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**THE EFFECT OF WARM-UP ON VO<sub>2</sub> KINETICS DURING HEAVY EXERCISE**

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THE EFFECT OF WARM-UP ON VO<sub>2</sub> KINETICS DURING HEAVY EXERCISE

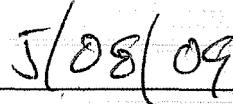
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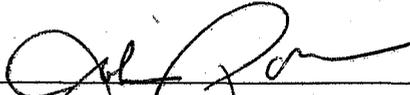
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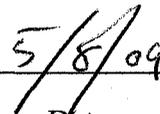
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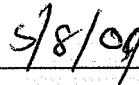
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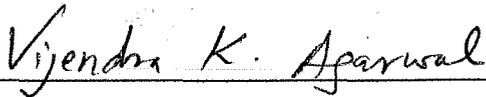


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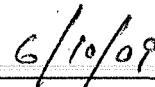


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## ABSTRACT

Stockman, M.T. The effect of warm up on VO<sub>2</sub> kinetics during heavy exercise. MS in Clinical Exercise Physiology, December 2009, 51 pp. (C. Foster)

Warm-up (WU) is considered an essential part of exercise training in athletic, preventive and rehabilitative exercise programs. Exactly how WU augments exercise performance is unclear. Previous studies during free range exercise have shown down-regulation of power output (PO) and slower oxygen uptake (VO<sub>2</sub>) kinetics in the absence of WU. However, since PO early during an exercise bout is known to drive VO<sub>2</sub> kinetics, it is unclear whether these results are primary or responsive to the lower PO in the absence of WU. Eight healthy, physically active volunteers ( $23.1 \pm 2.1$  years) performed two 3 km time trials (TT), the first with WU to define mean PO ( $279 \pm 84$  watts), the second without WU, and two square wave (SW) exercise tests to fatigue at a PO equal to the mean achieved on the TT with WU; one was performed with no WU and the other followed a 15 min WU protocol. Repeated measures ANOVA was used to analyze performance time, VO<sub>2</sub> kinetics as measured by Mean Response Time (MRT), peak power output (PPO), % PPO, heart rate (HR) response, ventilation, and rating of perceived exertion. Following WU, performance time in the SW tests was not significantly greater ( $371.3 \pm 165.7$  s vs.  $338.3 \pm 155.1$  s), maximal VO<sub>2</sub> was significantly greater ( $4.2 \pm 1.1$  vs.  $3.9 \pm 0.9$  L/min), HR was significantly greater ( $178 \pm 9$  vs.  $171 \pm 10$  bpm), but MRT was not different ( $48.0 \pm 13.6$ s vs.  $45.1 \pm 7.9$  s). Thus, the improved performance and overall aerobic response to exercise following WU could not be attributed to differences in MRT. These results fail to support the hypothesis that acceleration of VO<sub>2</sub> kinetics, independent of differences in muscle PO, is an outcome of WU. These results suggest that the down-regulation of PO previously observed in studies of WU were responsible for the slower VO<sub>2</sub> kinetics.

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## INTRODUCTION

In the transition from rest to exercise our bodies increase the oxygen supply to our muscles by the complex orchestration of pulmonary, cardiovascular and muscular processes. As exercise begins, the anaerobic systems are the first to respond. Oxidative phosphorylation, while capable of producing larger amounts of energy, takes longer to increase its contribution. However, the prolonged use of anaerobic processes can lead to hydrogen ion accumulation and to substrate (glycogen and phosphocreatine [PCr]) depletion which is felt to be related to increased fatigue and reduced exercise tolerance. Individuals who can adapt to the demands of exercise with a quicker rise in oxygen uptake ( $\text{VO}_2$ ) will reduce this "oxygen deficit" and preserve anaerobic reserves leading to greater endurance and exercise tolerance. The dynamics of  $\text{VO}_2$  kinetics are affected positively by exercise training and negatively by aging and many disease states. A.V. Hill first demonstrated the exponential nature of the  $\text{VO}_2$  response at the onset of exercise in the 1920s (18). Since that time the number of studies involving  $\text{VO}_2$  kinetics have increased as researchers are now investigating how oxygen uptake kinetics are affected by various disease states and by athletic training.

$\text{VO}_2$  kinetics are typically measured through pulmonary gas exchange analysis either using breath-by-breath analysis (7, 8, 17) or open-circuit spirometry (14). Pulmonary  $\text{VO}_2$  measurements reflect not only muscle  $\text{VO}_2$ , but also  $\text{VO}_2$  from the rest of the body, as well as oxygen transport and changes in lung, muscle and blood gas stores

(3). While direct measurement of muscle  $\text{VO}_2$  is possible (1, 11), obtaining muscle  $\text{VO}_2$  is technically difficult, invasive, and disruptive to normal activities. Additionally, the stimulation and direct observation of muscle tissues and/or individual muscle fibers is not able to be performed in-vivo. Non-invasive ways that have been used to measure muscle  $\text{VO}_2$  include Doppler ultrasound (16), and magnetic resonance spectroscopy (P-MRS) (2). P-MRS requires that any equipment and exercising body part be inside the confines of the magnet which requires difficult technical adaptations. As such, the majority of the current research being conducted on  $\text{VO}_2$  kinetics utilizes measurements of pulmonary  $\text{VO}_2$ . It has been shown that the kinetics of pulmonary  $\text{VO}_2$  during Phase II can be used to estimate muscle  $\text{VO}_2$  kinetics (3, 12). However, inferences to muscle physiology need to be critically interpreted for validity, especially when considering  $\text{VO}_2$  kinetic behavior in certain disease states (e.g. congestive heart failure) where both central and muscle  $\text{VO}_2$  kinetics may be altered by the pathophysiologic process.

The response of pulmonary  $\text{VO}_2$  to exercise has been widely described in the literature since Whipp and Wasserman's pioneering work in the late 1960s and 1970s (25). Three phases have been identified. Phase I represents the duration of the initial increase in cardiac output and pulmonary blood flow. Phase II represents the venous blood arriving at the lungs from the exercising muscles. It is also sometimes labeled the "fast component," the "primary component," or the "fundamental component." Therefore, the pulmonary  $\text{VO}_2$  kinetics during Phase II most closely reflect that of the exercising muscles. At the onset of Phase III, during moderate intensity exercise, a steady state will have been achieved and the  $\text{VO}_2$  will plateau within 3 minutes (18). At higher exercise intensities, the steady state may be delayed or absent and what is known

as the “slow component” emerges, which is described as the increase in  $VO_2$  from 3 minutes until the end of exercise (19). During very heavy exercise the slow component can cause the  $VO_2$  to increase to nearly maximal levels by the time exercise is terminated. It has been posited that the slow component may be a determinant of exercise tolerance as exercise training is known to decrease the amplitude of the slow component (26). The use of warm-up (“priming exercise”) has been the subject of several recent studies particularly as it relates to the reduction of the slow component.

It is commonly believed that warming up prior to exercise enhances performance. The effect of warm-up (WU) on exercise performance has been widely studied, but the results have been inconclusive. Passive WU strategies such as hot packs, paraffin mixtures or warm baths have been studied as well. Koppo (21) and Burnley (7) found evidence that these passive methods do not have an effect on performance as reflected in the  $VO_2$  slow component in heavy exercise. Gerbino et al. (11) demonstrated a speeding of  $VO_2$  kinetics associated with a smaller increase in blood lactate accumulation following a heavy WU, suggesting that the residual metabolic acidemia may lead to improved muscle perfusion during subsequent exercise. This study did not evaluate the effect on the slow component. In a review article summarizing the recent literature related to the use of WU, Bishop (5) states that there has been a lack of carefully controlled studies as well as a lack of standardized WU protocols and testing criteria. Grodjinovsky and Magel (13) studied the effect of two WU procedures (one regular, one vigorous) on running performance (60 yd, 440 yd, 1 mile) and on a 5 minute treadmill test with  $VO_2$  measurement. Times improved for the 60 yd and 440 yd runs when preceded by a WU. A vigorous WU improved the time for the 1 mile run when

compared to the regular and no warm-up (NWU). Hajoglou et al. (14) studied the effect of WU on cycling performance utilizing a 3 km time trial. She found improved 3 km times with both easy and hard WU when compared to NWU.  $\text{VO}_2$  kinetics, as measured by Mean Response Time (MRT), were also faster when preceded by either an easy or hard WU when compared to NWU. Power output (PO) in the NWU trial was noted to be decreased in the first 500 meters when compared to the two WU trials. It was unclear whether the relatively slower  $\text{VO}_2$  kinetics were due to the lack of WU or the lower PO. Hettinga et al. (15) studied the  $\text{VO}_2$  response during 4 maximal effort time trials ( $\text{PO} > 100\% \text{VO}_{2 \text{ max}}$ ) of 750 m, 1500 m, 2500 m and 4000 m with a standardized WU protocol. Subjects were instructed to finish as fast as possible. POs were observed to exhibit an initial burst in the first 15 s of all time trials. Faster  $\text{VO}_2$  kinetics were observed in the 750 m trial as evidenced by a reduction in the MRT compared to all other trials. A significantly higher mean  $\text{VO}_2$  was found for 750 m and 1500 m compared to 4000 m. Bishop et al. (6) found similar results using a “fast-start” strategy in kayaking. The fast start over the first 10 s followed by a transition to an even pacing strategy resulted in greater average power and greater average total  $\text{VO}_2$  in a 2 minute kayak ergometer test. Thus it is unclear whether the faster  $\text{VO}_2$  kinetics observed by Hajoglou et al. were a primary change in the cardiorespiratory response to exercise following WU or were secondary to a larger “muscle need” driven by the higher PO during the trial.

## METHODS

Eight (3 male, 5 female) healthy, well-trained cyclists volunteered to participate in this study. Some characteristics of the subjects are presented in Table 1. The subjects received an explanation of the study protocol and provided written informed consent.

The protocol was approved by the University of Wisconsin - La Crosse Institutional Review Board for the Protection of Human Subjects.

Table 1 - Subject Characteristics

	Total n = 8	Male n = 3	Female n = 5
Age (years)	23.1 ± 2.1	23.7 ± 2.9	22.8 ± 1.8
Height (cm)	176.5 ± 9.3	186.3 ± 3.9	170.7 ± 5.5
Weight (kg)	73.1 ± 12.0	85.5 ± 4.0	65.7 ± 7.8
Peak Power Output (PPO) (W)	316 ± 80.1	400 ± 50.0	265 ± 37.9
PPO/kg (W/kg)	4.3 ± 0.6	4.7 ± 0.8	4.0 ± 0.3
VO <sub>2</sub> at VT (L/min)	1.9 ± 0.3	2.2 ± 0.1	1.7 ± 0.2
VO <sub>2max</sub> (L/min)	3.9 ± 1.2	5.2 ± 0.4	3.1 ± 0.5
VO <sub>2max</sub> (ml/kg/min)	52.8 ± 8.3	61.7 ± 6.4	47.5 ± 2.2

### Testing Protocol

Each subject performed an incremental test on an electronically-braked cycle ergometer (Lode, Groningen, Netherlands) to determine peak power output (PPO), ventilatory thresholds (VT) and maximal oxygen consumption (VO<sub>2 max</sub>). Respiratory

gas exchange was measured by open-circuit spirometry (Moxus, AEI Technologies, Pittsburgh, PA, USA). Before each test the gas analyzers were calibrated with a known gas and room air. The pneumotachometer was calibrated with a 3 L syringe. Each subject also performed a preliminary habituation 3 km trial to become familiarized with the racing bicycle and the breathing apparatus under time trial (TT) conditions.

Each subject subsequently performed two 3 km TTs and two square wave (SW) tests, one under each warm up (WU) vs. no warm up (NWU) condition. The TTs took place on an electronic ergometer (Racermate, Seattle, WA, USA) fitted with a heavy (55 lb), large diameter flywheel with an internal freewheel, a fixed ratio chain drive, and electronic shifting. The system measured speed, distance, power output (PO), time, cadence and heart rate (HR) of the subject. The SW tests took place on the same electronically braked ergometer as for the incremental test. The subject performed the TT with WU (TT+WU) first to establish mean PO over the trial; this value was used as the PO on both the SW with WU (SW+WU) and the SW without WU (SW+NWU) tests. The second TT (with NWU) and the two SW tests were performed in random order. Trials were scheduled over a two-week period, on non-consecutive days, to allow adequate rest for the subjects.

For the exercise bouts which were preceded by a WU, the WU protocol was standardized at a total of fifteen minutes and began with five minutes of cycling at 100 watts (W), followed by five minutes of cycling at 50% of PPO obtained during the subject's incremental test, then three minutes of cycling at 75% of PPO, and lastly two minutes of cycling at 50% of PPO. The subject then remained on the bicycle, but rested without pedaling for five minutes. Ventilatory data collection began during this five

minute rest period and continued during the subsequent exercise bout. For exercise bouts which were performed without WU, the subject was attached to the respiratory equipment and monitored for five minutes to establish resting  $VO_2$  prior to beginning the test.

For the TTs, the subjects were instructed to finish as fast as possible. Figure 1 presents a schematic of a TT including WU for a representative subject. During the TTs heart rate (HR) and rating of perceived exertion (RPE) were recorded at the completion of each 10% segment of the trial (every 300 m).

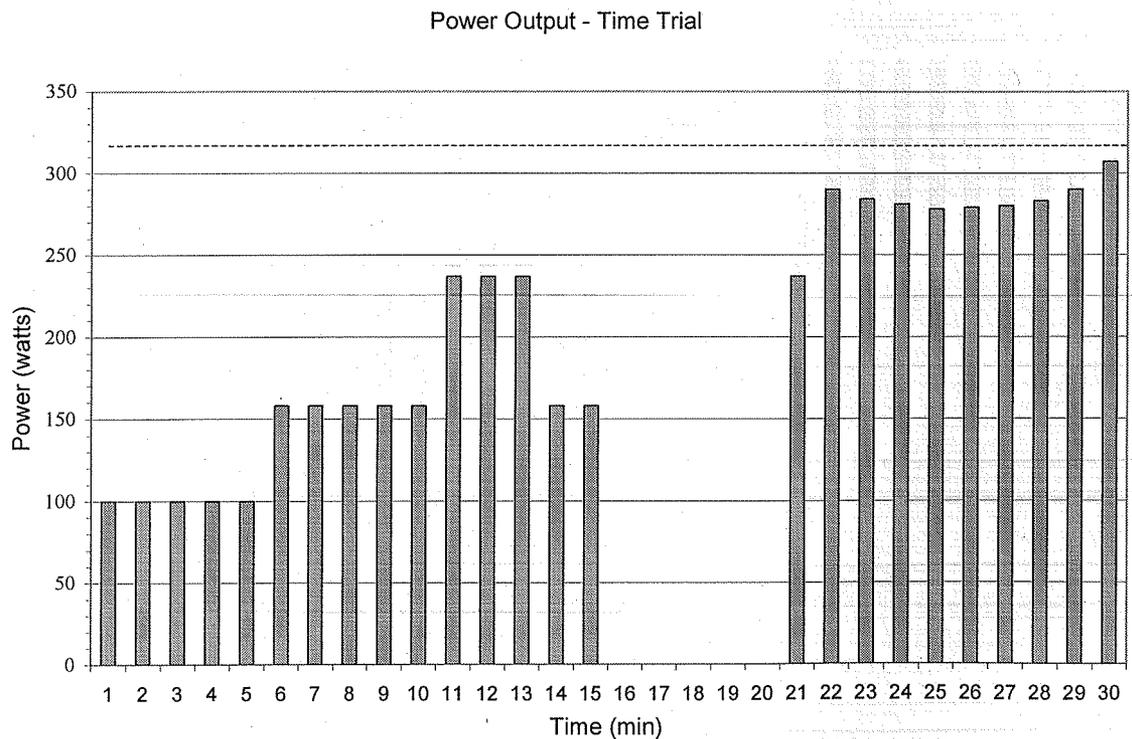


Figure 1 - Schematic of a TT for a representative subject including 15 minute WU and 5 minute rest periods. WU protocol assumes a max of 316 watts achieved on the ramp test. Once the ride begins at minute 21 the subject then completed a 3 km TT. Each bar represents consecutive 10% (300 m) segments of the ride.

For the SW tests, the subjects were instructed to ride to exhaustion. During the 5 minute rest period prior to each SW test the PO was pre-set at 100 W. Once the subject began pedaling, the PO was raised as quickly as possible to the mean PO value achieved during the first TT, and the amount of time needed to achieve the desired PO was recorded. Figure 2 presents a schematic of the PO for a representative subject during a SW test. During the SW tests HR and RPE data were collected each minute. RPE was measured using the category-ratio RPE (1-10) scale.

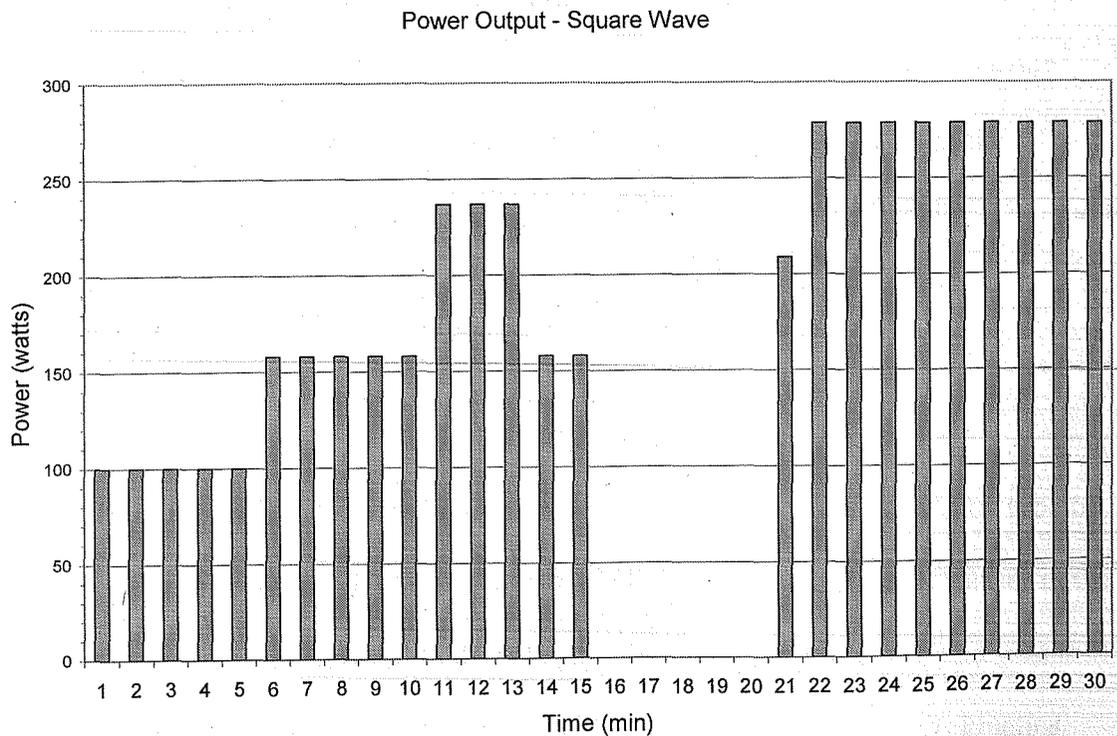


Figure 2 - Schematic of SW test progression for a representative subject, including 15 minute WU and 5 minute rest periods prior to ride. Beginning at minute 21 the subject rode to exhaustion at a fixed PO equal to the mean achieved during the TT+WU (279 watts). The first test minute (minute 21) has been adjusted down to reflect the lower mean PO due to the lag in reaching the assigned power.

A 1-component, 4-parameter model, conceptually similar to Bell (4) was used to calculate  $\text{VO}_2$  kinetics. A least squares fit of the  $\text{VO}_2$  response during each exercise bout was calculated.  $\text{VO}_2$  was modeled as:

$$\text{VO}_2 = \text{VO}_2_{\text{rest}} + A \cdot (1 - e^{-\lambda \cdot (\text{time} - t_d)})$$

where  $\text{VO}_2$  = oxygen uptake,  $\text{VO}_2_{\text{rest}}$  = mean  $\text{VO}_2$  during the last minute preceding the exercise bout,  $A$  = the calculated max of the  $\text{VO}_2$ ,  $\lambda$  = time constant,  $t_d$  = time delay (calculated). The Mean Response Time (MRT) was calculated as the time at which the fitted  $\text{VO}_2$  response was equal to 63% of the maximum response ( $A$ ), plus the time delay of the system. The variable  $\tau = 1/\lambda$ , which is 63% of the response time. Therefore,  $\text{MRT} = \tau + t_d$ . To determine the MRT,  $\text{VO}_2$  was plotted on a 6-second basis. After exclusion of Phase I by visual inspection, the resulting curve of the  $\text{VO}_2$  response was described using an exponential curve. From this curve, the time required to reach two-thirds of the value achieved during the TT was identified.

### Statistical Analysis

The MRT was analyzed using repeated measures ANOVA for a WU by type of exercise design to test the hypotheses that (1) MRT would be significantly shorter in the TT with WU condition compared to the TT with NWU and SW with WU, and would be slowest in the SW with NWU bout and (2) that WU would drive faster  $\text{VO}_2$  kinetics during the SW tests. Statistical significance was set at  $p < 0.05$ .

## RESULTS

### Time Trials

A summary of selected time trial (TT) results is presented in Table 2.

Table 2 - Selected TT results. Resting values are the average from the minute preceding the test.

	Warm Up	No Warm Up
Ride Time (sec)	294.9 ± 32.9	294.3 ± 31.8
VO <sub>2</sub> max (L/min)	4.2 ± 1.0 *	4.0 ± 1.0
VO <sub>2</sub> max (ml/kg/min)	56.4 ± 5.3	54.6 ± 5.6
HR <sub>max</sub> (bpm)	180 ± 9.7 *	172 ± 6.3
VE <sub>max</sub> (L/min)	139.8 ± 27.9	138.1 ± 31.4
MRT (sec)	38.6 ± 5.8	42.4 ± 7.9
Mean PO (W)	279 ± 84.3	280 ± 86.5
Rest VO <sub>2</sub> (ml/kg/min)	9.5 ± 2.3 *	6.8 ± 1.0
Rest HR (bpm)	99 ± 10.7 *	77 ± 7.4
Rest VE (L/min)	23.6 ± 10.0 *	17.1 ± 8.4

\*  $p \leq 0.05$  WU vs. NWU

There was no significant difference in the time to complete the TTs in the NWU condition compared to the WU condition ( $294.3 \pm 31.8$  v.  $294.9 \pm 32.9$ ). Split times for each 300 m segment are depicted in Figure 3. During the first two 300 m segments the split times were faster in the NWU condition than the WU condition, although not significantly so; thereafter there were only minor differences in split times with the exception of the final split (3000 m) which was significantly faster in the WU trial compared to the NWU trial ( $28.9 \pm 3.8$  seconds vs.  $28.4 \pm 3.8$  seconds,  $p=0.018$ ).

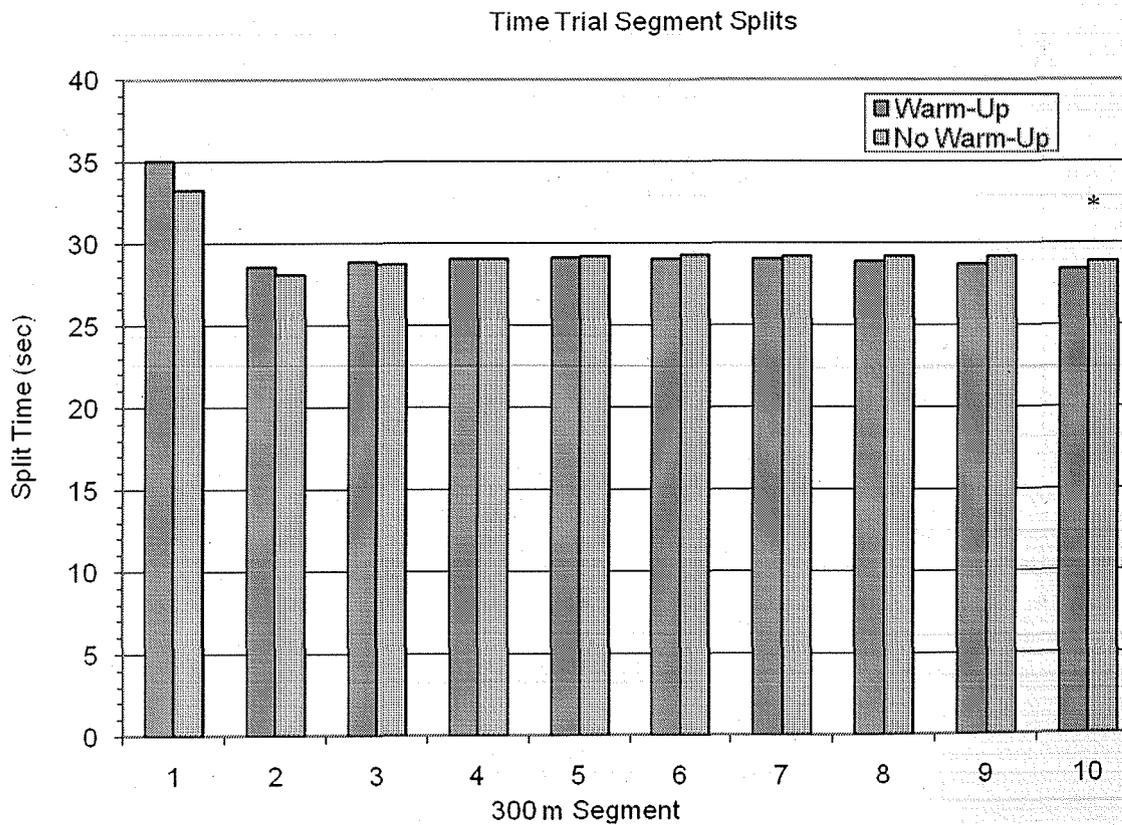


Figure 3 - Split times for each 10% segment (300 m) of the 3 km TT, comparing the WU to NWU condition.

\*  $p \leq 0.05$  WU vs. NWU

Figure 4 displays mean segment-by-segment PO data comparing NWU to WU. Mean PO with NWU ranged from 95%-114% of that with WU. PO in the first 300 m segment of the NWU trial was significantly higher than in the WU trial ( $270 \pm 108.7$  W vs.  $237 \pm 95.8$  W,  $p=0.015$ ). Conversely, in the final 300 m segment PO in the WU trial was significantly higher compared to the NWU trial ( $307 \pm 106.7$  W vs.  $291 \pm 101.9$  W,  $p=0.014$ ).

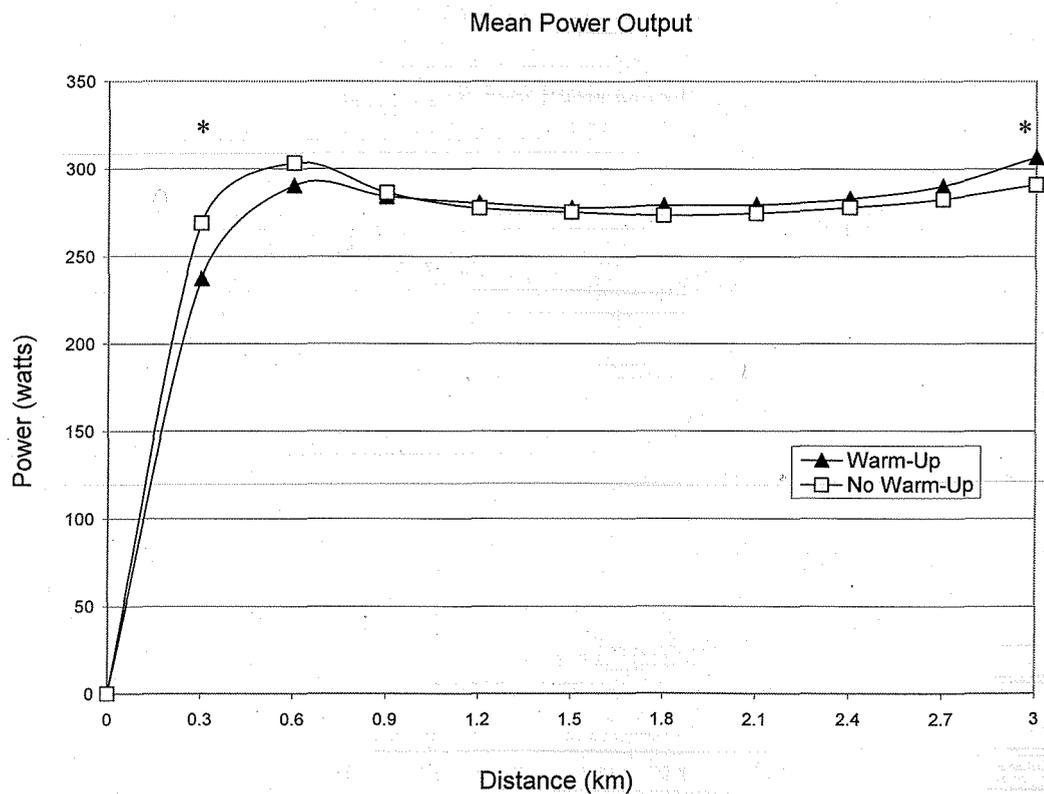


Figure 4 - Mean PO over the 3 km TT in WU and NWU conditions.

\*  $p \leq 0.05$

The PO achieved was calculated as a percent of the peak value achieved on the subjects' incremental tests; this data is displayed in Figure 5. A greater percentage of peak power output (%PPO) is achieved in the NWU condition vs. the WU condition for the first three 300 m segments, the differences is significant at 300 m ( $83\% \pm 18\%$  vs.  $73\% \pm 14\%$ ,  $p=0.015$ ). Thereafter the %PPO in the NWU condition remains below that of the WU condition and is significantly lower at 3000 m ( $90\% \pm 11\%$  vs.  $95\% \pm 13\%$ ,  $p=0.015$ ).

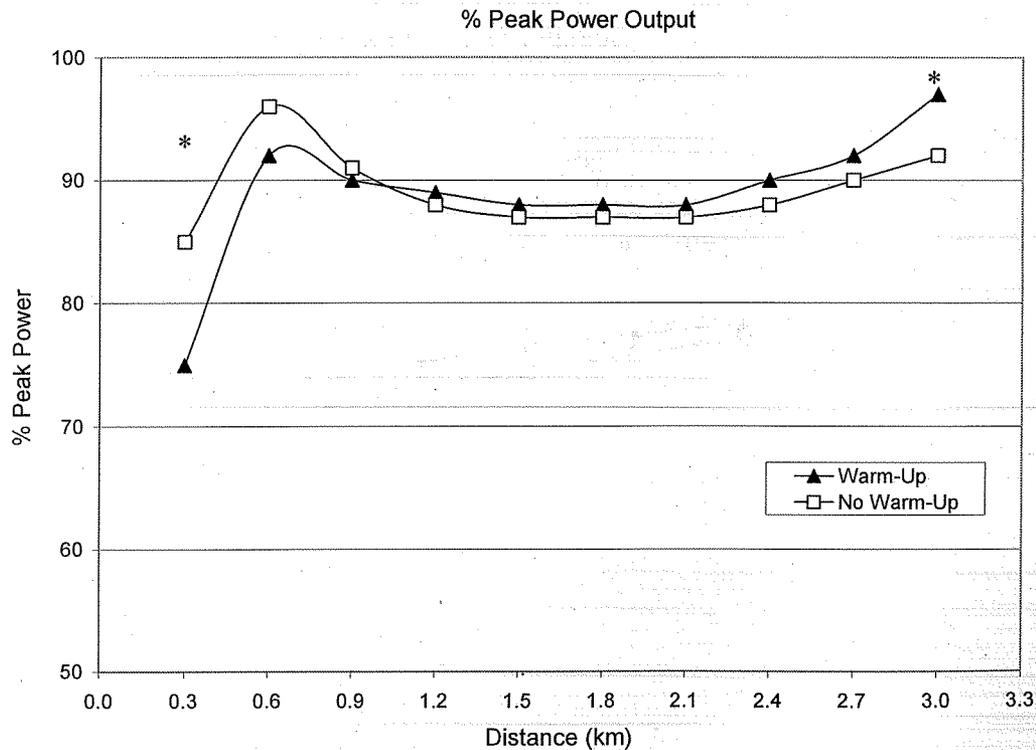


Figure 5 - PO shown as %PPO for each 300 m segment of the TTs under WU vs. NWU condition.

\*  $p \leq 0.05$  WU vs. NWU

The  $VO_{2max}$  achieved during the TTs is statistically significantly higher for trials in the WU condition compared to the NWU condition ( $4.2 \pm 1.0$  vs.  $4.0 \pm 1.0$  L/min,  $p=0.03$ ). However, when these values are normalized to the weight of the subjects the difference ( $56.4 \pm 5.3$  ml/kg/min for WU vs.  $54.6 \pm 5.6$  ml/kg/min for NWU) is not significant. The average  $VO_2$  for each 300 m segment of the TTs is depicted in Figure 6. Segmental differences between WU and NWU that achieved statistical significance occurred at 900 m, 1200 m, 1500m, 1800 m, 2400 m, 2700 m, and 3000m. The increase in  $VO_2$  in the WU condition is accounted for by the increase in resting  $VO_2$  following the WU ( $9.5 \pm 2.3$  vs.  $6.8 \pm 1.0$  ml/kg/min,  $p=0.006$ ).

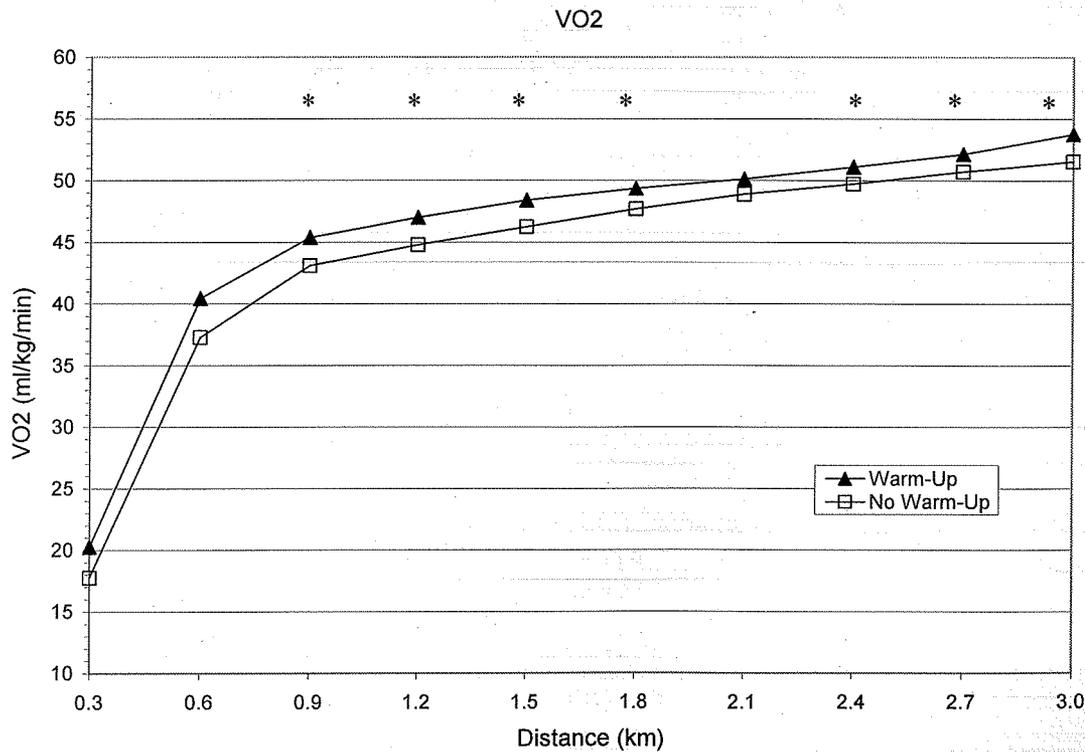


Figure 6 -  $VO_2$  response under WU and NWU conditions during 3 km TTs for each 300 m segment of the trial.

\*  $p \leq 0.05$  WU vs. NWU

The HR<sub>max</sub> achieved during the TTs was found to be significantly higher in the WU condition than in the NWU condition ( $180 \pm 9.7$  vs.  $172 \pm 6.3$  bpm,  $p=0.007$ ).

Average HR<sub>max</sub> values for each 300 m segment of the TTs are presented in Figure 7.

Average HR starts lower in the NWU condition and remains lower for the duration of the trial when compared to the WU condition reflecting the lower HR before the start in the NWU condition. WU trial values were significantly higher at 1500 m, 1800 m, 2100 m, 2400 m, 2700 m, and 3000 m compared to NWU values.

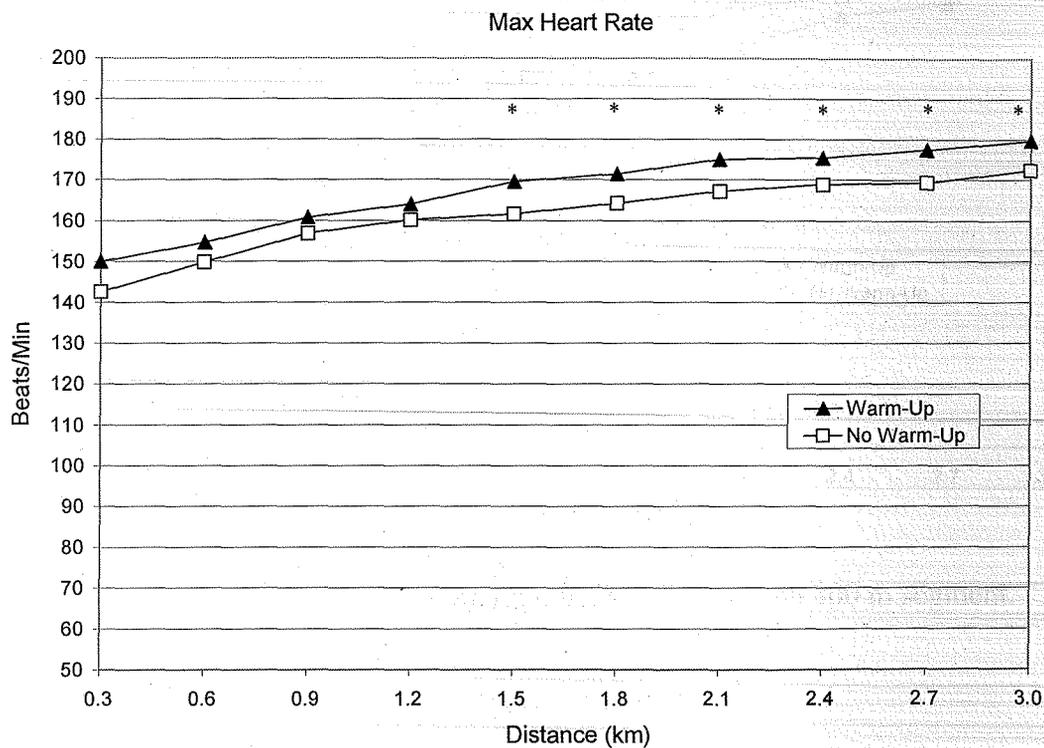


Figure 7 - HR<sub>max</sub> achieved for each 300 m of the TTs comparing WU to NWU condition.  
\*  $p \leq 0.05$

The rating of perceived exertion (RPE) for each 300 m segment of the TTs is depicted in Figure 8. Average segmental RPEs with NWU are 2-20% higher than with WU. The segmental value at 2700 m was significantly higher in the NWU trial compared to the WU trial ( $p=0.049$ ).

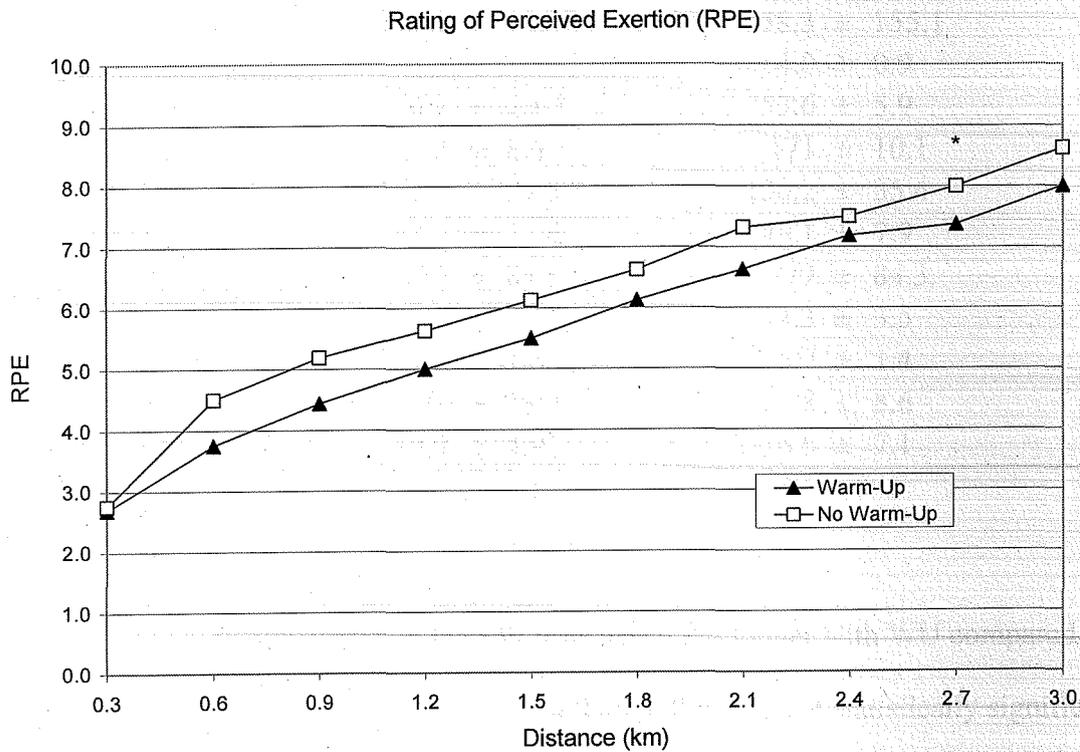


Figure 8 - RPE for WU vs. NWU condition in 3km TT for each 300 m segment.

\*  $p \leq 0.05$

## Square Wave Tests

Selected data from the square wave (SW) tests is depicted in Table 3.

Table 3 - Selected SW test results

	Warm Up	No Warm Up
Ride Time (sec)	371.3 ± 165.7	338.3 ± 155.1
VO <sub>2 max</sub> (L/min)	4.2 ± 1.1 *	4.0 ± 0.9
VO <sub>2 max</sub> (ml/kg/min)	56.7 ± 7.1 *	53.6 ± 5.9
HR <sub>max</sub> (bpm)	178 ± 8.6 *	171 ± 10.1
VE <sub>max</sub> (L/min)	137.2 ± 37.5 *	130.2 ± 35.4
MRT (sec)	48.0 ± 13.6	45.1 ± 7.9
Mean PO (watts)	279 ± 84.3	279 ± 84.3
Time to Reach PO (sec)	13.8 ± 3.1	14.3 ± 3.5
Rest VO <sub>2</sub> (ml/kg/min)	7.6 ± 1.6	6.7 ± 1.4
Rest HR (bpm)	98 ± 13.2 *	78 ± 8.6
Rest VE (L/min)	19.9 ± 4.9	16.6 ± 7.1

\* p ≤ 0.05

The time to exhaustion in the SW tests was 9.8% longer with WU compared to NWU (371.3 ± 165.7 vs. 338.3 ± 155.1 s), although this was not statistically significant. MRT was 45.1 ± 7.9 s for NWU compared to 48.0 ± 13.6 s for WU which was not statistically significant.

Selected SW data is graphically depicted in Figure 9. In this series of graphs the final data point connected by a line represents the last time where all subjects had data, the end point represents the average of all subjects' final value for the test regardless of time to finish.

A statistically significant difference was observed in HR<sub>max</sub> during the SW test in the NWU condition when compared to the WU condition (178 ± 8.6 vs. 171 ± 10.1 bpm,

p=0.01). Peak HR for each minute of the SW tests showed a significant difference at rest, at minute 1, and at minute 2, as well as the mean ending HR.

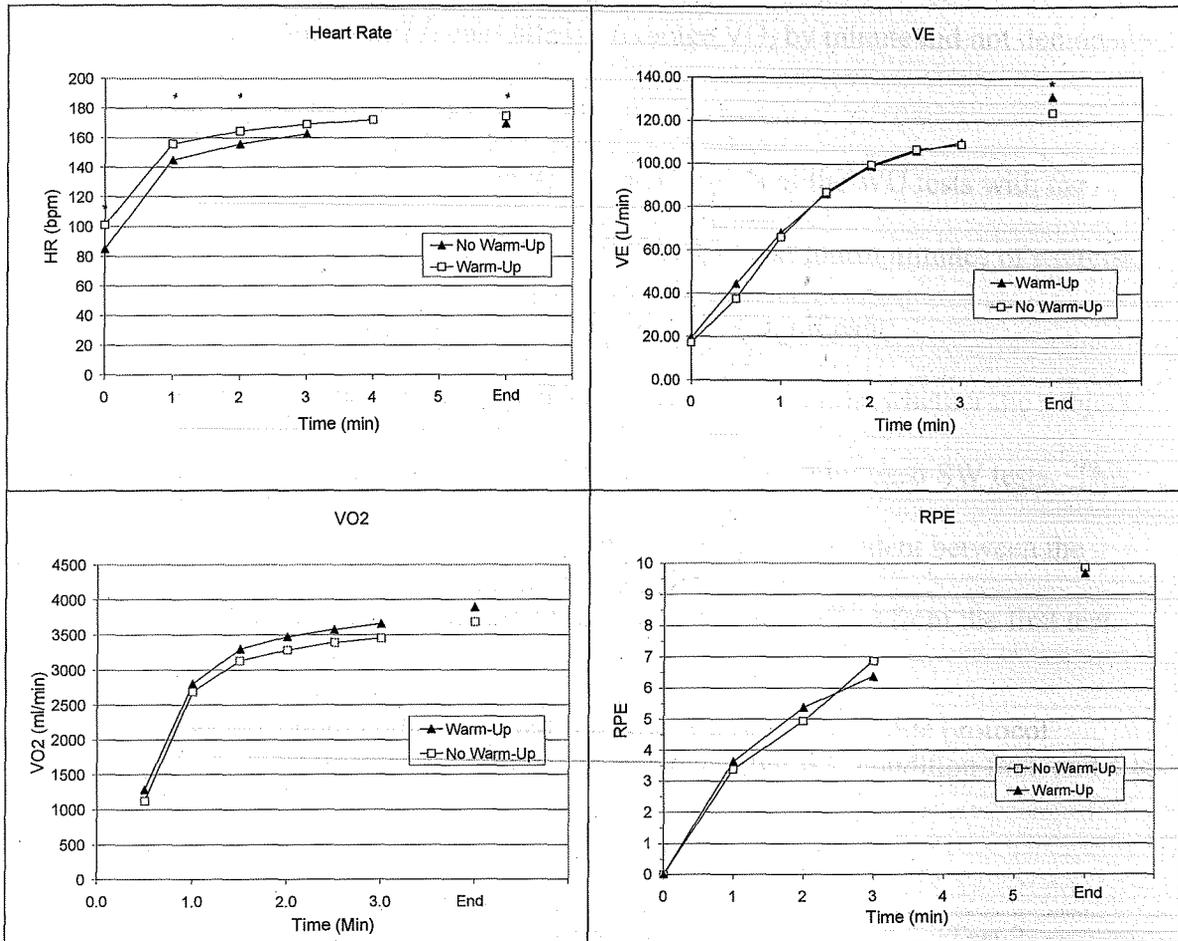


Figure 9 - HR, VE, VO<sub>2</sub>, and RPE data in SW, WU vs. NWU.

\*p ≤ 0.05

Maximal ventilation (VE<sub>max</sub>) was significantly higher in the WU condition compared to the NWU condition (137.2 ± 37.5 vs. 130.2 ± 35.4 L/min, p=0.002). VE is significantly higher in the WU condition for the final 30 seconds (131.5 ± 37.4 vs. 124.0 ± 38.0 L/min, p=.017).

A statistically significant difference was observed in absolute  $VO_{2max}$  with WU compared to NWU ( $4.2 \pm 1.1$  vs.  $4.0 \pm 0.9$  L/min,  $p=0.03$ , one-tailed). A significant difference was also seen in relative  $VO_{2max}$  for WU compared with NWU ( $56.7 \pm 7.1$  vs.  $53.6 \pm 5.9$  ml/kg/min,  $p=0.027$ , one-tailed). Average  $VO_2$  by minute did not demonstrate significant differences.

RPE during the NWU tests ranged from 91-107% of the WU tests with the greatest difference being experienced between the third and fourth minutes of exercise (107%) although these differences were not statistically significant.

Selected SW data was assembled by ride order to examine whether the subjects demonstrated improved performance on the latter of the 2 randomized SW tests. This data is displayed in Figure 10. No significant improvement is evident between the subjects' first and second tests that might indicate a learning effect from the first test.

Table 4 - Selected values comparing first SW test to second test in ride protocol sequence. Four subjects were randomized to ride under the WU condition first, and 4 to the NWU condition first.

	Test 1	Test 2
Mean Time (sec)	$356.3 \pm 168.2$	$353.3 \pm 154.5$
Peak $VO_2$ (ml/kg/min)	$55.1 \pm 4.7$	$55.4 \pm 8.0$
Peak Heart Rate (bpm)	$176 \pm 8.8$	$173 \pm 11.2$
Peak VE (L/min)	$133.1 \pm 34.5$	$134.3 \pm 38.7$
MRT (sec)	$43.7 \pm 9.4$	$49.3 \pm 12.1$

## DISCUSSION

This study was designed to test two hypotheses. The first was that mean response time (MRT) would be significantly shorter in the time trial with warm up (TT+WU) condition compared to square wave with warm up (SW+WU) or time trial with no warm up (TT+NWU) condition, and would be slowest in the square wave with no warm up (SW+NWU) bout. The second was that WU would drive faster  $VO_2$  kinetics during the SW tests. Neither hypothesis was supported.

While Grodjinovsky (13) reported improved performance times for a one-mile run following warm-up, and Hajoglou (14) reported improvement in 3 km cycling times with WU, TT+WU performance times in the present study did not improve, nor did the subjects ride significantly longer in the SW+WU tests as expected. While the overall  $VO_2$  was lower and the heart rate (HR) response was attenuated in the NWU condition, TT performance was not affected as evidenced by mean power output (PO), which was similar between the two TTs, and was actually higher in the first 300 m in the NWU condition. Thus, for reasons that are not intuitively obvious, the process of WU did not augment performance in this study.

The results in the SW test, where exactly the same muscle PO was performed, suggest that WU did not drive faster  $VO_2$  kinetics during Hajoglou's study, and thus the only viable explanation for the faster mean response time (MRT) by Hajoglou et al. is the higher early PO. However, since the subjects in this study did not use a higher PO early

in the TT+WU, we found no evidence of the accelerated  $\text{VO}_2$  kinetics reported by Hajoglou et al.

Compared to Hajoglou's study, the present study included subjects that were younger college-age students vs. relatively older community cyclists (age  $23.1 \pm 2.1$  vs.  $31 \pm 8$ ). While the subjects in the present data set were well-trained, they were generally less experienced in TT competitions than those in Hajoglou's study. It is unknown whether the differences seen in the outcomes of the two studies may have been the result of minor subject population differences. The significantly higher mean PO in the initial stages of the TT+NWU condition seems to indicate that lack of WU did not cause the subjects to hold back, as was suspected in Hajoglou's study. Recent studies have suggested that pre-exercise expectations regarding an exercise task can meaningfully influence the pacing strategy employed (10, 24). On this basis, it seems reasonable to suggest that the more experienced cyclists in the study of Hajoglou et al., who routinely perform extensive pre-competition warm-up may have purposely held back during the early part of the NWU trial simply because the thought of maximal exertion without WU was sufficiently abnormal as to prevent them from fully exerting themselves early in the NWU time trial. The less specialized subjects employed in the present study, having the benefit of a learning trial and no particular expectations about the value of warm-up, did not hold back during the NWU trial. Learning effects between the two sSW rides was ruled out as a potential influencing factor when ride results by sequence were examined.

Additionally, the subjects in the present study had a lower overall mean PO during the time trials with greater variability between subjects compared to Hajoglou's subjects ( $316 \pm 80\text{W}$  vs.  $334 \pm 42\text{W}$ ) and lower  $\text{VO}_2$  at VT ( $1.87 \pm 0.31$  vs.  $2.75 \pm 0.33$

L/min). Thus, it is possible that the overall strength and fitness of the riders and variability within the subject pool contributed to the lack of significant change in MRT which was expected following WU as  $\text{VO}_2$  kinetics have been shown to be faster as a result of training (26).

The general pre-test activity level of the subjects should also be considered as a potential factor confounding the results. The instructions to the subjects prior to both this and Hajoglou et al. were the same, to report to the laboratory well-rested and without WU. However, it is reasonable to suspect that the college-aged subjects in this study were potentially more active in the time leading up to their presentation at the lab, via normal activities of moving around campus, whereas the community subjects in Hajoglou's study were potentially more sedentary. Given that the effect of WU is at least somewhat persistent over time (20, 22, 23), it is possible that the student subjects performed an unintentional WU prior to reporting for a particular test. Given that even small amounts of WU may be sufficient to cause significant hemodynamic effect (9), it must be acknowledged that our control procedures (~15 minutes of rest in the laboratory before the 5 min pre-exercise gas collection period in the NWU condition) may have been inadequate to compensate for the more active lifestyle of younger students. We have no evidence that this occurred, but given the unexpected outcome of the results, we must acknowledge that inadvertent WU was a contributor to the results.

In summary, unexpected results were obtained in this study. The improvements in performance and  $\text{VO}_2$  kinetics seen in earlier studies were the rationale for extending the current study to include the fixed PO of the SW methodology. The TT performance and  $\text{VO}_2$  improvements were not replicated in this study. Further research needs to be

undertaken to clarify the relationship between WU,  $VO_2$  kinetics and performance in the heavy exercise realm. Subject selection and compliance should be carefully considered in future studies.

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**APPENDIX A**

**INFORMED CONSENT**

Protocol Title: Effect of Warm-Up on VO<sub>2</sub> Kinetics During Heavy Exercise

Principal Investigator: Marybeth Stockman  
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La Crosse, WI 54601  
240-644-4146

Emergency Contact: Carl Foster  
133 Mitchell Hall - UWL  
608-785-8687

#### Purpose and Procedure

- The purpose of this study is to determine the effect of warm-up on VO<sub>2</sub> kinetics during heavy exercise.
- My participation will involve six cycling tests, all of which will be very fatiguing. These tests will be both progressive (increasingly harder) and 3 km time trials (competitive simulations).
- The total time requirement is 6 hours over a four-week time period.
- Testing will take place in Room 225 Mitchell Hall, UW-L.
- During all tests I will wear a snorkel-like device to analyze my breathing, and a heart monitor, strapped around my chest, to monitor my heart rate.

#### Potential Risks

- I may experience muscle soreness and substantial fatigue.
- Individuals trained in CPR, Advanced Cardiac Life Support and First Aid will be present in the laboratory, and the test will be terminated if complications occur.
- The risk of serious or life-threatening complications for healthy persons, like myself, is near zero.

#### Rights & Confidentiality

- My participation is voluntary.
- I can withdraw from the study at any time for any reason without penalty.
- The results of this study may be published in scientific literature or presented at professional meetings using grouped data only.
- All information will be kept confidential. Only my professor and I will have access to raw data. My data will not be linked with any personally identifiable information. A notebook containing data will be kept in the laboratory.

#### Possible Benefits

- Other people and I may benefit by understanding how warm-up affects VO<sub>2</sub> kinetics during heavy exercise.

Questions regarding study procedures may be directed to student Marybeth Stockman (240-644-4146), the principal investigator, or the study advisor Dr. Carl Foster, Department of Exercise and Sport Science, UW-L (608-785-8687). Questions regarding

the protection of human subjects may be addressed to the UW-La Crosse Institutional Review Board for the Protection of Human Subjects at 608-785-8124 or [irb@uwlax.edu](mailto:irb@uwlax.edu).

Participant \_\_\_\_\_ Date \_\_\_\_\_

Researcher \_\_\_\_\_ Date \_\_\_\_\_

## APPENDIX B

### REVIEW OF LITERATURE

## REVIEW OF LITERATURE

The purpose of this paper is to review the literature related to the effect of warm up (WU) on  $\text{VO}_2$  kinetics during heavy exercise. Literature studies are divided into five categories. The first examines the development of a unified body of literature related to  $\text{VO}_2$  kinetics in general, and to discuss where controversy still exists. The second focuses specifically on  $\text{VO}_2$  kinetics in the heavy exercise domain. The third explores studies which utilize constant work rate exercise (square wave or "fast start"). The fourth considers the use of WU as it relates to  $\text{VO}_2$  kinetics in general, and heavy exercise in particular. The fifth examines the mean response time (MRT) as a measure of  $\text{VO}_2$  kinetics.

### $\text{VO}_2$ Kinetics

The response of pulmonary  $\text{VO}_2$  to exercise has been widely considered in the literature since Whipp and Wasserman's pioneering studies in the late 1960s and 1970s. Whipp et al. (31) describe three phases of the pulmonary response. Phase I (the cardiodynamic phase) represents the duration of the initial increase in cardiac output and pulmonary blood flow. Phase II represents the venous blood arriving at the lungs from the exercising muscles (it is also sometimes called the "fast component," the "primary component," or the "fundamental component" in the literature), therefore the pulmonary  $\text{VO}_2$  kinetics during Phase II most closely reflect that of the exercising muscles. At the onset of Phase III during moderate intensity exercise a steady state will have been

achieved and the  $\text{VO}_2$  will plateau within 3 minutes (28). At higher exercise intensities, a steady state of  $\text{VO}_2$  may be delayed or absent and what is known as the “slow component” may appear, which is described as the increase in  $\text{VO}_2$  from 3 minutes until the end of exercise (26). The physiological factors contributing to the slow component have been the focus of much of the current research on  $\text{VO}_2$  kinetics since they represent an “(increasing) metabolic inefficiency” which appears to be related to fatigue and exercise intolerance, and as such have implications for the study of  $\text{VO}_2$  kinetics in a multitude of disease states (27). Uncertainty remains as to whether the slow component is related to an imbalance in  $\text{O}_2$  supply and demand (a “feed-forward” mechanism) (22) vs. limitations in cellular processes (a “feedback” mechanism) (3). As such, factors known to affect either  $\text{O}_2$  availability or the intracellular metabolic processes have been the basis of much of the current research in  $\text{VO}_2$  kinetics and include such variables as muscle temperature, exercise position (supine vs. upright), muscle fiber recruitment, lactate accumulation (pH changes), exercise intensity, the effect of prior exercise including warm-up, and the age of subjects.

$\text{VO}_2$  kinetics are typically measured using pulmonary gas exchange analysis either using breath-by-breath (7, 9, 22) or open-circuit spirometry (19). Pulmonary  $\text{VO}_2$  measurements reflect not only muscle  $\text{VO}_2$  but also  $\text{VO}_2$  from the rest of the body, as well as  $\text{O}_2$  transport and changes in lung and muscle gas stores (4). While direct measurement of muscle  $\text{VO}_2$  is possible (1, 17), obtaining muscle  $\text{VO}_2$  is technically difficult and invasive. Additionally, the stimulation and direct observation of muscle tissues and/or individual muscle fibers is not able to be performed in-vivo. Non-invasive ways that have been used to measure muscle  $\text{VO}_2$  include Doppler ultrasound (21), and

magnetic resonance spectroscopy (P-MRS) (3). P-MRS requires that any equipment and exercising body part be inside the confines of the magnet leading to difficult technical adaptations. As such, the majority of the current research being conducted on  $\text{VO}_2$  kinetics utilizes measurements of pulmonary  $\text{VO}_2$ . It has been shown that the kinetics of oxygen consumption during Phase II can be used to estimate the kinetics of oxygen consumption in muscles (4, 17). However, any inferences to that of the muscles need to be critically interpreted for validity, especially when considering  $\text{VO}_2$  behavior in certain disease states (e.g. congestive heart failure) where muscle  $\text{VO}_2$  kinetics are altered.

### **$\text{VO}_2$ kinetics in the Heavy intensity domain**

At the onset of exercise pulmonary  $\text{VO}_2$  follows a predictable course. The speed of the kinetics is dependent on a variety of factors, one of which is the intensity of exercise. Instead of rising quickly to a steady state as in moderate exercise (i.e. below the ventilatory threshold), during heavy exercise above the lactate threshold,  $\text{VO}_2$  exhibits what is known as the slow component, a delay or failure to reach a steady state  $\text{VO}_2$ . As the slow component is first apparent at the heavy intensity range, studies in this exercise intensity domain often focus on the experimental response of the  $\text{VO}_2$  slow component. One such study was performed by Endo et al. (13). They investigated the effect of priming exercise at a variety of intensities on the  $\text{VO}_2$  response to cycle ergometry at a work rate of 50%  $\Delta$  (halfway between the lactate threshold and  $\text{VO}_{2\text{max}}$ ). The priming exercise consisted of either no warm up (NWU), a priming bout at sub-lactate threshold (80% LT), and three supra-lactate conditions (20%  $\Delta$ , 40%  $\Delta$ , and 60%  $\Delta$  intensities). They concluded that prior exercise just above, but not below, lactate threshold facilitated the  $\text{VO}_2$  response mainly due to reduced amplitude of the  $\text{VO}_2$  slow component. Jones et

al. (25) designed a study to test the effect of priming exercise on exercise in the supine position. They hypothesized that performance of a prior heavy exercise would speed  $\text{VO}_2$  kinetics during subsequent exercise in the supine position. The priming exercise and subsequent bouts were identical and consisted of 3 minutes of loadless peddling followed by 10 minutes at 50%  $\Delta$  work rate (calculated on the subject's incremental test). They found support for their hypothesis; prior heavy exercise accelerated  $\text{VO}_2$  kinetics in the supine studies, but not in the upright. They also found that in the upright position, prior heavy exercise had no effect on the phase II time constant ( $\tau$ ) but led to a substantial (50%) reduction in the slow component during subsequent heavy exercise. In contrast, in the supine studies, prior heavy exercise caused a significant reduction in the phase II  $\tau$  but had no effect on the amplitude of the slow component during subsequent heavy exercise. Their results related to upright exercise agree with others' such as Burnley (8). They speculate that these results lend support to the notion that  $\text{VO}_2$  kinetics are not limited by  $\text{O}_2$  availability but rather by cellular factors.

### **Studies Utilizing Fast Start**

Fast start or "all out" exercise is perhaps best exemplified in the non-laboratory setting by the firefighter who is awakened abruptly from sleep and whose body must immediately meet the demands of climbing ladders and carrying heavy loads, often in a smoke-filled environment. It was recently reported by the Centers for Disease Control (11) that between 1994 and 2004 heart attack was the leading cause of death for on-duty firefighters. Given the nature of the occupational hazards, it is not surprising that firefighters were the subjects of some of the early research conducted related to sudden strenuous exercise (SSE) in the 1970s. Barnard (2) reported EKG changes indicative of

subendocardial ischemia due to SSE in the absence of warm-up (WU). Further research was later conducted that examined ventricular function under SSE. Foster et al. (14) utilized first-pass radionuclide ventriculography to investigate left ventricular ejection fraction (LVEF) and ejection rate (LVER). He described an abnormal decrease in LVEF and inappropriately low LVER, as well as global hypokinesis possibly related to transient global ischemia. While his findings were suggestive of abnormal left ventricular (LV) behavior similar to that in coronary artery disease (CAD), he reported also that there was an absence of LV regional wall motion abnormalities which suggested an important difference from CAD. In addition, he did not find the same ST segment changes as Barnard (2), but that may have been affected either by the different duration periods of cardiac monitoring, or the general anxiety over catheter placement may have acted as a WU. Chesler et al. (12) also investigated SSE, her studies utilizing echocardiography to assess LV function as demonstrated by wall motion abnormalities and changes in LV dimensions. She found no significant differences in internal LV dimensions when SSE was performed with NWU when compared to the WU trial. Possible reasons cited were the technical difficulties inherent to and lack of comparative data for echocardiographic studies under upright exercise conditions and the fact that data was only collected for the last 10 seconds of exercise. Also she questioned whether handlebar-gripping had been controlled for in the earlier studies; it is possible that an isometric load may have contributed to LV dysfunction.

SSE is thought to fall under the realm of “severe” exercise and inferences to the “heavy” domain cannot necessarily be drawn. However, a study by Foster (15) demonstrating that WU mitigated, but did not fully eliminate, the LV abnormalities seen

in SSE is felt to be of significance to the current study as LVEF is a determinant of cardiac output and as such will factor into the speed of  $\text{VO}_2$  kinetics.

Commonly, studies of  $\text{VO}_2$  kinetics utilize a square wave (SW) design. Ozyener et al. (30) investigated the symmetry of the  $\text{VO}_2$  kinetics across a range of four physiologically defined exercise intensities ranging from moderate to severe. Subjects cycled at 4 different work rates (moderate = 90% lactate threshold [LT], heavy = LT+40%  $\Delta$ , very heavy = LT+80%  $\Delta$ , and severe = 110%  $\text{VO}_{2\text{peak}}$ ). Steady state was achieved in both moderate and (10 minutes into) heavy exercise. Steady state was not achieved in either very heavy or severe exercise consistent with the  $\text{VO}_2$  slow component. They did not find a significant influence of work intensity on the time constant ( $\tau$ ) for  $\text{VO}_2$  at the on-transient among moderate, heavy and very heavy exercise intensities. Casaburi et al. (10) examined whether there is a range of exercise intensities over which the responses of  $\text{VO}_2$ , carbon dioxide output ( $\text{VCO}_2$ ), and ventilation (VE) are substantially constant. They studied ventilatory and blood lactate data in the transition between loadless pedaling and higher work rates. Four subjects performed a total of 162 cycle ergometer studies exercising at a total of 7 different work rates. They found that  $\text{VO}_2$  kinetics correlated with work rate at intensities not associated with sustained lactate increase.  $\text{VO}_2$  kinetics at high work rates exhibited the addition of the slow component and  $\text{VCO}_2$  kinetics showed no consistent change with work rates probably related to the buffering of lactate and hyperventilation in response to lactic acidosis.

### **Effects of warm-up on $\text{VO}_2$ kinetics**

It is commonly believed that warming up prior to exercise enhances performance. The effect of WU on exercise performance as reflected in  $\text{VO}_2$  kinetics has been widely

studied in the literature. Faster overall  $\text{VO}_2$  kinetics in the second of 2 similar bouts of exercise have been reported by some authors (16, 29). The hypothetical bases of WU stem from the observation that prior exercise increases muscle blood flow,  $\text{O}_2$  and substrate delivery to the muscle, increases muscle temperature (24), can also potentially lead to increased lactate levels (16) and other metabolic changes that can impact performance. In a review article summarizing the recent literature related to the use of WU, Bishop (5) states that, while there is a multitude of data on WU, there has been a lack of carefully controlled studies as well as a lack of standardized WU protocols and testing criteria. It is therefore somewhat difficult to draw conclusions from the current body of knowledge as it relates to performance.

Studies utilizing passive WU strategies have failed to demonstrate much impact on the performance of heavy exercise. Specifically, in a study by Koga et al. (23) subjects wore water-perfused pants to raise muscle temperature to  $39^\circ\text{C}$ , then performed moderate intensity cycle exercise ( $\sim 60\text{-}70\%$  LT) and subsequently heavy intensity exercise at 50% of the difference ( $\Delta$ ) between LT and  $\text{VO}_{2\text{max}}$ . Temperature was measured rectally and four skin thermistors as well as by needle thermistor in the vastus lateralis. In the control trials exercise was performed with no water circulating through the pants. They found that there were no significant differences between the experimental and control results based on the amplitude of the  $\text{VO}_2$  fast (primary) component or of the Phase 2 time constant, but did find a reduction in the slow component during minutes 3-6. Similarly, Koppo et al. (24) utilized “parafango” and hot packs to passively warm the legs of subjects who subsequently performed 6 minutes of heavy cycling at power outputs corresponding to  $90\%$   $\text{VO}_{2\text{max}}$ . Intramuscular

temperature was measured continuously by indwelling thermistor. The subjects also performed a trial consisting of 2 identical 6-minute cycle exercise bouts at 90%  $VO_{2max}$  separated by a 6-minute recovery. In the trials following passive warming, there was no significant improvement in performance as evidenced by a reduction in the  $VO_2$  slow component. However, in the trials following prior exercise there was a significant reduction in the slow component. Koppo contrasts her study to Koga's by stating that Koga's use of 50%  $\Delta$  placed the exercise close to the boundary between heavy and severe intensity realm. An additional difference between the two studies was Koga's use of minutes 3-6 of the exercise bout as the period of the slow component. Koppo articulates that this could lead to an underestimation of the slow component as compared to their use of minutes 2-6. Koppo further speculates that the passively-attained muscle temperatures in Koga's trials reached 39 °C, which is higher than actual muscle temperatures normally attained during short duration, high intensity exercise. Overall, the design of the Koppo et al. study helps to demonstrate that the decrease in the  $VO_2$  slow component in the second of two consecutive bouts is not simply related to the increase in muscle temperature induced by prior exercise, but instead caused by factors that remain undetermined. In another related study, Burnley et al. (8) utilized passive warming (submersion in a warm water (42° C) bath for forty minutes) as well as the use of sprint and heavy prior exercise on the same subjects to test the effects on  $VO_2$  kinetics. Again, as in Koppo's study, they demonstrated that both prior sprint and heavy exercise influenced the  $VO_2$  kinetics during subsequent heavy exercise. Both of these resulted in an increase in the absolute amplitude of the primary  $VO_2$  response and reduced the amplitude of the  $VO_2$  slow component. Interestingly, Burnley et al., both in 2002 (8) as

well as in 2000 (7) used a 50%  $\Delta$  approach in determining workloads in the “heavy” exercise domain, in contrast to Koppo’s assertion.

An earlier seminal study by Gerbino et al. (16) demonstrated a speeding of  $VO_2$  kinetics in a second of two bouts of supra-LT exercise. This was associated with a smaller increase in blood lactate accumulation. They conjectured that it may have been a result of vasodilation and elevated muscle blood flow at the start of the second work bout or that possibly acidemia may have improved the  $O_2$  diffusion between the capillary blood and the mitochondria of the exercising muscles. In a subsequent study by MacDonald et al. (29) subjects performed heavy cycling exercise while breathing a hyperoxic gas mixture ( $FIO_2 = 0.70$ ) when compared to those breathing normoxic air ( $FIO_2 = 0.21$ ). Part two of her study involved studying the effect of prior exercise. She found that both hyperoxia and prior exercise caused an acceleration of  $VO_2$  kinetics as measured by MRT, reduced the  $O_2$  deficit and reduced the  $VO_2$  slow component for work rates above ventilatory threshold. Grodjinovsky and Magel (18) studied the effect of two WU procedures (one regular, one vigorous) on running performance (60 yd, 440 yd, 1 mile) and on a 5 minute treadmill test with  $VO_2$  measurement. Times improved for the 60 yd and 440 yd runs when preceded by a WU. A vigorous WU improved the time for the 1 mile run when compared to the regular and NWU. Hajoglou et al. (19) studied the effect of WU on cycling performance utilizing a 3 km time trial (TT). She found improved 3km times with both easy and hard WUs when compared to NWU.  $VO_2$  kinetics, as measured by MRT, also were speeded when preceded by either an easy or hard WU when compared to NWU. PO in the NWU trial was noted to be decreased in the first 500 meters when compared to the two WU trials. It was unclear whether the

relatively slower  $\text{VO}_2$  kinetics were due to the lack of WU or the lower PO. Hettinga et al. (20) studied the  $\text{VO}_2$  response during 4 maximal effort time trials (PO > 100%  $\text{VO}_{2\text{max}}$ ) of 750 m, 1500 m, 2500 m and 4000 m with a standardized WU protocol. Subjects were instructed to finish as fast as possible. POs were observed to exhibit an initial burst in the first 15 seconds of all TTs. Faster  $\text{VO}_2$  kinetics were observed in the 750 m trial as evidenced by a reduction in the MRT compared to all other trials. A significantly higher mean  $\text{VO}_2$  was found for 750 m and 1500 m compared to 4000 m. Bishop et al. (6) found similar results using a “fast-start” strategy in kayaking. The fast start over the first 10 seconds followed by a transition to an even pacing strategy resulted in greater average power and greater average total  $\text{VO}_2$  in a 2 minute kayak ergometer test.

### **Studies Utilizing Mean Response Time (MRT) as a Measure of $\text{VO}_2$ Kinetics**

In the transition from rest to exercise, as  $\text{VO}_2$  begins to rise, “the vertical distance between the actual  $\text{VO}_2$  at a given moment and that required in the steady state represents the energy requirement that must be met from energy stores within the muscle” (28), primarily from the phosphocreatine and anaerobic glycolysis processes. The oxygen equivalent that cannot be met by oxidative processes is called the “oxygen deficit.” It is represented graphically by the area above the curve delineating the rise in  $\text{VO}_2$  until it reaches a plateau (in moderate, steady-state exercise). In studies of  $\text{VO}_2$  the speed of the kinetics is often measured by the use of a time constant ( $\tau$ ) which denotes the time it takes to reach 63% of the response. It is the Phase II  $\tau$  that approximates muscle  $\text{VO}_2$ . Alternatively, the sum of the duration of Phase I and the  $\tau$  in Phase II can be used to estimate the oxygen deficit. This measurement is termed the MRT and was characterized

by Whipp et al. (31). In this study subjects performed a series of SW cycling tests at a work rate under the anaerobic threshold. Eight repetitions of each test were performed. Ventilatory data were collected and modeled according to three different models. Model 1, an exponential response constrained to start at the onset of 100 watt exercise; model 2, an exponential response not constrained as in model 1 and, as such, incorporated a time delay; model 3, exponential response constrained to start only at the inflection point of the response. Statistical analyses were performed to see which model provided the best fit to the data. They concluded that the early phase of  $\text{VO}_2$  response to constant load work is easily distinguishable from the subsequent pattern of increase to the steady state. While their model 3 time constants were shorter than reported in previous studies they cite they state that it is due to the whole response typically being included and leads to an erroneously long time constant. They conclude that utilizing model 3 (time constant data) is the most accurate description of the exponential behavior of gas exchange in Phase II, but also state that model 1, MRT is a useful way to examine oxygen deficit.

### Summary

After reviewing the literature, the following conclusions can be drawn: (1) There is widespread agreement that the use of WU or priming exercise can speed  $\text{VO}_2$  kinetics in heavy intensity upright cycling exercise, (2) Priming exercise should be of sufficient intensity to slightly raise the blood lactate level, (3) Fast-start exercise has been characterized by a burst of power that may be significant to the speed of  $\text{VO}_2$  kinetics, (4) Lack of WU is correlated in one study with a decreased  $\dot{V}\text{O}_2$  that may be related to slower  $\text{VO}_2$  kinetics, (5) MRT is useful when describing the oxygen deficit, while using  $\tau$  is recommended when describing the behavior of gas exchange.

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