THE EFFECT OF MODE AND INTENSITY ON VO$_2$ KINETICS IN THE
SEVERE INTENSITY DOMAIN

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The purpose of this study was to evaluate the effect of mode and intensity on VO$_2$ kinetics in the severe intensity domain. Seventeen participants completed 3-7 tests each on a cycle ergometer and treadmill. For each test, T$_{\text{FATIGUE}}$, VO$_{2\text{max}}$, T$_{\text{MEAN RESPONSE}}$, VO$_{2\text{GAIN}}$, T$_{\text{VO2max}}$ and T$_{\text{@VO2max}}$ were determined. Linear regression techniques were used to describe the relationship between T$_{\text{VO2max}}$ and T$_{\text{FATIGUE}}$.

VO$_{2\text{max}}$ values were higher in running. The VO$_2$ response profile was faster for running than cycling and faster at higher intensities.

The faster VO$_2$ response in running may be associated with larger active muscle mass or differences in muscle activation patterns. The faster response at higher intensities may suggest that VO$_2$ response is driven by O$_2$ demand.
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CHAPTER 1

INTRODUCTION

With respect to their metabolic (blood lactate) and gas exchange (VO$_2$) profiles, three domains of exercise intensity have been characterized. The three domains are moderate, heavy, and severe (Gaesser & Poole, 1996; Whipp & Mahler, 1980).

The moderate intensity domain comprises work rates that can be sustained without inducing a blood lactate response. During exercise at moderate intensity, VO$_2$ demonstrates rapid kinetics, rising exponentially to reach a steady state in approximately 3 minutes in healthy individuals (Whipp & Wasserman, 1972).

The lower boundary of the heavy intensity domain is the work rate at which blood lactate accumulates faster than it can be removed, i.e., the lactate threshold. The upper boundary is the critical power, the asymptote of the relationship between power and time to fatigue (Gaesser & Poole, 1996; Hill, Poole, & Smith, unpublished; Poole, Ward, Gardner, & Whipp, 1988). This is the highest work rate that can be sustained without a progressive increase in blood lactate (Gaesser & Poole, 1996; Whipp & Ward, 1990). During exercise at heavy intensity, an additional slow component is superimposed over the rapid phase of VO$_2$ kinetics, resulting in a delayed steady state. The slow component
represents an additional oxygen (O₂) demand over that predicted from values at submaximal work rates, thus, the O₂ cost per unit of work is greater at heavy intensity than at moderate intensity (Gaesser & Poole, 1996; Poole et al., 1988; Poole & Richardson, 1997).

The severe intensity domain comprises the range of work rates over which VO₂max can be elicited. The lower boundary is critical power and the upper boundary is the highest power output where tolerable duration is just sufficient for VO₂ to reach its maximum (Hill et al., unpublished). During exercise at severe intensity, pH continues to drop, blood lactate continues to rise without stabilizing, and VO₂ appears to rise to its maximum (Poole et al., 1988; Poole, Ward, & Whipp, 1990; Gaesser & Poole, 1996; Poole & Richardson, 1997). At work rates close to critical power, a particularly large slow component is observed and VO₂ continues to increase throughout exercise. VO₂ in the severe intensity domain is a function of power and time (Poole et al., 1988; Poole et al., 1990).

VO₂ kinetics within the severe intensity domain are not well understood. However, studies indicate that the time to achieve VO₂max is shorter at higher intensities within the severe intensity domain (Betik, O’Leary & Hughson, 1998; Whipp, 1994), and that there is a hyperbolic relationship between power and time to reach VO₂max in this domain (Hill & Ferguson, 1999; Hill & Smith, 1999).

Studies have demonstrated that exercise mode can have an effect on VO₂max and VO₂ kinetics. It is generally accepted that treadmill running elicits
higher \( VO_{2\text{max}} \) values than cycling. \( VO_2 \) kinetics have been shown to be faster in walking than in cycling (Chilibeck, Paterson, Smith, & Cunningham, 1996) and slower in arm cranking than in leg cycling (Koga, Shiojiri, Shibasaki, Fukuba, Fukuoka, & Kondo, 1996), which suggests that kinetics are faster during exercise engaging a larger muscle mass.

A challenge in interpreting the results of the few studies that have compared \( VO_2 \) kinetics across exercise modes is the inherent difficulty of equating intensities across modes. One method of circumventing this issue is to define intensity in terms of time to fatigue (\( T_{\text{FATIGUE}} \)). With this method, it is assumed that, regardless of exercise mode, \( VO_2 \) responses to exercise resulting in similar times to fatigue should be similar.

The \( VO_2 \) response profile can be described in terms of mean response times determined by fitting test data to the following model:

\[
VO_2(T) = VO_{2\text{INITIAL}} + (VO_{2\text{maxFINAL}} - VO_{2\text{INITIAL}}) \cdot (1 - e^{-(T-D) / \tau})
\]

where \( VO_2(T) \) is the value for \( VO_2 \) at time = \( T \), \( VO_{2\text{INITIAL}} \) is the pre-exercise \( VO_2 \), \( VO_{2\text{maxFINAL}} \) is a parameter which reflects the asymptote value to which the \( VO_2 \) is projecting, \( D \) represents an early delay like phase which precedes the exponential rise to the steady state, \( \tau \) is a time constant and \( T_{\text{MEAN RESPONSE}} = D + \tau \). \( T_{\text{MEAN RESPONSE}} \) is a parameter that provides a measure of the rate of response. If there is no delay, \( T_{\text{MEAN RESPONSE}} \) is equal to \( \tau \). Time to reach \( VO_{2\text{max}} \) (\( T_{VO2\text{max}} \)) can be directly determined from \( T_{\text{MEAN RESPONSE}} \).
Purpose

The purpose of this study was to evaluate the effect of mode and intensity on VO$_2$ kinetics in the severe intensity domain. All intensities were within the severe domain and exercise modes were treadmill running and ergometer cycling.

Null Hypothesis

There is no difference in the slope and y-intercept of the relationship between $T_{\text{FATIGUE}}$ and $T_{\text{VO2max}}$ in cycling and in running.

Working Hypothesis

There is an effect of mode and an effect of intensity on VO$_2$ kinetics, specifically on the relationships between $T_{\text{FATIGUE}}$ and $T_{\text{VO2max}}$ in cycling and running. It is anticipated that, at a given $T_{\text{FATIGUE}}$, $T_{\text{VO2max}}$ will be faster in treadmill running than in cycling.

Delimitations

Listed below are the factors incorporated in this study by the investigator for delimiting the investigation to help ensure meaningful results.

1. The subject pool consisted of kinesiology students aged 18 to 35 years.
2. All exercise tests were performed on both a cycle ergometer and a treadmill.
Limitations

Listed below are factors that could potentially confound the results of this investigation.

1. The subject pool consisted of kinesiology students aged 18 to 35 years. Therefore, results may not be generalizeable to other populations.

2. Mode comparisons were limited to modes that utilize relatively large muscle mass. The active muscle mass engaged may be too similar to detect a mode effect.

3. Data were forced to a monoexponential model. It is possible, however, that another model may provide a better description of the VO$_2$ profile.

4. Conclusions based on the results assume maximal effort on all tests.
Whipp and Mahler (1980) and Gaesser and Poole (1996) have defined three domains of exercise intensity based upon their distinct metabolic profiles. During exercise in the *moderate* domain, which encompasses all work rates at or below the lactate threshold, VO$_2$ demonstrates rapid kinetics, rising with a gain of ~10 ml to achieve a steady state within 3 min in healthy young individuals (Whipp & Wasserman, 1972). During exercise in the *heavy* domain, which includes work rates above the lactate threshold, a slow component of the VO$_2$ kinetics is superimposed upon the rapid response, resulting in a delayed steady state. The slow component represents an excess VO$_2$ that elevates the gain (Henson, Poole, & Whipp, 1989; Roston, Whipp, Davis, Cunningham, Effros, & Wasserman, 1987; Whipp & Mahler, 1980). During fatiguing exercise at yet higher work rates, in the *severe* domain, the VO$_2$ increases to VO$_2$max (Gaesser & Poole, 1996; Poole et al., 1988; Poole et al., 1990; Sloniger, Cureton, Carrasco, Prior, Rowe, & Thompson, 1996; Whipp, 1994).

VO$_2$ kinetics at various intensities within the severe intensity domain have not been well characterized. However, there is evidence that they may be faster at higher intensities and faster in exercise involving larger muscle mass.
Therefore, the purpose of this study was to evaluate the effect of mode and intensity on VO2 kinetics in the severe intensity domain.

In this chapter, literature describing VO2 kinetics with various exercise modes is discussed. Possible effects of intensity (within the severe domain) and mode on kinetics are also discussed. Detailed descriptions of especially pertinent studies are provided.

**VO2 Kinetics in the Moderate Intensity Domain**

The moderate intensity domain comprises work rates that can be sustained without inducing a blood lactate response i.e., below the lactate threshold (Gaesser & Poole, 1996; Poole et al., 1990). The monoexponential VO2 response can be fit to the following model:

\[
\text{VO}_2(T) = \text{VO}_2^{\text{INITIAL}} + (\text{VO}_2^{\text{maxFINAL}} - \text{VO}_2^{\text{INITIAL}}) \cdot (1 - e^{-\frac{(T-D)}{\tau}})
\]

where \(\text{VO}_2(T)\) is the value for \(\text{VO}_2\) at time = \(T\), \(\text{VO}_2^{\text{INITIAL}}\) is the pre-exercise \(\text{VO}_2\), \(\text{VO}_2^{\text{maxFINAL}}\) is a parameter which reflects the asymptote value to which the \(\text{VO}_2\) is projecting, \(D\) represents an early delay like phase which precedes the exponential rise to the steady state, \(\tau\) is a time constant and \(T_{\text{MEAN RESPONSE}} = D + \tau\). \(T_{\text{MEAN RESPONSE}}\) is a parameter that provides a measure of the rate of response. If there is no delay, \(T_{\text{MEAN RESPONSE}}\) is equal to \(\tau\).

While the monoexponential model provides a simple description of the \(\text{VO}_2\) profile in many cases, several investigators have described the response using more complex models. In fact, in some circumstances, three phases of \(\text{VO}_2\) kinetics have been identified (Barstow, 1994; Gaesser & Poole, 1996; Poole...
Phase 1 consists of an early delay-like component at exercise onset (first 15-25s of exercise). The phase 1 rise in VO₂ is generally attributed to augmented cardiac output and pulmonary blood flow. Other contributing factors are changes in lung gas stores and mixed venous O₂ content (Barstow, 1994; Gaesser & Poole, 1996; Poole & Richardson, 1997).

In phase 2, VO₂ rises exponentially to a steady state, with a time constant (tau) of 30-45 s. Steady state VO₂ is a linear function of work rate (Barstow, 1994; Gaesser & Poole, 1996). Phase 2 is initiated when venous blood from the exercising muscle arrives at the lung. This phase reflects the progressive desaturation of venous blood and continued increase in cardiac output. Within the moderate intensity domain, the phase 2 tau remains uniform across intensities (Barstow, 1994; Gaesser & Poole, 1996; Poole & Richardson, 1997). Modeling studies have shown that, in phase 2, pulmonary VO₂ mirrors muscle VO₂. Tau appears to reflect muscle O₂ kinetics, as there is a close temporal association between pulmonary VO₂ changes and phosphocreatine changes within the exercising muscle (Barstow, 1994; Gaesser & Poole, 1996; Poole & Richardson, 1997).

In Phase 3, at intensities within the moderate domain, the steady state level of VO₂ is achieved within ~ 3 minutes. Phase 3 represents the plateau of cardiac output and the nadir of the decrease in venous O₂ saturation (Barstow, 1994; Gaesser & Poole, 1996; Poole & Richardson, 1997).
At moderate intensity, VO₂ kinetics can be reduced with training and are usually faster in participants that are highly fit (Barstow, 1994; Gaesser & Poole, 1996; Poole & Richardson, 1997).

VO₂ Kinetics in the Heavy Intensity Domain

The lower boundary of the heavy intensity domain is the lactate threshold or the work rate at which blood lactate accumulates faster than it can be removed. At this work rate, blood lactate remains elevated for the duration of exercise. The upper boundary of heavy intensity is the highest work rate that can be sustained without a progressive increase in blood lactate (Gaesser & Poole, 1996; Whipp & Ward, 1990). At this work rate, although blood lactate remains elevated, blood lactate production and removal are balanced. This upper boundary of the heavy intensity domain is the critical power (PITICAL), the asymptote of the relationship between power output (P) and time to fatigue (T_FATIGUE) (Gaesser & Poole, 1996; Hill, Poole, & Smith, unpublished; Poole et al., 1988). Regardless of training status or absolute VO₂max, P_critical is the highest power at which blood lactate can be stabilized (Poole et al., 1990).

Barstow (1994) explained that phase 2 kinetics of the VO₂ response are more complex at heavy intensity. At the onset of exercise, VO₂ rises exponentially with a tau similar to that observed at moderate intensity, and blood lactate is elevated. After the first 100-200s into exercise, an additional slow component is superimposed over the rapid phase of O₂ kinetics. The slow component is evident only at work rates above the lactate threshold (Gaesser &
Poole, 1996; Poole & Richardson, 1997). At work rates between the lactate threshold and $P_{\text{CRITICAL}}$, $VO_2$ and blood lactate stabilize within 10-20 minutes, $VO_2$ can reach a steady state, and exercise can be sustained for a prolonged duration (Gaesser & Poole, 1996; Poole et al., 1988). Since the slow component represents an additional $O_2$ demand over that predicted from values at submaximal work rates, the $O_2$ cost per unit of work is greater at heavy intensity than at moderate intensity (Gaesser & Poole, 1996; Poole et al., 1988; Poole & Richardson, 1997). The increased $VO_2$ is the result of an increase in blood flow and $O_2$ extraction, which contribute equally to the excess $VO_2$ above the predicted value (Gaesser & Poole, 1996).

$VO_2$ kinetics in the heavy intensity domain can be speeded by training and, at a given work rate, end-exercise $VO_2$ can be reduced due to a reduction in the $VO_2$ slow component. Thus, observed $VO_2$ at a given work rate could be higher in an unfit subject than in a fitter subject (Poole & Richardson, 1997).

**$VO_2$ Kinetics in the Severe Intensity Domain**

$P_{\text{CRITICAL}}$ differentiates heavy intensity, where blood lactate is elevated but exercise can be sustained, from severe intensity, where blood lactate continues to increase and $VO_2$ rises to maximum (Poole et al., 1988; Poole et al., 1990). Hill et al., (unpublished) defined the severe intensity domain as that range of work rates over which $VO_{2\text{max}}$ can be elicited. The lower boundary is $P_{\text{CRITICAL}}$, and the upper boundary is the highest power output where tolerable duration is just sufficient for $VO_2$ to reach $VO_{2\text{max}}$. 

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Kinetics within the severe intensity domain are complex and not well defined (Poole et al., 1990; Whipp & Ward, 1990). At work rates within the severe intensity domain, VO$_2$ appears to increase linearly to its maximum after the rapid exponential increase in the first 3 minutes. A particularly large slow component is observable at work rates close to $P_{\text{CRITICAL}}$. At these work rates, the slow component increases throughout exercise (Gaesser & Poole, 1996).

According to Poole et al. (1990), work rates in the severe intensity domain result in VO$_{2\text{max}}$ if exercise is continued to fatigue. During exercise at intensities above $P_{\text{CRITICAL}}$, pH continues to drop, blood lactate continues to rise without stabilizing, and VO$_2$ continues to increase until a maximum value is reached (Poole et al., 1988; Poole et al., 1990; Gaesser & Poole, 1996; Poole & Richardson, 1997). No gas exchange or metabolic steady state can be achieved within the severe intensity domain (Gaesser & Poole, 1996). VO$_2$ acts as a function of power and time and, consequently, cannot be defined by a distinct power (Poole et al., 1988; Poole et al., 1990).

VO$_2$ kinetics within the severe domain can most likely be speeded by training. Training would also result in a higher VO$_{2\text{max}}$ at fatigue (Poole & Richardson, 1997).

**Testing in the Severe Domain**

Poole and Richardson (1997) explained that, when the goal of constant power testing is to determine VO$_{2\text{max}}$, there is no need to select a precise work rate. As long as exercise is within the severe intensity domain, the VO$_2$ attained
at fatigue in the severe domain is equal to $\text{VO}_{2}\text{max}$ and is not affected by work rate. A broad range of work rates can elicit $\text{VO}_{2}\text{max}$ as long as exercise is continued to fatigue.

Poole et al., (1990) concluded that during constant-load exercise tests performed at work rates above $P_{\text{CRITICAL}}$, $\text{VO}_2$ will increase to $\text{VO}_{2}\text{max}$ and a limit of work tolerance. They attribute this slow, continuous increase primarily to mechanisms related to the sites and routes of lactate metabolism. They believe that, due to the inability to stabilize $\text{VO}_2$ at these supra-$P_{\text{CRITICAL}}$ work rates, it may not be possible to demonstrate a “required” $\text{VO}_2$ within the severe domain.

**Effect of Intensity on Kinetics within the Severe Intensity Domain**

Pulmonary gas exchange responses within the severe domain have not been as well characterized as those within the moderate and heavy domains. However, Whipp (1994) has stated that $T_{\text{VO}_2\text{max}}$ is shorter ($\text{VO}_{2}\text{max}$ is achieved faster) at higher intensities and, by definition, these intensities must fall within the severe domain. Recently, Betik et al. (1998) reported that the tau of the $\text{VO}_2$ response is faster at 130% of $\text{VO}_{2}\text{max}$ than at 100% of $\text{VO}_{2}\text{max}$. In two other studies evaluating responses in the severe intensity domain, it was reported that $T_{\text{VO}_2\text{max}}$ is faster at higher intensities for both running (Hill & Ferguson, 1999) and cycle ergometer (Hill & Smith, 1999) exercise. In addition, in both these studies, there was a hyperbolic relationship between $T_{\text{VO}_2\text{max}}$ and power output.

Hill et al. (unpublished) conducted a study to investigate the $\text{VO}_2$ response to fatiguing exercise bouts within the severe intensity domain (for
details, see section titled Individual Studies). They described a relationship between power and VO\textsubscript{2} kinetics over a range of intensities within the severe domain, where T\textsubscript{MEAN RESPONSE} was faster at higher work rates. Calculating T\textsubscript{VO2max} from T\textsubscript{MEAN RESPONSE}, they were able to confirm the existence of a hyperbolic relationship between power and T\textsubscript{VO2max}. In addition, they determined that there is a linear relationship between T\textsubscript{FATIGUE} and T\textsubscript{VO2max}.

**Effect of Mode on VO\textsubscript{2max} and VO\textsubscript{2} kinetics**

It is generally accepted that there are differences between VO\textsubscript{2} responses to cycling and treadmill exercise. In an early study, Hermansen and Saltin (1969) found that, at maximal workloads, VO\textsubscript{2max} was higher (7%) on the treadmill. VO\textsubscript{2} differences were most significant at treadmill inclinations of greater than 3°. At submaximal workloads, they found VO\textsubscript{2} was higher, and HR, pulmonary ventilation, and blood lactate were lower, on the treadmill. Martinez, Santos, Grijalba, Santesteban, and Gorostiaga (1993), found work time was longer, VO\textsubscript{2max} was higher (8%), and blood lactate after final stage (exhaustion) was lower, at maximal workloads, in treadmill running than in other modes.

As these studies suggest that VO\textsubscript{2max} is a function of mode, other studies have demonstrated that VO\textsubscript{2} kinetics may also be a function of mode. When comparing responses to exercise within older participants, Chilibeck et al. (1996) found VO\textsubscript{2} kinetics in walking were significantly faster than VO\textsubscript{2} kinetics in cycling. In another study, Koga et al. (1996) examined VO\textsubscript{2} kinetics and cardiac output in arm cranking and leg cycling. They found that VO\textsubscript{2} kinetics at the onset
of exercise are slower for arm than for leg exercise. \( T_{\text{MEAN RESPONSE}} \) for \( \text{VO}_2 \), tissue \( \text{O}_2 \) consumption and cardiac output were longer in arm exercise. (For details on the above referenced studies, see the section titled “Individual Studies”).

**Individual Studies**

The purposes of the study by Hill et al. (unpublished) were 1) to re-evaluate \( P_{\text{CRITICAL}} \) as the demarcation between the heavy and severe intensity domains and 2) to evaluate a method for estimating the upper bound of the severe intensity domain. The direct measure of \( T_{\text{VO2max}} \) requires an assumption that \( \text{VO}_2_{\text{max}} \) was achieved during every test and that the exact moment it was achieved can be detected by examination of breath-by-breath responses. Rather than relying on this assumption, Hill et al. based their determination of \( T_{\text{VO2max}} \) on the kinetics of the \( \text{VO}_2 \) response at different intensities. They hypothesized that \( T_{\text{VO2max}} \) is a hyperbolic function of power with an asymptote equal to \( P_{\text{CRITICAL}} \) and, thus, \( P_{\text{CRITICAL}} \) is the lower boundary of the severe intensity domain. A second hypothesis was that \( T_{\text{VO2max}} \) is a linear function of \( T_{\text{FATIGUE}} \) and the relationship between \( T_{\text{VO2max}} \) and \( T_{\text{FATIGUE}} \) can be used to estimate the upper bound of severe intensity.

Eleven participants (9 men, aged 25 ± 6 yr; 2 women aged 18 and 21 yr) each performed a total of nine tests on a cycle ergometer. The first test was an incremental test to determine \( \text{VO}_2_{\text{max}} \) and the highest power that could be sustained for 1 minute (\( P_{\text{MAX}} \)). The next five tests were constant power tests performed at 95%, 100%, 110% and 135% of \( P_{\text{MAX}} \), and a practice trial performed
Values for $P_{\text{CRITICAL}}$ and $P'_{\text{CRITICAL}}$, were defined based on $T_{\text{VO2max}}$ and $T_{\text{VO2}}$ at each power. In order to determine if $P_{\text{CRITICAL}}$ is truly the lower boundary of the severe domain, 8 participants performed two additional tests in which they exercised for 25 minutes at $P_{\text{CRITICAL}}$ and at $P_{\text{CRITICAL}} + 10$ W. In order to describe the upper bound of the severe intensity domain, estimates of the highest power that could elicit VO$_{2\text{max}}$ ($P_{\text{UPPER BOUND}}$) and the shortest time that could elicit VO$_{2\text{max}}$ ($T_{\text{UPPER BOUND}}$), were derived based on the relationships between $T_{\text{MEAN RESPONSE}}$ and $T_{\text{FATIGUE}}$, and between power and $T_{\text{VO2max}}$. Eight of the participants also performed an additional incremental test to determine if there had been a training effect over the course of the study.

Hill et al. (unpublished) found that the relationship between power output and $T_{\text{FATIGUE}}$ was well described by the hyperbolic model:

$$T_{\text{FATIGUE}} = AWC \cdot (\text{power} - P_{\text{CRITICAL}})^{-1}.$$  

Mean values obtained using three mathematically equivalent models were identical.

They also found a relationship between power output and VO$_2$ kinetics in the severe intensity domain. $T_{\text{MEAN RESPONSE}}$ was faster at higher work rates, while the VO$_{2\text{max}}$$_{\text{PROJECTED}}$ was constant across a range of power outputs. The same VO$_{2\text{max}}$ value was attained in all tests, but it was sustained longer in the longer tests.

Hill et al. (unpublished) used three mathematically equivalent versions of the following model to describe the relationship between power and $T_{\text{VO2max}}$:

$$T_{\text{VO2max}} = AWC' \cdot (\text{power} - P'_{\text{CRITICAL}})^{-1}.$$  

The generated parameter, $P'_{\text{CRITICAL}}$,
represents the intensity beyond which VO$_{2\text{max}}$ can be achieved. This $P'_{\text{CRITICAL}}$ denotes the upper boundary of the heavy intensity domain and is immediately adjacent to the lower boundary of the severe intensity domain. $P'_{\text{CRITICAL}}$ was found to be to equal to $P_{\text{CRITICAL}}$.

Consistent with previous descriptions of VO$_2$ kinetics in the severe intensity domain, Hill et al. (unpublished) found that VO$_2$ did reach its maximum at intensities from 95% to 135% of $P_{\text{max}}$. This seems to indicate that VO$_{2\text{max}}$ can be reached and sustained for a period of time during constant-power exercise at intensities close to those that elicit VO$_{2\text{max}}$ in an incremental test.

Hill et al. (unpublished) proposed that the definition of the severe intensity domain be simplified to “exercise intensities at which VO$_{2\text{max}}$ can be elicited”. They feel this definition makes no attempt to describe how VO$_2$ climbs to VO$_{2\text{max}}$, provides no stipulation that fatigue and VO$_{2\text{max}}$ occur together, and provides an upper limit to the domain.

**Effect of Mode on VO$_{2\text{max}}$**

The purpose of the study by Martinez et al. (1993) was to determine the differences in cardiorespiratory and metabolic responses to treadmill running, ergometer cycling and roller skating in nine (six male, three female) top level roller skaters. Preliminary tests were conducted in order to establish a testing protocol that would elicit similar heart rate kinetics across all three modes. A discontinuous graded test performed to exhaustion, with 3-min work stages and 2-min rest stages, was used for each exercise mode.
The first preliminary test was conducted to measure VO$_{2\text{max}}$ during treadmill running for each subject. In the second test, they measured heart rate (HR) in 3 participants in treadmill running, cycling on a cycle ergometer, and roller-skating. For each mode, they used energy cost equations to choose the power or velocity, at each submaximal and maximal stage that would elicit a “similar theoretical VO$_2$”.

The results indicated a linear relationship between heart rate (HR) and workload in all 3 modes. The same 3 participants performed a third preliminary test, which consisted of 1 test for each mode. The treadmill test protocol was the same as in the second preliminary test. The velocity or power chosen for each stage in the cycling and skating tests was that which would elicit a HR similar to the corresponding treadmill stage, based on the HR-workload relationships obtained in the second test. The results of the third preliminary test indicated similar HR kinetics throughout all stages across all modes.

The resulting details of the final testing protocols used were as follows: On the treadmill, the inclination was +1%. The theoretical maximum velocity ($v_{\text{max}}$) was equal to the treadmill running VO$_{2\text{max}}$/3.5 ml/kg/min per km/hr. The first workload was 25% of $v_{\text{max}}$. Each successive workload increased by 11% of $v_{\text{max}}$ until exhaustion, so the 8th workload corresponded to $v_{\text{max}}$. On the cycle ergometer, the pedal rate was 60 rev·min$^{-1}$. Each work rate was defined by the equation $y = 3.21 \cdot x + 1.32$, where $y =$ treadmill running test velocity.
(in km·h⁻¹), and \(x\) = cycling workload (W·kg body mass⁻¹). The first workload was an average 69 W (range 50 W to 90 W). Each successive workload was an average increase of 40 W (range 20 W to 60 W). In roller skating, workload was defined by the equation \(y = 0.17 \cdot x \cdot 3.11\), where \(y\) = treadmill velocity (in km·h⁻¹) corresponding to each workload and \(x\) = roller skating velocity (in m·s⁻¹). The first workload was an average of 11.2 (m·s⁻¹)² (range 9.2 to 12.5). Each successive workload was an average of 11 (m·s⁻¹)² (range 6 to 13).

Martinez et al. (1993) found no HR differences at submaximal workloads across modes. On the treadmill, blood lactate was significantly lower, and VO₂, at a given stage, was higher. There were no observed differences in HR as a % of VO₂max between running and cycling. At maximal workloads, work time was longer, VO₂max was higher (8%), and blood lactate after final stage (exhaustion) was lower in treadmill running than in other the other modes.

Regarding the differences in VO₂ treadmill running and cycling, Martinez et al. (1993) believed their findings support the assertion that during maximal cycling, local muscular fatigue limits performance before there is a maximal demand on the O₂ transport system. They stated that their results suggest that the estimated amount of muscle mass involved in running and cycling is similar, but more muscle mass is actually engaged in running. They also referred to a potential difference in the tension developed within the muscle. Peak tension developed during each pedal thrust is higher in maximal cycling than in running.
This tension difference implies a greater fast-twitch fiber recruitment and, hence, a greater anaerobic contribution.

The purpose of the study conducted by Hermansen and Saltin (1969) was to study the circulatory and respiratory responses to maximal cycle ergometer and treadmill exercise. Fifty-five male participants between the ages of 19 and 86 years performed maximal exercise on a treadmill running uphill (3° or 5.25%) and on a cycle ergometer at a pedal cadence of 50 revolutions per minute. Submaximal workloads were chosen to represent 25, 50, 75, and 90% of the subject’s VO$_2$max measured previously for each mode. Each subject performed two submaximal tests to establish the relationship between VO$_2$ and other physiological parameters. Maximal workloads were selected based on the relationships established at submaximal work. Additional tests were conducted in which treadmill inclination and cycle ergometer pedal cadence were varied when differences in maximal values between the two modes were observed.

Hermansen and Saltin (1969) compared mean values for VO$_2$, pulmonary ventilation, heart rate, and blood lactate. At maximal workloads, VO$_2$ was higher (7%) on the treadmill. VO$_2$ differences were most significant at treadmill inclinations of greater than 3°. They found no significant differences in corresponding mean values for pulmonary ventilation, HR, or blood lactate. At submaximal workloads, VO$_2$ was higher on the treadmill, and pulmonary ventilation, HR and blood lactate were higher on the cycle ergometer.
As a possible explanation for the observed differences, Hermansen and Saltin (1969) cited the generally accepted theory that muscle mass engaged in running is probably larger than that used in cycling (especially when running uphill).

**Effect of Mode on Kinetics**

Chilibeck et al. (1996) examined cardiorespiratory kinetics in groups of old and young participants during exercise of muscle groups that differed in habitual use, mass, and central blood flow requirements. Though their objective was to explore the effects of aging on kinetics, the results they obtained are quite applicable to the study of mode and VO$_2$ kinetics.

Two studies were conducted. In the first, VO$_2$ kinetics in cycling and unilateral plantar flexion were measured in 12 older participants (2 men, 10 women, aged $66.7 \pm 6.7$ yr) and compared to measures obtained for 16 younger participants (9 men, 7 women, aged $26.3 \pm 2.5$ yr). In the second study, VO$_2$ kinetics in cycling and treadmill exercise were measured in five older men (aged $69.6 \pm 4.3$ yr) and compared to measures obtained for five younger participants (two men, three women, aged $24.4 \pm 3.1$ yr).

In order to determine ventilatory threshold and VO$_{2\text{max}}$, all participants performed a graded exercise test on a cycle ergometer. In addition, participants in the first study performed graded exercise tests on a plantar flexion ergometer to determine peak work rate. Participants in the second study also had
ventilatory threshold and VO$_{2\text{max}}$ measured for graded exercise tests on a treadmill.

One week later, participants performed three repeats each of a square wave lasting either 6 min (study 1) or 5 min (study 2) on the cycle ergometer. The work rate was set to an intensity that would correspond to 90% of ventilatory threshold. Square wave tests were separated by 6 min of unloaded cycling. In order to minimize the phase 1 response and facilitate phase 2 modeling, transitions to square wave repeats were from loadless work rates to minimize anticipatory responses.

Study 1 participants performed an average of 12 square wave transitions to and from ankle plantar flexion. The exercise intensity was set at 45% of the subject’s peak work rate. Transitions were separated by 6 min of unloaded plantar flexion.

Study 2 participants performed three repeats each of a square wave lasting 5 min on the treadmill. The work rate was set to an intensity that would correspond to 90% of ventilatory threshold. Treadmill speed was set at 3.3 mph for everyone, and the degree of incline was increased or decreased to result in a work rate that would elicit a VO$_2$ similar to that of each subject’s cycling square wave. Square wave repeats were separated by 5 min of walking next to the running treadmill at a rate that would elicit a VO$_2$ similar to that of unloaded cycling.
The same monoexponential model described previously for moderate intensity exercise was used to fit the averaged responses for each subject. This model was used to fit data beginning at the start of phase 2 for the VO$_2$ response and beginning at time 0 for HR response. The first 20 s of data (phase 1) are excluded from this model.

Chilibeck et al. (1996) found that VO$_2$ kinetics in moderate intensity cycling and treadmill walking were slowed as a function of age. HR kinetics were also slower with age. There was evidence of a strong correlation between tau VO$_2$ and tau HR.

Compared to younger individuals, older individuals must rely on anaerobic systems to a greater extent to meet energy requirements at the onset of exercise. This increases the possibility of early fatigue. Kinetics were faster in trained old than in untrained old. In fact, they discovered that training can improve kinetics to the point where response rates are similar to young, untrained participants. Chilibeck et al. (1996) believed the correlation of training improvements in VO2 and tau HR suggests that VO$_2$ kinetics may be related to central O$_2$ delivery.

When comparing exercise modes within the same older participants, who were accustomed to walking, VO$_2$ kinetics in walking were significantly faster than VO$_2$ kinetics in cycling. In addition, VO$_2$ kinetics during plantar flexion were not slower in the older participants compared to the younger ones. Chilibeck et al. (1996) believed these findings suggest that slowed VO$_2$ kinetics occur only in
modes of exercise where the muscle groups are not accustomed to the activity. They suggested that this is related to the recruitment of different muscle fiber types during a frequently used movements pattern compared to recruitment patterns during a seldom used movement pattern.

Chilibeck et al. (1996) explained that the reason age affects VO$_2$ kinetics in cycling and treadmill walking but not in plantar flexion, may be due to different circulatory demands of large and small muscle mass. Cardiovascular function is not generally challenged in exercise of small muscle mass, as O$_2$ perfusion is in excess of metabolic demand. In whole body exercise, involving larger muscle mass, the demand for O$_2$ may exceed its supply. So O$_2$ supply may be limiting to VO$_2$ kinetics during leg cycling but not during plantar flexion. The slower kinetics of cardiac output in the old would cause VO$_2$ kinetics to be affected during exercise of large muscle mass (cycling or walking) but not a smaller mass (plantar flexion).

In another study comparing modes of exercise, Koga et al. (1996) compared VO$_2$, mean tissue VO$_2$ and cardiac output kinetics in arm cranking and leg cycling. They hypothesized that VO$_2$ kinetics at onset of exercise are slower for arm than for leg exercise.

Five men, between 20 and 40 years of age, performed an incremental test, for each mode, on a cycle ergometer positioned for both leg cycling and arm cranking in a sitting position. Each subject’s anaerobic threshold and VO$_{2\text{max}}$ was determined for both arm and leg exercise. Participants then performed a
series of constant-power tests at intensities which corresponded to 30, 50 and 75% of peak VO$_2$ (25, 38, and 50 W) for arm cranking, and 17, 30, 50, and 75% of peak VO$_2$ (25, 50, 100, and 150 W) for leg cycling. In order to compare mean tissue VO$_2$ and cardiac output kinetics, four participants also performed an additional test for each mode at the same absolute work rate of 38 W, and at the same relative work rate (~ 50% of peak VO$_2$). The flywheel of the cycle ergometer was spinning at 50 rpm at the onset of each test. At each work rate, participants performed four to eight repetitions of 5 min exercise periods, with between 15 and 60 min (for more intense) rest periods in between. $T_{\text{MEAN RESPONSE}}$ was described using a monoexponential model for response data from exercise onset to the end of exercise. A second monoexponential model was used to describe data omitting phase 1. For work rates over the anaerobic threshold, a third, two-component model was used which omitted phase 1 data and included a slow component.

Koga et al. (1996) found VO$_2$ $T_{\text{MEAN RESPONSE}}$ was significantly longer in arm exercise than leg exercise at the same absolute or relative work rate. Phase 1 VO$_2$ kinetics were slower for arm than for leg exercise at identical work rates. They believed these results suggest that immediate cardiac output adjustments to arm exercise are less pronounced. Phase 2 VO$_2$ kinetics and cardiac output kinetics were also significantly longer for arm exercise. $T_{\text{MEAN RESPONSE}}$ for VO$_2$, tissue VO$_2$, and cardiac output were longer in arm exercise. They believed the
delayed VO$_2$ kinetics in arm exercise may be due to delayed cardiac output kinetics and higher glycolysis occurring early during arm exercise.

**Summary**

Severe intensity exercise can be defined as encompassing a range of intensities at which VO$_{2\text{max}}$ can be elicited. There is evidence that, within this domain, the kinetics of the VO$_2$ response are directly related to intensity. There is also evidence that kinetics may be a function of exercise mode, with faster kinetics during exercise involving a larger muscle mass. The purpose of this study is to evaluate the effects of both intensity and mode on the VO$_2$ kinetics response within the severe domain.
CHAPTER 3
METHODS

It has been shown that VO\textsubscript{2} kinetics (i.e., as reflected by the derived value for $T_{MEAN\ RESPONSE}$ or $\tau$) are a function of intensity in running and cycling within the severe intensity domain. However, it is difficult to compare kinetics across modes because it is difficult to equate intensities across modes. One way to circumvent this problem is to take advantage of the relationship between $T_{MEAN\ RESPONSE}$ and $T_{FATIGUE}$. Regardless of exercise mode, for intensities within the severe domain, it would seem logical that the largest $T_{FATIGUE}$ (i.e., at $P_{CRITICAL}$ or $V_{CRITICAL}$) should be similar, as should the shortest $T_{FATIGUE}$ (i.e., the shortest time in which VO\textsubscript{2max} can be elicited). (It is acknowledged that no studies have actually attempted to verify if this is, in fact, true).

Participants

Participants were volunteers from courses in the Department of Kinesiology at the University of North Texas. All were involved in personal fitness activities but none was training for any type of competitive sport.

Overview

Each participant performed 3 to 7 exhaustive tests on an electronically-braked cycle ergometer (Mijnhart 800S, The Netherlands), and 3 to 7 exhaustive tests on a treadmill (Model 24-72 Quinton Instruments, Seattle, WA). All tests
were conducted under similar conditions in a temperature-controlled laboratory (20 to 22 °C). For each mode, fatiguing constant power tests were performed at powers or velocities selected to induce fatigue in 3 to 10 minutes. Participants performed as many tests as were necessary to complete one test for each mode with approximate durations of 3, 5 and 8 minutes.

During each exercise test, and during the preceding warm-up period, expired gases were collected and analyzed using a MedGraphics (St. Paul, Minnesota, USA) CPX metabolic cart. The cart was calibrated before and after each test according to the manufacturer's instructions using precision-analyzed gases that span the expected \( \text{O}_2 \) and \( \text{CO}_2 \) concentration ranges and a 3-L syringe. Testing sessions were separated by at least 24 hours, and were scheduled at approximately the same time of day for each participant. On testing days, the participants were instructed not to exercise prior to arriving at the laboratory. Participants were directed to arrive well rested for the testing sessions, not to smoke, not to drink beverages containing alcohol, caffeine, or carbonation prior to tests, and to eat only lightly in the hours before testing. Adherence was verified before each test.

**Constant Power Tests**

All participants performed fatiguing constant power tests at intensities selected by the investigator. Each test was preceded by a 5-min warm-up at a work rate selected to elicit a heart rate of 120 to 140 b·min\(^{-1}\), and a 5-min recovery. Participants received verbal encouragement throughout the tests.
$T_{\text{FATIGUE}}$ was measured to the nearest second and VO$_2$max for that test was determined as the highest 30-breath value determined from a rolling average. The participants were not given information about the work rate or their elapsed time for any test until they were debriefed after all data collection had been completed.

**Determination of VO$_2$ Kinetics**

For each participant, for each test, values for $T_{\text{MEAN RESPONSE}}$ and VO$_2$GAIN were determined using nonlinear regression techniques on Kaleidagraph (Ver. 3.09) by fitting breath-by-breath VO$_2$ responses to the following equation:

$$\text{VO}_2(T) = \text{VO}_2^{\text{INITIAL}} + \text{VO}_2^{\text{GAIN}} \cdot (1 - e^{\left(-T / T_{\text{MEAN RESPONSE}}\right)})$$

where VO$_2(T)$ is the value for VO$_2$ at time $= T$, VO$_2^{\text{INITIAL}}$ is the pre-exercise VO$_2$, VO$_2^{\text{GAIN}}$ is the difference between VO$_2^{\text{INITIAL}}$ and the asymptote value to which the VO$_2$ is projecting, and $T_{\text{MEAN RESPONSE}}$ is a parameter that describes the rate of response. This is the equation that was presented in Chapter 2 assuming $D=0$. As there is no delay in this model, $T_{\text{MEAN RESPONSE}}$ is equal to tau and can replace tau in the equation. Time to reach VO$_2$max ($T_{\text{VO2max}}$) can be directly determined from $T_{\text{MEAN RESPONSE}}$.

**Estimation of the Time to Achieve VO$_2$max ($T_{\text{VO2max}}$)**

VO$_2$max is essentially attained when the increase in VO$_2$ is 99% of the projected gain (VO$_2$GAIN). This occurs when the value of $(-T / T_{\text{MEAN RESPONSE}})$ from the above equation is 0.99. When $T = (4.6 \cdot T_{\text{MEAN RESPONSE}})$, the value of the expression would be $(1 - e^{-4.6}) = 0.99$ or 99%. For each participant, for each
test, \( T_{\text{VO2max}} \) was determined as \((4.6 \cdot T_{\text{MEAN RESPONSE}})\). Time spent at \( \text{VO2max} \) was determined by subtracting the \( T_{\text{VO2max}} \) from \( T_{\text{FATIGUE}} \).

**Description of the Relationship Between \( T_{\text{VO2max}} \) and \( T_{\text{FATIGUE}} \)**

For both running and cycling data, linear regression techniques on SPSS were used to describe the relationship between the \( T_{\text{VO2max}} \) (calculated from \( T_{\text{MEAN RESPONSE}} \)) and \( T_{\text{FATIGUE}} \). With \( T_{\text{VO2max}} \) expressed as a function of \( T_{\text{FATIGUE}} \), it was possible to solve for the unique \( T_{\text{FATIGUE}} \), namely the upper bound of the severe intensity domain \( (T_{\text{UPPER BOUND}}) \), at which \( \text{VO2max} \) is achieved at the point of fatigue.

**Statistical Analyses**

An average \( \text{VO2max} \) value was calculated for all completed constant power tests on the cycle ergometer and on the treadmill. The average values were compared using a paired-means t-test to determine differences within and between modes. Mean values for \( T_{\text{UPPER BOUND}} \) and/or the means, slopes and y-intercepts of the regressions of \( T_{\text{VO2max}} \) and \( T_{\text{FATIGUE}} \) for cycling and running were compared using paired-means t-tests.

Another method of comparing the \( \text{VO2} \) responses was to match tests of similar duration. Mean values were calculated and a two-way ANOVA with repeated measures across exercise mode and exercise duration was run to determine main and interaction effects.

All results are reported as mean ± SD. Statistical significance was preset at an alpha \( \leq 0.05 \) for all analyses.
CHAPTER 4

RESULTS

The purpose of this study was to evaluate the effect of mode and intensity on VO$_2$ kinetics in the severe intensity domain. The results of this study are presented in this chapter.

Participant Demographics

Mean values for age, height and weight of the 17 participants, by gender, are presented in Table 1.

<table>
<thead>
<tr>
<th>Table 1. Participant Demographics</th>
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<tbody>
<tr>
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<tr>
<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Male (N=7)</td>
</tr>
<tr>
<td>Age 25 ± 6</td>
</tr>
<tr>
<td>Height (cm.) 177.1 ± 5.2</td>
</tr>
<tr>
<td>Weight (kg.) 77.8 ± 9.7</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Female (N=10)</td>
</tr>
<tr>
<td>Age 24 ± 4</td>
</tr>
<tr>
<td>Height (cm.) 170.4 ± 10.6</td>
</tr>
<tr>
<td>Weight (kg.) 65.3 ± 11.2</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation

Maximal Aerobic Power

The highest rolling 30-breath average VO$_{2\text{max}}$ value for each test was recorded. The mean of these values was determined, for each participant, for each mode of exercise. The 30-breath VO$_{2\text{max}}$ was 12% higher ($p < 0.01$) in running than cycling (2.75 ± 0.62 l·min$^{-1}$ vs 2.45 ± 0.60 l·min$^{-1}$). Mean VO$_{2\text{max}}$ values are presented in Table 2.
Table 2. Maximal Aerobic Power

<table>
<thead>
<tr>
<th></th>
<th>Male (N=7)</th>
<th>Female (N=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cycling VO(_{2\text{max}}) (ml·min(^{-1}))</td>
<td>3002 ± 321</td>
<td>2070 ± 412</td>
</tr>
<tr>
<td>Running VO(_{2\text{max}}) (ml·min(^{-1}))</td>
<td>3386 ± 189</td>
<td>2302 ± 355</td>
</tr>
<tr>
<td>Cycling VO(_{2\text{max}}) (ml/kg·min(^{-1}))</td>
<td>39.1 ± 6.0</td>
<td>31.7 ± 3.2</td>
</tr>
<tr>
<td>Running VO(_{2\text{max}}) (ml/kg·min(^{-1}))</td>
<td>44.0 ± 4.9</td>
<td>36.3 ± 1.8</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation

**VO\(_{2}\) Response Profile**

VO\(_{2}\) kinetics were described by three parameters, VO\(_{2\text{INITIAL}}\), T\(_{\text{MEAN RESPONSE}}\), and VO\(_{2\text{GAIN}}\). T\(_{\text{VO2max}}\) was determined as 4.6 · T\(_{\text{MEAN RESPONSE}}\). Overall, mean response times were faster in treadmill running than in cycling. Mean values are presented in Table 3.

Table 3. VO\(_{2}\) Kinetics Parameters

<table>
<thead>
<tr>
<th></th>
<th>Cycling (N=77)</th>
<th>Running (N=79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO(_{2\text{INITIAL}}) (ml·min(^{-1}))</td>
<td>391 ± 114</td>
<td>480 ± 223</td>
</tr>
<tr>
<td>VO(_{2\text{GAIN}}) (ml·min(^{-1}))</td>
<td>1957 ± 633</td>
<td>2205 ± 635</td>
</tr>
<tr>
<td>T(_{\text{MEAN RESPONSE}}) (s)</td>
<td>49 ± 14</td>
<td>35 ± 10</td>
</tr>
<tr>
<td>T(_{\text{VO2max}}) (s)</td>
<td>226 ± 65</td>
<td>160 ± 48</td>
</tr>
<tr>
<td>T(_{\text{FATIGUE}}) (s)</td>
<td>347 ± 177</td>
<td>326 ± 115</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation
Individual regressions of $T_{\text{VO2max}}$ versus $T_{\text{FATIGUE}}$ were calculated for each participant, for each exercise mode. Group means for the slope and y-intercept were calculated. Because there was no reason to believe that the effects of mode and intensity would be different in men and women, means were not calculated separately by gender. For the group, the regressions ($T_{\text{VO2max}} = y$-intercept + (slope $\cdot T_{\text{FATIGUE}}$) were:

- **Treadmill**: $T_{\text{VO2max}} = 101 + (0.17 \cdot T_{\text{FATIGUE}})$
- **Cycle ergometer**: $T_{\text{VO2max}} = 144 + (0.26 \cdot T_{\text{FATIGUE}})$

Results of one-sample t-tests revealed that the slopes were significantly different from zero in both cycling ($t_{16} = 5.53, P < 0.01$) and in treadmill running ($t_{16} = 3.69, P < 0.01$). Results of one-sample t-tests revealed that the y-intercepts were also significantly different from zero in both cycling ($t_{16} = 8.26, P < 0.01$) and in treadmill running ($t_{16} = 6.02, P < 0.01$). Thus, the $T_{\text{VO2max}}$ was longer when the $T_{\text{FATIGUE}}$ was longer, meaning that the $\text{VO}_2$ response was faster at higher intensities.

Results of paired-means t-tests revealed that the slopes in cycling and treadmill running were not statistically different ($t_{16} = 1.29, P = 0.22$). In addition, paired-means t-tests indicated that the y-intercepts in treadmill running and cycling were not statistically different ($t_{16} = 1.72, P = 0.10$). Thus, the effect of intensity on the $\text{VO}_2$ response profile within the severe intensity domain appeared to be similar in cycling and treadmill running. Although the slope and y-intercepts for treadmill running and cycling did not differ statistically, there was
a tendency for both to be higher in treadmill running. Using each participant’s individual regression equations, \( T_{\text{VO}_2\text{max}} \) was calculated for each exercise mode using values for \( T_{\text{FATIGUE}} \) of 183 seconds and longer. The minimum \( T_{\text{FATIGUE}} \) of 183 was chosen because it was the \( T_{\text{UPPER BOUND}} \) for cycle ergometer exercise (see below). Although, the slopes and y-intercepts in treadmill running and cycling did not differ, results of paired-means t-tests revealed in every case that the \( T_{\text{VO}_2\text{max}} \) values were higher in cycling than in treadmill running. Although there was not a statistically significant effect of exercise mode on the y-intercept of the \( T_{\text{VO}_2\text{max}} - T_{\text{FATIGUE}} \) relationship, these results suggest that there was a mode effect on the \( \text{VO}_2 \) response profile, as reflected by \( T_{\text{VO}_2\text{max}} \). Results are presented in Appendix B.

For each participant, for each exercise mode, \( T_{\text{UPPER BOUND}} \), the unique time where \( T_{\text{FATIGUE}} = T_{\text{VO}_2\text{max}} \) (i.e., \( \text{VO}_2\text{max} \) is achieved at the point of fatigue), was calculated based on the individual relationships between \( T_{\text{VO}_2\text{max}} \) and \( T_{\text{FATIGUE}} \). In cycling, the group mean value was 183 ± 65 s and, in treadmill running, the value was 114 ± 48 s. These means were significantly different \((t_{16} = 3.82, P < 0.01)\). The calculation of \( T_{\text{UPPER BOUND}} \) is presented in Appendix C. Relationships between \( T_{\text{VO}_2\text{max}} \) and \( T_{\text{FATIGUE}} \) in treadmill running and cycling, along with the \( T_{\text{UPPER BOUND}} \), are graphically presented in Appendix D.

Another method of comparing the \( \text{VO}_2 \) responses in treadmill running and cycling was to match tests of similar duration. Each participant had a cycling test and a running test of ~200 s and ~400 s. Mean Values are presented in Table 4.
Table 4. Mean Values for Tests of Similar Duration

<table>
<thead>
<tr>
<th></th>
<th>Short Bike</th>
<th>Short Run</th>
<th>Long Bike</th>
<th>Long Run</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{\text{FATIGUE}}$ (s)</td>
<td>220 ± 33</td>
<td>215 ± 32</td>
<td>362 ± 51</td>
<td>357 ± 51</td>
</tr>
<tr>
<td>$T_{\text{MEAN RESPONSE}}$</td>
<td>43 ± 9</td>
<td>29 ± 7</td>
<td>53 ± 8</td>
<td>35 ± 5</td>
</tr>
<tr>
<td>$T_{\text{VO2max}}$</td>
<td>197 ± 40</td>
<td>133 ± 33</td>
<td>244 ± 37</td>
<td>162 ± 25</td>
</tr>
<tr>
<td>$T_{\text{@VO2max}}$</td>
<td>23 ± 37</td>
<td>82 ± 41</td>
<td>118 ± 54</td>
<td>195 ± 45</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation

Results of a two-way ANOVA with repeated measures across exercise mode (running, cycling) and exercise duration (200 s, 400 s) revealed significant main effects for exercise mode ($F_{1,16} = 80.28$, $P < 0.01$) and for test duration ($F_{1,16} = 85.55$, $P < 0.01$). The mode by duration interaction effect did not reach statistical significance ($F_{1,16} = 2.75$, $P = 0.12$).
CHAPTER 5
DISCUSSION AND CONCLUSIONS

The purpose of this study was to evaluate the effect of mode and intensity on VO₂ kinetics in the severe intensity domain. For each mode, participants performed fatiguing, constant power tests at powers or velocities selected to induce fatigue in 3 to 10 minutes. Participants performed as many tests as were necessary to complete one test for each mode with approximate durations of 3, 5 and 8 minutes. T\text{FATIGUE} and VO₂max were determined for each test. VO₂ responses were fit to a nonlinear regression equation to determine T\text{MEAN RESPONSE} and VO₂\text{GAIN}. T\text{VO2max} was calculated as \(4.6 \cdot T\text{MEAN RESPONSE}\), and time spent at VO₂max was determined by subtracting T\text{VO2max} from T\text{FATIGUE}. For each test, linear regression techniques were used to describe the relationship between the T\text{VO2max} and T\text{FATIGUE} and solve for T\text{UPPER BOUND}.

As expected, VO₂max was found to be higher in treadmill running than in cycling. This is similar to findings reported by Hermansen and Saltin (1969) and Martinez et al. (1993). It is generally accepted that muscle mass engaged in running is greater than muscle mass engaged in cycling (Hermansen & Saltin, 1969; Martinez et al., 1993). Faulkner, Roberts, Elk, and Conway (1971) and Hermansen, Ekblom, and Saltin (1970) believed that VO₂max values were lower in cycling due to a smaller cardiac output resulting from a smaller stroke volume.
(SV). Faulkner et al. (1971) believed the decrease in SV at maximal work is likely due to either an impaired venous return, insufficient diastolic filling time, or both. Twisting, kinking, or local compression of blood vessels as they pass through the working muscle may result in decreased venous return.

In the present study, VO$_{2\text{max}}$ was higher and the mean response time was faster in running. It seems unlikely that the mechanism responsible for the higher VO$_{2\text{max}}$ is exactly the same as the mechanism responsible for the faster kinetics. VO$_{2\text{max}}$ was 12% higher, but the $T_{\text{MEAN RESPONSE}}$ was 29% faster (35 versus 49 s). The faster response means that after 60 s of exercise, the running VO$_2$ was 1808 ml/min which is 82% of the VO$_{2\text{GAIN}}$ of 2205 ml/min. After 60 s of cycling, the VO$_2$ was 1389 ml/min which is 71% of the VO$_{2\text{GAIN}}$ of 1957 ml/min. If the ultimate VO$_{2\text{max}}$ (or gain) were the only factor, the percentage of gain covered would be the same at any given time in the exercise (and there would be no difference in $T_{\text{MEAN RESPONSE}}$).

Kinetics were faster at higher intensities in both cycling and treadmill running. This was especially evident when an ANOVA was used to compare the results of ~200 s tests with those of ~400 s tests. The same conclusion was supported by the significant positive slopes of the $T_{\text{VO2max}}$-$T_{\text{FATIGUE}}$ relationships in both treadmill running and cycling. VO$_{2\text{max}}$ was reached faster but sustained for a shorter period of time in the shorter tests (at higher intensities). In shorter cycling tests, $T_{\text{VO2max}}$ had a mean value of 197 s versus 244 s in longer tests. VO$_{2\text{max}}$ was sustained for a mean value of 23 s in the short tests and 118 s in the longer
tests. Similar results were found comparing relatively short and long treadmill running tests. VO$_{2\text{max}}$ was reached in an average of 133 s in the short tests versus 162 s in the longer tests. VO$_{2\text{max}}$ was sustained for a mean value of 82 s in the short tests and 195 s in the longer tests. These results are consistent with previous findings for cycle ergometer and treadmill exercise. In two studies evaluating responses in the severe intensity domain, T$_{VO2\text{max}}$ was found to be faster at higher intensities for both treadmill running (Hill & Ferguson, 1999) and cycle ergometer exercise (Hill & Smith, 1999). Betik et al. (1998) reported that the tau of the VO$_2$ response was faster at 130% of VO$_{2\text{max}}$ than at 100% of VO$_{2\text{max}}$. Whipp (1994) stated that the time to achieve VO$_{2\text{max}}$ (T$_{VO2\text{max}}$) is shorter (VO$_{2\text{max}}$ is achieved faster) at higher intensities. Hill et al. (unpublished) described a relationship between power and VO$_2$ kinetics over a range of intensities within the severe domain, where T$_{\text{MEAN RESPONSE}}$ was faster at higher work rates.

The physiological mechanisms that result in faster kinetics at higher intensities are not clear. The results of this and other aforementioned studies suggest that the VO$_2$ response is driven by O$_2$ demand (even if it is ultimately limited by VO$_{2\text{max}}$). It is possible there is some feedback from the muscles to accelerate the response of the cardiovascular system. Perhaps the increased ATP breakdown (and release of metabolic intermediates) enhances local vasodilation. Alternatively, the feedback may have a central effect, and stimulate cardiovascular kinetics.
In the present study, kinetics were faster in treadmill running than in cycling. This was clearly indicated by the results of the two-way ANOVA. In addition, $T_{\text{VO2max}}$ calculated using the regression equations for cycling ($T_{\text{VO2max}} = 101 + (0.17 \cdot T_{\text{FATIGUE}}$) and treadmill running ($T_{\text{VO2max}} = 144 + (0.26 \cdot T_{\text{FATIGUE}}$), for all $T_{\text{FATIGUE}} > 183$ s, were higher in running than cycling. It is noted, however, that the difference between the y-intercepts of the $T_{\text{VO2max}}$–$T_{\text{FATIGUE}}$ relationships for treadmill running (144 s) and cycling (101 s) did not reach statistical significance.

If muscle mass engaged in running is greater than that engaged in cycling, there may be a greater demand for O$_2$ in running. The greater muscle mass may permit relatively greater O$_2$ extraction.

It has been reported that kinetics are faster in walking than in cycling (Chilibeck et al., 1996) and faster in leg cycling than in arm cranking (Koga et al., 1996). The purpose of the present study was to compare kinetics in running and cycling. Chilibeck et al. (1996) suggested that kinetics are slowed in exercise modes to which an individual is not accustomed, and that the slowed kinetics are associated with muscle recruitment patterns. Koga et al. (1996) also suggested that a contributing factor to slowed kinetics in arm exercise is the untrained state of arm muscle relative to leg muscle in the average person.

In the present study, kinetics were “slowed” in cycling compared to treadmill running. Informal interviews with the participants of this study suggested they were at least as accustomed to cycling as to running. The
difference in VO$_{2\text{max}}$ was 12% which is consistent with other studies (Hermansen & Saltin, 1969; Martinez et al., 1993) and indicates that prior experience had little effect on the VO$_2$ responses.

Koga et al. (1996) speculated that the slower kinetics in arm cranking may be due to a delayed increase in cardiac output, higher glycolysis occurring early during arm exercise, and greater transient lactate production in the smaller muscle mass of the arm. They found VO$_2$ $T_{\text{MEAN RESPONSE}}$ was significantly longer in arm exercise than leg exercise at the same relative and absolute work rate. At 50 W, $T_{\text{MEAN RESPONSE}}$ was 57 ± 8 s in arm exercise and 23 ± 3 s in leg exercise. The difference in active muscle mass is far greater for arm-versus-leg exercise than for cycling-versus-running. In the present study, $T_{\text{MEAN RESPONSE}}$ was 49 ± 14 s in cycling and 35 ± 10 s in treadmill running.

Faster kinetics in running versus cycling may be explained by a different muscle contraction regimen. Perhaps the response is slowed in cycling due a compression of vessels that doesn’t occur in running. Martinez et al. (1993) speculated that, during maximal cycling, high intramural pressures may occur which restrict the muscle blood flow to that area. They found that peak tension developed during each pedal thrust was higher in maximal cycling than in running. This tension difference implies a greater fast-twitch fiber recruitment and, hence, a greater anaerobic contribution. Faulkner et al. (1971) also suggested that biomechanical factors may contribute to greater impairment in skeletal muscle blood flow in cycling than in running. They explained that the
contraction portion of the contraction-relaxation cycle is prolonged in cycling. Running, in contrast, is more of a ballistic movement with a very short contraction phase and less impairment to muscle blood flow.

In summary, the purpose of this study was to evaluate the effects of intensity and mode on VO₂ kinetics during exercise at intensities in the severe intensity domain. First, across the range of severe intensities used in the present study, the VO₂ responded faster (i.e., T\text{MEAN RESPONSE} was shorter) in higher intensity exercise. This influence of intensity on kinetics was evidenced by the results of the two-way ANOVA, which revealed a significant main effect of intensity, and by the fact that the slopes of the relationships between T\text{VO₂max} and T\text{FATIGUE} were positive for both running and cycling. Second, within the range of intensities used in the present study, the VO₂ responded faster in running than in cycling. This influence of exercise mode on kinetics was evidenced by the results of the two-way ANOVA, which revealed a significant main effect of mode, and by the fact that T\text{UPPER BOUND} was shorter in running than in cycling, and by the fact that the calculated values for T\text{VO₂max} were shorter in running than cycling for all intensities for which T\text{FATIGUE} equaled or exceeded 183 s, the T\text{UPPER BOUND} for cycling. In conclusion, the VO₂ response profile during severe intensity exercise is faster for running than cycling and faster at higher intensities within the severe intensity domain.
APPENDIX A

PARTICIPANT INFORMED CONSENT
UNIVERSITY OF NORTH TEXAS

DEPARTMENT OF KINESIOLOGY, HEALTH PROMOTION, AND
RECREATION

CONSENT TO ACT AS A HUMAN SUBJECT

Subject's Name (print): ____________________________ Date:_____________

1. I hereby volunteer to participate as a subject in laboratory testing. I understand that this testing is part of a study titled "Defining the severe exercise intensity domain." The purpose of this study is to use mathematical modeling of responses to cycle ergometer exercise in order to predict the lowest and highest exercise intensities that will elicit a maximal aerobic response.

I hereby authorize David W. Hill and/or assistants as may be selected by him to perform on me the following procedures, on different days:

a) to have me run on a motorized treadmill until I feel I cannot continue (7 times, once per day on 7 different days); [I understand that it is up to me to try to continue as long as I can, and that the tests are at different work rates and they will last approximately 1 minute to 12 minutes];

b) to have me pedal at a fixed work rate on a cycle ergometer until I feel I cannot continue (7 times, once per day on 7 different days); [I understand that it is up to me to try to continue as long as I can, and that the tests are at different work rates and they will last approximately 1 minute to 12 minutes];

I understand that during all exercise tests I will be breathing through a mouthpiece and that my nose will be pinched shut.

1. The procedures outlined in paragraph 1 [(a) and (b), above] have been explained to me by David W. Hill.

2. I understand that the procedures involve the following risks and discomforts: temporary muscle pain is expected during exercise, and nausea and lightheadedness is common after exercise; there is the possibility of abnormal changes in my heart beat or blood pressure or even of a heart attack during the tests. However, I understand that heart rate, blood pressure, and EKG will be taken before testing, that my cardiovascular and metabolic responses to exercise will be monitored throughout all tests, and that I can terminate any test at any time at my discretion.
3. I have been advised that the following benefits will be derived from my participation in this study: aside from learning about my aerobic and anaerobic fitness level, there are no direct benefits to me.

4. I understand that David W. Hill and/or appropriate assistants as may be selected by him will answer any inquiries that I may have at any time concerning these procedures and/or investigations.

5. I understand that all data concerning myself will be kept confidential and available only upon my written request. I further understand that in the event of publication, no association will be made between the reported data and myself.

6. I understand that there is no monetary compensation for my participation in this study.

7. I understand that, in the event of physical injury directly resulting from participation in this study, compensation cannot be provided. Medical treatment will be available at the University Health Center for UNT students; the laboratory has an outside telephone line to contact emergency medical services (911).

8. I understand that I may terminate participation in this study at any time without prejudice to future care or any possible reimbursement of expenses, compensation, employment status, or course grade, and that, owing to the scientific nature of the study, the investigator may terminate the procedures and/or investigation at any time.

9. I understand that I may contact the chairperson of the KHPR Committee on the Use of Human Subjects, Dr. Noreen Goggin (PEB 112, 940-565-2212) on any matters concerning my participation in this study if I feel that there is infringement on my rights.

Subject’s Signature: ______________________________________________________

Witness: ___________________________________________________ Date: ____________

This project has been reviewed by the University of North Texas Institutional Review Board for the Protection of Human Subjects in Research (940-565-3940)
APPENDIX B

RESULTS OF $T_{\text{UPPER BOUND}}$

PAIRED-MEANS TESTS
Calculation of $T_{VO2max}$ with $T_{FATIGUE} \geq T_{UPPER \ BOUND}$

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>($N=17$)</td>
</tr>
<tr>
<td>Cycling $T_{VO2max}$ with $T_{FATIGUE} = 183$ s</td>
<td>$192 \pm 46$</td>
</tr>
<tr>
<td>Running $T_{VO2max}$ with $T_{FATIGUE} = 183$ s</td>
<td>$132 \pm 42$</td>
</tr>
<tr>
<td>Cycling $T_{VO2max}$ with $T_{FATIGUE} = 300$ s</td>
<td>$222 \pm 39$</td>
</tr>
<tr>
<td>Running $T_{VO2max}$ with $T_{FATIGUE} = 300$ s</td>
<td>$152 \pm 33$</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation

Paired Samples Test

<table>
<thead>
<tr>
<th></th>
<th>$t_{16}$</th>
<th>Sig. (2-tailed)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pair 1 Cycling $T_{VO2max}^{183}$ – Running $T_{VO2max}^{183}$</td>
<td>4.07</td>
<td>$P = .001$</td>
</tr>
<tr>
<td>Pair 1 Cycling $T_{VO2max}^{300}$ – Running $T_{VO2max}^{300}$</td>
<td>6.48</td>
<td>$P = .000$</td>
</tr>
</tbody>
</table>

* $P < 0.01$
APPENDIX C

CALCULATION OF $T_{UPPER BOUND}$
For each participant, for each exercise mode, $T_{\text{UPPER BOUND}}$, the unique time where $T_{\text{FATIGUE}} = T_{\text{VO2max}}$ was calculated based on the individual relationships between $T_{\text{VO2max}}$ and $T_{\text{FATIGUE}}$.

At $T_{\text{UPPER BOUND}}$, :

\[ T_{\text{VO2max}} = T_{\text{FATIGUE}} = T_{\text{UPPER BOUND}} = \]

\[ T_{\text{VO2max}} = y\text{-intercept} + (\text{slope} \cdot T_{\text{FATIGUE}}) \]

Therefore,

\[ T_{\text{UPPER BOUND}} = y\text{-intercept} / (1 - \text{slope}) \]

For example, an individual’s $y$-intercept and slope for cycling are 121 and 0.25 respectively

\[ T_{\text{VO2max}} = 121 + (0.25 \cdot T_{\text{FATIGUE}}) \]

\[ T_{\text{UPPER BOUND}} = 121 / (1 - 0.25) \]

\[ T_{\text{UPPER BOUND}} = 121 / 0.75 \]

\[ T_{\text{UPPER BOUND}} = 161 \]
APPENDIX D

RELATIONSHIPS BETWEEN $T_{\text{VO2max}}$, $T_{\text{FATIGUE}}$ and $T_{\text{UPPER BOUND}}$
REFERENCES


Hill, D.W., Poole, D.C., & Smith, J.C. The relationship between power and the time to achieve VO$_{2\text{max}}$. Unpublished.


