unfortunately, Hamilton et al. (1991) did not measure the fast component per se and Bell et al. (1997) did not quantify the magnitude of fast EPOC following repeated bouts. Furthermore, since Gitto (1995) found a significant relationship between $\dot{V}O_2max$ and fast EPOC after

only one of the 3 different single sprint protocols, more studies are needed to clarify the relationships between EPOC and high intensity intermittent exercise.

PCr replenishment has been linked to both fast EPOC (Hultman et al., 1967) and power recovery in repeated efforts (Bogdanis et al., 1996). Whereas ^{31}P-MRS studies appear to support a relationship between endurance training and PCr recovery following submaximal work (McCully et al., 1989; McCully et al., 1992; McCully and Posner, 1992), Cooke (1993) and Petersen and Cooke (1994) failed to confirm a relationship between $\dot{V}O_2$max and PCr resynthesis following a single bout of high intensity exercise while Takahashi et al. (1995) found $\dot{V}O_2$max to be significantly correlated to PCr recovery following exercises ranging from light to exhaustive. Furthermore, Yoshida and Watari (1993) clearly showed PCr recovery to be faster in endurance trained subjects following repeated bouts of moderately high intensity exercise. It is likely that some of the differences among these studies can be explained by different PCr resynthesis assessment techniques, however more research is needed if a clear model of PCr resynthesis as it relates to aerobic fitness is to emerge.

Bogdanis et al. (1996) found a strong relationship between power recovery and endurance fitness, as measured by the % $\dot{V}O_2$max corresponding to a blood lactate concentration of 4 mmol·L^{-1}. While this couples PCr resynthesis to both power recovery and endurance fitness, $\dot{V}O_2$max was not found to be associated with either. The results of most studies examining power or force recovery and aerobic fitness seem to suggest that endurance training and/or a higher $\dot{V}O_2$max results in superior power/force recovery across repeated bouts of high intensity intermittent exercise (Bogdanis et al., 1996; Dawson et al., 1993; Gaiga and Docherty, 1995; Hakkinen and Myllyla, 1990; Hamilton et al., 1991; McMahon, in press). However, conclusions based on the findings may be complicated by the use of % decrement when subjects have different initial peak power. Future research should attempt to compare aerobic fitness and power or force recovery over repeated sprints in subjects who are matched

on initial peak power yet differ in $\dot{V}O_2max$ so that % decrement is a more meaningful index of fatigue resistance.

Some studies support an association between aerobic fitness and lactate removal following a single bout of high intensity exercise (Freund et al., 1992; Gisolfi et al., 1966; Oyono-Enguelle et al., 1990) yet others failed to confirm an association for a single bout (Evans and Cureton, 1983) or repeated bouts of exercise (Schreiner, 1988). Unfortunately, all but Schreiner (1988) relied on blood lactate measures, which may be inadequate for making conclusions on lactate removal, as discussed.

While the literature suggests that aerobic fitness may enhance recovery from high intensity intermittent exercise, more carefully planned and executed research is needed to elucidate the relationship between aerobic fitness and recovery from intense intermittent exercise.

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