Changes in cycling efficiency and performance after endurance exercise

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ABSTRACT

PASSFIELD, L., and J. H. DOUST. Changes in cycling efficiency and performance after endurance exercise. *Med Sci. Sports Exerc.*, Vol. 32, No. 11, pp. 1935–1941, 2000. **Purpose:** This study was designed to examine the effects of moderate-intensity endurance exercise on cycling performance, gross efficiency, and 30-s sprint power output. **Methods:** Two separate experiments were conducted. After a controlled warm-up, subjects completed as much work as possible in a 5-min performance test (EXP1) or a maximal 30-s sprint test (EXP2). These initial exercise bouts were followed by ~60 min of cycling at ~60% $\dot{V}O_{2peak}$ or an equivalent period of rest (control) before repeating the warm-up exercise and either the 5-min performance or 30-s sprint test. Expired gas for calculation of cycling gross efficiency was collected over the last minute of each warm-up period. **Results:** Average 5-min performance power output was significantly reduced (12 W) after exercise in EXP1, and in EXP2 both peak and mean power output were significantly lower (26 and 35 W, respectively). Gross efficiency decreased significantly with exercise in both EXP1 and EXP2. Moreover, the change in gross efficiency was correlated with the change in 5-min performance ($r = 0.91$, $P < 0.01$), but not with the change in mean or peak 30-s sprint power output. **Conclusions:** After sustained moderate-intensity cycling significant reductions in 5-min performance, gross efficiency and sprint power output were observed in endurance trained cyclists. The reduction in 5-min performance was related to the exercise induced decrease in gross efficiency. **Key Words:** BLOOD LACTATE, GROSS EFFICIENCY, PEAK POWER OUTPUT, PERFORMANCE

Gross efficiency, defined as the ratio of power output to power input, has been demonstrated to influence cycling performance (21). Horowitz et al. (21) compared two groups of cyclists with a similar mean performance $\dot{V}O_2$ (4.46 vs 4.48 $L \cdot min^{-1}$) but significantly different gross efficiencies (20.4% vs 21.9%). They found average power output in a 1-h cycling performance test to be significantly better (342 vs 315 W) for the group with the higher efficiency. Gross efficiency may also be reduced by prior exercise, as sustained moderate-intensity cycling has been shown to result in an unexplained increase in $\dot{V}O_2$ (15,16). These observations raise the possibility that, after moderate-intensity endurance cycling, gross efficiency may be reduced and in turn impair performance. However, the effects of moderate-intensity endurance cycling on performance and gross efficiency have not been established, particularly in trained cyclists who regularly perform exercise of this nature.

During repeated submaximal isometric exercise, a marked increase in $\dot{V}O_2$ is observed, which is accompanied by a decline in maximum voluntary force (36). It is possible that the increase in $\dot{V}O_2$ during sustained cycling exercise (15,16) may be related to a reduction in the power-generating capacity of the active muscle mass in a similar manner. Previous research investigating the effects of dynamic exercise on subsequent sprint performance has focused primarily on short duration prior exercise (4,32) or high-intensity endurance exercise (28,29). Consequently, the effects of moderate-intensity endurance cycling on peak power output have not been clearly established.

The present study was undertaken to determine the effects of moderate-intensity endurance cycling on gross efficiency, a 5-min performance test, and sprint power output.

METHODS

Subjects. This study consisted of two separate experiments. Subjects for both experiments were men engaged in regular endurance cycle exercise at least 3–4 times per week. Most of the subjects were competitive cyclists in pre-season training, routinely exercising for more than 90 min. Subject details for the two experiments are presented in Table 1. Ten subjects gave written informed consent to take part in experiment 1 (EXP1) and nine in experiment 2 (EXP2).

Preliminary testing. To determine their blood lactate threshold and power output for the subsequent exercise condition, the subjects performed a stepwise incremental test. Each subject completed five or six stages lasting 5 min estimated to elicit from 40 to 80% $\dot{V}O_{2peak}$. Blood lactate

0195-9131/00/3211-1935/0
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Submitted for publication August 1999.
Accepted for publication February 2000.

TABLE 1. Subject details.

	Mass (kg)	Height (m)	MPO (W)	$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	% $\dot{V}O_{2peak}$ at LT
EXP1						
Mean	69.0	1.79	347	4.11	60.4	70%
SD	13.4	0.07	45	0.50	7.4	6%
EXP2						
Mean	69.0	1.78	381	4.42	64.3	69%
SD	6.5	0.04	43	0.36	4.7	6%

LT, lactate threshold.

threshold was calculated as described by Coyle et al. (7) as the work rate eliciting an increase in blood lactate concentration of 1 $mmol \cdot L^{-1}$ above exercising baseline. Expired gas and a blood sample were collected during the last minute of each stage. Maximal oxygen consumption ($\dot{V}O_{2peak}$) and associated maximal power output (MPO) were measured in a separate continuous incremental cycle test to exhaustion. During this test, work rate was incremented every 30 s by approximately 10 W, with expired gas and power output measured throughout. Expired gas samples were collected in Douglas bags for a minimum of 40 s, and $\dot{V}O_{2peak}$ was taken as the highest value. The MPO was recorded as the highest power output averaged over 60 s.

Cycle ergometer. All pre and experimental testing was performed on a friction braked cycle ergometer (model 818E. Monark, Varberg, Sweden). This ergometer had been modified with an infinitely adjustable saddle height, a racing saddle, the cyclists' own pedals, and power-measuring cranks (SRM, Julich, Germany). This power-measuring system calculated power output continuously from crank torque and angular velocity. Before this study, the precision and reliability of the power-measuring cranks were verified dynamically by comparison with a motor driven friction brake up to 625 W (23). In agreement with the findings of others (25), an $R^2 > 0.99$ and close agreement was found between the two systems.

Study design. In both EXP1 and EXP2, subjects completed an exercise and a control condition in a randomly ordered, cross-over manner. For both the exercise and control conditions, subjects completed a controlled warm-up immediately followed by either a 5-min performance test (EXP1) or a 30-s sprint test (EXP2). In both experiments, the initial tests were followed by ~60 min of cycling at ~60% $\dot{V}O_{2peak}$ (exercise) or an equivalent period of rest (control) after which the controlled warm-up exercise and 5-min or 30-s sprint tests were repeated. All subjects completed at least two familiarization tests before the experimental work in order to ensure they were accustomed to its strenuous nature.

EXP1 protocol. In EXP1, the subjects warmed up by cycling at their preferred cadence for 6 min at 60% $\dot{V}O_{2peak}$. The warm-up was immediately followed by a 5-min performance test in which the subjects completed as much work as possible during 5 min. Subjects were then allowed 3-min recovery before either continuing at 60% $\dot{V}O_{2peak}$ (exercise) or resting (control) for 69 min. Immediately after the exercise and control conditions, the subjects repeated the 6 min warm-up and 5-min test. The braking load on the ergometer for the 5-min tests was set to elicit 90% of each subject's MPO at a cadence of 95 $rev \cdot min^{-1}$. During the familiarization trials, subjects were encouraged to produce an evenly paced effort for maximum performance (10). Power output was recorded at 1-s intervals throughout the test, and performance was measured as the average power output for 5 min.

EXP2 protocol. Warm-up in EXP2 consisted of 10 min at the same power output as the exercise condition but at a fixed cadence of 100 $rev \cdot min^{-1}$. The warm-up was extended from EXP1 to allow more time for muscle temperature to stabilize as variations are known to alter the peak power output that can be generated (31). Muscle temperature has been shown to increase exponentially when exercising at 60% VO_{2peak}, tending to plateau at 10 min (20). Cadence was standardized at 100 $rev \cdot min^{-1}$ during the warm-up to allow the subjects to start sprinting at close to their likely optimal for peak power output (33). Immediately on completion of the warm-up, subjects sprinted against a braking load of 9% body weight, as this has been reported to be optimal for peak power generation in male subjects (9). The subjects were instructed to generate their peak power output as quickly as possible and to maintain this effort for the remainder of the 30 s. After the sprint test, the subjects either recovered for 3 min before continuing at approximately 60% $\dot{V}O_{2peak}$ using their preferred cadence (90, SD 7 $rev \cdot min^{-1}$) for 60 min (exercise) or rested for an equivalent time (control). After the exercise and control conditions, the subjects repeated the warm-up exercise and sprint test. Power output was recorded at 1-s intervals throughout the sprint test and peak power output (highest 1-s value), mean power output (30-s average), and fatigue index (% change from peak power to end of sprint) were calculated. The cadence at peak power output, peak cadence, and the time taken to reach peak power output were also recorded.

Data collection. Expired gas was collected during the final minute of warm-up periods in both EXP1 and EXP2 for the calculation of gross efficiency. In EXP1 expired gas was also collected from 45 s during the 5-min performance tests and at 25 and 50 min of the exercise condition. A blood sample was obtained during the last min of all warm-up periods and 1 or 3 min after the 5-min and 30-s performance tests respectively, for the determination of blood lactate concentration. In EXP1, seven subjects consented to the measurement of rectal temperature (T_R) during exercise which was measured with a rectal thermistor inserted 10 cm beyond the anal sphincter and recorded at 10-min intervals.

TABLE 2. Mean data for warm-up exercise before and after each condition in EXP1 and EXP2.

	Control			Exercise		
	Before	After	Change	Before	After	Change
EXP1 warm-up						
$\dot{V}O_2$ ($L \cdot min^{-1}$)	2.45	2.56	0.11	2.37	2.62	0.25**
SD	(0.27)	(0.22)	(0.17)	(0.32)	(0.33)	(0.13)
RER	0.97	0.98	0.01	0.97	0.94	−0.03**
SD	(0.02)	(0.03)	(0.02)	(0.04)	(0.05)	(0.03)
Gross efficiency %	22.2	21.6	−0.6	22.6	20.7	−1.8**
SD	(1.64)	(1.61)	(0.5)	(0.91)	(1.07)	(0.8)
Blood lactate ($mmol \cdot L^{-1}$)	1.6	2.1	0.5	1.3	1.4	0.1
SD	(0.5)	(0.5)	(0.6)	(0.6)	(1.0)	(0.7)
EXP2 warm-up						
$\dot{V}O_2$ ($L \cdot min^{-1}$)	2.93	2.94	0.01	2.97	3.11	0.14*
SD	(0.33)	(0.34)	(0.09)	(0.35)	(0.31)	(0.12)
RER	0.99	0.99	0.0	0.97	0.94	−0.03*
SD	(0.04)	(0.04)	(0.02)	(0.03)	(0.04)	(0.03)
Gross efficiency %	21.8	21.7	−0.1	21.8	20.8	−1.0**
SD	(0.7)	(0.9)	(0.5)	(0.8)	(1.4)	(0.8)
Blood lactate ($mmol \cdot L^{-1}$)	2.2	2.1	−0.1	2.1	1.5	−0.6**
SD	(1.0)	(0.7)	(0.7)	(0.7)	(0.5)	(0.5)

* $P < 0.05$, ** $P < 0.01$, for exercise compared with control conditions.

Laboratory conditions. All the exercise was conducted in a well-ventilated laboratory maintained at 19°C (SD 1), with the subjects cooled by a fan. During exercise conditions in EXP1 and EXP2, subjects consumed 10 $mL \cdot kg^{-1}$ body $mass \cdot h^{-1}$ of an 8% glucose polymer solution. A standard drink volume of 500 mL, with a similar carbohydrate content to the exercise condition, was consumed during the control condition.

Analytical techniques. Gross efficiency was calculated as the ratio of power output to power input and expressed as a percentage. Power input was determined as the rate of energy expenditure using indirect calorimetry. The rate of energy expenditure was calculated from $\dot{V}O_2$ and respiratory exchange ratio (RER) using the formula of Lusk (24). All $\dot{V}O_2$ and RER measurements were made from expired gas samples collected in 200-L Douglas bags. The Douglas bags were flushed with expirate before use, and gas collections were made over a whole number of respiratory cycles via a rubber mouthpiece and low-resistance T-shaped valve box (University of Brighton, UK). The O_2 and CO_2 concentrations were determined directly from the Douglas bag with paramagnetic and infrared analyzers, respectively (series 1100 and 1490, Servomex, Crowborough, UK). The gas analyzers were calibrated before use with certified calibration gases and outside air. All gases were passed through a condenser (Bühler PKE 3, Ratingen, Germany) to control water vapor pressure before analysis. The gas volume was measured by evacuating the Douglas bag through a dry gas volume meter (Harvard Apparatus Ltd., Edenbridge, UK) that had previously been calibrated with a 7-L gas syringe (Hans Rudolph Inc., Kansas City, MO). Power output, cadence, and heart rate were recorded continuously and stored at 1-s intervals using the power-measuring cranks. All blood lactate concentrations were determined from 20-μL thumb-prick capillary samples enzymatically (YSI 2300, Yellow Springs Instruments, OH).

Statistical analysis. Shapiro-Wilks' tests were conducted before statistical analysis and confirmed that all data were normally distributed. To maximize statistical power, an *a priori* planned comparison of the change resulting from exercise and control conditions was employed. The change in variables was calculated as the difference between before and after each condition. A paired *t*-test was used to detect significant differences between the change in exercise and control conditions. Based upon preliminary testing, eight subjects were required for 80% statistical power with $P < 0.05$ considered significant. Additionally, in EXP2 differences in cadence and time to peak power output between sprint tests were examined using a two-way ANOVA with repeated measures. Where appropriate, the relation between variables was examined by Pearson's correlation coefficient. Statistical significance was accepted if $P < 0.05$ was found. All data are presented as mean and SD.

RESULTS

EXP1 warm-up. The mean data for warm-up exercise and the change for both exercise and control conditions in EXP1 are presented in Table 2. The $\dot{V}O_2$ during warm-up before the exercise condition was 2.37 $L \cdot min^{-1}$ representing 58 (SD 4)% $\dot{V}O_{2peak}$. After exercise, $\dot{V}O_2$ had increased (0.24 $L \cdot min^{-1}$) and RER was lower; these changes being significantly greater than for the control condition ($P < 0.01$). Power output was similar at all times in both conditions. Consequently, the fall in gross efficiency was significantly greater for exercise compared with the control condition ($P < 0.01$). The change in blood lactate concentration between the warm-up periods was not significantly different for exercise and control conditions. During exercise, gross efficiency declined from 21.5% (SD 1.1%) at 25 min to 21.1% (SD 1.1%) at 50 min ($P < 0.01$), whereas T_R rose initially and then leveled out at approximately 38.5°C for the last 25 min.

Five-minute performance test. Average 5-min performance power output (Fig. 1) was reduced by 12 W after exercise but was identical before and after the control condition ($P < 0.01$ for the change in exercise vs control conditions). No significant differences in average and peak

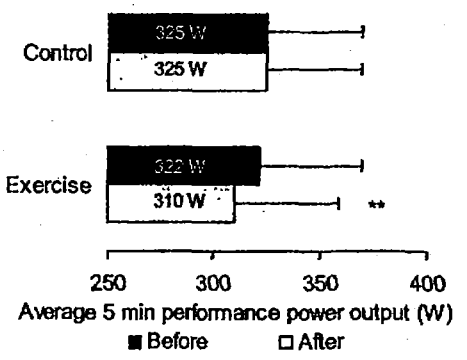

Figure 1—Average 5-min performance power output before and after exercise and control conditions in EXP1. Data are mean and SD; ** $P < 0.01$ for the change in exercise compared with control.

values of heart rate and $\dot{V}O_2$, measured during the 5-min performance tests or post test blood lactate concentrations were found between exercise and control conditions (data not shown). A significant correlation between the change in gross efficiency and the change in average 5-min performance power output was found for the exercise condition ($r = 0.73$, $P < 0.05$). This correlation was strengthened by the removal of a significant outlier (34) to $r = 0.91$ ($P < 0.01$, Fig. 2). No significant correlation was found between the change in average 5-min performance power output for the exercise condition and $\dot{V}O_{2peak}$, MPO, blood lactate threshold, or RER measured during exercise. However, average 5-min performance power output was strongly correlated with MPO ($r = 0.95$, $P < 0.01$), and $\dot{V}O_{2peak}$ ($r = 0.93$, $P < 0.01$). A weaker correlation was observed between average 5-min performance power output and $\dot{V}O_2$ at blood lactate threshold ($r = 0.80$, $P < 0.01$).

EXP2 warm-up. The data for the warm-up exercise for exercise and control conditions in EXP2 are also presented

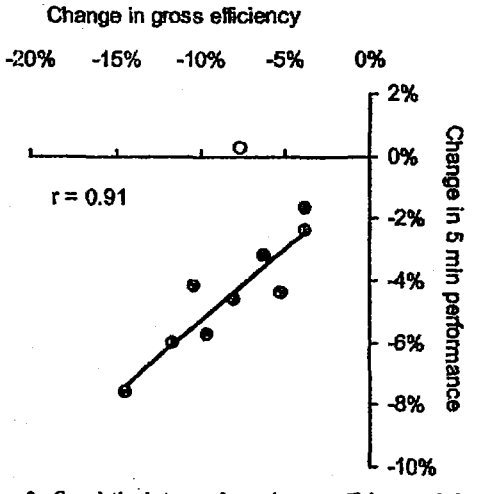

Figure 2—Correlation between change in gross efficiency and change in average 5-min performance power output for the exercise condition in EXP1. Regression line is shown for data with the outlying point (O) omitted.

in Table 2. The $\dot{V}O_2$ during the first warm-up for exercise and control conditions was equivalent to 67 (SD 4.5)% $\dot{V}O_{2peak}$. The changes between the first and second warm-up in the control condition were minor for $\dot{V}O_2$, RER, gross efficiency, and blood lactate. After exercise, $\dot{V}O_2$ increased while RER, gross efficiency, and blood lactate all decreased. Consequently, the changes in $\dot{V}O_2$ ($P < 0.05$), RER ($P < 0.05$), gross efficiency ($P = 0.01$), and blood lactate ($P = 0.01$) were all greater for exercise compared with the control condition.

Thirty-second sprint test. Data for the 30-s sprint test are shown in Table 3. Peak and mean power output were similar for the two sprints in the control condition but were reduced by 26 W and 35 W, respectively, after exercise. Accordingly, exercise resulted in a significant decrement in peak power output ($P < 0.05$) and mean power output ($P = 0.01$) compared with the control condition. The fatigue index of the sprints was similar at all times ($P = 0.4$). No difference was found in cadence at peak power output, maximum sprint cadence, or in time to peak power output between any of the sprints. A greater reduction in postsprint blood lactate concentration was found after exercise compared with the control condition ($P < 0.01$). No significant correlation was found between the change in either peak or mean sprint power output and the change in gross efficiency for the exercise condition.

DISCUSSION

Gross efficiency. In agreement with previous research (15,16), the present study found moderate-intensity endurance cycling consistently resulted in an increase in $\dot{V}O_2$. This increase in $\dot{V}O_2$ was associated with a significant decrease in gross efficiency. The cause of this reduction in gross efficiency is not known but may arise directly from the active muscle mass (12). No significant correlation was found between the change in gross efficiency and the change in peak power output. Thus, the reduction in gross efficiency does not seem to be related to the loss in maximal muscle function as reported for isometric exercise (36). It is noted, however, that the decline in gross efficiency and mean sprint power output were of a similar magnitude (~5%). A significant reduction in gross efficiency appears to occur regardless of cycling fitness, as the $\dot{V}O_{2peak}$ of the subjects in the present experiments (60 and 64 $mL \cdot kg^{-1} \cdot min^{-1}$) was appreciably higher than other studies (52 and 55 $mL \cdot kg^{-1} \cdot min^{-1}$) reporting a similar effect (15,16). Previous research has concluded that increases in fat metabolism, ventilation, body temperature, and lactate metabolism do not account for the increased oxygen cost of work after sustained moderate-intensity exercise (15,16). The present data support this conclusion. The calculation of gross efficiency takes into account changes in substrate utilization and variations in power output. The increased ventilation observed in this study (mean 8 $L \cdot min^{-1}$) was estimated to increase $\dot{V}O_2$ by a negligible 14 $mL \cdot min^{-1}$ O_2 (1). Neither changes in T_R nor blood lactate concentration were

TABLE 3. Mean sprint test data before and after each condition in EXP2.

Condition	Control			Exercise		
	Before	After	Change	Before	After	Change
Peak power output (W)	991	998	7	985	959	−27*
SD	(177)	(178)	(29)	(150)	(169)	(38)
Mean power output (W)	667	668	1	663	628	−35**
SD	(85)	(80)	(11)	(94)	(93)	(24)
Fatigue index (%)	58	58	0	55	56	1
SD	(17)	(17)	(4)	(14)	(13)	(3)
Lactate ($mmol \cdot L^{-1}$)	8.5	8.2	−0.3	8.6	6.6	−2.0**
SD	(1.6)	(1.3)	(1.1)	(1.2)	(1.5)	(1.3)
Peak $rev \cdot min^{-1}$	139	139	—	142	137	—
SD	(15)	(14)		(16)	(17)	
Time to peak power output (s)	3	4	—	4	3	—
SD	(1)	(1)		(1)	(1)	
Peak power output ($rev \cdot min^{-1}$)	123	122	—	125	124	—
SD	(8.7)	(7.8)		(13.9)	(9.1)	

* $P < 0.05$, ** $P = 0.01$, for exercise compared with control conditions.

consistent with the progressive decrease in gross efficiency observed during and after the exercise condition in EXP1.

Five-minute performance test. Average 5-min performance power output was reduced after exercise for 9 of the 10 subjects, with no change apparent in the control condition. This 12-W (4%) reduction in 5-min performance power output was observed despite the moderate-intensity and the trained status of the subjects. It is estimated using data from a recent study (3) that this change in performance corresponds to a reduction in cycling speed of 0.56 $km \cdot h^{-1}$ in a 1-h race. Notably, the subject for whom a reduction was not found reported his performance test before exercise might not have been maximal. Peak heart rate, $\dot{V}O_2$ and, post exercise blood lactate concentration for this performance test were all appreciably lower than in this subject's other tests. These lower responses before exercise were in contrast to those of the other subjects who showed little difference between tests. Indeed, the low coefficient of variation of 1.7% for the 5-min performance tests suggests this test was reliable. However, it is recommended that researchers employing this experimental protocol in the future brief subjects carefully on the need for a maximal effort in every test.

A new finding of this study was the significant correlation between the change in performance and gross efficiency ($r = 0.73$). This significant correlation was found despite the influence of the outlying data point caused by the subject discussed above. An outlier test (34) yielded a significant result ($P < 0.05$), and when the data were replotted with this point omitted, the correlation increased considerably to $r = 0.91$ ($P < 0.01$). It is noted, however, that the mean exercise induced change in 5-min performance (4%) was much less than the mean change in gross efficiency (8%). This difference may be due to a reduction in gross efficiency caused by the first 5-min performance test. Although the influence of gross efficiency on cycling performance has been previously demonstrated (21), this is the first study to demonstrate that an acute exercise-induced reduction in gross efficiency is associated with a concomitant decrease in performance. As average and peak $\dot{V}O_2$ during the 5-min performance test were not lower after exercise, a reduction in efficiency would be expected to cause a parallel decline in performance. These observations indicate the fatigue observed during moderate-intensity endurance cycling was related to a reduction in gross efficiency.

Thirty-second sprint test. The reason for the exercise-induced reductions in peak and mean power output observed in EXP2 remain unclear but are consistent with previous findings after higher-intensity endurance exercise (28,29). A force-velocity–mediated reduction in power output seems unlikely as no difference between sprints was found in either cadence at peak power output or peak sprint cadence (Table 3). The significant reduction in peak power output may indicate a substrate independent effect, as much of the energy for this part of the sprint is thought to be derived from ATP and phosphocreatine (11,27). Muscle ATP and phosphocreatine have been found to be reduced significantly by 12% and 63%, respectively, after submaximal endurance cycling in some studies (5,13,30), but not all (2,17). However, those studies that report a reduction in phosphocreatine were all conducted at a higher exercise intensity than EXP2. As muscle biopsy data were not collected in the present study, the cause of the exercise-induced reduction in peak power output cannot be further elucidated.

The reduction in post-sprint blood lactate concentration observed after exercise may indicate that glycogen depletion was responsible for the lower sprint mean power output. A combination of diet and exercise to reduce muscle glycogen content has been demonstrated to compromise high-intensity exercise and lactate production (22). However, there is no correspondence between the reduction in mean sprint power output (5%) and blood lactate concentration (23%) in the present data. A similar inconsistency between the decreased work done and muscle lactate concentrations has also been reported (22). This suggests the lower lactate concentrations are associated with, rather than causative in, the reduced performance. Furthermore, research into the effects of glycogen availability on maximal muscle function and sprint performance is equivocal (cf. 6,18,22,35). Muscle glycogen depletion to between 150 and 365 $mmol \cdot kg^{-1}$ dry mass does not alter sprint power output (18,35), although a reduced mean sprint power output has been demonstrated after extensive depletion to 25 $mmol \cdot kg^{-1}$ dry mass (6). However, the studies that find glycogen depletion does not

compromise high-intensity exercise tend to have controlled more carefully for the fatiguing effects of the depleting exercise by employing fully randomized crossover experimental designs. Muscle glycogen concentrations were not measured in the present study, but it is suggested that extensive depletion was unlikely. Cycling for 2 h at 60% $\dot{V}O_{2peak}$ has been reported to deplete muscle glycogen by 50% (17), whereas a 30-s sprint utilizes approximately 20% (14,26). Thus, the mechanisms responsible for the reduced sprint performance observed after moderate-intensity endurance cycling require further investigation.

Aerobic power and endurance performance. The importance of a high aerobic power as a determinant of cycling performance was evident in EXP1 from the strong correlation between the average 5-min performance power output and the preliminary measures of $\dot{V}O_{2peak}$, MPO, and blood lactate threshold. Similarly high correlations between indices of aerobic power and endurance cycling performance have been previously documented (7,8,19). In contrast, the lack of correlation between the exercise-induced reduction in performance and indices of maximal aerobic power is a new observation. This finding indicates that a high aerobic power is not associated with a superior ability to resist fatigue induced by moderate-intensity endurance cycling. Thus, endurance performance may be influenced not only by an individual's aerobic power but also the fatigue occurring during exercise. This fatigue may not be less marked in those with a high maximal aerobic power. Accordingly, future research may wish to consider separately the contributions of resistance to fatigue and maximal aerobic power to endurance performance.

CONCLUSIONS

This study has found that trained cyclists undertaking a bout of moderate-intensity endurance cycling experience a significant decrease in gross efficiency, 5-min performance, and sprint power output. The reduction in 5-min performance after exercise was associated with the decline in gross efficiency. However, the reasons for the exercise-induced reduction in gross efficiency and sprint power output require further investigation. A high maximal aerobic power was not related to superior resistance to fatigue during moderate-intensity endurance cycling but was associated with a higher average 5-min performance power output.

The authors gratefully acknowledge the cooperation of the cyclists who took part in this study. This study was supported by a University of Brighton research studentship.

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