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This study examined the effect of high-intensity interval training on the VO2 response during severe, constant-load exercise. Prior to, and following training, ten females (VO\textsubscript{2}\text{peak} 37.4 ± 6.0 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) performed a graded exercise test to determine VO\textsubscript{2}\text{peak} and lactate threshold (LT) and a 6-min cycle test (CT) at the pre-training VO\textsubscript{2}\text{peak} intensity. Training involved high-intensity intervals (2 min work, 1 min rest) performed 3 x week for 8 weeks. Breath-by-breath data from 0 to 6 min during the CT were smoothed using 5-s averages and fit to a bi-exponential model starting from 20 s. Training resulted in significant improvements in VO\textsubscript{2}\text{max} (2.34 ± 0.37 – 2.78 ± 0.30 L·min\textsuperscript{-1}), power at VO\textsubscript{2}\text{max} (170 ± 26 – 204 ± 25 W) and power at LT (113 ± 17 – 136 ± 20 W) (p<0.05). Following training, the VO\textsubscript{2} response showed a significant increase in the amplitude of the primary phase (A\textsubscript{1}) (1396 ± 103 – 1695 ± 100 mL·min\textsuperscript{-1}; p<0.05) and end-exercise VO\textsubscript{2} (VO\textsubscript{2}\text{EE}), with no difference (p>0.05) in the time constants of either phase or the amplitude of the slow component (318 ± 67 – 380 ± 48 mL; p= 0.15). In conjunction, accumulated oxygen deficit (AOD) (43.7 ± 9.8 – 17.2 ± 2.8 mL O\textsubscript{2} eq·kg\textsuperscript{-1}) and anaerobic contribution to the CT (19.4 ± 4.4 – 7.2 ± 1.2 %) were significantly reduced. In contrast to previous moderate-intensity research, a high-intensity interval training program increased A\textsubscript{1} and VO\textsubscript{2}\text{EE} for the same absolute exercise intensity, decreasing the AOD during a severe-intensity CT.

**Keywords:** AOD, high-intensity exercise, primary phase amplitude, slow component, VO\textsubscript{2} kinetics

**Introduction:**
It is well documented that the characteristics of the on-transient VO\(_2\) response to step-changes in exercise are exercise intensity specific \([1,2]\). The VO\(_2\) response to moderate, constant-load exercise below the lactate threshold (LT), following an initial cardio-dynamic phase, increases in a mono-exponential fashion until a steady-state is reached. For exercise above the LT, following the cardio-dynamic and primary phases, the VO\(_2\) response continues to rise exponentially either delaying the attainment of steady-state or continuing to rise until VO\(_2\) \(_{\text{peak}}\) or exercise termination, termed the slow component (SC) \([2,3]\). While the description of the characteristics of the VO\(_2\) response to supra-VO\(_2\) \(_{\text{peak}}\) exercise intensities (associated with or above VO\(_2\) \(_{\text{peak}}\)) has received growing attention \([4]\), the effect of high-intensity training on the VO\(_2\) response characteristics of severe-exercise intensities is less well documented.

Numerous studies have reported the effects of both sub-maximal interval \([5,6,7]\) and continuous \([8,9,10]\) endurance training (below VO\(_2\) \(_{\text{peak}}\)) on the VO\(_2\) response to constant-load exercise intensities that correspond to, at most, 80% of the difference (6) between the LT and VO\(_2\) \(_{\text{peak}}\) \([9]\). Following both continuous and interval sub-maximal endurance training, research has reported a faster VO\(_2\) response (representing muscle \(\text{O}_2\) uptake), as measured by tau (\(\tau\)) during the primary phase \([11,12,13]\), either no change or a reduced end-exercise VO\(_2\) (VO\(_2\) \(_{\text{EE}}\)) \([6,8,14]\) and either an unchanged or attenuated SC \([6,8,15]\). However, there is a paucity of reported data on the effects of training at VO\(_2\) \(_{\text{peak}}\), as research attention has focussed on training programs that elicit VO\(_2\) responses below VO\(_2\) \(_{\text{peak}}\).
Currently, only Demarle et al. [16] and Billat et al. [17] have reported the effect of exercise training intensities approaching $V\dot{O}_2$ peak on the $V\dot{O}_2$ response to constant-load, severe-intensity exercise. While both studies incorporated training exercise intensities that were still below $V\dot{O}_2$ peak ($60 – 90\% V\dot{O}_2$ peak), the exercise intensities of their constant-load tests were much closer to $V\dot{O}_2$ peak than previous research ($90 – 95\% V\dot{O}_2$ peak). Both studies reported a decrease in the primary phase $\tau$, while neither study reported any significant change in $V\dot{O}_2$ EE following training. Furthermore, both studies reported a decrease in the accumulated oxygen deficit (AOD) of the constant-load exercise test post-training ($p<0.05$). No other significant differences in the primary phase amplitude, SC or time delays were evident after training in either study.

While both studies extend our understanding of the effects of high-intensity training on the $V\dot{O}_2$ response to severe-exercise, to date research is limited on the effects of training at or above $V\dot{O}_2$ peak on the $V\dot{O}_2$ response to such intensities. This is surprising, as there are many athletic events that are performed above $V\dot{O}_2$ peak intensity and incorporate severe-intensity interval training sessions within the training program. Particularly where high intensity exercise (from the start of exercise) will cause greater fatigue inducing metabolic perturbations, and as such, starting $V\dot{O}_2$ kinetics ($\tau$) may be important to promote improved performance and limit the effect of anaerobic metabolic perturbations of these higher intensities. Based on the submaximal model of exercise, an expected response would be to observe a reduction in the respective phase amplitudes, given that the training program is likely to improve aerobic fitness and economy of effort; however, this is unconfirmed. Therefore, the aim of the current study was to investigate the effect of severe-intensity interval
training (at intensities associated with or above VO$_2$ peak) on the VO$_2$ response to severe, constant-load, cycle ergometer exercise (at absolute VO$_2$ peak).

**Methods:**

**Participants:**

Ten physically active females (mean ± SD: age 20 ± 4 y, mass 61.5 ± 9.4 kg, VO$_2$ max 2.30 ± 0.37 L min$^{-1}$) volunteered to participate in this study. Participants were informed of the study requirements, benefits and risks before giving written informed consent. Approval for the study’s procedures was granted by the Institutional Research Ethics Committee.

**Experimental overview:**

All participants completed a familiarisation trial of both the graded exercise test (GXT) and the 6-min constant-load cycle test (CT) before baseline testing. The GXT and CT were then performed by each participant before and after the training intervention, with post-training tests conducted in the same order as pre-training tests. At least 48 h separated each testing session and pre- and post-training tests were conducted at the same time of day. Participants were asked to maintain their normal diet and training throughout the study. Participants were required to consume no food or beverages (other than water) 2 h prior to testing and were asked not to consume alcohol or to perform vigorous exercise in the 24 h prior to testing. Food diaries were given to each participant to record food and fluid consumption two days prior to each test and participants were asked to replicate this during post-training testing. During both the pre- and post-training GXT and CT, pulmonary gas exchange was
determined breath-by-breath for O₂ and CO₂ concentrations and ventilation using a portable gas analysis system (K4b², Cosmed, Rome, Italy). The gas analysers were calibrated immediately before and verified after each test using a certified gravimetric gas mixture (BOC Gases, Chatswood, Australia), while the ventilometer was calibrated pre-exercise and verified post-exercise using a three litre syringe in accordance with the manufacturer's instructions.

Graded exercise test

Pre and post-training, the GXT was performed on an electronically-braked cycle ergometer (Lode, Gronigen, The Netherlands) and consisted of graded exercise steps (4-min stages), using an intermittent protocol (1-min break between stages). The test commenced at 50 W and thereafter, intensity was increased by 25 W every 4 min until volitional exhaustion. The test was stopped when the participant could no longer maintain the required power output. Strong verbal encouragement was provided to each participant as they came to the end of the test. Capillary blood samples were taken at rest and immediately following each 4-min stage of the GXT. A hyperaemic ointment (Finalgon, Boehringer Ingelheim, Germany) was applied to the earlobe 5 - 7 min prior to initial blood sampling. Glass capillary tubes were used to collect 50 µL of blood during the GXT and capillary blood lactate was measured using a blood-gas analyser (ABL 625, Radiometer, Copenhagen, Denmark). Both LT and VO₂ max were determined from the GXT. The LT was calculated using the modified Dₘₐₓ method [18], while VO₂ max was determined as the highest 30-s rolling average during the GXT and the power output at which VO₂ max occurred was accordingly used in the CT.
Constant-load cycle test:

Prior to and following the training intervention, participants performed a constant-load (80 rpm), 6-min cycle test at the absolute power output associated with pre-training VO₂ max (same ergometer as the GXT). Participants performed 4 min of unloaded cycling before a square-wave transition to the individually determined exercise intensity (power output associated with VO₂ max for 6 min). Transition times from unloaded to VO₂ max intensity cycling took 2 – 5 s. Following removal of outliers (± 3SD), breath-by-breath data from 0 to 6 min during the CT was smoothed using standing 5-s averages to enhance the underlying characteristics. A two-component exponential model was fit from 20 s (equation 1) for both pre- and post-training CT VO₂ data using an iterative, non-linear regression (customised program, Microsoft Excel 2002). Support for this two-component (one-trial) modelling method has recently been demonstrated [19] and previously used [17]. Zoladz et al. [19] state that control and replication of the power increase in the first seconds of high-intensity exercise is difficult and this can be avoided by ignoring the first 20-s of data and furthermore, that if an intervention effect is strong enough, it should be detectable even with a single exercise trial.

\[ VO₂(t) = VO₂(b) + A₁ \cdot (1- e^{-(t-td₁)/τ₁}) + A₂ \cdot (1- e^{-(t-td₂)/τ₂}) \]  

\((eq \ 1)\)

Note: \( VO₂(t) \) represents the VO₂ at any given time; \( VO₂(b) \) is the unloaded cycling VO₂ baseline value; \( A₁ \) and \( A₂ \) are the asymptotic amplitudes; \( τ₁ \) and \( τ₂ \) are the time constants and \( td₁ \) and \( td₂ \) are the time delays for each respective phase (primary and slow).
The AOD was calculated based on individual VO₂ – power output regression equations determined from the GXT for each participant for both the pre and post-training CT according to Medbø et al. [20].

Training intervention

All participants commenced the supervised, progressive training program within 4 to 7 days of their final pre-training test. Training intensity was set as a percentage of LT, rather than VO₂ peak as metabolic and cardiac stresses are similar when individuals of differing fitness levels exercise at a percent of LT, but can vary significantly when training at a percent of VO₂ peak [21]. The participants performed three high-intensity interval-training sessions per week for eight consecutive weeks. All training sessions were completed on mechanically-braked cycle ergometers (818E, Monark, Stockholm, Sweden) and were preceded by a 5 min warm up at 50 W. Training programs involved a periodised increase in volume and intensity with a taper in the final week so as to simulate athletic training programs; thus allowing progression and preventing over-training and participant attrition. All participants performed high-intensity interval training at an intensity of 130% (week one), 140% (week two), 150% (weeks three and four), 160% (week five), 170 - 180% (weeks six and seven) and 170% (week eight - with reduced volume) of their individually determined power at LT. A representation of the 8-week training intervention is presented in Figure 1. Intervals were of 2-min duration, with a 1-min recovery (work-to-rest ratio of 2:1). Participants were required to maintain a pedal frequency of 80 rpm during each 2-min interval and to rest completely during the recovery period. Increments in training
progression were controlled by increasing the workload (altering resistance) and the number of intervals performed in a training session.

**Statistical Analysis**

A one-way, repeated-measures ANOVA with Tukey’s post-hoc tests was used to determine any significant differences in the measured variables before and after the training intervention. The alpha level for statistical significance was set at 0.05. All results are reported as mean ± SEM unless otherwise stated.

**Results:**

Changes in physiological variables associated with the GXT, as a result of the high-intensity interval-training program, are presented in Table 1. Following training, there was a significant increase in VO$_2$ max, power at VO$_2$ max and power at LT (p<0.05). Pre and post-training VO$_2$ baseline (VO$_2$(b)), phase amplitudes (A$_1$, A$_2$), time constants ($\tau_1$, $\tau_2$), time delays (td$_1$, td$_2$), primary phase gain, VO$_2$ EE, peak heart rate, AOD and anaerobic contribution (%) for the CT are presented in Table 2. Mean Square Error measures for both pre- and post-training VO$_2$ curve models were not significantly different (8682 ± 4666 v 9006 ± 4561; p>0.05)

The severe-intensity interval training significantly increased A$_1$ and VO$_2$ EE, while decreasing the AOD and anaerobic contribution in the post-training CT at the pre-training peak VO$_2$ intensity (Table 2; p<0.05). While modelled SC (A$_2$) results are also presented in Table 2, Poole [3] defines the SC as the additional O$_2$ cost elevating the VO$_2$ above that predicted by the sub-LT power-VO$_2$ relationship. The predicted pre-training VO$_2$ at VO$_2$ max intensity calculated from the sub-LT power-VO$_2$
relationship was 2.43 ± 0.31 L·min⁻¹. This was significantly greater than the measured pre-training \(\text{VO}_2\max\) (2.30 ± 0.22 L·min⁻¹); however, was not significantly different from the measured post-training \(\text{VO}_2\) (2.55 ± 0.26 L·min⁻¹) at the same absolute intensity (pre-training peak power). No other \(\text{VO}_2\) response variable showed significant changes following training. The average pre and post-training \(\text{VO}_2\) response during the CT for all participants is presented in Figure 2 and shows the significant increases in the phase amplitudes and \(\text{VO}_2\text{peak}\) in the post-training CT.

**Discussion:**

The aim of the current study was to investigate the effects of severe-intensity interval training on the \(\text{VO}_2\) response to severe, constant-load cycle, ergometer exercise (pre-training 100% \(\text{VO}_2\max\)). Results show that severe-intensity interval training significantly increased \(A_1\) and \(\text{VO}_2\text{EE}\). Furthermore, the training program also resulted in a significant decrease in the AOD and anaerobic contribution in the post-training CT. These results hold significance as they contrast with the results reported for moderate – heavy intensity exercise (50 – 80% \(\text{VO}_2\text{peak}\)) following sub-maximal endurance training, where \(\text{VO}_2\text{peak}\) is not likely to be attained.

In the present study, an increase in \(\text{VO}_2\text{EE}\) was evident following high-intensity interval training, principally via an increase in \(A_1\). Previous training studies, incorporating constant-load exercise intensities below the LT, without the development of a SC, have not reported changes in \(\text{VO}_2\text{EE}\) [6,8]. However, for exercise intensities above the LT and below \(\text{VO}_2\text{peak}\), endurance training has been
reported to decrease \( V\dot{O}_2 \text{EE} \) at the same absolute intensity [14,22]. An increase in \( V\dot{O}_2 \text{EE} \) for the same absolute intensity, particularly following an 8-week training program, contrasts with previous research reporting an improvement in economy (decrease in \( V\dot{O}_2 \) at an absolute intensity) with endurance training [23], however studies have not involved training and testing at intensities that are associated with \( V\dot{O}_2 \text{peak} \). The measured pre-training \( V\dot{O}_2 \text{max} \) (2.30 ± 0.37 L·min\(^{-1}\)) was significantly lower than that predicted (2.43 ± 0.31 L·min\(^{-1}\)) from the linear extrapolation of the sub-LT \( V\dot{O}_2 \) – work rate relationship. This is consistent with previous research that has described a loss of linearity of the \( V\dot{O}_2 \) – work rate relationship for exercise intensities in excess of 85% \( V\dot{O}_2 \text{peak} \) [24]. As a result, it is likely that the metabolic demands of the workload at \( V\dot{O}_2 \text{max} \) may have exceeded the pre-training \( V\dot{O}_2 \text{peak} \). However, with improvements in \( O_2 \) delivery and utilisation following training (increased \( V\dot{O}_2 \text{max} \)), an increased post-training \( V\dot{O}_2 \) reduced these differences (predicted vs measured) during the CT, regardless of the change in relative exercise intensity. As such, the present study suggests that intense interval training, resulting in a significant increase in \( V\dot{O}_2 \text{peak} \), can increase the \( V\dot{O}_2 \text{EE} \) at absolute, severe-intensity, constant workloads and as such improve aerobic functioning to these high intensity workloads.

The increase in the primary gain following training due to a greater \( A_1 \) for the same workload also represents a reduction in the economy of effort. Again, this seems contradictory to previous research and common assumptions of the effects of aerobic training [23]. Apart from the potential inability of subjects to attain the required pre-training aerobic metabolic workload, Krustrup et al. [25] have reported higher muscle
VO2 in an isolated quadriceps muscle during high-intensity exercise after high-intensity interval training when compared to a control condition. While no differences were present at low exercise intensities, due to a larger muscle blood flow and better extraction following training, a larger muscle VO2 was measured at the higher intensities in the trained muscle. This fits with the whole body data reported in the present study and would account for the increased A1 and VO2EE reported following high-intensity interval training.

The principal increase in VO2 during the CT following training resulted from a significantly increased A1, in conjunction with a significantly improved VO2 peak. Research involving severe-intensity exercise has also not reported an increase in A1 following training [16,17]; however, no increase in VO2 peak was reported in these studies. This may not be that surprising given that improving VO2 peak in well-trained participants can be difficult [26] and that the aerobic capacity of these respective groups in this study was relatively well developed pre-training. As A1 represents the primary increase in muscle oxygen uptake [2], it is likely that the mechanisms responsible for improvements in 'aerobic fitness' (as seen from the increase in VO2 max and LT), are also responsible for the increase in A1. The major mechanisms responsible for increased muscle VO2 are a combination of O2 transport and/or O2 uptake factors [27]. Interval training has been previously shown to be an effective method of improving factors associated with O2 transport (blood flow, vascular conductance and capillarisation) and also muscle O2 uptake (citrate synthase activity) [25]. Accordingly, both factors are central to improving VO2max and the primary phase of the VO2 response and are likely to be responsible for the increased A1 in the current
study. Further evidence for a link between an increased $A_1$ and VO$_2$ peak is provided by Barstow et al. [28]. They reported an association between the amplitude of the VO$_2$ response over the first 2 – 3 min (phases I and II) and fitness level (VO$_2$ peak), with a greater VO$_2$ peak linked to a greater rise in VO$_2$ (at 80 rpm). In the current study, the increase in post-training VO$_2$ peak allowed to meet the aerobic demands of the exercise bout, predominantly via a significant increase in $A_1$ and therefore VO$_2$ EE.

While a significant increase in $A_1$ was reported, there was no significant change in the speed of the VO$_2$ response ($\tau_1$) in the present study. Demarle et al. [16] and Billat et al. [17] have previously reported a faster $\tau_1$ (to a non-significantly reduced $A_1$) for the same absolute exercise intensity following training. Demarle et al. [16] linked the faster $\tau_1$ to either an O$_2$ delivery explanation due to a faster heart rate response (possible circulatory limitation of VO$_2$ peak [29]) or an O$_2$ utilisation explanation via improved muscle O$_2$ usage as evident in the decreased [La$^-$]$_b$ post-training. The discrepancy between the speeding of $\tau_1$ reported in previous research [16,17] and the current study may result from the fact that $\tau_1$ and $A_1$ are not independent of each other. Tau represents the time required for the VO$_2$ to reach ~63% of the phase amplitude. Therefore, a large increase in $A_1$ will increase (slow) the $\tau$ value, even though the speed of the VO$_2$ response may not have effectively changed (Figure 2). Changes in $A_1$ may therefore result in changes in $\tau_1$ that reflect a relative, rather than absolute, change in the speed of the VO$_2$ response. Furthermore, the large increase in $A_1$ in the present study may have contributed to the absence of a significant change in $\tau_1$, despite the significant increase in aerobic fitness.
As a result of an increased VO₂ during the post-training CT, particularly after the first 40 s, the AOD was significantly reduced (12%) during the post-training CT. Demarle et al. [16] have also reported a reduction (34%) in the AOD following their training intervention, which they linked to reductions in τ₁. Similarly, Hagberg et al. [11] reported a 25% reduction in the AOD following endurance training, which was also linked to a faster increase to steady-state VO₂ following training. In contrast to these two studies however, the decrease in AOD in the present study was not the result of a lower τ₁, but rather the result of an increase in both A₁ and VO₂ EE.

Previous research incorporating sub-maximal endurance training interventions has also reported an attenuated SC during moderate - heavy exercise following training [6,8, 14]. Both Demarle et al. [16] and Billat et al. [17] reported non-significant reductions in the SC during severe-intensity runs to exhaustion (90 – 95% VO₂ peak) following training. The current study found no significant difference in the SC during severe-intensity exercise following training; however, there was a tendency for an elevated SC following training (p= 0.15). It is possible therefore, that increases in VO₂ peak following training may allow a greater SC to be expressed during severe-intensity exercise. This is supported by the observation that the SC was quite similar when expressed as a percentage of VO₂ EE following the high-intensity training program. Currently the favored mechanism to explain the SC is an increase in the recruitment of type II fibres [30]. Potentially, the high-intensity interval training is likely to have incorporated significant training of type II fibers and as such may be expressed in the tendency for an increase in the SC.
In conclusion, a severe-intensity, interval-training program significantly increased \( V\bar{O}_2 \) peak and power at both VO\(_2\)peak and the LT in active females via a significantly increased amplitude of the primary phase, without altering the speed of the VO\(_2\) response to severe-intensity exercise. As such, following training, a greater VO\(_2\) response decreased the AOD, which resulted in a significantly reduced reliance on anaerobic metabolism at the same absolute intensity. This increase in amplitude of the primary phase may be associated with the pre-training VO\(_2\) peak limiting the rise in VO\(_2\) to sufficiently meet the metabolic demands of the severe, pre-training exercise intensity. However, post-training, a significantly greater VO\(_2\) peak may have allowed for a greater rise in CT VO\(_2\), reducing the anaerobic energy contribution for the same given exercise intensity.

**Practical Implications:**

- Training at severe-exercise intensities can increase oxygen consumption at maximal exercise intensities without slowing the speed of the aerobic response.
- Oxygen consumption at maximal aerobic exercise intensities may be restricted in less well trained individuals and can be increased at the same intensity post-severe intensity interval training.
- A concomitant reduction in aerobic energy system contribution at severe exercise intensities can be gained following high-intensity interval training.
References:


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