



VO₂max during successive maximal efforts

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Carl Foster · Erin KuVel · Nicole Bradley · **Rebecca A. Battista** · Glenn Wright · John P. Porcari ·
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The concept of VO₂max has been a defining paradigm in exercise physiology for >75 years. Within the last decade, this concept has been both challenged and defended. The purpose of this study was to test the concept of VO₂max by comparing VO₂ during a second exercise bout following a preliminary maximal effort exercise bout. The study had two parts. In Study #1, physically active non-athletes performed incremental cycle exercise. After 1-min recovery, a second bout was performed at a higher power output. In Study #2, competitive runners performed incremental treadmill exercise and, after 3-min recovery, a second bout at a higher speed. In Study #1 the highest VO₂ (bout 1 vs. bout 2) was not significantly different (3.95 + 0.75 vs. 4.06 + 0.75 l min⁻¹). Maximal heart rate was not different (179 + 14 vs. 180 + 13 bpm) although maximal VE was higher in the second bout (141 + 36 vs. 151 + 34 l min⁻¹). In Study #2 the highest VO₂ (bout 1 vs. bout 2) was not significantly different (4.09 + 0.97 vs. 4.03 + 1.16 l min⁻¹), nor was maximal heart rate (184 + 6 vs. 181 + 10 bpm) or maximal VE (126 + 29 vs. 126 + 34 l min⁻¹). The results support the concept that the highest VO₂ during a maximal incremental exercise bout is unlikely to change during a subsequent exercise bout,

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despite higher muscular power output. As such, the results support the “classical” view of VO₂max.

Keywords Exercise testing · Oxygen uptake · Cycle exercise · Treadmill exercise

Introduction

The concept of the maximal oxygen uptake (VO₂max) has been one of the defining paradigms in exercise physiology for >75 years, ever since the classical work of Hill and Lupton (1923). In the 1950s Mitchell et al. (1958) and Taylor et al. (1955) provided support for the concept of a plateau in VO₂ during heavy exercise by demonstrating a slowing of the increase in VO₂ (to <50% of the previous rate of increase) with progressive work. The wide acceptance of their work formalized the concept of VO₂max as an international reference for cardiorespiratory fitness (Shephard et al. 1968). During the next generation, the concept that VO₂max was limited by the ability of the central circulation to “offer O₂” to the peripheral tissues became the primary line of thinking about VO₂max (Saltin 1985, di Prampero 2003), an issue that remains at the forefront of physiological discussions (Gonzalez-Alonzo et al. 2003). Slightly more than a decade ago, Noakes (1997, 1998) presented a systematic challenge of the underlying concept of VO₂max, and has continued (Noakes et al. 2004) to argue that the concept is based on a faulty interpretation of the original data of Hill and Lupton (1923), and is indeed biologically implausible. One of Noakes’ central arguments against the concept of the plateau phenomenon, is that in the early experimental studies the subjects did not perform a subsequent exercise stage with a greater O₂ requirement while demonstrating either no change or an inappropriately small change in VO₂, a neces-

sary step in demonstrating the validity of the plateau phenomenon. Given that Noakes' arguments have meaningfully challenged a fundamental paradigm in exercise physiology, it is reasonable that a number of papers have been published defending the plateau dependent concept of $\dot{V}O_2\text{max}$ (Bassett and Howley 1997, 2000; Bergh et al. 2000). Recent experimental studies using incremental exercise bouts followed by a subsequent higher intensity exercise bout, have demonstrated no change in $\dot{V}O_2$ despite performing a heavier workload (Day et al. 2003; Rossiter et al. 2006; Snell et al. 2007). These studies have provided important support for the "classical" concept of $\dot{V}O_2\text{max}$. However, given the importance of these recent findings to the central paradigm of $\dot{V}O_2\text{max}$, we felt that it was worthwhile to replicate this repeat exercise bout model with multiple approaches to the exercise stimulus. We hypothesized that there would be no change in $\dot{V}O_2$ during a second exercise bout following a preliminary incremental maximal effort test, despite a higher rate of external muscular work.

Methods

The study was conducted in two parts: Study #1 used cycle ergometer exercise with a very short recovery period between the first and second bouts; Study #2 used treadmill exercise with a longer recovery period between the first and second bouts. The subjects for Study #1 were healthy volunteers ($N = 20$, $m = 16$, $f = 4$). All were physically active, although none were athletes systematically training for competition. The subjects for Study #2 were well-trained athletes ($N = 20$, $m = 12$, $f = 8$), members of a university cross country team or local triathletes. Descriptive characteristics are presented in Table 1. All subjects provided written informed consent prior to participation and the protocol had been approved by university human subjects committee.

Study #1

Exercise was performed on an electrically braked cycle ergometer (Lode Excalibur, Groningen, NL, USA). Following a general warm-up, exercise was begun at a power

output of 25 W and was increased by 25 W every minute. Exercise continued until the subjects could not maintain a pedaling rate within the range of 60–90 rpm. The power output was immediately reduced to 25 W, and the subject continued pedaling for 60 s. At this point, the power output was increased to 25 W greater than that during the last stage (regardless of whether the subject had completed the full 60 s of the stage), and the subject exercised until they could no longer maintain pedaling within the range of 60–90 rpm. At this point, the power output was reduced to 25 W and the study was concluded. In order to ensure that there was no question about whether the primary exercise bout was "maximal", only subjects in whom the rate of increase in $\dot{V}O_2$ during the last minute was <50% of the rate of increase during the mid portion of the test [e.g. meeting widely accepted criteria (Howley et al. 1995)] were included in the analysis.

Study #2

After a general warm-up and habituation to treadmill running, the subjects performed incremental exercise to fatigue on a motor driven treadmill. Because of speed limitations on the treadmill, the grade was set at 4% for the male subjects and 3% for the female subjects. The initial speed was 2.23 m s^{-1} , and increased by 0.45 m s^{-1} every 3-min until the subject indicated they could no longer continue. At this point, the speed/grade of the treadmill was decreased to $0.89 \text{ m s}^{-1}/0\%$ grade for 3 min. At the end of this recovery period, and depending on the investigator's assessment of the subject's relative recovery, the grade was increased to 4 or 3% for males or females respectively, and the speed was increased to either 0.22 or 0.45 m s^{-1} greater than the velocity during the last stage. This speed was maintained for 3-min or until the subject indicated they were unable to continue. The speed and grade of the treadmill were then decreased to 0.89 m s^{-1} and 0% for 3-min. According to protocol, the subjects could have performed an additional stage with a similar progression strategy. However, none were willing to start an additional stage. Unlike Study #1, there was no selection based on a slowing of the rate of increase in $\dot{V}O_2$ during the primary bout.

Table 1 Characteristics of the subjects (mean \pm SD)

Study	1 (males, $n = 16$)	1 (females, $n = 4$)	2 (males, $n = 12$)	2 (females, $n = 8$)
Age (year)	31.5 ± 14.5	28.0 ± 12.4	21.6 ± 3.0	21.0 ± 4.5
Height (cm)	181 ± 6	170 ± 4	178 ± 5	168 ± 5
Body mass (kg)	78.6 ± 8.5	62.3 ± 6.4	72.7 ± 7.3	58.0 ± 7.8
$\dot{V}O_2\text{max}$ bout 1 (l min^{-1})	$3.95 \pm 0.75^*$		$4.09 \pm 0.97^*$	
$\dot{V}O_2\text{max}$ bout 2 (l min^{-1})	$4.06 \pm 0.75^*$		$4.03 \pm 1.16^*$	
HRmax bout 1	$179 \pm 14^*$		$184 \pm 6^*$	
HRmax bout 2	$180 \pm 13^*$		$181 \pm 10^*$	

For maximal $\dot{V}O_2$ and HR, the results of male and female subjects are combined. None of the differences between bout 1 and bout 2 were significantly different ($P < 0.05$).

Respiratory gas exchange was measured using open circuit spirometry using an automated metabolic cart (Applied Electrochemistry Inc., Pittsburgh, PA, USA). Pulmonary ventilation was measured using a turbine, calibrated against a 3 l syringe. Expired gas concentrations were continuously sampled from a mixing chamber. The gas analyzers were calibrated against a reference gas verified by micro-Schölander and room air. Gas analysis was integrated over 30 s.

Statistical comparisons were made between the highest VO_2 (30 s) during the primary incremental exercise bout, and the highest VO_2 (30 s) during the second exercise bout using repeated measures ANOVA. For comparison purposes, the ventilation ($V_{E\max}$) and heart rate (HR_{\max}) during the highest VO_2 samples were also compared using repeated measures ANOVA. A $P < 0.05$ was accepted as statistically significant.

Results

In Study #1, the highest VO_2 during the first and second exercise bouts (regardless of whether it occurred during the last 30 s or earlier) was not significantly different (3.95 ± 0.75 vs. 4.06 ± 0.75 l min^{-1}) (Table 1). This is rejected in the serial VO_2 responses (Fig. 1) which rejects the changes during each 30 s interval during the first and second exercise bouts. In the serial data, the subjects did not all exercise for exactly 2 min into the second exercise bout (range 90–210 s). In order to make the data more easily understandable, the data in the second exercise bout were plotted backwards from end exercise during the last 2 min of exercise for each subject. The HR_{\max} during the two exercise bouts was not significantly different (179 ± 14 vs. 180 ± 13 bpm). The $V_{E\max}$ was significantly larger in the second exercise bout (141 ± 36 vs. 151 ± 34 l min^{-1}).

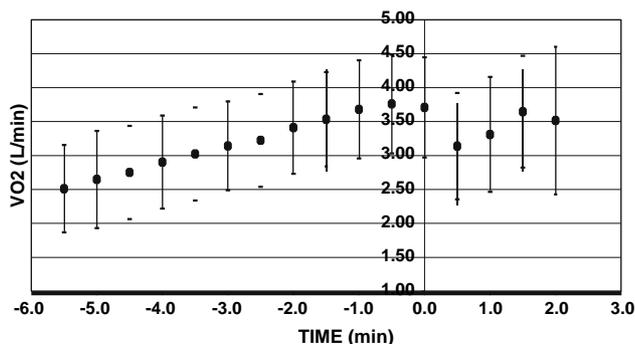


Fig. 1 Serial VO_2 responses during Study #1. To promote individual comparability, the results of the primary exercise bout are plotted backwards from the end of the W_{rst} exercise bout (time = 0.0), and the results of the second exercise bout are plotted backwards from an arbitrary time of 2.0 min, regardless of how long the subject actually continued during the second exercise bout

In Study #2, the highest VO_2 in the first and second exercise bouts (regardless of whether it occurred during the last 30 s or earlier) was not significantly different (4.09 ± 0.97 vs. 4.03 ± 1.16 l min^{-1}). This is rejected in the serial VO_2 responses (Fig. 2) which reject changes during each 30 s interval. As with Study #1, the data in the second bout have been plotted backward from end exercise during the last 2 min of exercise for each subject. The HR during the two exercise bouts was not significantly different (184 ± 6 vs. 181 ± 10 bpm), nor was the maximal V_E (126 ± 29 vs. 126 ± 34 l min^{-1}).

Discussion

The major finding of these studies was that the VO_2 during a second exercise bout, at a higher workload than that causing fatigue during a primary incremental bout, was not different than observed during the primary bout, in both running and cycling. As such, the results support our experimental hypothesis and refute one of the primary arguments by Noakes (1997, 1998) against the classical findings of Hill and Lupton (1923), on which the central paradigm of $VO_{2\max}$ is based. The present results are in agreement with similar studies published recently (Day et al. 2003; Rossiter et al. 2006; Snell et al. 2007), using variations of the experimental design used in this study.

The results are also supported by previous work from our laboratory indicating that the highest VO_2 during a 1-mile running time trial was not different than $VO_{2\max}$ measured during incremental treadmill running (Crouter et al. 2001). However, other observations from our laboratory have shown that the highest VO_2 during cycle time trials may be higher than the $VO_{2\max}$ during incremental cycle exercise, in either competitive athletes (Foster et al. 1993)

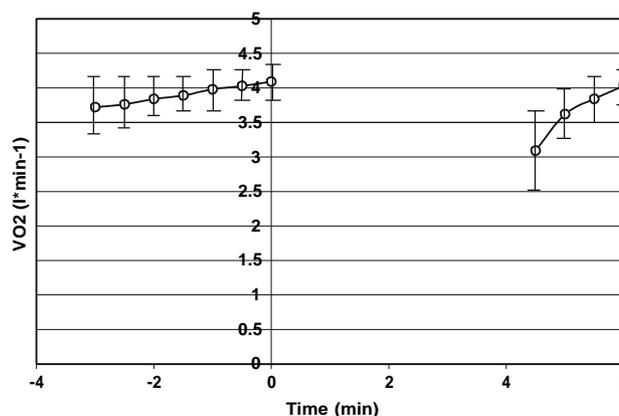


Fig. 2 Serial VO_2 responses during Study #2. To promote individual comparability, the results of the primary exercise bout are plotted backwards from the end of the W_{rst} exercise bout (time = 0.0), and the results of the second exercise bout are plotted backwards from an arbitrary time of 6.0 min

or trained non-athletes (Foster et al. 1997). To account for this, we were conservative in our selection of observations during Study #1, selecting only subjects who could demonstrate a plateau during the primary exercise bout, a finding that is normally observed only observed in about 15% of non-athletes (Day et al. 2003) and 50% of athletes (Doherty et al. 2003; Lucia et al. 2006). The data of Rossiter et al. (2006) suggest that the response may be the same regardless of whether the subjects actually demonstrate a plateau.

We varied the recovery interval in the two studies. In Study #1, the recovery period was only 1 min. This had the advantage of not allowing $\dot{V}O_2$ to decrease very much from the value at the end of the primary exercise bout, but the disadvantage of not allowing adequate time for metabolites to clear from the active musculature, for restoration of normal levels of muscle phosphagens or recovery of short term muscle power output, particularly during active recovery as was done in Study #1 (Spencer et al. 2006; Dupont et al. 2007). In the second study, we allowed a 3 min recovery period, which presented an opposite set of advantages and disadvantages. What is remarkable is that regardless of whether the recovery period is very short (1 min) as in Study #1, intermediate as in the data reported by Day et al. (2003) (2 min) or in Study #2 (3 min), or very long ($\gg 24$ h) (Snell et al. 2007) there is no evidence of difference in the $\dot{V}O_2$ during the first and second exercise bouts. These findings suggest that one of the primary objections (Noakes 1997, 1998) to the classic work of Hill and Lupton (1923), that Hill and Lupton did not perform an additional exercise bout, is not a critically relevant objection to the overall concept which developed from the work of Hill and Lupton (1923).

An underlying element in the arguments of Noakes (1997, 1998) is that a proposed central governor acts to protect the exerciser from creating an *internal milieu* that might be capable of causing injury or death. A central prediction of this model is that neural output is reduced as fatigue is approached, rather than the muscles becoming less responsive to neural output (Ansley et al. 2004; St Clair Gibson et al. 2001a, b). However, recent data from our laboratory (Hettinga et al. 2006) and elsewhere (Brink-Elfegoun et al. 2007b) demonstrates that neural output is increased during periods when power output is falling or $\dot{V}O_2$ is unchanging. We have interpreted these findings as evidence supporting a peripheral regulation of fatigue (Hettinga et al. 2006). This peripheral limitation is presumably related to either inorganic phosphate accumulation (Westerblad et al. 2002) or proton accumulation and phosphagen depletion (Karlsson and Saltin 1970) in the muscles, secondary to a failure of aerobic metabolism to provide for adequate ATP regeneration. Recent studies have shown, using an arm + leg model of maximal exercise, that $\dot{V}O_{2max}$ and cardiac output do not increase during supramaximal exercise despite a 10%

increase in muscular power output (Brink-Elfegoun et al. 2007a). These data have been interpreted as supporting the hypothesis that $\dot{V}O_2$ during heavy exercise is limited by central circulatory ability. Accepting that there are limited and conflicting data regarding downregulation of neural outflow during fatiguing exercise (Ansley et al. 2004; StClair Gibson et al. 2001; Hettinga et al. 2006), the present results can be taken to suggest that fatigue is likely associated with a failure of the muscles to respond to neural outflow as intermuscular conditions approach some critical degree of disturbance in homeostasis. Efferent feedback from such homeostatic disturbance, perhaps an increasing muscle fiber recruitment to maintain power output, and/or in the venous effluent to the circulation may still provide a sense of fatigue and cause the subject to stop exercising well before the disturbance in homeostasis reaches a level capable of causing damage to the organism. Recent evidence suggests that the likelihood of observing a plateau during heavy exercise is related to the pattern of lactate accumulation (Lacour et al. 2007). Individuals with a delayed pattern of lactate accumulation (implying a smaller disturbance in intermuscular conditions) during the sub-maximal portion of an incremental exercise test, are more likely to demonstrate a plateau of $\dot{V}O_2$. This suggests that sparing intermuscular homeostasis delays achieving a potentially critical disturbance in homeostasis within the muscle to the point where O_2 transport can become limiting. In any case, the usual experience of individuals at the end of maximal incremental tests is that they do not want to continue exercising, rather than that they are incapable of continuing exercise. St Clair Gibson et al. (2001a, b) and Amann et al. (2006) have demonstrated that the musculature is capable of responding to external stimuli even at the end of nominally maximal efforts. Thus, it can be argued that the sense of fatigue and the failure to continue may be just as reasonably attributed to a conscious unwillingness to continue exercise as to a subconscious downregulation of neural output.

One of the unacknowledged issues in the discussion about whether or not $\dot{V}O_{2max}$ represents a central parameter during exercise is that incremental exercise is an activity that humans never ordinarily do. It represents a variation of an open-loop exercise model, requiring the subject to continue exercising at a fixed workload for an indefinite amount of time. In their elegant presentation of the concept of a central governor, Noakes and St Clair Gibson (2004) and Lambert et al. (2004) have argued primarily from the standpoint of how fatigue is experienced during exercise involving a deWned task (e.g. running a race). In this case, the individual has some knowledge of the task demand from previous experience, has a plan (e.g. template) for executing the task, and modiWes the execution of the template based on eVerent feedback from a variety of

receptors. As a result, there is little evidence of the physiological catastrophe that Noakes and St Clair Gibson (2004) have argued is a central prediction of the physiological model grounded in the concept of VO_2max . However, although there is little evidence for catastrophic outcomes, there are clearly great disturbances in homeostasis at the end of a fatiguing exercise bout. In time trial exercise, where the individual only has to optimize their performance of the task, there is little evidence of competitive catastrophes (e.g. sudden, massive and uncontrolled decreases in power output) (Foster et al. 2003, 2004) as changes in power output often seem to be strategic (Rauch et al. 2005). In head to head competitive events, amongst more or less equal competitors, there are frequent examples of competitive catastrophes (sudden, massive, and uncontrolled decreases in power output, e.g. as when a rider suddenly drops back from the leading group during a mountain ascent during the Tour de France). However, with the exception of heat injuries, these departures from a controlled pattern of energy expenditure are not usually threatening to the organism, which satisfies a fundamental prediction of the central governor model, that competitive catastrophes are designed to protect the organism from achieving potentially dangerous disturbances in homeostasis. There is ample evidence that fatiguing exercise is scaled to the task (Eston et al. 2007), and that the willingness to exercise is influenced, both by a pre-exercise anticipation of the task to be performed, by the percent of the perceived task remaining to be completed, and by feedback from various internal receptors. Thus, the intrinsic nature of incremental exercise tests may to some degree foster the emergence of a “catastrophic” end point, at which the individual simply becomes unwilling to further exert themselves. That this might occur shortly after the athlete loses the ability to maintain homeostasis relative to phosphagen concentrations because aerobic ATP generation has reached its’ limits, in our view agrees with the concept that the proposed central governor is acting to protect the organism. However, rather than the action of the central governor being subconscious, via downregulation of motor outflow, the control appears to operate at a higher level, perhaps via the internal dialogue that has been proposed by St Clair Gibson and Foster (2007).

In summary, the results of the present studies of a higher intensity exercise bout following a primary fatiguing exercise bout, resulting in no increase over the VO_2 observed during the primary exercise bout, supports the “classical” view of VO_2 max first suggested by Hill and Lupton (1923), and supported in several recent experimental studies (Day et al. 2003; Rossiter et al. 2006; Snell et al. 2007). Taken together, these several studies support the idea that a proposed central governor designed to protect the organism from unreasonable disturbances in homeostasis may not be

employed until after the capacity of the oxygen transport system has been exceeded.

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