VO₂ RESPONSE PROFILE IN HEAVY INTENSITY CYCLING AFTER
HEAVY INTENSITY ARM OR LEG EXERCISE

April Louise Ptak, B.S.

Thesis Prepared for the Degree of

MASTER OF SCIENCE

UNIVERSITY OF NORTH TEXAS

December 2006

APPROVED:

David W. Hill, Major Professor
Noreen Goggin, Committee Member and
Program Coordinator
Jon Williamson, Committee Member
Jeff E. Goodwin, Chair of the Department of
Kinesiology, Health Promotion and
Recreation
M. Jean Keller, Dean of the College of
Education
Sandra L. Terrell, Dean of the Robert B.
Toulouse School of Graduate Studies
Ptak, April Louise, \textit{VO$_2$ response profile in heavy intensity cycling after heavy intensity arm or leg exercise}. Master of Science (Kinesiology), December 2006, 34 pp., 3 tables, 3 illustrations, references, 43 titles.

The elevated CO$_2$ levels, elevated temperature, and lower blood pH that may occur during exercise should enhance O$_2$ delivery to the exercising muscles. It was hypothesized that performance of prior exercise (PE) would result in a faster VO$_2$ response, as well as a reduced slow component contribution, in subsequent exercise bouts. Five women (21 ± 1 yr) and 10 men (23 ± 2 yr) performed nine 6-min bouts of heavy intensity cycle ergometer exercise (i.e., above the ventilatory threshold, individually determined by an incremental test). Three bouts were performed without prior heavy exercise (noPE), three were performed 6 min after a 6-min bout of heavy intensity arm cranking (PE$_A$), and three were performed 6 min after a 6-min bout of heavy intensity cycle ergometer exercise (PE$_L$). Breath-by-breath VO$_2$ data from each of the three sets of three tests were combined and fitted to a two-component model, which ignores the cardiodynamic phase. The primary and slow component amplitudes were truncated to reflect actual increases in VO$_2$ in each phase. The effects of PE on the time constant of the primary component were inconsistent. As hypothesized, the amplitude of the slow component was reduced by PE (noPE vs PE$_A$ vs. PE$_L$: 25% > 16% < 14%; \(p < .05\)). It is concluded that heavy intensity PE affects characteristics of the VO$_2$ profile in a subsequent bout of heavy intensity leg exercise.
ACKNOWLEDGEMENTS

First and foremost, I would like to thank Dr. David W. Hill for providing essential support, encouraging the pursuit of knowledge and showing me countless opportunities throughout my graduate career at the University of North Texas. Without him my career in academia might not have blossomed into what it has become today and what I hope my career will continue to evolve into. I would also like to thank Dr. Noreen Goggin and Dr. Jon Williamson for their patience and service on my committee as well as their invaluable input on my manuscript. I also extend a special thanks to the staff in the Department of Kinesiology, Health Promotion and Recreation for the helping hand they lend daily and unequivocally to those of us pursuing higher education careers. I would also like to thank every investigator and participant in the exercise physiology lab over the last two years. Their service to science has provided the opportunity for UNT to continue to contribute cutting edge research to the scientific community. Finally, I would like to thank Chelsie Rowell, my colleague and friend. Without her expertise paving the way for me to follow, I would have faced many hardships and set backs without the knowledge with which to persevere.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>ii</td>
</tr>
<tr>
<td>LIST OF TABLES AND ILLUSTRATIONS</td>
<td>iv</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Exercise Intensities and Modeling</td>
<td>2</td>
</tr>
<tr>
<td>Prior Exercise Affects VO₂ Kinetics</td>
<td>5</td>
</tr>
<tr>
<td>Effects of Prior Exercise on Primary Phase Kinetics</td>
<td>6</td>
</tr>
<tr>
<td>Effects of Prior Exercise Intensities on VO₂ Kinetics</td>
<td>7</td>
</tr>
<tr>
<td>Effects of Prior Exercise Modes on VO₂ Kinetics</td>
<td>9</td>
</tr>
<tr>
<td>Possible Mechanisms</td>
<td>10</td>
</tr>
<tr>
<td>Purposes of the Study</td>
<td>12</td>
</tr>
<tr>
<td>METHODOLOGY</td>
<td>13</td>
</tr>
<tr>
<td>Participants</td>
<td>13</td>
</tr>
<tr>
<td>Procedures</td>
<td>13</td>
</tr>
<tr>
<td>Incremental Tests</td>
<td>13</td>
</tr>
<tr>
<td>Constant-Power Tests</td>
<td>15</td>
</tr>
<tr>
<td>Description of the VO₂ Response Profile</td>
<td>16</td>
</tr>
<tr>
<td>Data Analysis</td>
<td>18</td>
</tr>
<tr>
<td>RESULTS</td>
<td>19</td>
</tr>
<tr>
<td>DISCUSSION AND CONCLUSIONS</td>
<td>22</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>31</td>
</tr>
</tbody>
</table>
LIST OF TABLES AND ILLUSTRATIONS

Page

Tables
1. Exercise Responses to Prior Exercise Conditions .......................................................... 28
2. Prior Exercise (Warm-up) Descriptives ........................................................................ 29
3. Incremental Test Descriptives ..................................................................................... 29

Figures
1. Example of constant power PE_A trial ........................................................................... 27
2. Examples of the two remaining constant power protocols ........................................... 27
3. Breath by breath data from one participant modeled as a bi-exponential curve ........... 30
INTRODUCTION

Aerobic physical exercise positively increases the activity of skeletal muscle and the cardiovascular system. The body’s ability to sustain muscular exercise is highly dependent on the body’s ability to transport O₂ from the environment to be used in the mitochondrial electron transport system. It is believed that oxygen uptake (VO₂) and carbon dioxide production at the mouth reflects oxidative metabolism happening in the active muscles. Therefore, the rate of VO₂ response to aerobic exercise (VO₂ kinetics) is valuable as an index reflecting adjustments of both systemic O₂ transport and muscle metabolism (Caputo, Mello & Denadai, 2003; Carter, Grice, Dekerle, Brickley, Hammond & Pringle, 2005). It has been well described that VO₂ responses to exercise differ with varying intensities. Exercise intensities can be categorized into 4 major domains. In order of increasing intensity, these domains are moderate, heavy, severe and extreme (Gaesser & Poole, 1996; Whipp, 1987, Hill & Stevens, 2005). Each exercise intensity has a specific and mathematically mapable VO₂ response profile. Factors other than exercise mode that influence the characteristics of the individual VO₂ response profiles might include exercise work rates within a domain, age and training state of the individual, prior exercise, time of day and gender. Exercise work rates will determine if lactate will be produced, if pH will drop, if there is enough time for a slow component to emerge, the duration of the exercise and how fast VO₂max or VO₂peak will be attained. Exercise mode determines what skeletal musculature is recruited (including fiber type) and thereby how much oxygen will be consumed by the working muscles. Age and training status may influence the body’s oxygen carrying capacity, its ability to extract oxygen at the working muscles and what the overall work rate will be
for the specific exercise intensities (Phillips, Green, MacDonald, & Hughson, 1995). For example, Armon et al. (1991) found that, over a range of workrates, the O$_2$ cost of exercise was greater in children than in adults. Prior exercise, or warm-up, could speed overall VO$_2$ kinetics or change the magnitude of certain kinetic components such as, for example, the additional gain in VO$_2$ of the slow component (Jones, Wilkerson, Burnley, & Koppo, 2003; Perrey, Scott, Mourot, & Rouillon, 2003; Koppo, Jones, & Bourckaert, 2003; Koppo, & Bouckaert, 2002, 2001 and 2000; Burnley, Doust, Carter, & Jones, 2001).

Exercise Intensities and Modeling

The lowest exercise intensity domain, moderate intensity, comprises work rates at or below the lactate or ventilatory threshold (VT). Exercise in this intensity domain can be sustained ~90 minutes and can be as long as 2 hours or more. Fatigue is caused by dehydration, insufficient glucose, or boredom. Moderate exercise intensity is so low that VO$_{2\text{max}}$ cannot be reached. However, VO$_2$ will increase monoexponentially and plateau at a steady state (VO$_{2\text{ss}}$) after ~3 minutes. For moderate intensity exercise, the VO$_2$ response is characterized by two distinct phases. The first phase is a cardiodynamic phase, in which the early fast increase in VO$_2$ measured at the mouth reflects mainly an increase in cardiac output (Burnley, et al., 2001; Hill & Stevens, 2005; Casaburi, Barstow, Robinson, & Wasserman, 1989). The duration of the cardiodynamic phase is 15-20 seconds after the onset of exercise (Gerbino, Ward, & Whipp, 1996; Caputo, Mello, & Denadai, 2003). When modeling the cardiodynamic phase of the VO$_2$ response, either a monoexponential curve is used or the phase is deleted altogether.
and modeling begins using data from ~20 seconds after exercise commencement (Jones, Koppo, Burnley, 2003). After the cardiodynamic phase, VO₂ rises in an exponential fashion and appears to attain a new VO₂ss within 2-3 minutes (Carter, Grice, Dekerle, Brickley, Hammond, & Pringle, 2005; Xu & Rhodes, 1999). It is in this second phase, or primary response, that the exponential rise in VO₂ reflects muscle oxygen uptake. Therefore this phase may be analyzed to deduce the rate of oxygen uptake in the exercising muscles as the uptake is linearly related to the work rate (Jones, et al., 2003). Furthermore, the VO₂ measured can be extrapolated to estimate the VO₂ of other work rates.

Heavy intensity exercise is associated with work rates above the VT but below critical power (CP) or critical velocity (CV). Critical power is the asymptote of the mathematical relationship between time to fatigue and work-rate (Hill & Stevens, 2005). Prior research in cycling, running and swimming suggests that CP is at an intensity beyond a metabolic steady state (Carter, et al., 2005). Historically, CP was believed to represent an intensity that could be sustained “for a very long time without fatigue” (Monod & Scherrer, 1965). More recently, time to exhaustion at this intensity has been shown to be limited (Pringle & Jones, 2002). Fatigue is caused by the body’s inability to continue to buffer excess H⁺. At CP, there is a steady state until the buffer runs out and therefore it may be considered a maximal lactate steady state. Exercise in this domain can be sustained 60-90 minutes or more.

Heavy intensity exercise includes the two phases present in moderate intensity exercise (although only the primary phase is modeled) as well as an additional third phase. Unlike in moderate intensity exercise, here a delayed slow progressive rise in
VO₂ (slow component) is superimposed on the underlying monoexponential process (primary phase). For this reason, heavy exercise kinetics are often modeled using a biexponential or two-component model (Fukuba, Hayashi, Koga, & Yoshida, 2002; Koppo, Jones & Bouckaert, 2003). This third phase to the VO₂ response emerges after about 90-110 seconds of exercise and drives the VO₂ above what would be predicted based on the submaximal relationship between VO₂ and work prior to exhaustion (Åstrand & Saltin, 1961; Gaesser & Poole, 1996; Poole et al., 1988; Poole et al., 1990; Whipp et al., 1982). Thus the attainment of VO₂ss is delayed and the value of VO₂ss is greater than predicted from and demonstrated in moderate intensity exercise.

Severe intensity exercise is defined as work rates above CP or CV, or above the maximal lactate steady state (Carter, Grice, Dekerle, Brickely, Hammond, & Pringle, 2005). Exercise within the severe intensity domain can last between ~5 and 45 minutes (Carter, et al., 2005). The highest intensity at which VO₂max can be elicited is at the upper limit of the severe intensity domain (Hill & Stevens, 2005). Fatigue in this domain is caused by an inability to buffer fast enough to prevent lactic acidosis from dropping the blood pH. As in heavy intensity exercise, the VO₂ response in severe exercise also has three components (cardiodynamic, primary and slow). However, in severe intensities, the additional rise of the slow component in generally accounts for a smaller percentage of the overall peak oxygen utilization when compared with its percentage contribution in heavy exercise intensities. The magnitude of the slow component in severe intensity exercise also depends on the duration of the exercise. That is, the longer the duration of activity, the smaller the slow component amplitude.
The fourth exercise intensity domain is the extreme domain. Extreme exercise is so intense that there is not enough time to reach VO$_{2\text{max}}$ or for a slow component to emerge. The blood pH drops too fast and all of the creatine phosphate has been used for energy, causing localized fatigue to set in before VO$_2$ can reach a steady state.

Prior Exercise Affects VO$_2$ Kinetics

Aerobic exercise of varying intensities “primes” the O$_2$ transport system (Panganelli, Pendergast, Koness, & Cerretelli, 1989). The elevated CO$_2$ levels, elevated temperature, and lower blood pH that may occur during exercise should enhance O$_2$ delivery to the exercising muscles. Yet, while the overall VO$_2$ response appears faster after prior exercise, it is not clear whether it is the primary or slow component (or both) that is affected. It has been shown that the overall VO$_2$ response in heavy cycle ergometer exercise is faster, that is, shorter mean response time to VO$_{2\text{peak}}$ and reduced slow component, following a prior exercise bout on the cycle ergometer (Gerbino et al., 1996; MacDonald, Pedersen & Hughson, 1997). Mean response time (MRT) is measured as a parameter of the overall exponential response. That is, if all three phases were modeled as one monoexponential phase from the onset of exercise until exercise termination. By definition, MRT = $\tau$ + TD where $\tau$ is the time constant of the overall response and TD represents the time delay for the response after the onset of exercise. MRT is also described as the time to complete $\frac{2}{3}$ of the total change of VO$_2$ amplitude from the onset of exercise. Furthermore, the slow component in heavy leg exercise preceded by heavy leg exercise is reduced when compared with heavy leg exercise not preceded by heavy exercise. This slow component amplitude is
sometimes expressed as the difference in VO₂ between the sixth minute (or end of exercise) and the third minute of exercise [ΔVO₂ (6-3)] (Xu & Rhodes, 1999). In a related study, Bohnert, Ward & Whipp (1998) report that the speeding of VO₂ kinetics in leg exercise is less prominent, but no less significant, after arm crank prior exercise than after leg cycle prior exercise. This study also reports that, on average, VO₂ kinetics 50% above LT after a similar bout of exercise arespeeded by 32%. However, Burnley, Jones, Carter & Doust (2000) reports that prior heavy leg exercise does not “speed” VO₂ kinetics in the primary phase, but rather, reduces the magnitude of the slow component in subsequent heavy leg exercise. Also, prior heavy exercise reduces the MRT and the effective time constant of the overall VO₂ response.

However, few groups have shown that prior exercise does influence the amplitude of the primary response. For example, Burnley and colleagues (2001) noted that, if the baseline O₂ consumption is elevated prior to the second bout of exercise, then in fact the amplitude of the primary response was greater following a bout of prior exercise. More recently, Perrey and colleagues (2003) found both a reduction in the amplitude of the slow component and an increase in the amplitude of the primary response. Thus, the primary response may be affected by prior exercise.

Effects of Prior Exercise on Primary Phase Kinetics

While most studies have reported no effect of prior exercise on the amplitude of the primary response, and all studies have reported no effect on the primary phase time constant, a pattern of non-significant effects is noted. In most studies, the time constant of the primary response appears faster (or smaller) after prior exercise. Unless noted
otherwise, exercise was at heavy intensities that were approximately midway between
the VT and VO2max. For example, Koppo et al. (2003) calculated time constants of 34 s,
33 s and 31 s (NS) for no prior exercise, after prior arm exercise and after prior leg
exercise respectively. A different study by Fukuba et al. (2002), reported a time
constant of 35 s without prior exercise, 34 s after arm exercise, and 32 s after leg
exercise (NS). A different study by Koppo & Bouckaert (2001) yielded primary time
constants of 35 s and 33 s (NS) without prior exercise and after leg exercise
respectively. Jones et al. (2003) reported a time constant of 41 s at 100% of VO2max
without prior exercise and 33 s after leg exercise. Burnley et al. (2000) reported 44 s
without prior exercise and 44 s after leg exercise (the cardiodynamic phase time
constants were 15 s and 17 s respectively). Perrey et al. (2003), showed 47 s without
prior exercise and 44 s after leg exercise (the cardiodynamic phase time constants were
8 s and 15 s respectively).

Effects of Prior Exercise Intensities on VO2 Kinetics

Metabolic changes resulting from prior heavy intensity exercise have been shown
to speed VO2 kinetics in subsequent exercise above LT, but not below LT (Gerbino et
al., 1996). In the referenced study, the effective time constant was shortened by 30%,
partial O2 deficit was reduced by a similar amount, and the slow component was 60%
smaller. Gerbino also reported that moderate intensity exercise does not speed VO2
kinetics in heavy intensity and neither moderate nor heavy intensity prior exercise had
an effect on a subsequent moderate intensity exercise bout. Burnley and colleagues
(2000) also determined that prior heavy intensity exercise (but not prior moderate
intensity exercise) affected the overall VO₂ response to heavy exercise, in which a slow component is engendered, but did not affect the VO₂ response to moderate intensity exercise. They reported that the overall response was quicker after heavy intensity exercise (MRT 28% when compared to no prior exercise and 24% faster when compared to prior moderate exercise). The observed 11% reduction in the slow component amplitude following moderate intensity exercise was not statistically significant. The amplitude of the slow component in subsequent heavy exercise (increase in VO₂ from the start of the slow component until the end of exercise) was smaller by 63% and 58% when compared to no prior exercise and prior moderate exercise respectively. The amplitude and time constant of the primary response were unaffected by prior exercise.

Koppo and Bouckaert (2001) used a two component kinetics model while Burnley and colleagues (2000) used a 3 component model (including the cardiodynamic phase). Both studies reported no change in characteristics of the primary response in heavy intensity cycling exercise following a bout of heavy intensity cycling. Koppo calculated a smaller amplitude of the slow component (49% faster), which they estimated by the difference in VO₂ from minute 3 to the end of exercise at minute 6. In another study from the same laboratory, Koppo and Bouckaert (2002) reported that a similar reduction in the amplitude of the slow component could be induced by prior moderate intensity exercise. In this study, the prior moderate intensity exercise lasting 12 minutes resulted in a larger (29%) reduction than when the same intensity was engaged for 6 minutes (17% reduction in slow component amplitude) prior to exercise.
Jones and colleagues (2003) evaluated the effect of prior heavy exercise on responses to severe intensity exercise, specifically, calculated as 100%, 110% and 120% of VO$_{2peak}$. Although this study did not quantify the slow component based on their mathematical model of the two-component response, they did provide values for VO$_2$ at one minute and at the end of exercise. The VO$_2$ values at exhaustion and at one minute into exercise were significantly higher at 120% VO$_{2peak}$ after prior heavy exercise. Only VO$_2$ at one minute into exercise was higher in the 110% VO$_{2peak}$ workrate, and there were no significant differences between VO$_2$ at one minute or at exhaustion in the 100% VO$_{2peak}$ workrate. The highest intensity, for which mean times to exhaustion were 139s or 180s (after prior exercise), was close to the upper boundary of the severe intensity domain, at which no slow component would be expected (Hill & Stevens, 2005). In severe intensity exercise preceded by heavy intensity exercise, VO$_{2max}$ was attained sooner at higher workrates within severe intensity exercise domain, while the speed of the primary response was unaffected (Hill & Stevens, 2005).

Effects of Prior Exercise Modes on VO$_2$ Kinetics

Studies have also investigated whether or not the prior exercise must be performed with the same muscles as the subsequent bout of exercise to elicit a change in the VO$_2$ kinetic profile. Bohnert and colleagues (1998) reported that both prior heavy arm exercise and prior heavy leg exercise quickened the overall VO$_2$ response in subsequent leg exercise, with the effect of prior leg exercise being greater. Yoshida Kamiya, & Hishimoto (1995) reported that the VO$_2$ response profile during one-legged cycling exercise was affected only when prior exercise was performed with the same
leg. Koppo and colleagues (2003) had 10 participants perform 6-min bouts of leg ergometer exercise following 6 minutes of arm cranking or leg cycling. They found no significant change in the characteristics of the primary response. However, the amplitude of the slow component was significantly reduced by prior heavy leg exercise and, to a lesser degree, by prior heavy arm exercise (41% smaller after leg versus 16% after arm when compared to no prior exercise conditions). Fukuba and colleagues (2002) found the same pattern, although the effect of the arm exercise did not reach statistical significance. Although, both arm cranking and leg cycling prior exercise did produce similar degrees of residual lactic acidosis. This finding led to the conclusion that the changes in VO2 kinetics in supra-LT exercise preceded by heavy warm up is not only due to lactic acidosis. Instead, mechanisms that induce hyperemia at the site of active muscles are necessary to induce such changes in kinetics (Fukuba, Hayashi, Koga, & Yoshida, 2002). Thus, the consensus among researchers has been that the primary response in heavy exercise is a function of workrate and, otherwise, is immutable.

Possible Mechanisms

Why does prior exercise have a priming effect and how does it benefit exercise tolerance? The Bohr Effect (the affinity level of hemoglobin for O2) enhances O2 delivery, which has no potential effect on the cardiodynamic phase, but might speed or increase the primary phase (Boning, Hollnage, Boecker, & Goke, 1991). If the primary phase is affected, the slow component may change to accommodate changes in the primary phase. If the amplitude of the primary component is increased while the
amplitude of the slow component is reduced as a result of prior exercise, then it appears to suggest that the slow component is not an entity in and of itself but, rather, that the overall amplitude (primary gain + slow gain) is affected by prior exercise. In this case, the slow component is secondary to how well the primary component succeeds in reaching the final VO$_{2ss}$. Bohnert et al. (1998) suggests that the slow component in heavy VO$_2$ kinetics is likely a blood flow limitation to muscles and thereby affecting O$_2$ availability. MacDonald and colleagues (1997) also agree with the theory of an O$_2$ transport limitation at the onset of exercise for workrates above VT as a factor in speeding the VO$_2$ response.

Improvements in performance, time to exhaustion or exercise tolerance may be explained by residual lactic acidosis. Metabolic acidosis leads to vasodilation of active muscle tissue, thus improving perfusion and oxygen availability (Gerbino, et al., 1996; Carter, et al., 2005). Elevated blood lactate levels after heavy intensity exercise remained equally elevated after 6 minutes and 12 minutes of recovery before onset of second exercise bout (Burnley, et al., 2001). This supports the point that metabolic acidosis is the major influence on subsequent exercise responses. Koppo, Jones, Bossche & Bourckaert (2002) found that improved O$_2$ availability at the start of the second exercise bout is due to either metabolic acidosis and/or changes in muscle fibre recruitment. Type II muscle fibres are more efficient in power generation and their contribution to heavy exercise may be related to the reduction in VO$_2$ slow component. Type II fibres have relatively greater glycolytic capacity, greater O$_2$ diffusion distances and presumably slower VO$_2$ kinetics (Fukuba, Hayashi, Koga, & Yoshida, 2002). Thus
the change in the VO$_2$ slow component may be influenced by the relative amount of Type II fibre recruitment.

More recently, exercise tolerance is thought to be improved by a reduction in O$_2$ deficit as a result of prior exercise. This allows for an increased aerobic contribution to exercise (Carter, et al., 2005). Prior heavy exercise also limits the depletion of intramuscular phosphocreatine concentrations and the production of lactic acid as a means of benefiting exercise tolerance (Jones, et al., 2003; Koppo, Jones, & Bouckaert, 2003).

**Purposes of the Study**

The present study mathematically modeled the VO$_2$ response profile during heavy intensity cycling and running where a VO$_{2ss}$ would be elicited, but a VO$_{2max}$ would not be achieved. The focus was to examine the effect of prior heavy intensity arm or leg exercise on the VO$_2$ response profile during heavy intensity leg exercise. As in previous studies, the emphasis was on the quickness of the primary response (which is described by the time constant of the exponential increase in VO$_2$) and the contributions of the primary and slow components to the overall response. The hypothesis was that prior leg exercise and, to a lesser extent, prior arm exercise would cause an apparent speeding of the VO$_2$ response in a subsequent bout of exercise, and that the speeding could be attributed to changes in both the primary and slow components of the response. Also, it was hypothesized that there is a reduced contribution of the slow component to the overall VO$_2$ gain after prior exercise.
METHODOLOGY

Participants

Prior to any data collection, the study was approved the Institutional Review Board for the Protection of Human Subjects at the University of North Texas. The purpose, methods, risks and possible benefits of the study were explained to potential participants, and voluntary written informed consent was obtained. There were fifteen participants: 10 men (mean ± SD; age 23 ± 2 yr, height 179 ± 6 cm, and weight 80 ± 10 kg) and 5 women (21 ± 1 yr, 165 ± 5 cm, and 63 ± 7 kg). All were undergraduate kinesiology majors at the University of North Texas.

Procedures

Incremental Tests

First, each participant performed an incremental maximal arm cranking test and an incremental maximal leg cycling test. The tests were performed in random order, on different days, at the same time of day, under similar conditions in a temperature-controlled laboratory (20-22º C). On testing days, participants were instructed to abstain from strenuous exercise for at least 24 hours; they were to not smoke or consume beverages containing alcohol, caffeine or carbonation for at least 4 hours prior to tests. Test administrators verified adherence verbally prior to each test.

The arm cranking tests were performed on an Uppercycle (Engineering Dynamics Corp., Lowell MA, USA) upper body ergometer (UBE). Both the cycle ergometer and the UBE were on the same platform. The participant stood, straddling the cycle ergometer, while the test administrator adjusted the UBE fulcrum to the
shoulder height of the participant. This height was recorded and was readjusted for each participant prior to subsequent tests. Administrators informed the participant to use only arms and shoulders to maintain arm crank speed in an effort to reduce lower body and trunk contributions to exercise. The incremental arm crank test began at a workrate of 30 W for the men and 20 W for the women. The workrate increased 10 W each minute for the men and 5 W each minute for the women until exhaustion. The difference in base workrate as well as the per-minute increase was decided based on pilot testing. Heart rate (HR) and rate of perceived exertion (RPE) on Borg’s 6-20 scale were recorded at the end of each minute of exercise. Five seconds prior to the end of each stage, the participant pointed to the RPE the corresponded with the respective intensity. A large poster describing Borg’s 6-20 scale was held within arms reach of the participant to facilitate pointing to an intensity. Heart rate was attained from a Polar HR monitor watch (Polar B1, Kempele, Finland) with telemetric readings from a Polar HR monitor chest strap (Polar T31transmitter and belt, non coded, Kempele, Finland) that was worn by the participant for the duration of the test.

The lower body leg cycle ergometer tests were performed on an Ergomedic 828 E cycle ergometer (Monark Exercise, Varberg, Sweden). Seat height and handlebar position were individually adjusted to suit each participant. For the men, the incremental leg test began at a work-rate of 80 W while the women’s incremental leg test began at 40 W. The increase each minute was 20 W for both men and women. Again, HR and RPE were recorded at the completion of each minute of exercise and at exhaustion.
For arm and leg tests (incremental and constant power), participants were to maintain a cadence of 80 revolutions per minute (rev·min⁻¹). Participants were required to exercise in time to a metronome, and they were instructed to continue as long as possible. Tests were terminated either when the participant volitionally stopped exercising or when the cadence fell below 75 rev·min⁻¹ and the participant could not return cadence to 80 rev·min⁻¹, despite strong verbal encouragement. VO₂ and other metabolic parameters were obtained on a breath-by-breath basis using a Medical Graphics Express system (St. Paul, MN, USA), which was calibrated according to the manufacturer’s instructions immediately prior to each test. Breath-by-breath data were reduced to 15 s averages. VO₂max was determined as the highest average of consecutive 15 s values.

The VT was determined as the intensity associated with an increase in the ratio between minute ventilation (VE) and VO₂ (Caiozzo et al., 1982). Other markers included consistent increases in the pattern of response of VE, decreases in carbon dioxide production (FE CO₂), and increases in the fraction of oxygen in the expired air (FE O₂). VO₂ at the VT was determined as the average of the two 15 s values that immediately preceded the changes in responses that signaled the attainment of the VT.

Constant-Power Tests

Work rates were selected for the constant-power exercise tests based on the results of the incremental tests. Constant power for each mode was determined by 25% increase above the participants’ power sustained at VT in the incremental tests.

\[ P_{VT} + 0.25(P_{VO2\text{max}} - P_{VT}) = P_{\text{constant power}}. \]

After completing one incremental arm trial and
one incremental leg trial, each participant performed three test sessions per week for three weeks. Participants were assigned a testing schedule of Monday-Wednesday-Friday or Tuesday-Thursday-Friday depending on availability. Participants were tested at the same time of day for all 11 tests (or trials). The trials were 24 minutes in length divided into 6 segments. Two of the first 3 segments (stages 2 and 3) constitute the warm-up (or prior-exercise) phase of each trial. The prior-exercise mode was either leg cycle (PE_L) or arm crank (PE_A). Similarly, the exercise mode (stages 5 and 6) was leg cycling. In one week, participants completed three trials: one PE_L trial, one PE_A trial and one PE_L-A trial (where stages 2 and 3 were leg cycle and stages 5 and 6 were arm crank). The PE_L-A trials were not analyzed in this study. Stages 1 and 4 are rest periods involving no exercise to allow the subject to regain a pre-exercise baseline starting point.

The prior-exercise stages 1 and 2 were each three minutes in length. The first three minutes involved no exercise and only necessary movements to allow for breathing adjustments to the mouthpiece. The next three minutes (stage 2) were spent in a moderate intensity. If the mode was leg cycle, the unloaded intensity was 60W for men and 40W for women. If the mode was arm crank, the unloaded intensity was 30W for men and 20W for women. Stage 2 was followed by an immediate (square wave) increase to the desired intensity (stage 3). This lasted six minutes at a work rate of Δ25% above VT work rate. These three stages were repeated in stages 4, 5 and 6. During the rest intervals (stage 1 and stage 4), participants stood for 3 minutes, straddling the cycle ergometer. Verbal encouragement from the test administrators was used when necessary to keep the participants motivated and to keep them focused on
their cadence. The directions and conditions employed in the incremental tests were also used in each of the 9 trials.

Description of the VO₂ Response Profile

For each prior-exercise test and each constant-power exercise test, parameters of the VO₂ response profile were determined using nonlinear regression techniques on KaleidaGraph 3.50 (Reading, PA, USA), by fitting breath-by-breath VO₂ responses to a model with two exponential terms (primary and slow components). Data from the first 20 seconds of exercise (representing the cardiodynamic phase) were excluded from the analysis. The two-component model is computed as follows:

\[
\text{VO}_2(t) = A_{\text{baseline}} + (A_{\text{primary}} \cdot (1 - e^{-(t - T_{D\text{primary}})} / \tau_{\text{primary}})) + (A_{\text{slow}} \cdot (1 - e^{-(t - T_{D\text{slow}})} / \tau_{\text{slow}}))
\]

\(\text{(1).}\)

\(\text{VO}_2(t)\) is the value for VO₂ at time = t. \(A_{\text{baseline}}\) is the measured average VO₂ value over the last 60 seconds of the resting baseline; \(A_{\text{primary}}\) and \(A_{\text{slow}}\) are the asymptotic amplitudes for the two exponential terms; \(\tau_{\text{primary}}\) and \(\tau_{\text{slow}}\) are the time constants for the respective exponential terms; and \(T_{D\text{primary}}\) and \(T_{D\text{slow}}\) are the time delays for the commencement of each phase. In this model, the primary phase is considered terminated at the onset of the slow phase (i.e., at \(T_{D\text{slow}}\)) and was assigned an oxygen uptake value for that time (\(A'_{\text{primary}}\))

\[
A'_{\text{primary}} = A_{\text{primary}} \cdot (1 - e^{T_{D2} / \tau_{\text{primary}}})
\]

\(\text{(2).}\)
Similarly, the slow component was terminated at the end of exercise and its achieved amplitude was calculated ($A'_{\text{slow}}$). A time equal 0 was assigned to the beginning of both stage 3 and stage 6 so that the end time of each phase was 360 seconds.

$$A'_{\text{slow}} = A_{\text{slow}} \cdot (1 - e^{(\text{time to fatigue} - T_{D_{\text{slow}}}) / \tau_{\text{slow}}})$$  \hspace{1cm} (3).$$

Finally, the overall time constant ($\tau_{\text{overall}}$) of the response in each test was determined using a simple monoexponential equation with no delay,

$$VO_2(t) = A_{\text{baseline}} + (A_{\text{overall}} \cdot (1 - e^{-t / \tau_{\text{overall}}}))$$  \hspace{1cm} (4).$$

Data Analysis

Parameters of the $VO_2$ responses were calculated for stage 3 of the L-L tests (no PE), from stage 6 of the L-L tests (PE_L) and from stage 6 of the A-L tests (PE_A). Using a two-way ANOVA (type of PE by sex) with repeated measures across type of PE, it was concluded that there was no effect of sex aside from the difference in amplitude of the responses. These responses were then reanalyzed using one-way repeated measures ANOVA in SPSS version 12.0 (SPSS Inc., Chicago, IL, USA) to view the effects of PE_L and PE_A to a control of no PE. For all analysis, the 0.05 probability level was used to denote statistical significance. All values reported in this paper are given as the mean ± standard deviation.
RESULTS

As expected, the VO\textsubscript{2max} that was achieved in the lower body incremental exercise tests (39.37 ± 8.5 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} or 2874.7 ± 695.5 ml/min) was higher (by 26%, \textit{p} < .001) than the value achieved in upper body tests (29.07 ± 5.6 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} or 2124.4 ± 478.4 ml/min). Maximal heart rate was slightly higher (+ 3.7 %, \textit{p} < .001) in lower body exercise (190 ± 10 bt·min\textsuperscript{-1}) than in upper body exercise (183 ± 11 bt·min\textsuperscript{-1}). Workrates corresponding to the power at VT + \Delta25\% of the difference between power at VT and maximal power from the incremental tests averaged 137 ± 48 W for arm cranking and 223 ± 54 W for leg cycling. These workrates produced different metabolic demands in both arm exercise and leg exercise. Heavy exercise performed with the arms resulted in a lower relative VO\textsubscript{2peak} (21.66 ± 0.85 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} versus 34.55 ± 2.08 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}, \textit{p} < .001) and a lower absolute VO\textsubscript{2peak} (1598.4 ± 73.8 ml·min\textsuperscript{-1} versus 2540.47 ± 156.36 ml·min\textsuperscript{-1}, \textit{p} < .001) when compared with heavy leg exercise. Despite the different metabolic demands of the prior exercise conditions, the VO\textsubscript{2peak} in PEA (34.88 ± 2.0 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} or 2572.47 ± 156.4 ml·min\textsuperscript{-1}) was not significantly different than in PEL (34.69 ± 7.64 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} or 2555.4 ± 152.7 ml·min\textsuperscript{-1}). Consequently, participants reported on Borg’s RPE scale that PEA felt no more demanding than PEL (15.6 ± 1.76 versus 16.3 ± 1.80) while only PEL felt more demanding than the control condition (13.9 ± 2.5, \textit{p} = .008). The absolute VO\textsubscript{2} achieved under the three constant power conditions did not differ (\textit{p} > .05 ) with values being 2540 ± 605 ml·min\textsuperscript{-1} (control), 2572 ± 605 ml·min\textsuperscript{-1} (PE\textsubscript{A}), and 2555 ± 591 ml·min\textsuperscript{-1} (PE\textsubscript{L}).
In short, only one of the three hypotheses can be accepted in this research study. It was found that the slow component amplitude was significantly smaller after PEL and tended to be smaller after PEA.

Descriptors of the VO$_2$ response profile that were generated using equations 1 to 4 are presented in Table 1. In several cases, when the slow component began, the VO$_2$ increased in a linear fashion until the end of exercise. In these cases, the values for $A_{\text{slow}}$ and $\tau_{\text{slow}}$ were exceptionally large (i.e., $A_{\text{slow}}$ greatly exceeded the actual increase ($A'_{\text{slow}}$) observed in exercise). Therefore, means for $A_{\text{slow}}$ and $\tau_{\text{slow}}$ are not provided. Rather, the more meaningful $A'_{\text{primary}}$ and $A'_{\text{slow}}$ are given in Table 1.

Neither prior heavy arm cranking nor prior heavy leg cycling had an effect on the overall VO$_2$ response. It was hypothesized that heavy PE would speed the overall VO$_2$ response. A non-significant trend appears, that $\tau_{\text{overall}}$ is smaller after PE and even smaller in PEL than PEA (Table 1; $p = .22$).

Similarly, neither prior exercise condition had an effect on the primary phase parameters when compared to the control condition. There were no significant differences in $\tau_{\text{primary}}$, TD$_{\text{primary}}$, $A_{\text{primary}}$ or $A'_{\text{primary}}$ (Table 1). The PEL condition resulted in a 7.2% reduction in $\tau_{\text{primary}}$ when compared to the control, but this was not significant ($p = .058$). If sphericity is assumed, then $\tau_{\text{primary}}$ is 27.2% faster in PEL than PEA ($p = .004$).

Both heavy arm and heavy leg exercise had significant effects on slow component kinetics. The TD$_{\text{slow}}$ was 37% slower PEA compared to no PE ($p = .008$) and 29% slower in PEL when compared to no PE ($p = .007$). As hypothesized, $A'_{\text{slow}}$ was reduced after both prior heavy exercise conditions. The slow component was reduced
by 51.5% by prior heavy cycling (p = .032) and reduced by a similar amount, but not a significantly (p = .052), by prior heavy arm crank exercise. Also, the amplitude of the slow component tended to contribute a smaller percentage to the overall VO₂ amplitude after heavy leg exercise (p = .216) and did contribute a significantly smaller percentage after heavy arm exercise (p = .047).
DISCUSSION

The principal findings of this study were that (1) neither prior heavy exercise in arm or leg affected the overall VO₂ response; (2) prior heavy exercise had no effect on the time constant, the amplitude or the time delay of the VO₂ primary phase irrespective of whether the prior exercise is performed by the arms or the legs when compared to the control; and (3) the slow component amplitude was smaller after heavy exercise using the same muscle group ($p = .032$) and tended to be smaller after heavy exercise with a remote muscle group ($p = .052$).

Others often report that the overall response of VO₂ during heavy/severe intensity exercise is slower in small muscle exercise, whether the comparison is between arm exercise and leg exercise (Casaburi et al., 1992; Koga et al., 1996; Schneider et al., 2002), cycle ergometer and treadmill exercise (Caputo & Denedai, 2004; Hill et al., 2002, 2003), or one-legged and two-legged exercise (Koga et al., 2001). It is not clear whether the overall faster response in large muscles is a function of the role of muscle mass per se or a function of other factors associated with different forms of exercise, such as cadence, duty cycle, or extent of associated isometric muscle actions.

It is also not clear from the research whether differences in the overall response of VO₂ are related to differences in the primary response or differences in the slow component. Only studies which use a comprehensive mathematical model to determine characteristics of both the primary and slow components can address this issue. The most current consensus is that the apparent speeding of the overall VO₂ response results from a reduction in the slow component amplitude along with a similar increase
in the primary component amplitude without a change in the MRT or time constant of
the primary phase or the overall VO2 response (Jones, Wilkerson, Burnley & Koppo,

Early research suggested that prior heavy exercise “speeded” the VO2 response
via reduction in MRT and/or the overall time constant in the subsequent heavy exercise
bout (Gerbino, et al., 1996; MacDonald, et al., 1997). The recent studies that use more
advanced mathematical models demonstrate that the apparent speeding of the VO2
response is a result of a reduction in the slow component amplitude without much if any
effect on the primary phase of the VO2 kinetics (Burnley, et al., 2001; Koppo, &
Bouckaert, 2002; Fukuba et al., 2002). These more advanced mathematical models
provide a better fit to the data, and thereby a better representation of the VO2 response
than a mono-exponential model (Barstow, et al., 1993; Koppo & Bouckaert, 2001;
Koppo, et al., 2003). Following this research, the present study used a two-component
(or bi-exponential) mathematical model to describe the kinetic parameters of VO2
primary and slow components more precisely (Figure 3). The estimated kinetic
parameters from the two-component model showed that the main affects on overall VO2
kinetics between PEL and the control reside in the slow component and not the primary
phase. The slow component was significantly reduced (i.e. A_{slow}) when compared to no
PE. The amplitude in the primary phase remained virtually the same in PEL as in PE_A
and in no PE.

Barstow et al. (1993) reported markedly slower primary response in one group of
untrained participants performing heavy intensity arm exercise than in another group of
participants performing heavy intensity leg exercise. Our results follow this pattern in
that the $\tau_{\text{primary}}$ is significantly slower in $\text{PE}_A$ than in $\text{PE}_L$ (-27.2%) and with the same group of subjects. This was the only significant difference in the primary phase to be reported from the present study. Koga and colleagues (1996) reported that the overall response was slower in arm exercise ($\tau_{\text{overall}} = 57$ s) than in leg exercise ($\tau_{\text{overall}} = 41$ s) at ~75% of $\text{VO}_2\text{max}$ and without any prior exercise. However, there was no difference in $\tau_{\text{primary}}$ in arm exercise when compared to leg exercise. This may further support the idea that prior exercise affects the slow component.

Gerbino and colleagues (1996) hypothesized that a facilitated $\text{VO}_2$ response during subsequent heavy leg exercise was caused by lactic acidosis since the facilitated response was not observed in exercise below VT. Lactic acidemia is assumed to induce vasodilation and thereby increase muscle blood flow for both improved $\text{O}_2$ transport to the exercising muscles and $\text{O}_2$ utilization by the same exercising muscles (Fukuba, et al., 2002). Results of Fukuba et al. (2002) suggest that hyperemia induced by a warm-up using the same mode as to be used in the performance exercise bout may contribute to the facilitation of $\text{VO}_2$ kinetics, leaving systemic lactic acidosis a less important role to play.

With respect to $\text{O}_2$ delivery to active skeletal muscles, studies indicate that oxygen delivery to muscular sites plays no major role in priming of $\text{VO}_2$ kinetics. Instead, the intrinsic inertia of oxidative metabolism in muscle cells may be the central focus regulating $\text{VO}_2$ kinetics at the onset of heavy exercise (Fukuba, et al., 2002).

With respect to $\text{O}_2$ utilization within the muscle, it makes sense that this would depend on the recruitment of less efficient type II muscle fibers during heavy exercise as suggested by Gaesser and Poole (1996). In fact, the magnitude of the slow
component response to a single heavy exercise bout was significantly related to the percentage of type II muscle fibers recruited in the vastus lateralis (Barstow, Jones, Nguyen & Casaburi, 1996). Type II fibers have a greater glycolytic capacity, greater O₂ diffusion distance and possibly slower VO₂ kinetics (Walsh, 1992). After a warm-up involving the same muscle group, the matching of O₂ delivery to O₂ demand (i.e. a reduction of the O₂ deficit) may be more rapidly established due to a greater availability of O₂. Thus, after a warm-up, a more aerobic metabolic profile will recruit a greater amount of type I fibers and reduce the need for type II muscle fibers in the second exercise bout. This reduced recruitment for type II muscle fibers may explain the smaller slow component after prior exercise (Fukuba, et al., 2002).

While the muscle fiber recruitment hypothesis might explain the effect of prior heavy leg exercise on the VO₂ response profile in subsequent heavy leg exercise, it is more difficult to explain how this mechanism might operate when heavy arm exercise precedes a bout of heavy leg exercise. Koppo, et al. (2003) speculate that the accumulation of H⁺, lactate and K⁺ in the non-working leg muscle following heavy arm exercise could cause fatigue or affect motor unit recruitment patterns when the non-working muscle is put to use in the subsequent exercise bout. In the present study, however, the only significant effect of prior heavy arm exercise on VO₂ kinetics in heavy leg exercise was seen in the reduction of the slow component as a percentage of the combined primary and slow VO₂ amplitude when compared to the control condition (44.5% smaller, p = .047).

The reduction in the VO₂ slow component amplitude in PE_L led to a small but significant decrease in overall VO₂ when compared to the no PE condition (A_{overall} =
12.4% smaller in PE_L than no PE). It is possible that a higher baseline VO_2 was the starting point for the second bout of exercise. This elevated residual metabolic level may have obscured the decrease in A_{overall}. Also of importance to note is that the slow component emerged after an average of 191.9 s in PE_L and 216.6 s in PE_A. These values are a bit longer than expected based on prior literature (Koppo, et al., 2003; Carter, et al., 2000) in which TD_{slow} of 60-156 s and 65-155 s are reported respectively. This could be a result of the rest period in the present protocol. The participants did not engage in any activity for 3 minutes before beginning a moderate intensity warm up for 3 minutes. Or perhaps the relative work rates were not as high as in previous studies. The work rates in the present study were based on power outputs that yielded 60% of VO_{2max} in leg exercise and 53% of VO_{2max} in arm exercise in the incremental tests.

In conclusion, prior exercise must be heavy or severe to facilitate or prime the VO_2 response profile in subsequent exercise bouts. The subsequent exercise bouts must also be in the heavy or severe exercise intensity domain to see such priming effects. The mode of prior exercise does matter, however prior arm exercise may not always affect subsequent responses in leg exercise. This observation allows for the argument that facilitation of the VO_2 response as elicited by prior heavy exercise may have both a local affect on the skeletal musculature recruited and a central affect of speeding O_2 delivery.
Figure 1. Example of constant power $P_{EA}$ trial. Exercise only stages are labeled (Stages 1 and 4 are rest periods where only standing took place). $VO_2$ data is continually recorded in rest stages.

Figure 2 and 2a. Examples of the two remaining constant power protocols. Example constant power protocols of L-L trials (2) and L-A trials (2a). Exercise only stages are labeled (stages 1 and 4 are rest periods). $VO_2$ data is recorded in rest stages. Stage 3 from both of these protocols was analyzed as the control condition or “no PE”. Stage 6 from 2a was analyzed as “PE L”. The protocol from figure 2a was not analyzed for the present study.
Table 1

Exercise Responses to Prior Exercise Conditions

<table>
<thead>
<tr>
<th>Parameters</th>
<th>No PE</th>
<th>PE&lt;sub&gt;A&lt;/sub&gt;</th>
<th>PE&lt;sub&gt;L&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VO2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO&lt;sub&gt;2peak&lt;/sub&gt;, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>2540 ± 606</td>
<td>2573 ± 606</td>
<td>2555 ± 591</td>
</tr>
<tr>
<td>VO&lt;sub&gt;2peak&lt;/sub&gt;, m·kg&lt;sup&gt;-1&lt;/sup&gt;·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>34.6 ± 8.1</td>
<td>34.9 ± 7.7</td>
<td>34.7 ± 7.6</td>
</tr>
<tr>
<td>Baseline VO&lt;sub&gt;2&lt;/sub&gt;, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>1191 ± 193&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1283 ± 209&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1322 ± 245&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Baseline VO&lt;sub&gt;2&lt;/sub&gt;, m·kg&lt;sup&gt;-1&lt;/sup&gt;·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>16.1 ± 1.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.3 ± 1.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>17.8 ± 2.3&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>RPE</td>
<td>13.9 ± 2.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>15.6 ± 1.8&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>16.3 ± 1.8&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Heart Rate (b/min)</td>
<td>167 ± 13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>173 ± 13&lt;sup&gt;b&lt;/sup&gt;</td>
<td>175 ± 14&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

**Monoexponential modeling**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>No PE</th>
<th>PE&lt;sub&gt;A&lt;/sub&gt;</th>
<th>PE&lt;sub&gt;L&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>τ&lt;sub&gt;s&lt;/sub&gt;, s</td>
<td>103.8 ± 39</td>
<td>94.6 ± 37</td>
<td>84.8 ± 25</td>
</tr>
<tr>
<td>A&lt;sub&gt;s&lt;/sub&gt;, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>1350 ±541&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1222 ±533&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>1182 ±454&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>A&lt;sub&gt;s&lt;/sub&gt; + Baseline VO&lt;sub&gt;2&lt;/sub&gt;, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>2542 ± 596</td>
<td>2505 ± 669</td>
<td>2505 ± 585</td>
</tr>
</tbody>
</table>

**Two-component modeling**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>No PE</th>
<th>PE&lt;sub&gt;A&lt;/sub&gt;</th>
<th>PE&lt;sub&gt;L&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>TD&lt;sub&gt;p&lt;/sub&gt;, s</td>
<td>9.62 ±1.72</td>
<td>7.9 ±1.8</td>
<td>10.22 ±0.94</td>
</tr>
<tr>
<td>τ&lt;sub&gt;p&lt;/sub&gt;, s</td>
<td>54.05 ± 34.5&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>68.9 ± 7.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>50.17 ± 19.7&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>A&lt;sub&gt;p&lt;/sub&gt;, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>1011 ± 462</td>
<td>1106 ± 484</td>
<td>987 ± 501</td>
</tr>
<tr>
<td>A&lt;sub&gt;p&lt;/sub&gt;′, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>913 ± 440</td>
<td>1055 ± 488</td>
<td>956 ± 503</td>
</tr>
<tr>
<td>TD&lt;sub&gt;s&lt;/sub&gt;, s</td>
<td>136.3 ± 62.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>216.6 ± 73&lt;sup&gt;b&lt;/sup&gt;</td>
<td>191.9± 53.7&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>A&lt;sub&gt;s&lt;/sub&gt;′, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>324±249&lt;sup&gt;a&lt;/sup&gt;</td>
<td>156 ± 113&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>157 ± 78&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>%A&lt;sub&gt;s&lt;/sub&gt; of overall A, %</td>
<td>24.7±14.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>13.7 ± 9.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>16.3±8.9&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>VO&lt;sub&gt;2&lt;/sub&gt; Total, ml·min&lt;sup&gt;-1&lt;/sup&gt;</td>
<td>2429 ± 624</td>
<td>2437 ± 647</td>
<td>2494 ± 716</td>
</tr>
</tbody>
</table>

Note: VO<sub>2</sub> total is a sum of Baseline VO<sub>2</sub>, A<sub>p</sub> and A<sub>s</sub>′. When ANOVA revealed a significant difference, results of post hoc tests are provided; means sharing a similar superscript were not different. Significance level set at p < 0.05.
Table 2  
**Prior Exercise (Warm-up) Descriptives**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Arm Crank Prior Exercise</th>
<th>Leg Cycle Prior Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$, ml·min$^{-1}$</td>
<td>1598 ± 286 $^a$</td>
<td>2540 ± 606 $^b$</td>
</tr>
<tr>
<td>VO$_2$, ml·kg$^{-1}$·min$^{-1}$</td>
<td>21.7 ± 3.3 $^a$</td>
<td>34.6 ± 8.1 $^b$</td>
</tr>
<tr>
<td>% of VO$_{2peak}$ (from incremental)</td>
<td>75.8 ± 6.8</td>
<td>89.9 ± 8.6</td>
</tr>
<tr>
<td>Heart Rate, bpm</td>
<td>152 ± 13 $^a$</td>
<td>167 ± 13 $^b$</td>
</tr>
<tr>
<td>% Heart Rate max (from incremental)</td>
<td>83.1%</td>
<td>87.9%</td>
</tr>
<tr>
<td>RPE$_{peak}$</td>
<td>12.2 ± 1.9</td>
<td>13.9 ± 2.5</td>
</tr>
</tbody>
</table>

*Note:* Values in this table are taken from stage 3 of the PE$_{arm}$ and PE$_{leg}$ protocols respectively. When ANOVA revealed a significant difference, results of post hoc tests are provided; means sharing a similar superscript were not different. Significance level set at $p < 0.05$.

Table 3  
**Incremental Test Descriptives.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Arm Crank (UBE)</th>
<th>Leg Cycle (Monark)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO$_2$ @ VT, ml·min$^{-1}$</td>
<td>1400 ± 320</td>
<td>1868 ± 639</td>
</tr>
<tr>
<td>VO$_{2max}$, ml·min$^{-1}$</td>
<td>2124 ± 478</td>
<td>2875 ± 695</td>
</tr>
<tr>
<td>VO$_{2max}$, ml·kg$^{-1}$·min$^{-1}$</td>
<td>29.1 ± 5.7</td>
<td>39.4 ± 8.5</td>
</tr>
<tr>
<td>% VO$<em>2$ @ VT of VO$</em>{2max}$</td>
<td>66.4 ± 9</td>
<td>64.7 ± 10</td>
</tr>
<tr>
<td>Heart Rate @ VT, bpm</td>
<td>153 ± 18</td>
<td>155 ± 18</td>
</tr>
<tr>
<td>Heart Rate max, bpm</td>
<td>183 ± 11</td>
<td>190 ± 10</td>
</tr>
<tr>
<td>% VT Heart Rate of Heart Rate max</td>
<td>83.1 ± 5.8</td>
<td>81.3 ± 6.7</td>
</tr>
<tr>
<td>Power @ VT, Watts</td>
<td>58.8 ± 15.3</td>
<td>136.9 ± 47.5</td>
</tr>
<tr>
<td>Power$_{max}$, Watts</td>
<td>113.1 ± 30.2</td>
<td>223.1 ± 54.1</td>
</tr>
<tr>
<td>% Power @ VT of Power$_{max}$</td>
<td>53.0 ± 10</td>
<td>60.8 ± 10</td>
</tr>
<tr>
<td>RPE @ VT</td>
<td>12.7 ± 2.0</td>
<td>11.5 ± 2.3</td>
</tr>
</tbody>
</table>
Figure 3. Breath by breath data from one participant modeled as a bi-exponential curve. Three different graphs from the same participant on one coordinate plane. The $\tau_p$ (tau primary) appears to get faster after a warm up and even faster still after a warm up with larger muscles as well as the same muscles used in the exercise bout.
REFERENCES


Koppo, K., & Bouckaert, J. (2002). The decrease in the VO₂ slow component induced by prior exercise does not affect the time to exhaustion. *International Journal of Sports Medicine, 23*, 262-267.

Koppo, K., Jones, A.M., Bossche, L.V., & Bouckaert, J. (2002). Effect of prior exercise on VO₂ slow component is not related to muscle temperature. *Medicine & Science in Sport & Exercise, 34*(10), 1600-1604.

Koppo, K., & Bouckaert, J. (2000). In humans the oxygen uptake slow component is reduced by prior exercise of high as well as low intensity. *European Journal of Applied Physiology, 83*, 559-565.


