THE EFFECTS OF TRAINING ON AEROBIC POWER AND EXCESS POST EXERCISE OXYGEN CONSUMPTION

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Abstract. The purpose of this study was to investigate the effects of training on aerobic power and the relationship to excess post exercise oxygen consumption (EPOC) after supramaximal exercise. Ten untrained males participated in a six week training study. The subjects performed pre and post training VO\textsubscript{2max} tests and anaerobic speed tests (ASTs). EPOC volume and EPOC rate components (τ\textsubscript{1} and τ\textsubscript{2}) as well as blood lactate responses were measured following a two minute supramaximal exercise test. Significant differences were evident between pre and post training VO\textsubscript{2max} (46.38±3.74 ml kg\textsuperscript{-1} min\textsuperscript{-1} vs. 51.82±5.21 ml kg\textsuperscript{-1} min\textsuperscript{-1} and 3.61±0.42 l min\textsuperscript{-1} vs. 4.00±0.44 l min\textsuperscript{-1}; P<0.05). EPOC volume was significantly decreased following training (9.13±1.68 l vs. 7.49±1.73 l; P<0.05). Significant differences were found between pre and post training τ\textsubscript{1} (2.69±0.19 min vs. 2.29±0.33 min; P<0.05) and τ\textsubscript{2} (43.74±5.12 min vs. 39.63±4.24 min; P<0.05). Blood lactate response was significantly decreased following training (15.28±1.80 mmol l\textsuperscript{-1} vs. 13.36±1.55 mmol l\textsuperscript{-1}; P<0.05). A significant relationship was found between the change in VO\textsubscript{2max} and the change in blood lactate concentration (r=0.73; P<0.05). No significant relationships were evident between VO\textsubscript{2max}, EPOC volume, or EPOC recovery rates (P>0.05). Findings indicated that aerobic training could decrease the VO\textsubscript{2} recovery volume and rate, as well as decrease the blood lactate response associated with anaerobic exercise. However, the rate and magnitude of the recovery VO\textsubscript{2} from supramaximal work appear to be independent of VO\textsubscript{2max}.


Key words: EPOC - Aerobic power - Training - Recovery VO\textsubscript{2} - Supramaximal exercise

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Introduction

After exercise perturbation the recovery VO$_2$, or Excess Post Exercise Oxygen Consumption (EPOC) serves to restore an individual to homeostasis. The elevated VO$_2$ after exercise appears to be dependent upon a number of interrelated factors; which include: replenishment of the high energy phosphates ATP and CP, reloading the body’s oxygen stores in the blood and muscle, increased energy cost of ventilation and circulation, metabolism of lactate, repletion of glycogen, response to exercise induced catecholamine release, oxidation of fat, increased substrate cycling rate, elevation of body temperature (Q10 effect), compensatory increased protein synthesis, ionic redistribution, thermic effect of food, and an elevated physiological functioning [5,19,20].

Changes in aerobic power and changes in the rate of recovery of EPOC associated with endurance training have led to implications that aerobic power may indicate the nature of an individual’s recovery VO$_2$ ability. Physiological changes that take place with training are responsible for an increased aerobic power (VO$_{2\text{max}}$) as well as a smaller and more rapid rate of recovery VO$_2$ for the same absolute work load administered post training. There are, however, discrepancies in the literature pertaining to the exact relationship that exists between aerobic power (VO$_{2\text{max}}$) and EPOC. Some studies provide evidence to suggest that there is no relationship between the recovery VO$_2$ and maximal aerobic power [7,9,12,17]. However, Hagberg et al. [21] and Elliot et al. [15] have indicated a relationship between recovery VO$_2$ and an individual’s level of training.

Most research investigating training and the recovery VO$_2$ after exercise has focused on recovery from submaximal exercise conditions. In fact, there is a paucity of information on the effects of an endurance training program to the recovery VO$_2$ associated with supramaximal exercise.

The purpose of this study was 1) to determine if aerobic is related to the rate and magnitude of the recovery VO$_2$ following a supramaximal work bout 2) to determine whether an endurance training program can alter the rate and magnitude of the recovery VO$_2$ after supramaximal exercise and 3) to determine if training induced changes in aerobic power are related to changes in the rate and magnitude of the recovery VO$_2$ following endurance training.

Material and Methods
**Subjects:** Ten male subjects (19-25 yrs) volunteered to participate in this study. All subjects were untrained and had a pre-training VO$_{2\text{max}}$ of less than 50 ml$\text{ kg}^{-1}\text{ min}^{-1}$. The mean ($\pm$SD) age, height, and weight were 22.4$\pm$2.1 years, 176.5$\pm$8.5 cm, 79.5$\pm$12.3 kg (pre-training) and 78.7$\pm$11.5 kg (post-training). The study was approved by the University of British Columbia Clinical Screening Committee for research and other studies involving human subjects. All subjects completed a written informed consent form prior to their inclusion in the study.

**Experimental protocol:** Subjects performed six treadmill tests (four pre-intervention tests and two post-intervention tests). These tests were performed on separate days with a minimum of two days rest between each test. Subjects were post absorptive for the two hour period prior to the testing.

Subject’s anthropometric data (height and weight) was collected on the first testing day, prior to the VO$_{2\text{max}}$ test, in the pre and post-intervention phases of the study. Metabolic data acquisition was performed with a Beckman Metabolic Measurement Cart interfaced with a Hewlett Packard 3052A Data Acquisition System.

*a. Pre-intervention phase:* All subjects performed one VO$_{2\text{max}}$ test and three AST’s over a period of two weeks. VO$_{2\text{max}}$ was determined using a continuous treadmill protocol as described in Rhodes and McKenzie [33]. Subjects met two of three criteria in order to ensure attainment of VO$_{2\text{max}}$: (i) heart rate within 10% of age predicted maximum; (ii) leveling off of VO$_{2}$ with an increase in workload; (iii) RER greater than 1.15.

A RMR measurement was performed during both the pre-intervention and post-intervention phases of the study. After an overnight fast (minimum of 8 hrs), subjects reported to the lab. Ventilatory gasses were collected for 30 min while the subjects lay in a supine position. RMR was calculated as the mean rate of oxygen consumption (relative and absolute) for the last five min of the thirty min session.

In the pre-intervention phase of the study, subjects were required to perform three Anaerobic Speed Tests (AST’s). AST # 1 was performed at 8 mph and 20% grade and AST # 2 at 8 mph and 12% grade. Subjects were required to run until exhaustion. An intensity versus duration performance curve for each individual was established from AST # 1 and AST # 2 and used to extrapolate a treadmill grade that would exhaust the subject after two min of running (2 min AST). The 2 min AST was designed to assess the maximal attainable recovery VO$_{2}$ or EPOC [30].

Maximum EPOC was measured for 30 min following the completion of the 2 min AST. Immediately upon the cessation of the exercise, collection of expired metabolic gasses began and was sampled every 15 s for the next 30 min. Oxygen
consumption was averaged for each min from the four 15 s sampling intervals. Recovery VO$_2$ magnitude (EPOC) involved summing the volume of oxygen consumed above resting values for the 30 min data collection period.

Blood lactate measurements were made at 3 min after the 2 min AST. Blood lactate samples were taken by finger prick samples and analyzed with an Accusport portable blood lactate analyzer.

b. Intervention phase - endurance training program: The endurance training program consisted of a six week training period. Subjects were required to exercise on a treadmill three times a week for 30 min each session. All training was progressive with respect to each subject’s pre-training ventilatory threshold (VT). The VT was represented by a disproportionate (nonlinear) increase in the excess CO$_2$ (ExCO$_2$) elimination curve over time [16,33]. A progression in exercise intensity was as follows:

- week 1 - treadmill speed at 16% below VT.
- week 2 - treadmill speed at 12% below VT.
- week 3 - treadmill speed at 8% below VT.
- week 4 - treadmill speed at 5% below VT.
- week 5 - treadmill speed at 3% below VT.
- week 6 - treadmill speed at pre-training VT.

The training program was monitored in the Exercise Science Laboratory at the University of British Columbia. All subjects completed the six week training program.

c. Post intervention phase: The subjects were tested on one VO$_{2\max}$ test as well as one two min AST (2 min AST). The 2 min AST was performed at the same workload and time duration as in the pre-intervention phase.

Calculations and statistical methods: Individual recovery curves (VO$_2$ vs. time) were split into two components, fast and slow, and fit to separate exponentials ($y=Ae^{-t/\tau}$). The rate constants were determined by plotting individual min recovery VO$_2$ values in log form against time in a scatter plot, and then carrying out linear regressions (i.e. $y=mx+b$). The linear regression constants $m$ and $b$ were used to derive the exponential equations and provide rate constants ($\tau_1$ and $\tau_2$) for each component of each recovery curve.

EPOC was defined as the total oxygen consumed above pre-exercise values during the thirty min recovery period and was obtained by summing the net expenditure for each minute value of the post-exercise period [36]. EPOC was collected for thirty min and expressed as an absolute value (litres) and as a relative value (ml kg$^{-1}$).
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Hotelling’s $T^2$ test was used to determine mean differences between dependent variables and Pearson Product Moment Correlation Coefficient ($r$) was used to determine relationships between variables.

Results

Table 1
Physiological characteristics and differences after a set treadmill run between pre and post training values ($n = 10$) for $\text{VO}_{2\text{max}}$, $\tau_1$, $\tau_2$, EPOC, EPOCfast, EPOCslow, and blood lactate

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre training</th>
<th>Post training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>22.4 ± 2.1</td>
<td>-</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.5 ± 8.5</td>
<td>-</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.5 ± 12.3</td>
<td>78.7 ± 11.5</td>
</tr>
<tr>
<td>$\text{VO}_{2\text{max}}$ (l min$^{-1}$)</td>
<td>3.61 ± 0.42</td>
<td>4.00 ± 0.44 *</td>
</tr>
<tr>
<td>$\text{VO}_{2\text{max}}$ (ml kg$^{-1}$ min$^{-1}$)</td>
<td>46.38 ± 3.74</td>
<td>51.82 ± 5.21 *</td>
</tr>
<tr>
<td>$\tau_1$ (min$^{-1}$)</td>
<td>2.69 ± 0.19</td>
<td>2.29 ± 0.33 *</td>
</tr>
<tr>
<td>$\tau_2$ (min$^{-1}$)</td>
<td>43.74 ± 5.12</td>
<td>39.63 ± 4.24 *</td>
</tr>
<tr>
<td>EPOC (litres)</td>
<td>9.13 ± 1.68</td>
<td>7.49 ± 1.73 *</td>
</tr>
<tr>
<td>EPOCfast (ml kg$^{-1}$)</td>
<td>116.89 ± 18.21</td>
<td>95.38 ± 16.55 *</td>
</tr>
<tr>
<td>EPOCslow (ml kg$^{-1}$)</td>
<td>15.28 ± 1.80</td>
<td>13.36 ± 1.55 *</td>
</tr>
</tbody>
</table>

*Significant difference between pre and post training values ($P < 0.05$)
Table 1 reveals the physiological variables for all the subjects. Significant differences were evident between pre and post training absolute and relative VO\textsubscript{2max} values; 3.61±0.42 l min\(^{-1}\) vs. 4.00±0.44 l min\(^{-1}\) and 46.38±3.74 ml kg\(^{-1}\) min\(^{-1}\) vs. 51.82±5.21 ml kg\(^{-1}\) min\(^{-1}\) (P<0.05). Significant differences were also evident between pre and post training absolute and relative EPOC values; 9.13±1.68 litres vs. 7.49±1.73 litres and 43.74±5.12 ml kg\(^{-1}\) vs. 39.63±4.24 ml kg\(^{-1}\) (P<0.05). Significant differences were also found between pre and post training fast component recovery rates (\(\tau_1\)); 2.69±0.19 min vs. 2.29±0.33 min; and pre and post training slow component recovery rates (\(\tau_2\)); 43.74±5.12 min vs. 39.63±4.24 min (P<0.05). Blood lactate values were significantly different from pre to post training; 15.28±1.80 mmol l\(^{-1}\) vs. 13.36±1.55 mmol l\(^{-1}\) (P<0.05).

Table 2 illustrates the relationships between VO\textsubscript{2max} and the rate constant recovery variables. No significant correlations were found between the pre and post training relative VO\textsubscript{2max} and the pre and post training rate constants. Table 3 shows the correlations between the change in VO\textsubscript{2max} and the change in the measured recovery variables. No significant correlations were found between the change in VO\textsubscript{2max} and the changes in EPOC and changes in recovery rate constants between pre and post training. There was a significant correlation evident between
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the relative change in VO\(_{2\text{max}}\) and the change in blood lactate concentration (r=0.73; P<0.05).

Table 3
Correlation coefficients for the change in VO\(_{2\text{max}}\) and the changes in the measured recovery variables (n = 10)

<table>
<thead>
<tr>
<th>Variable</th>
<th>ΔVO(_{2\text{max}}) (relative)</th>
<th>ΔVO(_{2\text{max}}) (absolute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ EPOC (relative)</td>
<td>0.51 (P=0.07)</td>
<td>-</td>
</tr>
<tr>
<td>Δ EPOC (absolute)</td>
<td>-</td>
<td>0.33 (P=0.17)</td>
</tr>
<tr>
<td>Δ τ(_1)</td>
<td>0.24 (P=0.25)</td>
<td>0.06 (P=0.44)</td>
</tr>
<tr>
<td>Δ τ(_2)</td>
<td>0.30 (P=0.20)</td>
<td>0.08 (P=0.41)</td>
</tr>
<tr>
<td>Δ [BLa]</td>
<td>0.73 (P=0.01)*</td>
<td>0.49 (P=0.08)</td>
</tr>
</tbody>
</table>

*Significant difference between relative and absolute values (P<0.05)

Discussion

The major findings from this study suggest that endurance training improves the rate and magnitude of the recovery VO\(_2\) associated with supramaximal exercise. However, no relationships were evident between aerobic power and the recovery VO\(_2\), thus suggesting that the rate and magnitude of the recovery VO\(_2\) after supramaximal exercise are independent of VO\(_{2\text{max}}\).

Researchers have demonstrated that aerobic endurance training can improve the rate of the recovery VO\(_2\) [20]. Hagberg et al. [21] showed a decreased time to recovery at both the same absolute and relative workloads after training. These researchers analyzed the recovery curves as a single exponential and reported half-times of 23 to 35 s for work rates of 50% and 70% VO\(_{2\text{max}}\) before training and 58% (previously 70%) and 70% after training. Hagberg et al. [21] reported a 21.0% reduction in recovery rate half-times, from 31.0 s to 23.3 s, for the same absolute work performed pre and post training (50% of VO\(_{2\text{max}}\)). As well, a 24.8% reduction in recovery rate half times, from 35.5 s to 26.7 s, was found for the same absolute 70% VO\(_{2\text{max}}\) workload administered pre and post training. In the present study, the recovery curves were fit to double exponential equations. A decrease in the rates of both the fast component rate constant (τ\(_1\)) and the slow component rate constant
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(τ₂) were observed post-training in this study. The pre-training fast component rate constant (τ₁) was computed as 2.69±0.19 min (t₁/₂=1.86±0.13 min) and the post-training fast component rate constant was found to be 2.29±0.33 min (t₁/₂=1.59±0.23 min). These values are slightly higher than others reported in the literature; though, the majority of research on recovery VO₂ kinetics has focused on recovery from submaximal exercise conditions. In fact a trend can be found for longer (slower) fast component recovery rates for workloads which require an energy release higher than maximal VO₂ uptake and for those which also involve a sustained lactic acidosis. In the present study the subjects were working at supramaximal exercise intensities and induced a significant lactic acidosis. This may explain the longer recovery rates reported in the present study as compared to other values reported in the literature. In the present study, the changes in recovery rate after training correspond to a 14.9% decrease in τ₁ and a 9.4% decrease in τ₂. Generally, changes in VO₂ kinetics follow changes in chronic usage of the muscles, speeding with training [10,21] and slowing with bed rest [13]. A greater training stimulus would, theoretically, result in greater changes in VO₂ kinetics. This would explain the greater change in VO₂ kinetics in the study by Hagberg et al. [21], corresponding with a greater training stimulus. In the Hagberg study, subjects exercised at a high intensity for 30-40 min per session, 6 days per week, for 9 weeks. In the present study, subjects exercised at an intensity near threshold for 30 min per session, 3 days a week, for 6 weeks. The faster VO₂ kinetics observed after training are consistent with improved O₂ delivery to, and/or enhanced aerobic capacity of the contracting muscles during and after exercise. Endurance exercise training typically results in improvements in exercise tolerance and these results are thought to be due to a combination of central (cardiac) and peripheral (skeletal muscle) adaptations. These training adaptations allow for a greater capacity to deliver O₂ from the lungs to the mitochondria of the contracting muscles [6].

The majority of research on EPOC magnitude has focused on measuring VO₂ uptake after prolonged submaximal exercise. Few studies have measured the recovery VO₂ following short bouts of supramaximal exercise involving a significant anaerobic contribution and high levels of blood lactate [5,24,34]. Bahr et al. [5] compared EPOC values from three separate intermittent two min exercise bouts on a cycle ergometer at 108% VO₂max. They measured EPOC one hour into recovery and reported values of 7.8±0.7 l (3 * 2 min.), 6.7±0.4 l (2 * 2 min), and 5.6±0.4 l (1 * 2 min). These values are much lower than the EPOCs recorded in this study. In this study, pre-training EPOCs of 9.13±1.68 l and post training EPOCs of 7.49±1.73 l were recorded after 30 min of recovery from a
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supramaximal 2 min AST. The higher EPOCs in this study are most likely attributed to the larger muscle mass engaged in the AST running test as opposed to the smaller muscle mass involved in the cycling protocol used by Bahr et al. [5].

In comparison, Rhodes and Roberts [34] found larger EPOCs from four separate sprint conditions involving either an isokinetic device (Anaerobic Power Master - APM) or free sprinting. They reported 30 min EPOCs of 15.16±2.59 l, 11.38±2.72 l, 9.88±2.80 l, and 9.09±2.51 l for a 2 min AST, 5 APM, 10 free sprints, and 5 free sprints, respectively. The 2 min AST used in the study by Rhodes and Roberts [34] as well as the 2 min AST used in the present study were administered identically. The larger EPOCs found by Rhodes and Roberts [34] most likely are attributed to the subject pool; eight highly trained sprinters. These subjects would most likely have a larger muscle mass and a greater lactate tolerance in comparison to the subjects used in the present study.

Endurance training has been shown to have a significant effect on the volume of O2 consumed in recovery from exercise [21]. Hagberg et al. [21] compared pre and post training EPOC values after nine weeks of aerobic endurance training and found that total EPOC magnitude was reduced at the same absolute workload after training. They recorded ten min pre and post training EPOC values at 50% and 70% of VO2max and found values of 0.70±0.07 l and 1.41±0.15 l, respectively. Ten min EPOC values were then recorded at the same absolute work rates post training and yielded values of 0.49±0.04 l and 1.04±0.07 l. These post-training values correspond to a reduction in EPOC magnitude of 30% and 26.2%, respectively. The larger EPOC values recorded in the present study are probably linked to the longer time for which EPOC was recorded, the higher intensity of the preceding exercise condition and the running as opposed to a cycling protocol. In the present study, a reduction in EPOC magnitude from pre to post training of 18.0% was found. The larger percentage difference in EPOC values between pre and post training in the Hagberg study as compared to the present study may be attributed to a greater training stimulus. In the study by Hagberg et al. [21] subjects trained at a high intensity, six days a week for 30-40 min a session. The training program in the present study was not as frequent or intense as the one in the study by Hagberg et al. [21]. Subjects in the present study exercised at an intensity near threshold for three days a week at 30 min per session.

In very early studies researchers postulated that blood lactate was almost exclusively responsible for the elevated VO2 seen in recovery from exercise [25,31]. The “O2 debt” hypothesis explaining the recovery VO2 was based on the assumption that the primary fate of lactate in recovery from exercise was gluconeogenesis. More recent research suggests that lactate is not so
Inconsistencies in the literature still remain regarding the effects of lactate on the recovery VO\textsubscript{2}. In the present study, no significant relationships were found between lactate and EPOC. Several other investigators have also found no significant relationships between blood lactate and EPOC [3,4,27,35]. In fact, Roth et al. [35], found no significant effect on EPOC through manipulating end exercise blood lactate levels. Interestingly, Knuttgen [27], was able to show substantial slow component EPOC magnitudes in recovery from exercise eliciting small blood lactate changes. These results contradict early work, which indicated that blood lactate concentration was predominantly responsible for the recovery VO\textsubscript{2} after exercise. In the present study the mean peak blood lactates derived from the 2 min ASTs were 15.28 mmol\textsuperscript{l}\textsuperscript{-1} (pre training) and 13.36 mmol\textsuperscript{l}\textsuperscript{-1} (post training). These values are similar to those reported in other high intensity/short duration tests from other studies; Medbo et al. [30], 13.4 mmol\textsuperscript{l}\textsuperscript{-1}; Medbo and Burgers [29], 14.9 mmol\textsuperscript{l}\textsuperscript{-1}; and Rhodes and Roberts [34], 14.83 mmol\textsuperscript{l}\textsuperscript{-1}. In comparing data from this study and that of Rhodes and Roberts [34], lactate values were within 2.38 mmol\textsuperscript{l}\textsuperscript{-1} while there was a 7.67 litre difference in EPOC values.

More recent research on the recovery VO\textsubscript{2} has shown that EPOC reflects a general metabolic disturbance to the body and that a number of physiological mechanisms increase their energy demands during exercise and into recovery and these systems contribute to the recovery VO\textsubscript{2}. Bangsbo et al. [2] has indicated that other tissues as well as muscle must be responsible for the recovery VO\textsubscript{2} because whole body EPOC is much greater than can be accounted for by local muscle events.

Physiological adaptations take place during training which would correspond with lower blood lactate levels for a set absolute workload administered post training [28]. The decreased blood lactate concentration post training may reflect lower lactate production and/or a greater lactate removal rate. In fact, Karlsson et
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al. [26] has shown muscle lactate concentration to be decreased at the same absolute and relative submaximal work rates following training. These authors indicate that at the subcellular level one of the biochemical properties responsible for the recovery VO\textsubscript{2} is decreased in the trained state. In the present study a decrease in blood lactate concentration was observed post training. A pre-training blood lactate concentration of 15.28±1.80 mmol\textsubscript{l}\textsuperscript{-1} and a post training blood lactate concentration of 13.36±1.55 mmol\textsubscript{l}\textsuperscript{-1} were recorded after a two min supramaximal exercise test. This corresponds to a 12.6% change in blood lactate concentration from pre to post training.

Early research indicated that there were individual differences in the ability to recover from exercise. Berg [8] recognized that trained individuals exhibited faster VO\textsubscript{2} recovery rates from moderate exercise. Henry and Demoor [22,23] stated that the recovery rate constants exhibit reliable individual differences and are altered by factors such as athletic training. Since this time, cross sectional studies have provided inconsistent results on the training-recovery relationship. Some cross sectional studies have shown faster kinetics in trained vs. sedentary individuals [11], higher VO\textsubscript{2max} when examining individuals differing in VO\textsubscript{2max} [14,37], and athletes differing in VO\textsubscript{2max} [32]. Other cross sectional studies provide inconsistent results to the literature, suggesting there is no relationship between VO\textsubscript{2max} and recovery VO\textsubscript{2} kinetics. Chad and Quigley [12] failed to find a significant difference in the rate of the recovery VO\textsubscript{2} uptake between trained and untrained females after a 30 min cycle ergometer test. Bell et al. [7] examined the relationship between aerobic fitness and the recovery VO\textsubscript{2} kinetics from high intensity intermittent exercise in a cross sectional sample of endurance trained cyclists. They reported no significant correlations between aerobic power (VO\textsubscript{2max}) and between the fast (τ\textsubscript{1}) and slow (τ\textsubscript{2}) rates of recovery VO\textsubscript{2}. These authors indicate that the lack of relationships between the measurements of aerobic fitness and metabolic recovery after the high-intensity, intermittent exercise bouts suggests that the physiological factors underlying the different assessments of aerobic fitness are probably too diverse to be used as indicators for the ability to recover from high-intensity, intermittent exercise. In the present study there were no correlations found between VO\textsubscript{2max} and τ\textsubscript{1} and τ\textsubscript{2} when examining the data as a cross sectional sample. There was, however, a trend for higher aerobic powers being associated with faster recovery rates. It is possible that the relatively small sample size and the homogeneity of the group did not allow for this relationship to be found. The results from this study support some of the recent work on the recovery rate-training status relationship in failing to indicate a relationship between an individuals rate of recovery and their level of aerobic fitness (VO\textsubscript{2max}).
In the present study, correlations made between the change in VO$_{2\text{max}}$ and the change in the recovery rate constants also failed to provide any significant relationships. Although significant differences were found between pre and post training VO$_{2\text{max}}$ and pre and post training $\tau_1$ and $\tau_2$, those subjects who made the greatest change in VO$_{2\text{max}}$ did not necessarily have the greatest change in recovery rate. There was, however, a trend in the data for those individuals who made a larger change in VO$_{2\text{max}}$ from pre to post training to make a larger change in the recovery rates from pre to post training. Again, it is possible that the relatively small sample size and the homogeneity of the group did not allow for this relationship to be found. In the present study, there was a 10.2% increase in VO$_{2\text{max}}$, a 14.9% decrease in $\tau_1$, and a 9.4% decrease in $\tau_2$ for recovery oxygen consumption. The relatively small differences and small changes in VO$_{2\text{max}}$ and $\tau$’s in the present study may also have been another reason for finding no significant correlation’s between VO$_{2\text{max}}$ and $\tau$VO$_2$. The results from this study are consistent with some recent work on VO$_2$ kinetics and training. Recent work has focused on the VO$_2$ uptake at the start of exercise in specific populations. Babcock et al. [1] and Barstow et al. [6] documented large differences in both pre and post training VO$_{2\text{max}}$ and $\tau$VO$_2$ at the onset of exercise. Babcock et al. [1] reported a 20% increase in VO$_{2\text{max}}$ and showed a significant decrease (48.7%) in $\tau$ for O$_2$ uptake kinetics at the start of exercise. Barstow et al. [6] showed an 11% increase in VO$_{2\text{max}}$ and a 25.94% decrease in $\tau$VO$_2$ at the start of exercise. Although these differences were found in the data, no correlation’s were found between the decrease in $\tau$VO$_2$ and the increase in VO$_{2\text{max}}$. Barstow et al. [6] also examined the change in the recovery rate of VO$_2$ uptake in relation to the increase of VO$_{2\text{max}}$ after the training program. No correlations were found between the decrease in the recovery $\tau$VO$_2$ (19.6%) and the increase in VO$_{2\text{max}}$ (11.0%).

In conclusion, the present study demonstrated that aerobic training can decrease the rate and magnitude of the recovery VO$_2$ as well as decrease the blood lactate response associated with an absolute supramaximal work bout. These results suggest that the adaptive response(s) of the aerobic energy system with training allows for more efficient recovery from the preceding exercise condition. The results also demonstrate that the recovery VO$_2$ from supramaximal work appears to be independent of VO$_{2\text{max}}$. The findings from this study as well as those from other recent research suggests that the numerous physiological factors underlying the assessment of VO$_{2\text{max}}$ are probably too diverse to allow for this single measure of aerobic power to be used as an indicator of recovery VO$_2$ ability [6,7]. Changes in the rates and volume of the recovery VO$_2$ may be more closely related to changes
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in the oxidative capacity of mitochondria within specific muscles that to whole body oxygen consumption as reflected by VO$_{2\text{max}}$.

References


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