Effect of Prior Exercise above and below Critical Power on Exercise to Exhaustion

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ABSTRACT

CARTER, H., Y. GRICE, J. DEKERLE, G. BRICKLEY, A. J. P. HAMMOND, and J. S. M. PRINGLE. Effect of Prior Exercise above and below Critical Power on Exercise to Exhaustion. Med. Sci. Sports Exerc., Vol. 37, No. 5, pp. 775–781, 2005. Purpose: The aim of the present study was to ascertain whether the intensity of prior exercise altered the time to exhaustion at critical power (CP).

Methods: Eleven participants volunteered to take part in the study (mean ± SD: VO₂max 4.1 ± 0.5 L·min⁻¹; age 30.1 ± 7.2 yr; body mass 74.6 ± 9.1 kg) and completed three trials to exhaustion at their CP under differing prior exercise conditions: 1) a control trial (CON); 2) a trial preceded by three 60-s efforts at 110% CP (severe); and 3) a trial preceded by three 73-s efforts at 90% CP (heavy). All trials followed a 5-min baseline at 50 W. Results: Time to exhaustion was significantly lengthened after prior heavy exercise (1071 ± 18 s) when compared with CON (973 ± 16 s, F = 9.53, P = 0.006). However, there was no effect on TTE after prior severe exercise (967 ± 16 s). Oxygen deficit was significantly reduced from that in CON (3.8 ± 0.2 L) after prior heavy (3.2 ± 0.3 L) and prior severe exercise (3.1 ± 0.3 L, F = 10.95, P = 0.001). Concurrently, there was a significant reduction in the magnitude of the VO₂ slow component (SC) in the trials with prior exercise (197 ± 34 and 126 ± 19 mL·min⁻¹ after heavy and severe exercise, respectively) when compared with CON (223 ± 31 mL·min⁻¹, F = 9.62, P = 0.006). Conclusion: Prior heavy exercise does appear to improve the time to exhaustion at CP by ~10% and is associated with a reduction in the VO₂ SC. However, the reduction in the SC, with no change in performance after prior severe exercise, suggests that a reduced SC may not necessarily lead to improved TTE. Key Words: OXYGEN DEFICIT; VO₂ SLOW COMPONENT; INTENSITY DOMAINS; WARM-UP

It is widely recognized that warm-up exercise benefits performance in endurance events (17). However, scientific evidence explaining the underpinning physiological mechanisms of the ergogenic effect is limited (3). Traditionally, most research points to a temperature-related etiology through factors such as nerve-conduction rates, altered force–velocity relationships, and increased vasodilation (3,14). However, more recent evidence suggests that other systemic factors cause the ergogenic effect including increased acidemia and elevation of oxygen uptake (VO₂) (17). Research that has addressed the issue of warm-up exercise and the mechanisms underpinning its effects has focused on the measurement of the physiological response as opposed to performance. Because measurement of pulmonary VO₂ is known to provide an insight to muscle oxygen utilization (24), the oxygen uptake kinetic response to square-wave exercise has proved a useful model to understand the effects of warm-up on metabolism.

In the transition from rest to constant-load moderate exercise (i.e., below the lactate threshold (LT)), the pulmonary oxygen uptake (VO₂) response can be partitioned into three distinct phases. After the cardiodynamic phase (phase I), VO₂ rises in an approximately monoexponential fashion (phase II or primary component) to attain a new steady state (phase III) within 2–3 min (1). However, during “heavy” exercise (i.e., above the LT), the attainment of a steady state is delayed or absent due to the development of an additional VO₂ slow component, which is superimposed on the primary component (1).

Above the boundary of the heavy and severe exercise domains, that is, at the critical power (CP), the VO₂ kinetics are subject to further change. The CP is the asymptote of the hyperbolic power–time relationship and, therefore, theoretically represents the upper limit of sustained, tolerable work (23). Traditionally, the CP was thought to represent an intensity that could be maintained “for a very long time without fatigue” (22), but more recently it has been established that time to exhaustion at this intensity is limited (8,25), being higher than the individual’s maximal lactate steady state. What is certain is that exercise above CP (i.e., severe intensity) is tolerable for only a short, but predictable, duration (between ~5 and 45 min), and a VO₂ steady state is not attained because of an increasing development of the VO₂ slow component (9,25). It has been suggested that, in cycling exercise at least, VO₂max may be reached if exercise is continued long enough (15,23). Other consequences of exercising in this domain are sustained and inexorable in-
increases in \([\text{La}]\) and \([\text{Pi}]\) related to increased anaerobic energy turnover (8,10).

In the study of Gerbino et al. (14) and subsequent work by Burnley and colleagues (5,6), it was demonstrated that performance of heavy exercise leads to changes in the \(\text{VO}_2\) profile in a subsequent constant-load exercise bout. Prior heavy-intensity exercise leads to an overall “speeding” of the \(\text{VO}_2\) kinetic response, or more specifically an increase in the \(\text{VO}_2\) primary component amplitude and a reduction in the \(\text{VO}_2\) slow component. However, very little work has addressed the issue of whether prior exercise improves performance (17). It is suggested that because the prior exercise effect reduces the overall oxygen deficit, this would afford an increased aerobic contribution to exercise and could thus benefit exercise tolerance. Only two previous studies have attempted to examine the effect of prior exercise on performance alongside the changes in \(\text{VO}_2\) kinetics. However, both of these studies focused on performance trials at near (19) or supramaximal intensities (18) and used “priming” exercise not normally witnessed in the practical setting. Furthermore, no research has tested whether the prior exercise effect and proposed improvements in performance are dependent on intensity, or more specifically, its proximity to the CP.

Therefore, the aim of the present study was to ascertain whether prior exercise would improve time to exhaustion of an individual exercising at their CP. Furthermore, it was hypothesized that the intensity of the prior exercise (relative to the critical metabolic threshold of CP) would influence the time to exhaustion, causing it to be greater after heavy prior exercise (i.e., sub-CP) and shorter after severe prior exercise (i.e., supra-CP). The choice of interval-type prior exercise rather than constant-load exercise was used, because this is an accepted form of training in athletes, and would thus increase the ecological validity of the experimentation.

**METHODS**

**Experimental design and participants.** The experimental procedures used in this study were approved by the university’s ethics committee. Eleven participants (8 men, mean ± SD: \(\text{VO}_{2\text{max}}\) 4.13 ± 0.51 L·min\(^{-1}\); age 30.1 ± 7.2 yr; body mass 74.6 ± 9.1 kg) gave written informed consent after the experimental procedures, the associated risks, and the benefits of participation were explained. All participants were recreationally active and fully familiar with laboratory exercise testing procedures. The participants were instructed to arrive at the laboratory after an overnight fast, to be rested, and in a fully hydrated state, having abstained from the consumption of caffeine for the previous 24 h. The participants were also asked to avoid strenuous exercise in the 48 h preceding a test session and to record food intake for this period. The same diet was replicated before each session. Volunteers performed three stages of testing: 1) determination of the ventilatory threshold (VT) and \(\text{VO}_{2\text{max}}\); 2) estimation of the CP; and 3) three trials to exhaustion at their derived CP, each preceded by a different warm-up exercise, and each performed on separate days. The tests were performed at the same time of day for each individual. The study was completed within 2 wk for all subjects.

**Procedures.** All tests were conducted on an electrically braked cycle ergometer (Schrober Rad Messtechnik, Germany) with seat and handlebar height kept constant over the sessions for each participant. Volunteers were allowed to self-select pedal frequency during the first testing session and then asked to maintain this cadence within ± 5 rpm for all remaining tests. Pulmonary gas exchange was determined breath by breath using standard algorithms, allowing for the time delay between gas concentration and volume signals. Individuals breathed through a low dead space (90 mL), low resistance (0.65 mmH\(_2\)O·L\(^{-1}\)·s\(^{-1}\)) mouthpiece and turbine assembly. Gases were continuously drawn from the mouthpiece through a 2-m capillary line of small bore (0.5 mm) at a rate of 60 mL·min\(^{-1}\) and analyzed for \(\text{O}_2\), \(\text{CO}_2\), and \(\text{N}_2\) concentrations by a quadrupole mass spectrometer (CaSE EX670, Gillingham, Kent, UK), which was calibrated before each test using gases of known concentration. Expiratory volumes were determined using a turbine volume transducer (Interface Associates, CA). The volume and concentration signals were integrated by computer after analog-to-digital conversion. Respiratory gas exchange variables (\(\text{VO}_2\), \(\text{VCO}_2\), \(\text{VE}\)) were calculated and displayed for every breath. In all tests, pulmonary gas exchange was measured breath by breath, and subsequently interpolated to provide one value per second. Heart rate was recorded telemetrically throughout the exercise tests (Polar Electro Oy, Kempele, Finland). Fingertip capillary blood samples (~ 25 \(\mu\)L) were collected in capillary tubes and subsequently analyzed for lactate concentration using an automated analyzer (YSI 2300, Yellow Springs, OH).

At the beginning of the study, all participants performed a ramp exercise test to determine the VT and the \(\text{VO}_{2\text{max}}\). The initial power output was kept at 50 W for 5 min and then increased by 5 W every 12 s (equating to 25 W·min\(^{-1}\)) to volitional exhaustion. The highest 30-s average of the breath-by-breath \(\text{VO}_2\) data was taken to be the \(\text{VO}_{2\text{max}}\). Attainment of \(\text{VO}_{2\text{max}}\) was confirmed by the incidence of a plateau phenomenon in \(\text{VO}_2\) (i.e., 150 mL·min\(^{-1}\) between last and penultimate stage) RER values above 1.10, and heart rates within 5 beats·min\(^{-1}\) of age-predicted maximum. In all subjects, at least two of the three criteria were met. The VT was determined by two investigators as the point above which there was a non-linear increase in minute ventilation (\(\text{V}\_\text{E}\)) plotted against \(\text{VO}_2\), and an increase in \(\text{VE}/\text{VO}_2\) against \(\text{VO}_2\) with no increase in \(\text{VE}/\text{VO}_2\) against \(\text{VO}_2\) (28).

For the second stage of testing, extrapolation of the relationship between \(\text{VO}_2\) and power output for exercise below VT was used to estimate the power output at \(\text{VO}_{2\text{max}}\) (\(P\_\text{VO}_{2\text{max}}\)). Subsequently, a series of power outputs were calculated for the determination of CP; three trials to exhaustion at 90, 100, and 110% \(\text{VO}_{2\text{max}}\), performed with at least 4 h of recovery in between. The power outputs chosen were designed to elicit exhaustion in 2–15 min (16), ensuring that \(\text{VO}_{2\text{max}}\) was achieved during each exhaustion test (13). Subjects were instructed to cycle for as long as possible and were given strong verbal encouragement to con-
The physiological response to exercise at CP across the three conditions of prior exercise.

<table>
<thead>
<tr>
<th>Condition</th>
<th>CON</th>
<th>90% CP</th>
<th>110% CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL VO₂ (L·min⁻¹)</td>
<td>1.37 (0.05)</td>
<td>1.43 (0.07)</td>
<td>1.57 (0.08)*</td>
</tr>
<tr>
<td>Pretrial [La] (mM)</td>
<td>1.2 (0.1)</td>
<td>1.7 (0.2)</td>
<td>2.3 (0.2)* †</td>
</tr>
<tr>
<td>VO₂ at 3 min (L·min⁻¹)</td>
<td>3.48 (0.14)</td>
<td>3.55 (0.14)</td>
<td>3.68 (0.11)</td>
</tr>
<tr>
<td>O₂ deficit at 3 min (L)</td>
<td>3.8 (0.2)</td>
<td>3.2 (0.3)</td>
<td>3.1 (0.3)†</td>
</tr>
<tr>
<td>Total O₂ deficit (L)</td>
<td>5.3 (0.7)</td>
<td>8.4 (1.7)*</td>
<td>8.7 (2.0)*</td>
</tr>
<tr>
<td>VO₂ SC₆₋₃min (mL·min⁻¹)</td>
<td>223 (31)</td>
<td>197 (34)*</td>
<td>126 (19)* †</td>
</tr>
<tr>
<td>VO₂ at EE (L·min⁻¹)</td>
<td>3.99 (0.16)</td>
<td>3.95 (0.18)</td>
<td>3.96 (0.16)</td>
</tr>
<tr>
<td>VO₂ at EE (% VO₂max)</td>
<td>97 (0.02)</td>
<td>96 (0.03)</td>
<td>96 (0.03)</td>
</tr>
<tr>
<td>EE [La] (mM)</td>
<td>9.2 (0.5)</td>
<td>9.2 (0.5)</td>
<td>9.3 (0.5)</td>
</tr>
<tr>
<td>EE heart rate (beats-min⁻¹)</td>
<td>178 (1)</td>
<td>177 (2)</td>
<td>176 (1)</td>
</tr>
<tr>
<td>TTE (s)</td>
<td>973 (16)</td>
<td>1071 (18)*</td>
<td>967 (16)†</td>
</tr>
</tbody>
</table>

Values are means (± SEM). BL, baseline; EE, end exercise; TTE, time to exhaustion.
* Significantly different from control; † significantly different from 90% CP.

TABLE 1. The physiological response to exercise at CP across the three conditions of prior exercise.

RESULTS

On average, the VT across the subject group was 167 ± 7 W with the VO₂ at this parameter being 2.34 ± 0.3 L·min⁻¹ equating to 56.7 ± 4.4% VO₂max. The three exhaustive trials for the calculation of CP were performed at 337 ± 17, 314 ± 16, and 296 ± 14 W, and led to exhaustion in 216 ± 13.5, 339 ± 16, and 640 ± 48.1 s, respectively. The relationship between power and 1/t was linear (r² = 0.97). In all trials, the VO₂ attained at the end of each trial to exhaustion was at least 98% VO₂max. The mean CP of the group was 278 ± 12 W, and the anaerobic work capacity (AWC) was 13.4 ± 1.7 kJ. The work rate at the CP equated to 82.6 ± 1.7% VO₂max.

There existed much intersubject variation in the time to exhaustion (TTE) at CP in the control trial, which could not be explained by VO₂max, VT, or AWC, because no correlations existed between these parameters and performance (P > 0.05). However, some patterns emerged when the group was separated into those individuals that could maintain exercise at CP longer than (HIGH TTE) or somewhat less than (LOW TTE) the group mean of ~16.5 min. In the HIGH TTE group, where TTE was significantly longer (1204 ± 80 vs 737 ± 34 s, P < 0.001), VO₂max tended to be higher (~58 mL·kg⁻¹·min⁻¹) compared with the LOW TTE group (~51 mL·kg⁻¹·min⁻¹, P = 0.07). The AWC in the HIGH TTE group was also significantly higher than in the LOW TTE group (17.1 ± 1.3 vs 8.9 ± 0.1 kJ, P = 0.005). When the performance across the warm-up conditions was compared across these groupings, the magnitude and direction of the experimental effect was the same.

The mean data for the subject group is shown in Table 1.
was also significantly elevated compared with that after intensity exercise (967
However, there was no effect on the TTE after the severe-intensity exercise (1.43
had returned to baseline levels after the heavy-intensity exercise. This was in contrast to the O2 although there was no difference between prior heavy- or severe-intensity warm-up (control), a heavy-intensity warm-up (90% CP), and a severe-intensity warm-up (110% CP). The VO2max of the individual is represented by the dashed line.

Immediately before the experimental trials at CP, VO2 and blood [La] were significantly elevated after severe-intensity prior exercise compared with CON (F = 6.285, P = 0.020; and F = 15.18, P = 0.001, respectively). Pretrial blood [La] was also significantly elevated compared with that after heavy-intensity prior exercise (F = 8.63, P = 0.011). Both had returned to baseline levels after the heavy-intensity exercise (1.43 ± 0.07 L·min⁻¹ and 1.7 ± 0.2 mM, respectively). The time to exhaustion was significantly lengthened after prior heavy-intensity exercise (1071 ± 18 s) when compared with CON (973 ± 16 s, F = 9.53, P = 0.006). However, there was no effect on the TTE after the severe-intensity exercise (967 ± 16 s). The VO2 response of a typical subject in the three experimental conditions is shown in Figure 1.

Table 1 shows that, compared with the control condition, the oxygen deficit incurred in the first 3 min was significantly smaller (~17%) after prior high-intensity exercise, although there was no difference between prior heavy- or severe-intensity exercise. This was in contrast to the O2 deficit over the total exercise time, which was significantly greater in the trials with prior exercise (see Table 1). As illustrated in Figure 2, the change in the early VO2 response was accompanied by a significant reduction in the magnitude of the VO2 SC6 – 3min in the trials with prior exercise (197 ± 34 mL·min⁻¹ after heavy exercise and 126 ± 19 mL·min⁻¹ after severe exercise, the latter being significantly lower than heavy, F = 10.4, P = 0.003) when compared with CON (223 ± 31 mL·min⁻¹, F = 9.62, P = 0.006). The TTE was significantly related to the size of the slow component in all conditions, though these relationships were very weak (r ranging from 0.35 in the CON to 0.52 and 0.45 after prior heavy and severe exercise, respectively).

Despite the difference in TTE across trials, there were no differences in end exercise VO2, HR, or blood [La] at exhaustion. In all three conditions, the VO2 at exhaustion reached ~3.9 L·min⁻¹. This was equivalent to ~97% of the VO2 at VO2max. Heart rate reached 178 ± 1, 177 ± 2, and 176 ± 1 beats·min⁻¹ in the CON, after prior heavy and severe exercise, respectively, somewhat lower than the maximum measured in the ramp test (190 ± 3 beats·min⁻¹). Blood [lactate] at exhaustion was consistent across trials at ~9.2 mM.

The improvement in performance after heavy-intensity prior exercise was poorly related to the decrease in oxygen deficit (r = 0.28, P > 0.05) and the decrease in the slow component (r = 0.26, P > 0.05), and only moderately related to the preexercise lactate concentration (r = 0.44, P < 0.05).

**DISCUSSION**

The present study has shown that prior heavy exercise results in improved performance in a time to exhaustion trial at CP. The enhanced performance was accompanied by reductions in both the VO2 slow component and O2 deficit, as would be anticipated from the results of previous research. However, when preceded by severe-intensity exercise, time to exhaustion was not improved, even though similar changes in the VO2 kinetics response were observed. One consequence of this finding is that the often-cited causal link between the VO2 slow component and fatigue may be questionable. However, it cannot be ruled out that this relationship has been moderated by other factors.

The TTE at CP observed in this study was similar to previous reports. Although originally proposed as an intensity that should be sustainable “indefinitely,” and representing the upper boundary of the heavy exercise domain (23), exercise time at CP is reported to typically last between ~5 and 45 min (8,25). Research in running (27), cycling (11,25), and swimming (12) points to the CP being at an intensity beyond a metabolic steady state (25). In the present study, VO2 was also seen to rise over the course of the trial at CP, attaining near maximal values at exhaustion (~97%). The relatively short TTE at this parameter found in this study is therefore not surprising, and supports the contention that CP cannot be sustained indefinitely, presumably associated with an accumulation of muscle and blood metabolites (25).
The presence of a prior bout of heavy-intensity warm-up exercise lengthened TTE by some 10% from ~16 to ~18 min. This magnitude of effect was evident in 9 of the 11 subjects tested. It is generally accepted that a warm-up enhances performance (3), particularly in activities performed above the lactate threshold (19). The performance enhancements could be attributed largely to induced metabolic effects facilitating oxygen delivery (20,21). Prior exercise has been shown to increase the amplitude of the primary response while reducing the size of the VO₂ slow component (5,6,14,17). Mechanisms underpinning the altered VO₂ profile include enhanced vasodilation, changes in phosphorylation potential, and the availability of acetyl groups (3,14,26).

The present study only employed one exercise transition in each condition, so it is therefore necessary to explore the mechanisms for the warm-up effect using less sophisticated estimates of the kinetic profile. The prior exercise effect was present in this study as evidenced by a smaller O₂ deficit at 3 min, and reduced VO₂ SC₆–₃ min in the two conditions with prior exercise as compared with control. This would translate to less work being performed anaerobically at exercise onset, leaving more of this reserve until later into exercise, and thus explaining the increase in time to exhaustion (3). However, in the present data set (acknowledging the difficulties in using this parameter to estimate anaerobic contributions to exercise without knowing muscle lactate levels), blood lactate concentration was not different across the three conditions at 5 min into the performance trials (at 6 mM), nor was there a strong relationship between the reduction in O₂ deficit and improvement in TTE after heavy exercise (r = 0.28). This is more than likely a reflection of exercise duration, because 20 min of sustained exercise will be supported predominantly by aerobic metabolism, thus diluting the impact of the changes to O₂ deficit.

Previous work has proposed the extent of the metabolic acidosis being implicated in the ergogenic effect that prior exercise affords. Indeed, in the present study, there was a significant positive relationship between preexercise blood [La] and TTE after prior heavy-intensity exercise (r = 0.44). In the study of Jones et al. (18), slight preexercise elevations in blood lactate of 2.5 mM were associated with an increased TTE. These authors suggest that residual acidosis preserves muscle function and retards fatigue development. Gerbino et al. (14) have proposed that residual acidosis leads to vasodilation of the muscle tissue, improving perfusion and oxygen availability, which could help explain such improvements in performance. Furthermore, H⁺ ions may also increase muscle oxygen delivery via a shift in the oxyhemoglobin dissociation curve (4).

Although prior heavy-intensity exercise improved exercise tolerance in the present study, it was of great interest that prior severe-intensity exercise did not aid performance. In fact, for 8 of 11 subjects, TTE reduced after prior severe-intensity exercise compared with control. Coats et al. (10) used an interesting study design to illustrate that exercise in the severe-intensity domain is associated with a metabolic nonsteady state. In their study, they asked participants to exercise to exhaustion at an intensity that would lead to fatigue in approximately 6 min. At exhaustion, the workload was reduced to a new power output. They found that reducing the workload to moderate- or heavy-intensity exercise allowed further exercise to be continued. However, when the work rate was reduced to 110% CP (i.e., within the severe domain), no further work could be completed. The authors attributed this to the inability to reduce [La], stabilize pH, replenish high energy phosphate stores, and prevent Pi increases at intensities above CP. Likewise, Jones et al. (18) suggest that prior exercise of high intensity results in accumulation of many fatiguing metabolites that will affect performance. Indeed, previous authors have suggested the upper limit for metabolism lies at the MLSS, a parameter known to be somewhat lower than CP (8,11,25,27).

The intensity-specific nature of the warm-up/performance relationship has been alluded to previously. Jones et al. (18) demonstrated that 6 min of heavy constant-load exercise improved performance by up to 60% at exercise intensities around the VO₂max. However, in studies using prior exercise estimated as within the severe domain, little or no benefit has been observed (2,19). Our study indicates that the precise intensity of the warm-up bout (i.e., its proximity to the CP) is decisive from a performance enhancement perspective and in understanding its mechanistic basis. Jones et al. (17,18) emphasize that heavy exercise may represent an ideal warm-up intensity: moderate exercise does not enhance the aerobic contribution to subsequent exercise, whereas severe-intensity exercise does not allow sufficient time for the restoration of resting metabolism and/or removal of fatiguing metabolites. Blood lactate did not return to baseline after severe exercise, and thus residual acidosis was present as subjects entered the TTE trial at CP. After severe-intensity exercise, higher blood lactate concentrations were associated with a reduced TTE (r = −0.36).

As with blood lactate, the VO₂ also failed to reach the control baseline levels after the prior severe exercise. This observation makes it unclear as to whether the increase in absolute VO₂ at 3 min was dependent on the elevation in baseline under this condition. Burnley et al. (6) gave consideration to this issue by elongating the length of recovery between two heavy exercise bouts to 12 min, thus allowing baseline VO₂ to be restored. Interestingly, the authors found the prior exercise effect remained even with this longer recovery duration. Blood lactate was still slightly elevated at this point, supporting the level of acidosis being the major influence on subsequent exercise responses. In a review of existing research, Bishop (3) suggests that the warm-up effect is only observed when the intensity of prior exercise and the recovery duration after it combine to leave an elevated baseline VO₂. However, 6 min was enough to see VO₂ return to baseline after heavy exercise, yet still induce an ergogenic effect.

It is possible that other physiological events influenced the TTE at CP across the experimental conditions. Coats et al. (10) suggest that the performance of prior exercise may alter the CP itself. Other interventions are known to have an effect on this parameter including training status, inspired
O$_2$ fraction, and glycogen depletion. Thus, although the power output used in the control trial represents the CP in “nonfatigued” muscle, prior exercise above CP may have distorted the power–time relationship. Jones et al. (18) attempted to investigate this effect in their study by computing the CP from trials at 100, 110, and 120% VO$_{2max}$ with and without prior exercise. They found the CP did not differ, yet the estimate of AWC (W') did. Because W' represents a finite reserve, it is not possible to alter it without some ergogenic agent. Therefore, it is feasible that the differences in TTE caused by prior exercise in our subject group could have been due to an increased ability to utilize W'.

Both heavy and severe prior exercise resulted in an increase in the ability to utilize the W', as estimated by the oxygen deficit calculated over the entire exercise time in the exhaustive trials. In the control condition, the total oxygen deficit incurred was ~5 L compared with ~8 L after both warm-up interventions. The fact that the O$_2$ deficit was similar at exhaustion after the severe exercise, despite a shorter TTE, points to an increased rate of use of the W' in this condition. Considering the factors that might determine exercise time at CP (e.g., the balance between accumulation and removal of fatiguing metabolites, depletion of high-energy phosphate stores, and alteration in motor unit recruitment pattern), prior severe exercise may have adversely affected one, or all, of these processes. Certainly, performance of exercise above CP could have resulted in an increased recruitment of the Type II muscle fiber pool, explaining an increased rate of W' utilization. Of course, it is also feasible that some of the W' was used within the prior exercise bouts themselves. After the severe-intensity exercise, inadequate time for the complete removal of fatiguing metabolites and restoration of high-energy phosphates (10) may have led to the lack of improvement in TTE. This is indeed supported by our crude method of assessing VO$_2$ kinetics, which suggested the aerobic/anaerobic contributions to be similar early in exercise across both prior exercise conditions.

The VO$_2$ slow component has been suggested as a determinant of exercise tolerance (1,17). We therefore hypothesized that the reduced slow component anticipated after prior exercise would lead to performance enhancement and a lengthened TTE at CP. As reported, this was the case after heavy- but not severe-intensity exercise. This is in agreement with the work of Koppo and Bouckaert (19), who also dissociated the VO$_2$ kinetic response from performance. It is interesting to speculate as to why severe-intensity prior exercise reduced the VO$_2$ slow component but did not then lead to improved performance. Recently, Burnley et al. (7) reported that iEMG was significantly increased in the second of two severe exercise bouts, mirroring the altered VO$_2$ response. This change in iEMG may represent an increased motor unit recruitment after the first exercise bout, perhaps consisting predominantly of more Type II muscle fibers, although it must also be considered that similar changes in iEMG could be brought about by increased motor unit firing rates or action potential amplitudes. As discussed earlier, an increased rate of W' utilization and premature fatigue might ensue. Alternatively, the link between the VO$_2$ slow component and exercise tolerance may be more coincidental than previously thought. Indeed, when we compared the relationship between TTE and the size of the slow component in our present data set, we observed only very weak negative relationships. It is therefore necessary to systematically examine the VO$_2$ kinetic profile and performance rather than speculate on this relationship using parallel changes with experimental interventions, for example, endurance training.

In conclusion, provided the intensity does not exceed the CP, prior exercise does improve the time to exhaustion at CP by ~10%. This improvement is accompanied by a reduction in the VO$_2$ SC. However, the reduction in the SC with no change in performance in the 110% CP trial suggests the SC may not necessarily be causally linked to fatigue.

REFERENCES


