Effects of Prior Warm-up Regime on Severe-Intensity Cycling Performance

MARK BURNLEY[1], JONATHAN H. DOUST[1], and ANDREW M. JONES[2]

[1]*Department of Sport and Exercise Science, University of Wales, Aberystwyth, Ceredigion, UNITED KINGDOM; and*
[2]*Department of Exercise and Sport Science, Manchester Metropolitan University, Alsager, Cheshire, UNITED KINGDOM*

ABSTRACT

BURNLEY, M., J. H. DOUST, and A. M. JONES. Effects of Prior Warm-up Regime on Severe-Intensity Cycling Performance. *Med. Sci. Sports Exerc.*, Vol. 37, No. 5, pp. 838–845, 2005. **Purpose:** The purpose of the present study was to determine the effect of three different warm-up regimes on cycling work output during a 7-min performance trial. **Methods:** After habituation to the experimental methods, 12 well-trained cyclists completed a series of 7-min performance trials, involving 2 min of constant–work rate exercise at ~90% $\dot{V}O_{2max}$ and a further 5 min during which subjects attempted to maximize power output. This trial was performed without prior intervention and 10 min after bouts of moderate, heavy, or sprint exercise in a random order. Pulmonary gas exchange was measured breath by breath during all performance trials. **Results:** At the onset of the performance trial, baseline blood [lactate] was significantly elevated after heavy and sprint but not moderate exercise (mean ± SD: control, 1.0 ± 0.3 mM; moderate, 1.0 ± 0.2 mM; heavy, 3.0 ± 1.1 mM; sprint, 5.9 ± 1.5 mM). All three interventions significantly increased the amplitude of the primary $\dot{V}O_2$ response (control, 2.59 ± 0.28 L·min^{-1}; moderate, 2.69 ± 0.27 L·min^{-1}; heavy, 2.78 ± 0.26 L·min^{-1}; sprint, 2.78 ± 0.30 L·min^{-1}). Mean power output was significantly increased by prior moderate and heavy exercise but not significantly reduced after sprint exercise (control, 330 ± 42 W; moderate, 338 ± 39 W; heavy, 339 ± 42 W; sprint, 324 ± 45 W). **Conclusions:** These data indicate that priming exercise performed in the moderate- and heavy-intensity domains can improve severe-intensity cycling performance by ~2–3%, the latter condition doing so despite a mild lactacidosis being present at exercise onset. **Key Words:** O_2 UPTAKE KINETICS, LACTATE, TIME TRIAL, RELIABILITY, MODELING

It is widely accepted that prior warm-up exercise should be performed before the main bout of activity during both sport and exercise. The rationale for such practice is that the prior exercise may prevent musculoskeletal injury and improve performance, although there is limited evidence for either of these effects. The scientific investigation of prior warm-up exercise is extensive. However, studies have usually addressed the physiological effect of warm-up exercise, rather than addressing its potential performance benefit (5,11,23). How warm-up exercise should be structured in terms of its intensity, duration, mode, and the recovery period between the warm-up and performance will depend upon the physical requirements of the criterion task: sprint exercise requiring the generation of maximal muscle power might well benefit from a true "warm-up," whereas the performance benefit of increasing muscle and/or core temperature during endurance exercise in the heat is clearly questionable (4).

Address for correspondence: Dr. Mark Burnley, Department of Sport and Exercise Science, Carwyn James Building, University of Wales, Aberystwyth, Aberystwyth, Ceredigion, SY23 3FD, United Kingdom; E-mail: mhb@aber.ac.uk.
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There has been much recent interest in the effect of prior exercise on the time course of the $\dot{V}O_2$ response (i.e., the $\dot{V}O_2$ kinetics) after the onset of heavy submaximal exercise. It has been repeatedly demonstrated that prior exercise performed above the lactate threshold (heavy exercise) results in a speeding of the overall $\dot{V}O_2$ response and a reduction in blood lactate accumulation in an identical subsequent bout (9,11,18,21). This speeding of the $\dot{V}O_2$ response has been shown to be a consequence of an increase in the amplitude of the primary "fast" $\dot{V}O_2$ component and a reduction in the subsequent $\dot{V}O_2$ slow component (6,7). Prior sprint exercise, either in isolation (8) or when repeated (26), also results in similar alterations in the $\dot{V}O_2$ response to subsequent heavy exercise. The effect of prior intense exercise on the $\dot{V}O_2$ response has been convincingly shown to originate within the exercising muscle (1). In contrast, increasing exercising muscle temperature, either through the performance of moderate exercise (9,11) or by external heating (8,16,20), has not been shown to alter the primary $\dot{V}O_2$ response. Prior moderate exercise or muscle heating has been shown by some (16,17) but not others (7,8,20) to result in a small but significant reduction in the amplitude of the $\dot{V}O_2$ slow component.

Collectively, the above findings suggest that, as first proposed by Gerbino et al. (11), "warm-up" exercise is perhaps more appropriately termed "acid-up" exercise in the context of its effect on subsequent exercise metabolism. Whether an exercise-induced lactacidosis can improve physical performance has, until recently, been unclear. Although marked reductions in power output consequent to

repeated sprint exercise suggest that performance is diminished (15), a recent study has demonstrated a significant improvement in time to exhaustion (of 30–60%) during perimaximal leg cycling after a prior bout of heavy exercise (14). In contrast, Koppo and Bouckaert (19) showed no changes in time to exhaustion after either prior moderate or prior heavy exercise. One difference between those studies was the severity of the lactacidosis: the protocols employed by Jones et al. (14) yielded a baseline blood [lactate] of ~2.5 mM, whereas those of Koppo and Bouckaert (19) resulted in a baseline blood [lactate] of ~6 mM after prior heavy exercise. It is possible that these differing degrees of metabolic disturbance result in quite different performance effects. To date, however, this proposal has not been investigated. Furthermore, the use of time to exhaustion as a measure of athletic performance lacks ecological validity (12), and therefore the findings of Jones et al. (14) require corroboration using tests that detect changes in mean power output during severe-intensity exercise.

The purpose of the present study was to determine the effect of various warm-up regimes on cycling work output during a 7-min performance trial. The "warm-up" procedures chosen were prior moderate exercise, prior heavy exercise, and a prior 30-s sprint. These prior tasks were intended to result in marked differences in baseline blood [lactate] after a 10-min recovery period. It was hypothesized that prior heavy exercise that induced a mild acidosis would improve power output during the performance trial, that prior moderate exercise would have no effect, and that the 30-s sprint would result in a reduced power output.

METHODS

Twelve well-trained cyclists provided written informed consent to participate in the present study, which was approved by the ethics committee of the University of Wales, Aberystwyth. The physical characteristics of the subjects were (mean ± SD) age 34 ± 9 yr, body mass 74.6 ± 8.6 kg, and height 180.1 ± 9.0 m. Subjects were instructed to arrive at the laboratory at the same time of day (±2 h) in a rested (no heavy exercise in the preceding 24 h), well-hydrated state, having consumed no food or alcohol in the preceding 3 h. Care was taken to schedule the testing around the subjects' training and competition schedules. Throughout the study, the subjects showed an excellent motivation and willingness to produce a maximal effort during the performance trials on each visit to the laboratory.

Subjects visited the laboratory on six to eight occasions over a 2- to 3-wk period to complete the experimental work. The first visit was used to establish peak $\dot{V}O_2$ ($\dot{V}O_{2peak}$) and gas exchange threshold (GET) to determine the work rates for the subsequent protocols. On the second visit to the laboratory, subjects were familiarized with the performance trial, as detailed below. Subjects then performed four subsequent tests (control (no prior exercise), prior moderate, prior heavy, prior sprint) in a random order to complete the experimentation. A subgroup of six subjects performed two further performance trials on separate days to establish the trial's reliability.

All testing was performed on an electrically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) in a well-ventilated laboratory at a temperature of 21–25°C. Each cyclist adjusted the ergometer for comfort, including the fitting of their own pedals, and the adjustments were recorded and replicated during subsequent tests. After the measurement of height and body mass, subjects performed a ramp test to determine $\dot{V}O_{2peak}$ and the GET. This test consisted of 3 min of pedaling at 0 W, followed by a continuous (ramp) increase in power output of 30 $W \cdot min^{-1}$ with the ergometer in its hyperbolic mode. Pulmonary gas exchange was measured breath by breath as described below and averaged over 10-s intervals. Subjects were instructed to maintain a cadence of 90 ± 2 rpm throughout this test, and the test was terminated if the subject could no longer maintain 80 rpm despite strong verbal encouragement. In all cases, this fall in cadence was precipitous. The $\dot{V}O_{2peak}$ was determined as the highest $\dot{V}O_2$ measured over 30 s, and the GET was determined using the V-slope method (3), where the threshold is determined as the first disproportionate increase in $\dot{V}CO_2$ relative to $\dot{V}O_2$. The work rates for the subsequent trials at moderate (80% GET), heavy (at a power output designed to elicit a $\dot{V}O_2$ halfway between the GET and $\dot{V}O_{2peak}$), and severe (at a power output designed to elicit a $\dot{V}O_2$ that was 70% of the difference between GET and $\dot{V}O_{2peak}$ − 70% Δ) power outputs were calculated by linear regression of $\dot{V}O_2$ versus power output.

Three prior exercise interventions were performed by the subjects, each intended to result in a different baseline blood [lactate]. On separate days, subjects performed each intervention 10 min before the performance trial (Fig. 1). The interventions chosen were: 1) a 6-min bout of heavy exercise; 2) moderate exercise (performed for 10–12 min, to complete the same amount of external work as done during heavy exercise); and 3) a 30-s all-out sprint. Strong verbal encouragement and feedback on the time elapsed were provided during the sprint. At the end of each of these conditions, subjects were allowed to "spin" their legs at 0 W for

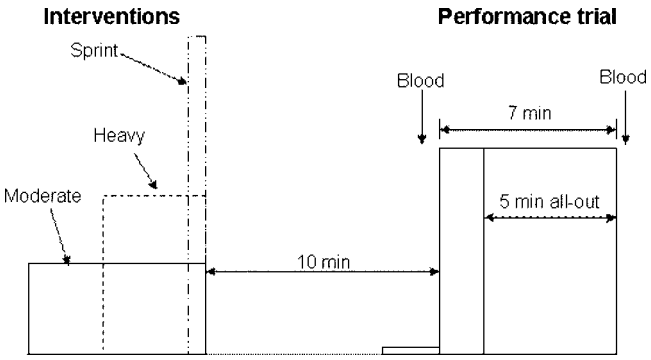

FIGURE 1—Schematic representation of the experimental protocol. The performance trial was preceded by 3 min of unloaded pedaling, followed by an abrupt increase in power output that was maintained constant for 2 min before the ergometer switched to its rpm-dependent mode for the next 5 min. The interventions shown were performed on separate days.

1 min after each intervention, and were then instructed to dismount the ergometer and recover passively. Subjects therefore chose to either sit or stand reasonably still during this period. After 7 min of recovery, the 3-min baseline-pedaling phase of the performance trial began. Power output abruptly increased to 70% Δ exactly 10 min after the cessation of the intervention, and thus the performance trial began.

The performance trial was 7 min in duration. It was designed to achieve two aims: 1) to collect relevant gas exchange data at a constant work rate and 2) to measure cycling performance during a short (5 min) self-paced effort. The first 2 min of the performance trial was performed at a work rate demanding a $\dot{V}O_2$ of 70% Δ (~90% $\dot{V}O_{2peak}$), wherein subjects were instructed to maintain a cadence of 90 ± 2 rpm. A 2-min period was chosen to be sufficient to estimate the parameters of the primary $\dot{V}O_2$ response we have previously measured, but short enough to prevent the development of a significant slow component that would distort these parameters using the modeling procedure described below. After this 2-min period, the ergometer was switched to a mode in which power output was dependent on subjects' cadence, and the subjects then attempted to maximize power output over the remaining 5 min. The resistance applied to the flywheel was adjusted so that a cadence of 90 rpm would still elicit 70% Δ after the switch in modes using the linear factor parameter in the Lode software (linear factor (L) = power × cadence^{-2}). Subjects were instructed to produce as much work as possible over the 5-min period and were given strong verbal encouragement throughout these trials, as well as feedback on the time elapsed every 30 s. The total work done was recorded every 30 s during the performance trial.

Throughout the preliminary tests and the performance trials, pulmonary gas exchange was measured breath by breath. Subjects wore a nose clip and breathed through a low–dead space (90 mL), low-resistance (0.75 mm Hg·L^{-1}·s^{-1} at 15 L·s^{-1}) mouthpiece and impeller turbine assembly (Jaeger Triple V). The inspired and expired gas volume and gas concentration signals were continuously sampled at 100 Hz, the latter using paramagnetic (O_2) and infrared (CO_2) analyzers (Jaeger Oxycon Pro, Hoechberg, Germany) via a capillary line connected to the mouthpiece. These analyzers were calibrated before each test with gases of known concentration, and the turbine volume transducer was calibrated using a 3-L syringe (Hans Rudolph, Kansas City, MO). The volume and concentration signals were time aligned by accounting for the delay in capillary gas transit and analyzer rise time relative to the volume signal. Oxygen uptake, carbon dioxide output, and minute ventilation were calculated using standard formulae (2) and displayed breath by breath. Heart rate was measured every 5 s using short-range radio telemetry (Polar S610, Polar Electro Oy, Kempele, Finland). At 1 min before and immediately after the trial, a fingertip blood sample (~25 μL) was taken and subsequently analyzed for blood [lactate] using an automated lactate analyzer (YSI Stat 2300, Yellow Springs, OH). The coefficient of variation of this analyzer was 3.8% for 26 samples of clinical control human sera measured in duplicate in the physiological range (5 mM). Mean power output for the performance trials was calculated from the cumulative work output data.

The breath-by-breath data from the performance trials were used to estimate the $\dot{V}O_2$ kinetics with and without prior exercise interventions. The data were first manually filtered to remove outlying breaths. These were defined as breaths deviating by more than three standard deviations from the preceding five breaths. In performing this filtering, care was taken not to remove breaths that were clearly part of the kinetic trends at the onset of exercise. After this filtering, the breath-by-breath data were interpolated to provide second-by-second values and modeled using a modification of the procedure recently used by Rossiter et al. (25) to analyze heavy exercise data. The first 20 s after the work rate transition was removed from the analysis to eliminate the influence of the cardiodynamic (phase I) component of the response. The first 2 min of data (20–120 s) were then modeled with monoexponential function of the form:

$$\dot{V}O_2(t) = \dot{V}O_2(b) + A \times (1 - e^{-(t-TD)/\tau}) \quad [1]$$

where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at time t; $\dot{V}O_2(b)$ is the baseline $\dot{V}O_2$ measured in the 60 s before the transition in work rate; A, TD, and τ are the amplitude, time delay, and the time constant of the primary (phase II) response, respectively (see Fig. 2).

A monoexponential function was chosen in preference to more complex procedures because: 1) the cardiodynamic component was not of interest in the present study, 2) more complex models applied to single transition data are usually associated with poor statistical confidence in the parameter estimates, and 3) the switch from constant–work rate to rpm-dependent work prevented the formal mathematical analysis of data beyond the 2-min fitting window. All data from the control trials were time aligned and averaged to maximize the confidence in the parameter estimates in the control condition.

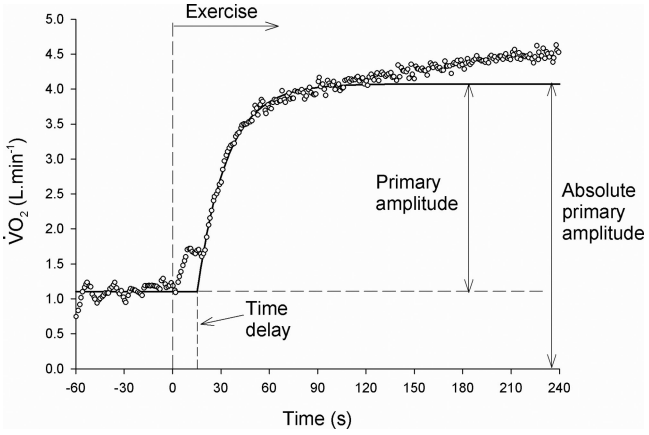

FIGURE 2—Modeled oxygen uptake response to severe cycle exercise in a representative subject labeled with the parameters of the $\dot{V}O_2$ response. The time constant (τ) represents the time taken to attain 63% of the primary amplitude, A, (because, from equation 1, when time (t) = τ, $\dot{V}O_2 = A \times (1 - e^{-1}) = A \times (0.63)$).

TABLE 1. Mean power output during each performance trial for all subjects.

Subject	Control (W)	After Moderate (W)	% Change Moderate vs Control	After Heavy (W)	% Change Heavy vs Control	After Sprint (W)	% Change Sprint vs Control
1	403	401	−0.3	403	0.1	394	−2.2
2	309	310	0.3	320	3.7	303	−1.9
3	395	400	1.4	410	3.8	378	−4.3
4	330	336	1.8	330	−0.1	316	−4.2
5	281	300	6.9	285	1.7	257	−8.3
6	304	309	1.9	325	6.9	311	2.5
7	360	360	0.0	360	0.0	365	1.3
8	311	321	3.4	316	1.6	304	−2.3
9	323	324	0.5	317	−1.8	302	−6.5
10	369	390	5.8	392	6.3	389	5.4
11	279	303	8.6	295	5.5	281	0.5
12	296	304	2.6	312	5.2	292	−1.6
Mean	330	338	2.7	339	2.7	324	−1.8
SD	42	39	2.9	42	2.9	45	3.8
95% confidence limits		13.9, 2.9*	4.6, 0.9*	14.7, 2.8*	4.6, 0.9*	2.3, −13.7	0.6, −4.2

Confidence limits of the same sign indicate a statistically significant difference between conditions, and are highlighted by an asterisk.

Estimates of differences between the experimental conditions were calculated using paired-samples 95% confidence intervals. The probability of Type I error was determined using a one-way repeated measures ANOVA. Results are therefore reported as means ± SD, followed by the paired-samples 95% confidence intervals of the differences between conditions, with intervals not including the null value indicating a statistically significant difference between conditions. The reliability of the performance trial in the subgroup of subjects was determined using the coefficient of variation ($(SD/\bar{x}) \times 100$), and the intraclass correlation coefficient. Data are presented as mean ± SD.

RESULTS

The subjects' $\dot{V}O_{2peak}$ (4.29 ± 0.40 $L \cdot min^{-1}$; 58 ± 4 $mL \cdot kg^{-1} \cdot min^{-1}$) GET (2.94 ± 0.33 $L \cdot min^{-1}$) and peak power output (395 ± 50 W) were indicative of their well-trained status and are typical of club-level cyclists in Great Britain (22). The work rates calculated for the moderate and heavy exercise bouts were 154 ± 24 and 282 ± 29 W, and the duration of the moderate exercise bout was 667 ± 50 s. Although the power output during the 30-s sprint exercise was not of specific interest, a mean power output of 600–700 W was typically recorded at the conclusion of the sprint.

The measured coefficient of variation for six subjects repeating the performance trial on three separate occasions suggests that an intervention yielding a performance change equal to or greater than 1.5 ± 0.7%, or ~5 ± 2 W at a control power output of 330 W, represents a measurable change. These data are supported by the intraclass correlation coefficient, which was r = 0.99 ($P < 0.001$, $N = 6$). Table 1 shows the mean performance of all subjects during both the control condition and after the three interventions. Significant main effects were observed for performance ($F_{3,11} = 10.41$, $P < 0.001$). Performance was improved after both moderate (by 8 W or 2.7%) and heavy exercise (by 9 W or 2.7%). However, performance was not significantly reduced after the prior sprint exercise (by −6 W or −1.8%; Table 1).

Figure 3 shows the change in individual performance after the interventions as a function of the blood [lactate] at the onset of the performance trials. The figure shows that performance generally declines if blood [lactate] is elevated above ~5 mM. In contrast, a blood [lactate] in the range of 1–4 mM was generally associated with a performance enhancement relative to the control condition.

In comparison with the control trial, baseline $\dot{V}O_2$ was similar after both moderate and heavy exercise but elevated after sprint exercise ($F_{3,11} = 8.36$, $P < 0.001$; Table 2). Both heavy and sprint exercise elevated baseline blood [lactate] to ~3 and ~6 mM, respectively ($F_{3,11} = 109.11$, $P < 0.001$), with the elevation after sprinting being significantly greater than after heavy exercise (mean difference 2.9 mM; 95% confidence intervals (3.6, 2.2 mM)). Thus, as intended, all three conditions yielded different baseline blood [lactate]. The baseline heart rate was elevated by both prior heavy and prior sprint exercise but not by prior moderate exercise ($F_{3,11} = 21.96$, $P < 0.001$; Table 2).

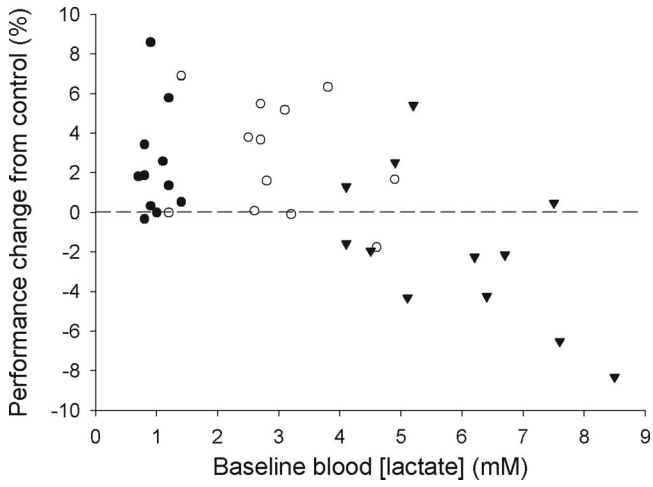

FIGURE 3—Cycling performance as a function of baseline blood [lactate] in all subjects. Notice that performance is improved by both prior moderate and heavy exercise in most subjects, despite blood [lactate] being elevated by up to 5 mM in the latter condition.

TABLE 2. Oxygen uptake, blood lactate, and HR responses to the performance trials.

Parameter	Control	After Moderate	After Heavy	After Sprint
Baseline				
Baseline $\dot{V}O_2$ ($L \cdot min^{-1}$)	1.11 ± 0.14	1.10 ± 0.15 (0.04, −0.06)	1.12 ± 0.15 (0.06, −0.05)	1.21 ± 0.10* (0.16, 0.05)
Baseline blood [lactate] (mM)	1.0 ± 0.3	1.0 ± 0.2 (0.17, −0.10)	3.0 ± 1.1* (2.6, 1.4)	5.9 ± 1.5* (5.8, 4.0)
Baseline HR ($b \cdot min^{-1}$)	93 ± 11	89 ± 13 (3, −10)	102 ± 13* (14, 4)	109 ± 16* (24, 8)
Primary response				
Primary amplitude ($L \cdot min^{-1}$)	2.59 ± 0.28	2.69 ± 0.27* (0.20, 0.01)	2.78 ± 0.26* (0.29, 0.09)	2.78 ± 0.30* (0.27, 0.11)
Absolute primary amplitude ($L \cdot min^{-1}$)	3.70 ± 0.34	3.79 ± 0.34* (0.15, 0.03)	3.90 ± 0.34* (0.28, 0.11)	4.00 ± 0.36* (0.39, 0.20)
Primary gain ($mL \cdot min^{-1} \cdot W^{-1}$)	8.7 ± 0.5	9.1 ± 0.6 (0.69, −0.001)	9.4 ± 0.7* (1.01, 0.29)	9.4 ± 0.7* (0.91, 0.38)
Primary τ (s)	23.5 ± 3.0	22.5 ± 3.5 (1.2, −3.2)	24.4 ± 4.9 (4.0, −2.2)	23.7 ± 4.5 (3.1, −2.7)
End-exercise responses				
Peak $\dot{V}O_2$ ($L \cdot min^{-1}$)	4.41 ± 0.37	4.46 ± 0.44 (0.14, −0.05)	4.40 ± 0.44 (0.10, −0.12)	4.45 ± 0.42 (0.12, −0.03)
End-exercise blood [lactate] (mM)	9.6 ± 1.6	10.0 ± 2.0 (1.3, −0.5)	10.0 ± 1.7 (1.3, −0.5)	10.2 ± 2.2 (1.5, −0.4)
End-exercise HR ($b \cdot min^{-1}$)	177 ± 10	178 ± 11 (3, −1)	181 ± 12* (6, 2)	177 ± 9 (2, −2)

Values are mean ± SD, and values in parentheses are the 95% paired-samples confidence limits of differences between control condition and each intervention. Confidence limits of the same sign indicate a statistically significant difference between conditions and are highlighted by an asterisk.

All three interventions resulted in an increase in the primary amplitude (both net ($F_{3,11} = 9.42$, $P < 0.001$) and absolute ($F_{3,11} = 27.59$, $P < 0.001$)). The effect of prior moderate exercise was smaller, though not significantly so, than after heavy or sprint exercise. None of the prior conditions altered the primary time constant ($F_{3,11} = 0.28$, $P = 0.61$; Table 2). The 95% confidence intervals for the parameter estimates of the $\dot{V}O_2$ response were (mean ± SD) control: amplitude, 42 ± 11 $mL \cdot min^{-1}$; τ, 2.3 ± 0.9 s; TD, 1.5 ± 0.5 s; after moderate: amplitude, 65 ± 23 $mL \cdot min^{-1}$; τ, 3.2 ± 1.2 s; TD, 2.0 ± 0.8 s; after heavy: amplitude, 70 ± 29 $mL \cdot min^{-1}$; τ, 3.8 ± 2.0 s; TD, 2.7 ± 1.5 s; after sprint: amplitude, 67 ± 27 $mL \cdot min^{-1}$; τ, 3.9 ± 2.2 s; TD, 2.9 ± 2.1 s. Representative $\dot{V}O_2$ responses are shown in Figure 4, which clearly illustrates that the effect of prior work was confined to the amplitude of the primary $\dot{V}O_2$ response.

Peak $\dot{V}O_2$, end-exercise HR, and end-exercise blood [lactate] responses were similar across conditions, with a small but significant elevation in the end-exercise HR observed after the performance trial after prior heavy exercise ($F_{3,11} = 5.82$, $P = 0.003$). As a result of these similarities, the change in blood [lactate] induced by the performance trial was substantially smaller after heavy and sprint exercise (Δ [lactate] control: 8.6 ± 1.5 mM; after moderate 9.0 ± 1.9 mM; after heavy 7.1 ± 0.8 mM; after sprint 4.3 ± 1.4 mM). The mean differences and paired-samples confidence inter-

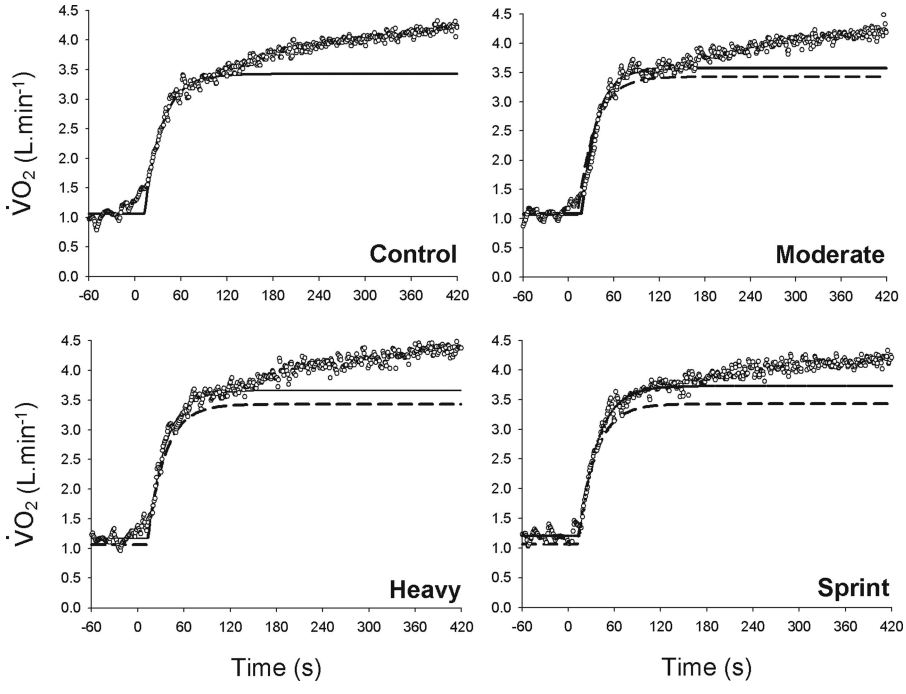

FIGURE 4—Oxygen uptake responses to each condition in a representative subject (subject 2). The control performance trial was performed with no prior exercise; moderate, heavy, and sprint performance trials were performed 10 min after moderate, heavy, and sprint exercise, respectively. The best-fit monoexponential function is superimposed on each response (*solid line*), with the best fit of the response to the control condition given as a *dashed line* in the moderate, heavy, and sprint plots. Note the increased amplitude of the $\dot{V}O_2$ response in each of the interventions.

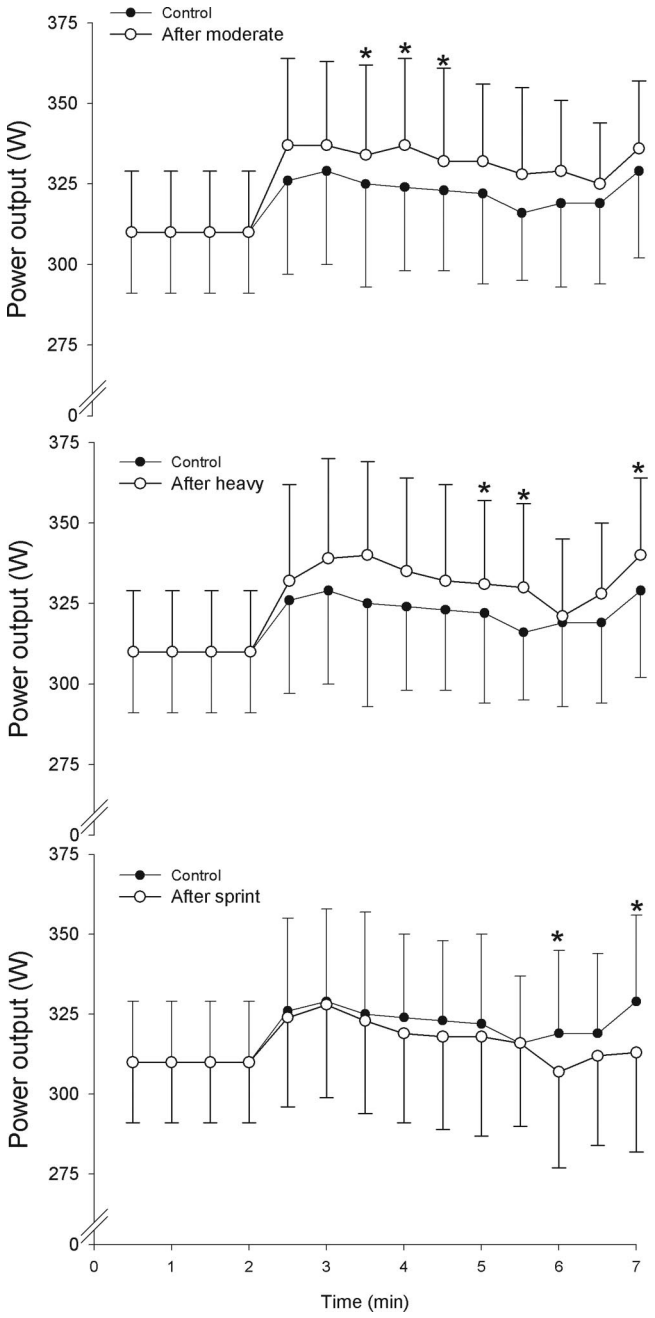

FIGURE 5—Power output profiles in each condition for 11 subjects (left panels, mean ± 95% confidence intervals). Note the higher power outputs after moderate and heavy exercise but the slightly lower power outputs in the after sprint condition; * significantly different from control condition (paired samples confidence intervals did not include zero).

vals for after heavy and after sprint compared with the control condition were −1.6 (−0.9, −2.3) mM and −4.4 (−3.5, −5.2) mM, respectively.

The power outputs measured over 30-s intervals in 11 subjects are presented in Figure 5 (technical difficulties prevented the cumulative work being recorded in one subject; in this case, only the mean power output was determined). The improved performance after both the moderate and heavy exercise interventions appeared to be due to the maintenance of a higher power output throughout the performance trial, rather than from the subjects adopting fundamentally different pacing strategies. In contrast, the mean performance data after the sprint showed a similar, if slightly lower, power output compared to the control condition until the last minute of the trial, at which point the subjects were unable to maintain or, as in case of all the other trials, raise the power output beyond control values in the last 30–60 s.

DISCUSSION

The principal original finding of the present investigation was that prior heavy exercise, which elevated baseline blood [lactate] to ~3 mM, and prior moderate exercise, which did not alter the baseline blood [lactate], improved severe-intensity cycling performance in a group of well-trained club cyclists. Prior sprint exercise, which resulted in an elevation of baseline blood [lactate] to ~6 mM, did not significantly diminish performance. All three interventions resulted in an elevation in the primary $\dot{V}O_2$ amplitude, without speeding the primary $\dot{V}O_2$ kinetics (i.e., τ was unaltered).

To test the experimental hypotheses required the design of a performance trial that would be sufficiently sensitive to detect performance changes of 1.5–2.0% (based on the assumption that the 30–60% change in time to exhaustion observed by Jones et al. (14) would result in an ~16-fold smaller change in mean power output (12)). Further, the performance trial was designed to allow the acquisition of breath-by-breath data to estimate the effect of each intervention on the primary $\dot{V}O_2$ kinetics. The results of the present work suggest that we were successful in achieving both of these aims: the coefficient of variation for three trials of the test (after habituation) was 1.5% on average, and the 95% confidence intervals for the estimation of the primary amplitude were sufficiently small (60–70 $mL \cdot min^{-1}$) to draw meaning from the changes we observed, despite using only one exercise transition. The reliability of the performance trial compares well with similar performance trials in the literature (cf. Hopkins et al. (12)). Passfield and Doust (22) demonstrated that a 5-min performance trial in a very similar group of subjects had a mean coefficient of variation of 1.7% and was sensitive to exercise-induced changes in gross efficiency. We did not intend to adopt a specific cycling event to measure "true" cycling performance. Instead, we adapted the performance trial of Passfield and Doust (22) because a 5-min effort after 2 min of constant–work rate exercise at 70% Δ (~90% $\dot{V}O_{2max}$) would most likely have placed the subjects in the severe-intensity domain during the performance, where it has been shown previously that prior heavy exercise is effective in altering both the $\dot{V}O_2$ response to exercise (26) and in extending time to exhaustion (14). The performance trial used here therefore seems to provide a reliable and sensitive means of determining the effect of an intervention on mean power output, while also allowing the investigator to collect meaningful gas exchange data for the assessment of the kinetic responses and to measure peak aerobic function.

The results of the present study provided partial support for our hypotheses. As predicted, prior heavy exercise, which led to a mild elevation in blood [lactate] at the onset of the performance trial, resulted in a substantial improvement in mean power output (of ~9 W, 2.7%). This finding is consistent with the work of Jones et al. (14), who demonstrated that prior heavy exercise, resulting in a similar baseline blood [lactate], extended the time to exhaustion during severe constant–work rate exercise by 30–60%. However, prior moderate exercise (in which the same total amount of external work was performed but without an elevation in blood [lactate]) also increased power output to a similar degree, in contrast to our hypothesis. The present study therefore suggests that, for performances that are dependent on maximal or perimaximal rates of aerobic metabolism, both moderate and heavy exercise are equally effective as "warm-up" procedures. Of importance in the present work is that many subjects benefit from a mild "acid-up," and in a few cases this "acid-up" may be even more effective than a classical moderate warm-up (Table 1 and Fig. 3, subjects 2, 3, 6, and 12).

The elevated primary $\dot{V}O_2$ amplitude has been consistently observed after prior exercise that induces a residual lactacidosis (6–9,14,26). A new and unexpected finding is that prior moderate exercise also led to a measurable increase in the primary amplitude. The mechanism(s) responsible for this effect is not clear, although an acidosis-mediated increase in O_2 delivery (11), increased substrate flux through the pyruvate dehydrogenase complex (PDC) (10), increased muscle temperature (20), and increased motor unit recruitment (6) have all been considered. However, neither elevated muscle temperature (20) nor increased substrate flux through the PDC (13,24) has been shown to increase the primary $\dot{V}O_2$ amplitude during heavy exercise. Burnley et al. (6) demonstrated that the increased primary $\dot{V}O_2$ amplitude after prior heavy exercise was associated with an increased leg muscle integrated electromyogram and evidence of vasodilatation in the first 2 min of heavy exercise. It is therefore possible that the increased primary $\dot{V}O_2$ amplitude observed in the present work reflects additional motor unit recruitment and/or improved homogeneity of muscle perfusion at exercise onset. The finding of a similar effect after prior moderate exercise in the present study suggests that a component of this increase in the primary $\dot{V}O_2$ amplitude may be independent of an exercise-induced acidosis. Additionally, that both prior moderate and heavy exercise improved performance to a similar extent despite markedly different baseline blood [lactate] in each condition suggests that the ergogenic effect of prior exercise may also be independent of a residual lactacidosis.

Cycling performance after prior sprint exercise, which elevated blood [lactate] to ~6 mM, was not significantly different from the control condition. However, 8 of the 12 subjects showed a diminished performance after sprint exercise (Table 1). A negative mean value (−6 W or −1.8%), coupled with a lower bound confidence interval of −14 W, suggests that prior sprint exercise may reduce performance relative to performing no prior work in some subjects. The effect of prior sprint exercise on endurance performance has not been previously measured. However, the present data are similar to the work of Koppo and Bouckaert (19), who demonstrated that prior exercise inducing a baseline plasma [lactate] of ~6.3 mM led to a nonsignificant reduction in time to exhaustion during constant–work rate exercise at 95% $\dot{V}O_{2max}$. More recently, Wilkerson et al. (26) demonstrated a significant reduction in time to exhaustion at 105% $\dot{V}O_{2max}$ after three bouts of maximal sprint exercise. In that study, blood [lactate] at the onset of exercise was ~7.7 mM. It therefore appears that prior exercise that induces a severe lactacidosis (baseline blood [lactate] above 5 mM, Fig. 3) may be associated with unchanged or reduced perimaximal exercise performance. However, this does not necessarily imply that lactate itself is causing fatigue: if it were, prior heavy exercise would also negatively affect performance, which was clearly not the case.

The present study has shown that the performance of both prior moderate exercise (which did not change the baseline blood [lactate]) and prior heavy exercise (which increased baseline blood [lactate] to ~3 mM) were equally effective in improving severe-intensity cycling performance in a sample of well-trained club-level cyclists. The improvement in performance after both prior moderate and heavy exercise was associated with an increase in the amplitude of the primary $\dot{V}O_2$ response. Although a similar $\dot{V}O_2$ response was evident after a 30-s bout of sprint exercise, this intervention, which elevated the baseline [lactate] to ~6 mM, did not significantly reduce exercise performance compared with control. This study has also shown that a protocol involving 2 min of constant–work rate exercise followed by a 5-min period of all-out cycling provides valid $\dot{V}O_2$ kinetic data and a reliable measure of severe-intensity cycling performance. The present study suggests that both moderate and heavy exercise can be considered as effective warm-up regimes and that some individuals may perform most effectively under conditions of a mild lactacidosis.

REFERENCES

1. Bangsbo, J., P. Krustrup, J. Gonzalez-Alonso, and B. Saltin. ATP production and efficiency of human skeletal muscle during intense exercise: effect of previous exercise. *Am. J. Physiol.* 280: E956–E964, 2001.
2. Beaver, W. L., K. Wasserman, and B. J. Whipp. On-line computer analysis and breath-by-breath display of exercise function tests. *J. Appl. Physiol.* 34:128–132, 1973.
3. Beaver, W. L., K. Wasserman, and B. J. Whipp. A new method for detecting anaerobic threshold by gas exchange. *J. Appl. Physiol.* 60:2020–2027, 1986.
4. Bishop, D. Warm up I: potential mechanisms and the effects of passive warm up on exercise performance. *Sports Med.* 33:439–454, 2003.
5. Bishop, D., D. Bonetti, and B. Dawson. The effect of three different warm-up intensities on kayak ergometer performance. *Med. Sci. Sports Exerc.* 33:1026–1032, 2001.
6. Burnley, M. J. H. Doust, D. Ball, and A. M. Jones. Effects of prior exercise on $\dot{V}O_2$ kinetics during the on-transient of heavy exercise are related to changes in muscle activity. *J. Appl. Physiol.* 93:167–174, 2002.

7. BURNLEY, M., J. H. DOUST, H. CARTER, and A. M. JONES. Effects of prior exercise and recovery duration on oxygen uptake kinetics during heavy exercise in humans. *Exp. Physiol.* 86:417–425, 2001.
8. BURNLEY, M., J. H. DOUST, and A. M. JONES. Effects of prior heavy exercise, prior sprint exercise and passive warming on oxygen uptake kinetics during heavy exercise in humans. *Eur. J. Appl. Physiol.* 87:424–432, 2002.
9. BURNLEY, M., A. M. JONES, H. CARTER, and J. H. DOUST. Effects of prior heavy exercise on phase II pulmonary oxygen uptake kinetics during heavy exercise. *J. Appl. Physiol.* 89:1387–1396, 2000.
10. CAMPBELL-O'SULLIVAN, S. P., D. CONSTANTIN-TEODOSIU, N. PEIRCE, and P. L. GREENHAFF. Low intensity exercise in humans accelerates mitochondrial ATP production and pulmonary oxygen kinetics during subsequent more intense exercise. *J. Physiol.* 538:931–939, 2002.
11. GERBINO, A., S. A. WARD, and B. J. WHIPP. Effects of prior exercise on pulmonary gas exchange kinetics during high-intensity exercise in humans. *J. Appl. Physiol.* 80:99–107, 1996.
12. HOPKINS, W. G., J. A. HAWLEY, and L. M. BURKE. Design and analysis of research on sport performance enhancement. *Med. Sci. Sports Exerc.* 31:472–485, 1999.
13. JONES, A. M., K. KOPPO, D. P. WILKERSON, S. WILMSHURST, and I. T. CAMPBELL. Dichloroacetate does not speed phase-II pulmonary $\dot{V}O_2$ kinetics following the onset of heavy intensity cycle exercise. *Pflugers Arch. Eur. J. Physiol.* 447:867–874.
14. JONES, A. M. D. P. WILKERSON, M. BURNLEY, and K. KOPPO. Prior heavy exercise enhances performance during subsequent peri-maximal exercise. *Med. Sci. Sports Exerc.* 35:2085–2092, 2003.
15. KARLSSON, J., F. BONDE-PETERSEN, J. HENRIKSSON, and H. G. KNUTTGEN. Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. *J. Appl. Physiol.* 38:763–767, 1975.
16. KOGA, S., T. SHIOJIRI, N. KONDO, and T. J. BARSTOW. Effects of increased muscle temperature on oxygen uptake kinetics during exercise. *J. Appl. Physiol.* 83:1333–1338, 1997.
17. KOPPO, K., and J. BOUCKAERT. In humans the oxygen uptake slow component is reduced by prior exercise of high as well as low intensity. *Eur. J. Appl. Physiol.* 83:559–565, 2000.
18. KOPPO, K., and J. BOUCKAERT. The effect of prior high-intensity cycling exercise on the $\dot{V}O_2$ kinetics during high-intensity cycling exercise is situated at the additional slow component. *Int. J. Sports Med.* 22:21–26, 2001.
19. KOPPO, K., and J. BOUCKAERT. The decrease in the $\dot{V}O_2$ slow component induced by prior exercise does not affect the time to exhaustion. *Int. J. Sports Med.* 23:262–267, 2002.
20. KOPPO, K., A. M. JONES, L. VANDEN BOSSCHE, and J. BOUCKAERT. Effect of prior exercise on $\dot{V}O_2$ slow component is not related to muscle temperature. *Med. Sci. Sports Exerc.* 34:1600–1604, 2002.
21. MACDONALD, M., P. K. PEDERSEN, and R. L. HUGHSON. Acceleration of $\dot{V}O_2$ kinetics in heavy submaximal exercise by hyperoxia and prior high-intensity exercise. *J. Appl. Physiol.* 83:1318–1325, 1997.
22. PASSFIELD, L., and J. H. DOUST. Changes in cycling efficiency and performance after endurance exercise. *Med. Sci. Sports Exerc.* 32:1935–1941, 2000.
23. ROBERGS, R. A., D. D. PASCOE, D. L. COSTILL, et al. Effects of warm-up on muscle glycogenolysis during intense exercise. *Med. Sci. Sports Exerc.* 23:37–43, 1991.
24. ROSSITER, H. B., S. A. WARD, F. A. HOWE, et al. Effects of dichloroacetate on $\dot{V}O_2$ and intramuscular ^{31}P metabolite kinetics during high-intensity exercise in humans. *J. Appl. Physiol.* 85: 1105–1115, 2003.
25. ROSSITER, H. B., S. A. WARD, J. M. KOWALCHUK, F. A. HOWE, J. R. GRIFFITHS, and B. J. WHIPP. Effects of prior exercise on oxygen uptake and phosphocreatine kinetics during high-intensity knee-extension exercise in humans. *J. Physiol.* 537:291–304, 2001.
26. WILKERSON, D. P., K. KOPPO, T. J. BARSTOW, and A. M. JONES. Effect of prior multiple sprint exercise on pulmonary O_2 uptake kinetics following the onset of peri-maximal exercise. *J. Appl. Physiol.* 97:1227–1236, 2004.