

# Metabolic and performance responses to constant-load vs. variable-intensity exercise in trained cyclists

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**Palmer, Garry S., Lars B. Borghouts, Timothy D. Noakes, and John A. Hawley.** Metabolic and performance responses to constant-load vs. variable-intensity exercise in trained cyclists. *J. Appl. Physiol.* 87(3): 1186–1196, 1999.—We studied glucose oxidation ( $\text{Glu}_{\text{ox}}$ ) and glycogen degradation during 140 min of constant-load [steady-state (SS)] and variable-intensity (VI) cycling of the same average power output, immediately followed by a 20-km performance ride [time trial (TT)]. Six trained cyclists each performed four trials: two experimental bouts (SS and VI) in which muscle biopsies were taken before and after 140 min of exercise for determination of glycogen and periodic acid-Schiff's staining; and two similar trials without biopsies but incorporating the TT. During two of the experimental rides, subjects ingested a 5 g/100 ml [ $U\text{-}^{14}\text{C}$ ]glucose solution to determine rates of  $\text{Glu}_{\text{ox}}$ . Values were similar between SS and VI trials:  $\text{O}_2$  consumption ( $3.08 \pm 0.02$  vs.  $3.15 \pm 0.03$  l/min), energy expenditure ( $901 \pm 40$  vs.  $904 \pm 58$  J·kg<sup>-1</sup>·min<sup>-1</sup>), heart rate ( $156 \pm 1$  vs.  $160 \pm 1$  beats/min), and rating of perceived exertion ( $12.6 \pm 0.6$  vs.  $12.7 \pm 0.7$ ). However, the area under the curve for plasma lactate concentration vs. time was significantly greater during VI than SS ( $29.1 \pm 3.9$  vs.  $24.6 \pm 3.7$  mM/140 min;  $P = 0.03$ ). VI resulted in a 49% reduction in total muscle glycogen utilization vs. 65% for SS, while total  $\text{Glu}_{\text{ox}}$  was higher ( $99.2 \pm 5.3$  vs.  $83.9 \pm 5.2$  g/140 min;  $P < 0.05$ ). The number of glycogen-depleted type I muscle fibers at the end of 140 min was 98% after SS but only 59% after VI. Conversely, the number of type II fibers that showed reduced periodic acid-Schiff's staining was 1% after SS vs. 10% after VI. Despite these metabolic differences, subsequent TT performance was similar ( $29.14 \pm 0.9$  vs.  $30.5 \pm 0.9$  min for SS vs. VI). These results indicate that whole body metabolic and cardiovascular responses to 140 min of either SS or VI exercise at the same average intensity are similar, despite differences in skeletal muscle carbohydrate metabolism and recruitment.

carbohydrate; glucagon; glucose; free fatty acids; insulin; muscle glycogen

THE METABOLIC RESPONSES to prolonged (>90 min), constant-load, submaximal [ $<75\%$  maximal  $\text{O}_2$  uptake ( $\dot{V}\text{O}_{2\text{max}}$ )] exercise have been extensively investigated (7, 9, 16, 26). Furthermore, there is substantial evi-

dence to show that the ingestion of carbohydrate (CHO) supplements throughout such exercise can postpone the onset of fatigue (see Ref. 5 for review). However, far less is known about the physiological and metabolic responses to variable-intensity (VI) exercise in which the work rate fluctuates in a random fashion. Although steady-state (SS) exercise conditions may prevail in long-distance running races such as the marathon, most mass-start endurance cycle races are characterized by multiple changes of pace and intensity throughout the duration of an event, as shown by stochastic or variable shifts in the frequency and amplitude of the heart rate (HR) responses to such races (23).

We have previously shown that 20-km time-trial (TT) performance that followed 150 min of either SS or VI cycling, undertaken at the same average power output, was 6% faster after SS (22). At the time, we speculated that the repeated work jumps during VI may have been associated with an increased muscle glycogen utilization compared with SS exercise, but we lacked metabolic measurements to evaluate this theory.

Accordingly, the aims of the present investigation were, first, to evaluate the whole body metabolic and hormonal responses to prolonged (140 min) cycling in well-trained men who ingested CHO throughout both SS [ $\sim 70\%$  peak  $\dot{V}\text{O}_2$  ( $\dot{V}\text{O}_{2\text{peak}}$ )] and VI (40–85%  $\dot{V}\text{O}_{2\text{peak}}$ ) cycling of the same average intensity. Second, we wished to determine the effects of these two different exercise preloads on muscle carbohydrate metabolism and subsequent 20-km TT performance.

## MATERIALS AND METHODS

**Subjects.** Six male cyclists were recruited to participate in this investigation, which was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town. Subject characteristics are displayed in Table 1. Because radiolabeled tracers would be used and blood and muscle biopsy samples would be taken, the procedures and risks were carefully explained to each subject, and their written, informed consent was obtained. Each subject was well trained and had been participating in regular endurance cycle training (>2 h/day) and competition for at least 3 yr before the study.

**Preliminary testing.** Before their participation in this investigation, all subjects were required to undertake a progressive, incremental, maximal exercise test to volitional fatigue on a Kingcycle air-braked cycle simulator (Kingcycle, High Wycombe, Buckinghamshire, UK) for the determination of

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Table 1. *Subject characteristics*

Subject	Age, yr	Mass, kg	Height, m	PPO, W	$\dot{V}O_{2\text{peak}}$ , l/min	HR <sub>peak</sub> , beats/min
1	17	65	1.81	363	4.07	196
2	26	79	1.76	347	3.90	182
3	19	60	1.78	387	4.34	198
4	41	90	1.78	457	5.06	197
5	19	82	1.81	449	5.01	195
6	25	70	1.81	402	4.52	193
Mean	24.5	74.3	1.79	401	4.48	193
SD	8.8	11.3	0.02	44.7	0.48	5.89

PPO, peak power output;  $\dot{V}O_{2\text{peak}}$ , peak  $O_2$  consumption; HR<sub>peak</sub>, peak heart rate.

peak sustained power output (PPO),  $\dot{V}O_{2\text{peak}}$ , and peak HR (HR<sub>peak</sub>). The calibration procedures, as well as the reliability and validity of the Kingcycle ergometer, have been described in detail previously (21).

After completing a warm up of self-selected duration and intensity, subjects commenced the maximal test at a work rate of ~200 W. This work rate was increased by 20 W/min until subjects were no longer able to maintain the desired workload. The subjects' PPO,  $\dot{V}O_{2\text{peak}}$ , and HR<sub>peak</sub> were taken as the highest values sustained for any 60 s of the maximal test.

In addition to completing the maximal test, all subjects undertook a familiarization ride on a electromagnetically braked cycle ergometer (Lode, Gronigen, The Netherlands) which was adapted with clip-in pedals and with low profile and TT handlebars to match the subject's own riding position. Power output on the Lode ergometer is independent of pedal frequency between 60 and 120 rpm. The familiarization ride consisted of 50 min of VI exercise at the same average intensity ( $58 \pm 11\%$  PPO) that the individual would complete in the experimental trials. This ride was immediately followed by a 20-km TT on the Kingcycle ergometer; this was performed under the same laboratory conditions as those for all of the experimental trials, with the exception that muscle biopsies and blood samples were not taken.

Throughout the maximal test and during sections of the subsequently described experimental rides, subjects wore a noseclip and breathed through a mouthpiece attached to an Oxycon Alpha automated gas analyzer (Mijnhardt, The Netherlands). Before each ride was performed, the gas analyzer was calibrated with a Hans-Rudolph 5530 3-liter syringe and a 5%  $CO_2$ -95%  $N_2$  gas mixture. Analyzer outputs were processed by an IBM-compatible computer which calculated liters/minute ventilation rates ( $\dot{V}E$ ),  $\dot{V}O_2$ , and  $CO_2$  production ( $\dot{V}CO_2$ ) by using conventional equations.

The subject's HR was measured by a Polar Sports Tester HR monitor (Polar Electro, Kempele, Finland). This monitor consists of a transmitter, an electrode belt worn around the chest, and a wrist-mounted receiver that records and stores momentary HR at predetermined intervals. A time interval of 5 s was chosen for the maximal test, and an interval of 15 s was chosen for all experimental trials.

**Dietary analysis.** Before all experimental trials, each subject completed a 4-day dietary record. Subjects were given precise written and verbal instructions on how to record all food and fluid consumed for this period, which always included 1 day of the weekend. Using a commercial computer program [Food Finder Diet Analysis, Medtech, Tygerberg, Cape Town, Republic of South Africa (RSA)], a registered dietician determined the energy content and nutritional composition of each subject's diet. This analysis revealed that

the average energy intake was  $13.94 \pm 0.31$  MJ, while the breakdown of macronutrients was  $398 \pm 40$  g of CHO ( $51 \pm 3\%$  of total energy),  $129 \pm 20$  g of fat ( $34 \pm 2\%$  of energy), and  $133 \pm 9$  g of protein ( $16 \pm 1\%$  of energy).

**Standardization of testing.** To ensure that subjects presented for each experimental trial in the same nutritional and physical state, their diet and training load was strictly controlled for the 3 days before each trial. This was undertaken by providing each subject with 3 days of food that was already prepared (Nutriferit, Cape Town, RSA) and consisted of the same total energy content and composition as each subject's habitual diets (described previously) and by requesting the subjects to maintain the same training pattern for this period. Compliance with the dietary control was facilitated by instructing subjects to return all previously prepared food that they had not consumed and having them record any additional fluid and food they ingested. To ensure the same training was undertaken, subjects were requested to maintain a diary for each 3-day period before a trial. It has been our experience that well-trained subjects will still ride moderately hard the day before a laboratory trial, even when instructed to the contrary. Therefore, subjects refrained from all heavy exercise for the 24 h preceding an experimental trial and were given a HR monitor to record all activity during this period. If these HR records showed the subject had trained or had been involved in vigorous physical activity, the subject was not allowed to participate in an experiment until appropriately rested.

**Exercise trials.** All subjects completed a random crossover of four trials that were separated by exactly 7 days and were conducted at the same time of day. Subjects reported to the laboratory for each ride 3 h after a standardized breakfast that was similar in size and composition to one that they would normally ingest before competition (1.5 g/kg body mass CHO: 2 slices of toast, 1 cup of cereal with 125 ml of milk). Immediately before each experimental trial, subjects ingested 4 ml/kg body mass of a 5 g/100 ml CHO solution and then underwent a 5-min incremental warm up on the Lode ergometer. The warm up commenced at an intensity of 29% PPO (~116 W) and was increased at a rate of ~6% PPO (~24 W) every minute until the desired intensity for that trial was reached.

Figure 1 shows a schematic diagram of the testing protocols. The exercise intensity for each of the rides is represented as a percentage of PPO. The first 140 min of each ride consisted of either SS or VI (experimental). During VI exercise, subjects rode five 20-min bouts of VI exercise interspersed with four 10-min periods of work at a constant power output (58% PPO or ~65%  $\dot{V}O_{2\text{peak}}$ ). The average work rate during each 20-min period was  $58 \pm 13$  (SD)% PPO, with a range in power between 35 and 77% of PPO (~40 and 85%  $\dot{V}O_{2\text{peak}}$ ; Fig. 1). By design, the mean power output throughout the two rides, as calculated by the area under the curve of power vs. time, was the same for each subject:  $58 \pm 11$  (SD)% PPO or  $232 \pm 44$  W. Such a range in VI was chosen because it was similar to that observed in the field during mass-start road races (23) and the same as in a previous study that examined the effects of stochastic exercise on subsequent exercise performance (22).

Because we wished to determine both the metabolic and subsequent performance responses to 140 min of SS and VI, two of the 140-min experimental rides (control) were followed by a 20-km performance ride on the Kingcycle ergometer (Fig. 1, top). During these 20-km performance rides, which commenced exactly 60 s after completion of the 140-min experimental control exercise bouts, subjects were instructed to ride "as fast as possible." The only feedback given during

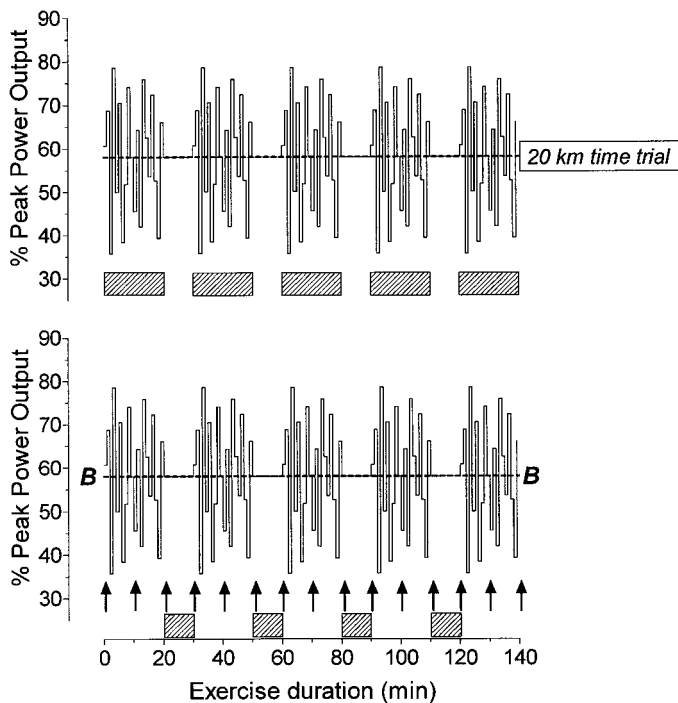


Fig. 1. Schematic diagram of the testing protocols employed during 2 control rides (*top*) and 2 invasive experimental rides (*bottom*). Solid lines, workload during the 140-min variable intensity (VI) rides; dashed lines, workload maintained during 140-min constant-load [steady-state (SS)] rides. Hatched boxes, times when subjects were on-line during control and invasive trials. *B*, time at which a muscle biopsy was taken before and after the invasive trials; arrows, collection of blood, rating of perceived exertion (RPE), and  $^{14}\text{CO}_2$  during invasive trials. See MATERIALS AND METHODS for further details.

the performance ride was the elapsed distance the subjects had cycled, indicated as a percentage of distance to go. All invasive metabolic data was collected during the other SS and VI 140-min experimental trials.

During the two control experimental trials, subjects breathed through the previously described gas-analysis system for five 20-min periods. The gas-collection periods during these two control experimental rides were between 0–20, 30–50, 60–80, 90–110, and 120–140 min of exercise. During the two invasive experimental rides, expired gas was collected for 10-min periods after 20, 50, 80, and 110 min of the ride. By using such a design, subjects were on-line for an entire (although not the same) 140-min VI or SS experimental ride (Fig. 1). Immediately before all experimental trials, subjects ingested 4 ml/kg body mass of a 5 g/100 ml CHO solution. During the two invasive experimental rides by the subjects, the CHO solution contained 18  $\mu\text{Ci}$  of U- $^{14}\text{C}$ -labeled glucose for the subsequent determination of the rates of blood glucose oxidation. After the first 15 min of each 140-min experimental ride and at subsequent 15-min intervals, subjects ingested the same solution for that ride at a rate of 10 ml  $\cdot$  kg body mass $^{-1} \cdot$  h $^{-1}$ .  $\dot{V}^{14}\text{CO}_2$  was collected for 2- to 3-min periods after 20, 50, 80, and 110 min by having subjects breathe through a Hans-Rudolph one-way valve to fill a 2-liter anesthesia bag with expired air. This trapped air was then passed through a solution that contained 1 ml of 1 N hyamine hydroxide (United Technologies, Packard, IL), 1 ml 96% ethanol, and one to two drops of phenolphthalein (SAARCHEM, Krugersdorp, RSA) until the phenolphthalein indicator showed that exactly 1 ml of  $\text{CO}_2$  had been trapped, as described previously (28). On completion of each ride, 10

ml of liquid scintillation cocktail (Ready Gel, Beckman, Fullerton, CA) was then added to this solution, and  $^{14}\text{CO}_2$  specific activity (sp. act.) [disintegrations/min (dpm)/mmol] was measured in an Inorb 460C automatic liquid scintillation counter (United Technologies). All counts were corrected for differences in quench and background. Throughout the performance ride, subjects had access to water ad libitum.

Before the two invasive experimental rides (Fig. 1, *bottom*), subjects rested in a supine position, and a muscle biopsy was taken from the vastus lateralis muscle according to the technique of Bergström (1), as modified by Evans et al. (11). At the same time, an incision was made in the contralateral leg for a postexercise biopsy, while a Jelco 18-gauge cannula (Critikon, Halfway House, RSA) was inserted in a forearm vein for blood sampling. A postexercise muscle biopsy sample was collected within 60–120 s of completion of the experimental rides. No TT was performed after the SS and VI invasive trials.

Ratings of perceived exertion (RPE) (2) were recorded, and venous blood samples (20 ml) were drawn at minutes 10, 21, and 30, and after each 10-min period thereafter, during the invasive experimental rides. (Fig. 1).

*Analytic techniques.* Whole body rates of instantaneous CHO and fat oxidation were calculated by indirect calorimetry, assuming a nonprotein respiratory exchange ratio (RER) by using the following equations (17)

$$\text{CHO oxidation} = 4.585\dot{V}\text{CO}_2 - 3.226\dot{V}\text{O}_2 \quad (1)$$

$$\text{Fat oxidation} = 1.695\dot{V}\text{O}_2 - 1.701\dot{V}\text{CO}_2 \quad (2)$$

Total CHO and fat oxidized during 140 min of either SS or VI exercise were estimated from the area under the CHO and fat oxidation vs. time curve for each subject.

*Equations 1 and 2* are based on the assumption that  $\dot{V}\text{O}_2$  and  $\dot{V}\text{CO}_2$  accurately reflect tissue  $\text{VO}_2$  and  $\text{VCO}_2$ . In well-trained subjects, like those in the present investigation, indirect calorimetry is a valid method for quantifying rates of substrate oxidation during strenuous exercise at 80–85%  $\dot{V}\text{O}_{2\text{max}}$  (27). Furthermore, pilot studies showed that rates of  $\dot{V}\text{E}$  were relatively constant during both experimental conditions. This suggests that respiratory compensations for increasing metabolic acidosis were negligible compared with the overall  $\dot{V}\text{CO}_2$  values at the higher exercise intensities during VI exercise. If we assume a non-steady-state lactate distribution volume of 100 ml/kg body mass (30), the resultant loss of  $\text{HCO}_3^-$  to  $\text{CO}_2$  would be expected to increase  $\dot{V}\text{CO}_2$  values by <0.08 l/min. Indeed, even the most rapid ( $\sim 1.5$  mM) increases in plasma lactate concentrations during VI would be expected to increase  $\dot{V}\text{CO}_2$  by, at most, 2% (20).

*Blood samples.* At the same time that expired gas was collected, blood samples (10 ml) were drawn into tubes that contained potassium oxalate and sodium fluoride. Blood samples were kept on ice until the completion of a trial and then were centrifuged at 750 g for 10 min at 4°C. Plasma glucose concentrations were subsequently determined by the glucose oxidase method with the use of a glucose analyzer (Glucose Analyzer 2, Beckman Instruments). Blood lactate concentrations were measured by spectrophotometric (model 35, Beckman Instruments) enzymatic assays (Lactate PAP, boiMerieux, Lyons, France).

Plasma insulin and glucagon concentrations were subsequently determined by using radioimmunoassay techniques (Coat-a-Count Insulin and Double Antibody Glucagon; Diagnostic Products, Los Angeles, CA), while serum free fatty acids (FFA) concentrations were measured by using an enzymatic colorimeter assay (29).

*Specific activities of plasma glucose and lactate.* A 1-ml sample of plasma, which had been collected for determination of plasma glucose, was used for this assay. Initially 70  $\mu$ l of 3.5 M HClO<sub>4</sub> were added to deproteinize each sample and to drive off any <sup>14</sup>C-bicarbonate as <sup>14</sup>CO<sub>2</sub>. The samples were then centrifuged at 5,000 *g* for 10 min at 4°C, and the protein-free supernatant was removed and kept refrigerated. The precipitate was then resuspended in 0.76 ml of 0.13 M HClO<sub>4</sub> and recentrifuged; the supernatant was added to that previously saved. This step was repeated an additional time. The pH of the combined supernatant of each sample was then neutralized with the addition of 136  $\mu$ l of 3 M K<sub>2</sub>CO<sub>3</sub> in 0.01 M Tris·HCl buffer and centrifuged again at 5,000 *g* for 20 min to remove the precipitate. The supernatant was then passed through an anion-exchange column (Extra-Sep RC SAX, Chromatography Research Supplies, Addison, IL) that had been conditioned with 2  $\times$  10-ml washes of ethanol and 2  $\times$  10-ml washes of distilled water. The void volume, which contained some glucose, was collected as the remaining glucose was eluted with distilled water (3  $\times$  1 ml). Lactate was then eluted with 2  $\times$  1 ml of 1 M CaCl<sub>2</sub>, pH 2.

Samples were then evaporated to near dryness at 60°C for ~20 h; after cooling, they were mixed with 15 ml of scintillation cocktail (Ready Gel, Beckman Instruments). <sup>14</sup>C radioactivity was measured in an Inorb 460C automatic liquid scintillation counter (United Technologies). Any losses in radioactivity during preparation of the sample were calculated from a control plasma sample, which had been spiked with a known amount of [U-<sup>14</sup>C]glucose and was run concurrently with the test samples. Such recoveries averaged 90  $\pm$  0.7%. After the corrections for losses of radioactivity had been made, the specific activity (in dpm/mmol glucose) could be calculated. Furthermore, because the 1-ml aliquot of plasma used for radiation counting was from the same plasma sample as was previously used for the determination of glucose concentration, total blood glucose oxidation was calculated from the equation

$$\text{Glu}_{\text{ox}} = (\text{sp. act.}_{\text{CO}_2} / \text{sp. act.}_{\text{Glu}}) \cdot \dot{V}\text{CO}_2 \quad (3)$$

In this equation, Glu<sub>ox</sub> is the rate of plasma glucose oxidation (in mmol/min); sp. act.<sub>CO<sub>2</sub></sub> is the specific radioactivity of the expired CO<sub>2</sub> (in dpm/mmol); sp. act.<sub>glu</sub> is the corresponding specific radioactivity of the plasma glucose (in dpm/mmol); and  $\dot{V}\text{CO}_2$  is the volume of expired CO<sub>2</sub> (in mmol/min), calculated from the  $\dot{V}\text{CO}_2$  (in l/min) and the 22.4 ml/mmol gas volume. Because the complete conversion of one molecule of [U-<sup>14</sup>C]glucose to six molecules of <sup>14</sup>CO<sub>2</sub> decreases the specific radioactivity (in dpm/mmol) by a factor of six, the  $\dot{V}\text{CO}_2$  values did not need to be divided by six to allow for six CO<sub>2</sub> molecules arising from the oxidation of one glucose molecule.

*Muscle samples.* Muscle biopsy samples were divided into two pieces. One piece was immediately frozen in liquid N<sub>2</sub> and stored at -70°C for subsequent determination of glycogen content by conventional methods (24). The second piece was oriented in mounting medium (Tissue Tech, Cape Town, RSA) and was rapidly frozen in isopentane maintained at its freezing point in liquid N<sub>2</sub>. Cryostat sections (10–15  $\mu$ m) were cut at -20°C. Serial sections of the sample were stained for determination of fiber type by using ATPase activity at pH 4.3 (4) and glycogen content by using periodic acid-Schiff's (PAS) reaction (25). Sections from each biopsy sample were magnified by using a Leica DRA microscope (Leica Technology, Rijswijk, The Netherlands) and were digitized with a Leica Quantimed 500 Image system. The intensity of the PAS staining in the individual muscle fibers was automatically rated by a gray-scale value by using Adobe Photoshop version

4.0 (Adobe Systems, Seattle, WA). Each section contained an average of 98  $\pm$  5 fibers.

*Statistical analysis.* All data, unless otherwise indicated, are presented as means  $\pm$  SE. Where appropriate, statistical significance between values was assessed with a paired Student's *t*-test or by using a two-way ANOVA for repeated measures. Where a significant difference was found by using the ANOVA, Scheffé's post hoc test was used to locate where this difference occurred. Differences were considered significant when *P* < 0.05.

## RESULTS

*$\dot{V}\text{O}_2$ , HR, RPE, rates of substrate oxidation, and energy expenditure.* Table 2 displays the  $\dot{V}\text{O}_2$ , HR, and the rates of CHO and fat oxidation averaged for each successive 10-min time period during the two 140-min experimental rides. During SS,  $\dot{V}\text{O}_2$  remained relatively constant, at ~3.0 l/min, throughout the 140 min of exercise. Despite the five bouts of stochastic work during VI, which totaled 100 of the 140 min of exercise,  $\dot{V}\text{O}_2$  also averaged ~3.1 l/min and was only significantly higher than SS between 111 and 120 min (3.22  $\pm$  0.16 vs. 3.13  $\pm$  0.15 l/min; *P* < 0.05). There was a gradual drift in HR during both trials, so that during the last 10 min of exercise, HRs for both SS and VI were ~25 beats/min higher than after the first 10 min (144  $\pm$  3 vs. 167  $\pm$  5 and 145  $\pm$  2 vs. 169  $\pm$  5 beats/min for SS and VI, respectively; *P* < 0.001). However, there were no differences in HR between the two experimental conditions at any time point. RPE rose progressively from 9.3  $\pm$  0.8 and 9.7  $\pm$  0.8 units after the first 10 min to 13.0  $\pm$  0.9 and 14.0  $\pm$  0.7 units during the last 10 min of exercise for SS and VI, respectively (*P* < 0.05). However, there were no differences in RPE between the two experimental conditions at any time during exercise, nor was there a difference in the average RPE throughout the entire 140-min bout (12.6  $\pm$  0.7 vs. 12.6  $\pm$  0.6 for SS and VI, respectively).

As would be expected with the higher work rate, CHO oxidation was significantly elevated during the initial 10 min of VI compared with SS (3.42  $\pm$  0.34 vs. 2.89  $\pm$  0.24 g/min; *P* = 0.03). CHO oxidation was still higher during the second bout of VI exercise (from 31 to 40 min) compared with SS (3.42  $\pm$  0.25 vs. 2.94  $\pm$  0.25 g/min; *P* = 0.007), but thereafter there were no differences between the two experimental conditions. Accordingly, the average rates of CHO oxidation for the entire 140 min of exercise for SS and VI were similar (2.89  $\pm$  0.03 vs. 3.08  $\pm$  0.10 g/min, respectively). Accompanying the accelerated CHO oxidation during the early stages of exercise, there was a concomitant reduction in the rate of fat oxidation during the first portion of VI (0.28  $\pm$  0.05 vs. 0.41  $\pm$  0.06 g/min; *P* < 0.05). Thereafter, fat oxidation between the two trials was not significantly different, averaging 0.46  $\pm$  0.02 and 0.42  $\pm$  0.04 g/min for SS and VI, respectively. The overall rate of total energy expenditure for the two experimental conditions was also very similar (901  $\pm$  40 and 904  $\pm$  58 J·kg<sup>-1</sup>·min<sup>-1</sup> for SS and VI, respectively).

*Circulating metabolites.* Figure 2 shows the plasma glucose, FFA, and lactate concentrations during the

two experimental conditions. Resting plasma glucose concentrations were the same for SS and VI exercise ( $4.7 \pm 0.1$  vs.  $4.7 \pm 0.2$  mM; Fig. 2, top). After subjects ingested CHO, plasma glucose concentration rose progressively; after 20 min of exercise, it was significantly higher in VI than SS ( $6.7 \pm 0.6$  vs.  $6.0 \pm 0.5$  mM;  $P < 0.05$ ). From 20–60 min of exercise, subjects' plasma glucose concentration declined to  $5.3 \pm 0.3$  mM in VI, although euglycemia ( $>5$  mM) was well maintained throughout the entire 140-min ride ( $5.7 \pm 0.5$  mM). During SS exercise, blood glucose concentration averaged  $5.6 \pm 0.2$  mM, and it was relatively constant for the entire exercise bout (Fig. 2, top). Plasma FFA concentrations were similar before exercise ( $0.18 \pm 0.05$  vs.  $0.22 \pm 0.05$  mM before VI and SS exercise, respectively) and rose progressively throughout both trials so that, by the end of 140 min, they had reached  $\sim 0.35$  mM for both VI and SS (Fig. 2, middle). As might be expected, plasma lactate concentration remained relatively constant during SS, averaging  $1.8 \pm 0.2$  mM for the entire ride (Fig. 2, bottom). On the other hand, plasma lactate concentration during VI exercise mirrored the changes in exercise intensity: with each increase in level of intensity, lactate concentration increased by  $\sim 1$  mM (from  $\sim 1.6$  to  $2.5$  mM). After the first hour of exercise was completed, lactate concentration during VI exercise rose progressively and was significantly higher than during SS exercise after 70, 100, and 110 min. It reached a peak of  $3.0 \pm 0.5$  mM after 130 min (all  $P < 0.05$ ; Fig. 2, bottom). The area under the curves for lactate vs. time was significantly greater for VI compared with SS exercise ( $29.1 \pm 3.9$  vs.  $24.6 \pm 3.7$  mM/140 min;  $P = 0.03$ ).

**Hormonal responses.** Figure 3 shows the concentrations of the circulating hormones (insulin and glucagon) in response to the two different experimental trials. Plasma insulin concentrations were similar at rest for the two experimental conditions ( $26 \pm 4$  vs.  $23 \pm 3$   $\mu$ U/ml for SS and VI, respectively), rose to between 35 and 40  $\mu$ U/ml after 30 min of exercise, and then declined progressively throughout the remainder of the work bout, so that by the end of 140 min of either VI or SS exercise they were  $\sim 20$   $\mu$ U/ml (Fig. 3, top). Plasma glucagon concentrations were the same at rest for the two trials ( $121 \pm 7$  vs.  $122 \pm 9$  pU/ml for SS and VI, respectively) and, apart from the values at 30 min ( $126 \pm 7$  and  $114 \pm 7$  pU/ml for SS and VI, respectively;  $P = 0.02$ ), were not significantly different between treatments (average,  $129 \pm 8$  vs.  $128 \pm 7$  pU/ml for SS and VI, respectively). There were no statistically significant differences in the area under curves for plasma insulin or plasma glucagon ( $368 \pm 58$  vs.  $351 \pm 53$   $\mu$ U·ml $^{-1}$ ·140 min $^{-1}$  and  $298 \pm 122$  vs.  $264 \pm 107$  pU·ml $^{-1}$ ·140 min $^{-1}$  for SS and VI, respectively).

**Blood glucose specific activity and rates of plasma glucose oxidation.** Figure 4 displays the blood glucose specific activity over time for the two experimental conditions, whereas the rates of plasma glucose oxidation and RER for the two experimental conditions are displayed in Fig. 5. During SS exercise, the rate of

Table 2. Oxygen uptake, heart rate, and substrate oxidation during prolonged constant-load or variable-intensity exercise

Exercise	Time, min													
	0–10	11–20	21–30	31–40	41–50	51–60	61–70	71–80	81–90	91–100	101–110	111–120	121–130	131–140
SS	2.97 ± 0.10	3.00 ± 0.10	3.04 ± 0.11	3.05 ± 0.11	3.04 ± 0.10	3.07 ± 0.13	3.07 ± 0.12	3.11 ± 0.14	3.10 ± 0.15	3.12 ± 0.15	3.14 ± 0.15	3.13 ± 0.15	3.16 ± 0.16	3.15 ± 0.16
VI	3.06 ± 0.13	3.08 ± 0.14	2.99 ± 0.10	3.11 ± 0.12	3.11 ± 0.14	3.08 ± 0.11	3.19 ± 0.15	3.16 ± 0.16	3.19 ± 0.16	3.24 ± 0.16	3.19 ± 0.16	3.22 ± 0.16*	3.23 ± 0.15	3.21 ± 0.14
SS	144 ± 3	150 ± 4	154 ± 4	156 ± 4	158 ± 4	160 ± 4	161 ± 4	160 ± 4	163 ± 4	163 ± 4	165 ± 4	167 ± 4	168 ± 4	167 ± 5
VI	145 ± 2	149 ± 4	154 ± 4	156 ± 4	155 ± 4	158 ± 4	158 ± 4	158 ± 4	162 ± 4	163 ± 4	163 ± 4	166 ± 5	168 ± 5	169 ± 5
SS	2.89 ± 0.25	2.98 ± 0.24	3.08 ± 0.18	2.94 ± 0.25	2.90 ± 0.24	3.00 ± 0.23	2.93 ± 0.25	2.94 ± 0.25	2.85 ± 0.18	2.89 ± 0.26	2.86 ± 0.25	2.82 ± 0.18	2.79 ± 0.27	2.74 ± 0.22
VI	3.42 ± 0.34†	3.39 ± 0.28	2.67 ± 0.34	3.43 ± 0.26*	3.29 ± 0.23	2.92 ± 0.14	3.27 ± 0.16	3.17 ± 0.18	2.86 ± 0.11	3.17 ± 0.18	2.98 ± 0.20	2.70 ± 0.12	2.94 ± 0.17	2.93 ± 0.21
SS	0.41 ± 0.06	0.41 ± 0.08	0.37 ± 0.06	0.43 ± 0.08	0.44 ± 0.08	0.42 ± 0.07	0.44 ± 0.08	0.46 ± 0.08	0.49 ± 0.06	0.48 ± 0.08	0.51 ± 0.08	0.51 ± 0.06	0.54 ± 0.08	0.55 ± 0.07
VI	0.28 ± 0.06†	0.28 ± 0.05	0.33 ± 0.06	0.31 ± 0.04	0.34 ± 0.03	0.45 ± 0.02	0.38 ± 0.03	0.40 ± 0.02	0.53 ± 0.03	0.44 ± 0.04	0.49 ± 0.04	0.60 ± 0.04	0.52 ± 0.03	0.51 ± 0.04

Values are means ± SE; n = 6 subjects; SS, steady state; VI, variable intensity. VI significantly greater than SS, \*  $P < 0.01$ , †  $P < 0.05$ .

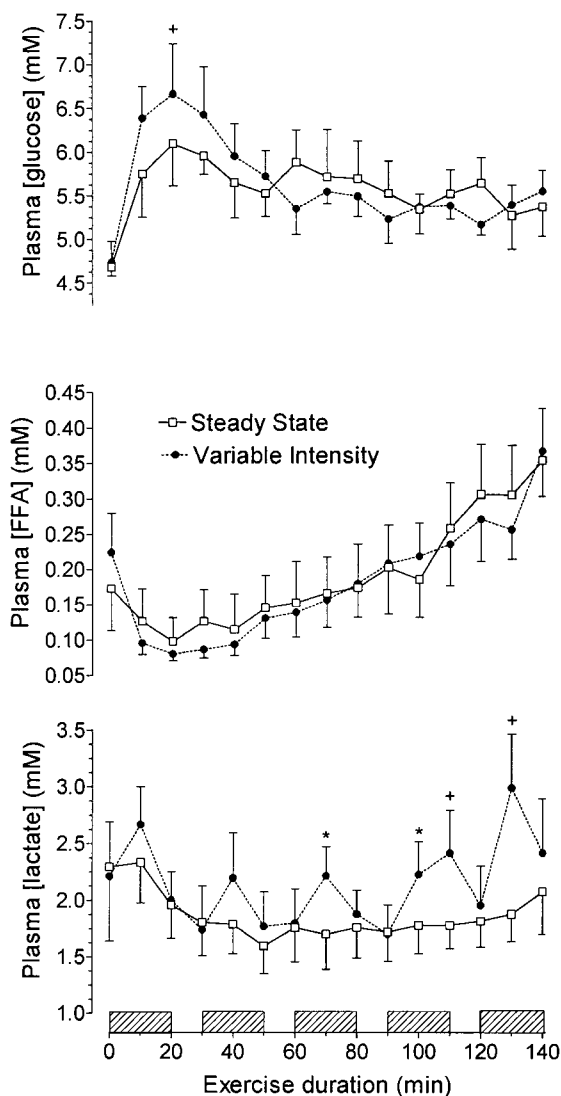


Fig. 2. Plasma glucose (top), free fatty acid (FFA, middle), and lactate concentrations (bottom) during 140-min experimental rides. Hatched boxes, periods of VI exercise. Area under the curve for plasma lactate during VI exercise (●) is significantly greater than that during SS exercise (□) bout ( $29.1 \pm 3.9$  vs.  $24.6 \pm 3.7$  mM/140 min;  $P = 0.03$ ). VI significantly greater than SS: \*  $P < 0.001$ , +  $P < 0.05$ .

plasma glucose oxidation rose progressively throughout exercise from  $0.56 \pm 0.08$  mmol/min ( $0.10 \pm 0.01$  g/min) at 10 min and peaked at  $5.11 \pm 0.35$  mmol/min ( $0.93 \pm 0.06$  g/min) after 130 min of the work bout. Rates of plasma glucose oxidation also rose over time during VI exercise [from  $0.46 \pm 0.11$  mmol/min ( $0.08 \pm 0.02$  g/min) at 10 min] and peaked at  $6.50 \pm 0.55$  mmol/min ( $1.18 \pm 0.10$  g/min) after 130 min of the work bout, with intermediate increases being directly related to the changes in exercise intensity, particularly during the latter stages of the ride. The rate of blood glucose oxidation was significantly higher in VI than in SS exercise at 90 min ( $5.12 \pm 0.30$  vs.  $4.23 \pm 0.15$  mmol/min;  $0.95 \pm 0.06$  vs.  $0.77 \pm 0.03$  g/min;  $P = 0.03$ ), 100 min ( $5.67 \pm 0.29$  vs.  $4.15 \pm 0.29$  mmol/min;  $1.03 \pm 0.53$  vs.  $0.75 \pm 0.05$  g/min;  $P = 0.005$ ), and after 130

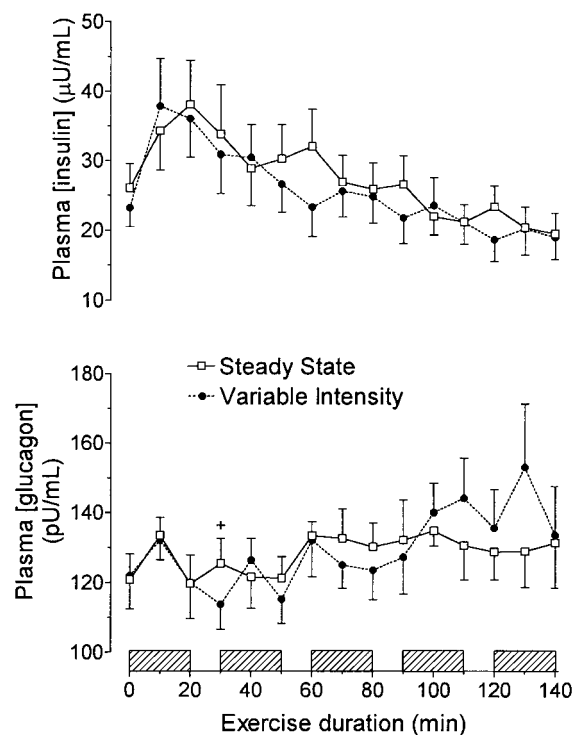


Fig. 3. Plasma insulin (top) and glucagon (bottom) concentrations during 140-min experimental rides. Hatched boxes, periods of VI exercise. + SS significantly greater than VI,  $P < 0.05$ .

min ( $6.50 \pm 0.55$  vs.  $5.11 \pm 0.35$  mmol/min;  $1.19 \pm 0.1$  vs.  $0.93 \pm 0.06$  g/min) (Fig. 5). The average rate of plasma glucose oxidation was  $0.7$  vs.  $0.6$  g/min for VI and SS, respectively. The total plasma glucose oxidized during the entire exercise bout (as calculated from the area under the curve for each subject) was greater in VI than SS exercise ( $99.2 \pm 5.3$  vs.  $83.9 \pm 5.2$  g/140 min;  $P < 0.05$ ).

*Muscle fiber type, glycogen utilization, and PAS staining.* The vastus lateralis muscle fiber composition was  $53.6 \pm 2.9\%$  type I and  $46.4 \pm 2.9\%$  type II fibers. In these subjects, the vastus lateralis glycogen concentration, before and after 140 min of either SS or VI

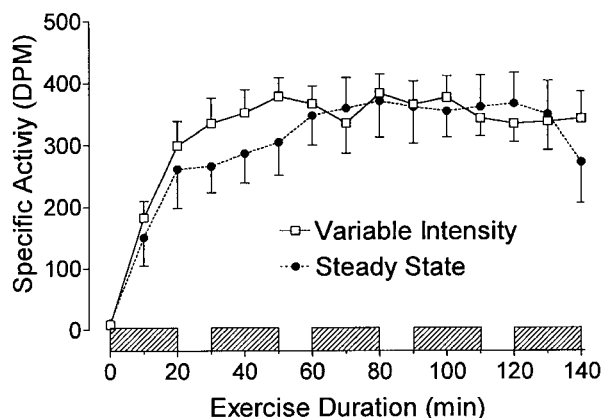


Fig. 4. Blood glucose specific activity over time during 140-min experimental rides. Hatched boxes, periods of VI exercise; DPM, disintegrations/min.

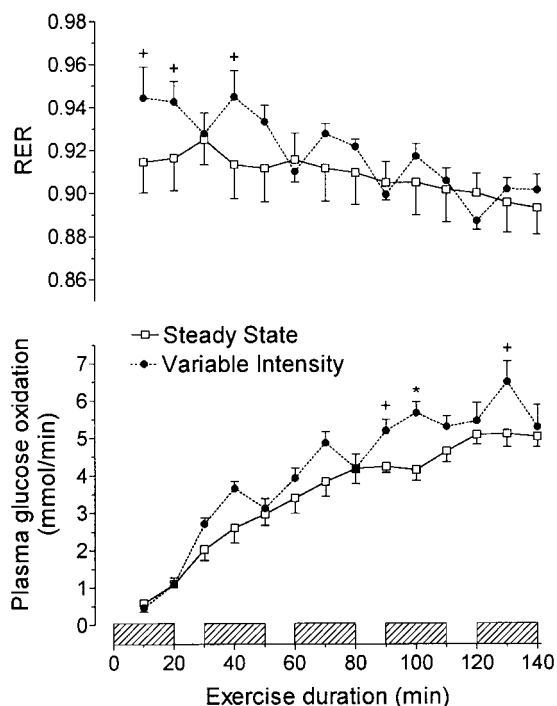


Fig. 5. Respiratory exchange ratio (RER; *top*) and rates of plasma glucose oxidation (*bottom*) during 140-min experimental rides. Hatched boxes, periods of VI exercise. VI significantly greater than SS: \* $P < 0.001$ , +  $P < 0.05$ .

exercise, is shown in Fig. 6. As intended, muscle glycogen content did not differ between SS or VI before exercise ( $156 \pm 14$  vs.  $148 \pm 23$  mmol/kg wet wt). Neither were there any differences in glycogen content after 140 min of exercise ( $54 \pm 14$  vs.  $75 \pm 6$  mmol/kg wet wt for SS and VI, respectively; not significant). Accordingly, SS exercise resulted in a 65% reduction in total muscle glycogen content compared with 49% for VI exercise.

Figure 7 shows the percentage of fibers stained for glycogen with PAS reagent after 140 min of either SS or VI exercise. All muscle fibers stained dark (4-5) for glycogen before exercise. However, there was a marked disappearance of glycogen from the type I fibers (as

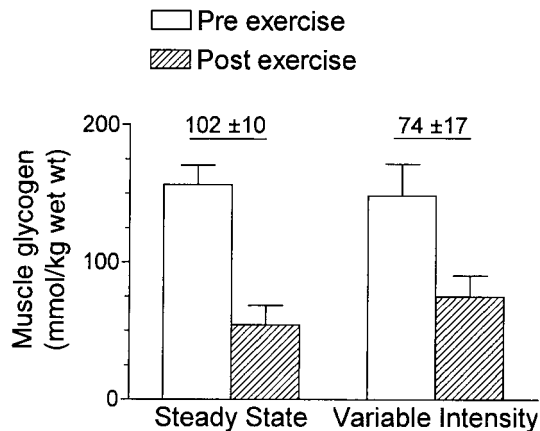


Fig. 6. Muscle glycogen concentration in vastus lateralis before and after 140-min experimental rides.

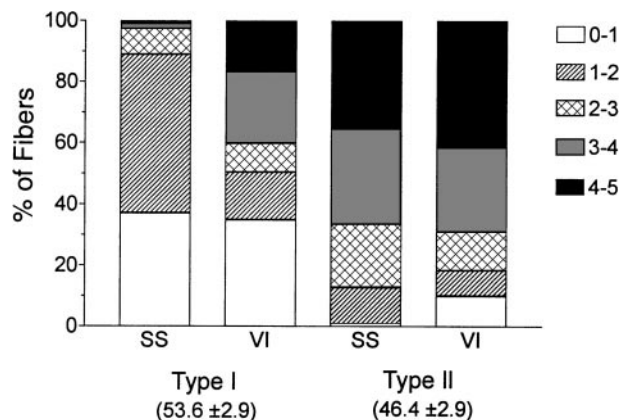


Fig. 7. Sections of muscle biopsy sample obtained from vastus lateralis after 140-min experimental rides were stained for glycogen content by using periodic acid-Schiff's reagent. Pattern of staining is displayed for both type I and type II muscle fibers. Nos. of each fiber type shown in parenthesis. Intensity of glycogen staining was automatically rated by computer on a gray scale: 0-1 = negative, 4-5 = darkly stained.

indicated by a low gray-scale score) after SS compared with VI (98 vs. 59% of fibers scoring 0-2 for SS and VI, respectively). Conversely, the density of type I fibers darkly stained (3-5) at the end of 140 min of exercise was only 2% after SS vs. ~40% after VI (Fig. 7). The number of type II fibers that showed a negative gray-scale score (0-1) was 10% after VI compared with just 1% after SS (Fig. 7). However, for both SS and VI, approximately two-thirds of type II fibers stained darkly (3-5) for glycogen at the end of the 140 min preload exercise bout.

**TT performance.** Figure 8 shows the power output (*top*) and HR (*bottom*) for each 5% segment of the 20-km TT after 140 min of either SS or VI exercise.

There was no difference in the average power output sustained during the two performance rides ( $283 \pm 22$  vs.  $256 \pm 18$  W for SS and VI, respectively). Accordingly, the 20-km TT performance was similar ( $29.14 \pm 0.9$  vs.  $30.5 \pm 0.9$  min for SS and VI, respectively). The HR responses were also similar between the two rides ( $171 \pm 4$  vs.  $172 \pm 4$  beats/min for SS and VI, respectively).

**DISCUSSION**

The physiological and metabolic responses of well-trained individuals to constant-load submaximal exercise (in particular, cycling) have been well documented (9, 26). Until recently, however, few studies have examined intense intermittent exercise or VI work in which power output or speed vary in a random or stochastic manner. Reasons for this may include 1) the lack of appropriate equipment, 2) concerns that non-SS conditions do not permit valid or reliable estimates of substrate metabolism, or 3) the belief that SS conditions are common in most sports.

Several recent investigations have used exercise models in which the work rate alternates between periods of low- and high-intensity exercise (45-60 and

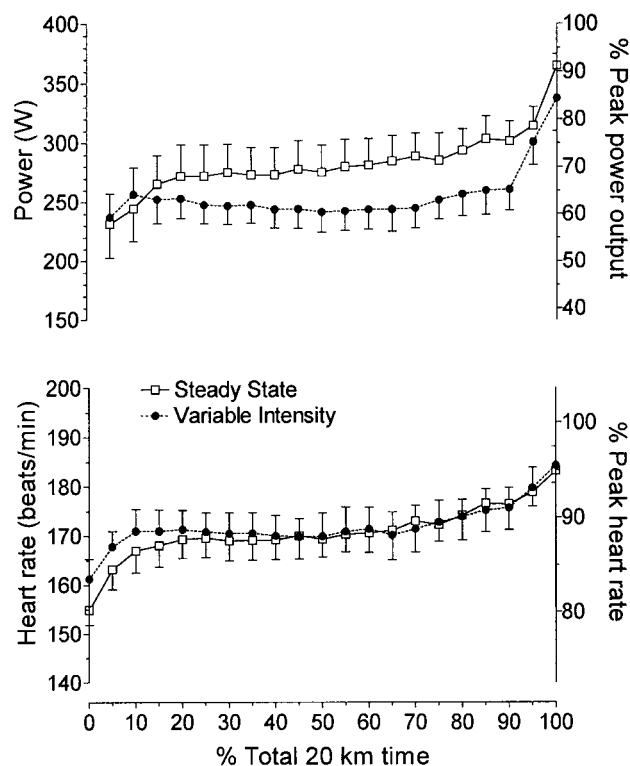


Fig. 8. Power output (*top*) and heart rate (*bottom*) for each 5% section of the 20-km performance time trial after 140 min of either SS or VI exercise.

75–85%  $\dot{V}O_{2\max}$ , respectively) and which are of sufficient duration to allow well-trained subjects to attain SS (6, 12, 36). Under these conditions, indirect calorimetry provides a valid measure of substrate oxidation in well-trained subjects who exercise at intensities of up to 85%  $\dot{V}O_{2\max}$  (27). We used a similar approach to compare the metabolic and hormonal responses to prolonged (140 min) cycling at either constant ( $232.5 \pm 10.6$  W,  $\sim 70\%$   $\dot{V}O_{2\text{peak}}$ ) or variable loads ( $143.1 \pm 6.5$  to  $314.7 \pm 14.3$  W,  $\sim 40\text{--}85\%$   $\dot{V}O_{2\text{peak}}$ ) but of the same average intensity. In addition, we wished to determine whether these two different exercise modes would affect performance during a subsequent cycling TT.

The first finding was that, despite five 20-min bouts of stochastic exercise that totaled  $\sim 70\%$  of the entire work bout, the average  $\dot{V}O_2$  was remarkably steady throughout both the constant-load and VI work (Table 2). Nor did the subjects perceive any differences in average effort during the two work bouts or at any time point during the 140-min rides. Yaspelkis et al. (36) reported that  $\dot{V}O_2$  was elevated  $\sim 40\%$  (from  $\sim 2.1$  to  $3.45$  l/min) when their well-trained subjects increased their work rate from low (45%  $\dot{V}O_{2\max}$ ) to moderate (75%  $\dot{V}O_{2\max}$ ) intensity and that their subjects' RPEs reflected the alterations in exercise intensity. A possible reason for discrepancies between their findings and ours could be that the VI exercise model we employed alternated rapidly between short bouts of low- and high-intensity work. This model was chosen because it is a more accurate simulation of real conditions in

competition (18). In contrast, Yaspelkis et al. (36) employed a less complex protocol in which subjects cycled for 30 min at 45%  $\dot{V}O_{2\max}$ , followed by six repeated 16-min periods of alternate cycling at 75 and 45%  $\dot{V}O_{2\max}$  (8 min each), followed by a rest period, then a further period of alternate intervals (3 min at 45%  $\dot{V}O_{2\max}$  3 min at 75%  $\dot{V}O_{2\max}$ ).

As might be expected from similar  $\dot{V}O_2$  values, the average HR responses during both trials were almost identical (Table 2). This finding emphasizes the difficulties of attempting to monitor exercise intensity by HR data alone. In cycling, for example, HR cannot be considered an accurate indicator of work rate (power output) or speed in situations in which a cyclist is riding in a pack or is free to choose his or her own pace. We (23) and others (18) have previously reported that, in mass-start cycling races, HR varies randomly, with frequent changes in amplitude and frequency, and that such perturbations are not related to speed, power output, or course profile. More to the point, when the duration of a work load is short ( $< 2$  min), the cardiovascular response will lag behind any changes in muscle power output.

Despite the similar whole body responses ( $\dot{V}O_2$ , HR, energy cost) to the two different experimental protocols, there were differences in the lactate profiles between trials (Fig. 2), with plasma lactate levels reflecting the VI exercise. During the stochastic exercise, lactate concentrations were  $\sim 1.5$  mM higher than values at the same time during the constant-load ride. Despite the periods of low-intensity exercise during the stochastic trial, plasma lactate concentrations tended to be higher than during the constant-load trial, particularly during the latter stages of the experimental ride, resulting in a greater area under the curve for lactate vs. time. The lactate concentrations measured in the present study are similar to those reported by Yaspelkis et al. (36). They are, however, somewhat lower than those measured by Coggan and Coyle (6) during intense cycling. The latter reported values of  $\sim 5$  mM when their highly trained subjects alternated every 15 min between 60 and 85%  $\dot{V}O_{2\max}$  (6).

A second finding of this study was the tendency for a reduction in total muscle glycogen utilization (16%) during 140 min of stochastic compared with constant-load exercise that produced the same total work (Fig. 6). However, this decrease was not statistically significant. The amount of glycogen remaining in the muscle ( $\sim 80$  mmol/kg wet wt) after 140 min was similar to the value reported by Yaspelkis et al. (36) after  $\sim 130$  min of VI cycling ( $\sim 90$  mmol/kg wet wt). The difference in whole muscle glycogen utilization between trials just failed to reach statistical significance. Although the total plasma glucose oxidized during the 140-min experimental rides was greater in VI than in SS (99 vs. 84 g/140 min, respectively), such a difference cannot explain the reduction in calculated glycogen degradation. If we assume an active muscle mass of 8 kg during cycling (19), the  $\sim 15$  g greater glucose oxidation during

VI would explain only 36% of the 42 g of glycogen sparing.

However, the true rate of glycogen utilization by contracting fibers cannot be accurately assessed by measurement of changes in the total glycogen of muscle samples (15). Accordingly, we subsequently performed PAS staining to determine whether there were similar patterns of glycogen depletion in the different fiber types (Fig. 7). Such analysis revealed that <5% of the total number of type I fibers stained dark (3–5) for glycogen at the end of 140 min of constant-load cycling, compared with >40% at the end of the VI exercise. Accordingly, ~95% of type I fibers stained negatively or light (0–2) for glycogen after constant-load exercise compared with ~60% in the VI trial. On the other hand, there was a marked loss of glycogen from the type II fibers (those staining 0–1) after VI exercise (~10%), with little or no loss occurring after the constant-load work bout.

The objectivity and reliability of the PAS-rating procedure has been questioned (15). In previous studies (8, 10, 14, 15, 35, 36), the intensity of the PAS staining in individual fibers was rated visually by one or more of the investigators. In the present study, an automated computer system scored the muscle samples, thus removing an element of observer bias. Furthermore, our results are in excellent agreement with previous studies of muscle glycogen-depletion patterns during prolonged, continuous, constant-load (10, 14, 34, 35), and severe (>80%  $\dot{V}O_{2\max}$ ) intermittent cycling (10). Those studies showed that, during moderate-intensity (<70%  $\dot{V}O_{2\max}$ ), constant-load exercise, type I fibers are the first to display reduced PAS staining, whereas intense VI exercise at close to 100%  $\dot{V}O_{2\max}$  recruits both type I and type II fibers (10, 14, 15, 35).

Compared with water ingestion, CHO supplementation has been shown to reduce muscle glycogen use during VI cycling. Glycogen sparing with CHO ingestion has also been reported by Tsintzas et al. (32) at the end of 60 min of constant-speed running at 70%  $\dot{V}O_{2\max}$  and during submaximal running to exhaustion (33), although others (3, 8) have not observed any differences in muscle glycogen utilization after several hours of submaximal constant-load cycling when subjects were fed either CHO or water (see Ref. 31 for review). To the best of our knowledge, there are no reports in the literature that compare muscle glycogen utilization during VI and SS exercise of the same average power output when subjects ingest CHO. However, the possibility remains that, in the present study, CHO ingestion resulted in a net glycogen synthesis in some active (and inactive) muscle fibers during the VI ride. In support of this hypothesis, Kuipers et al. (19) have previously reported that, after a ride to exhaustion designed to result in glycogen depletion, net glycogen synthesis occurred in the nonactive muscles of well-trained cyclists who ingested large (~500 g) amounts of CHO during a subsequent bout of prolonged (3 h) low-intensity (~50%  $\dot{V}O_{2\max}$ ) cycling. These workers found that muscle glycogen content was increased by an

average of ~30% after the low-intensity work bout compared with the value at exhaustion (199 vs. 136 mmol/kg dry wt). However, the amount of CHO incorporated into muscle was likely to be much higher, because these workers could not account for the fate of a large proportion of the CHO ingested by their subjects (~275 g). Although it is tempting to speculate that glycogen synthesis could explain the tendency for attenuated loss of glycogen during the VI ride in the present investigation, there was insufficient muscle biopsy tissue left to quantify whether there had been any incorporation of  $^{14}\text{C}$  into glycogen during both experimental rides.

The third finding of this investigation was that, despite differences in the 140-min preload exercise bout, subsequent 20-km TT performance was not statistically different between the two experimental trials (Fig. 8). This result seems surprising, given that subjects rode at a higher average power output throughout SS compared with VI (283 vs. 256 W, respectively). Indeed, the 27-W difference in average power between the two conditions would normally be expected to result in a significant performance effect for the two treatments. The main reason for such a finding was that three of the subjects went faster after the SS ride (with 1 subject riding considerably faster), whereas three rode slightly faster after VI exercise. It is tempting to speculate that, if the TT had been conducted over a longer distance (40 km, ~1 h), differences in power output between SS and VI exercise might have resulted in a significant performance enhancement.

The result of no performance difference is also at odds with our previous study (22) in which performance in a similar TT improved by 6% in well-trained cyclists who had completed 150 min of constant-load cycling at ~250 W (65%  $\dot{V}O_{2\max}$ ) compared with results when the same amount of work was undertaken as stochastic exercise in which the power output varied between 155 and 355 W. During the final 10 min of the 150-min stochastic ride, subjects sustained high work rates (>300 W), finishing with a bout of high-intensity (~340 W, >90%  $\dot{V}O_{2\max}$ ) cycling. Although no metabolic measures were taken in that study, such an intense bout of exercise could have resulted in cyclists' commencing the TT with high blood (and muscle) lactate concentrations compared with the constant-load exercise. Evidence for this contention comes from an analysis of the power outputs during the first three-quarters of the 20-km TT. After the stochastic ride, power was consistently lower than during constant-load exercise, although riders were able to increase their speed during the latter stages of both TT, finishing at similar (~400 W) workloads. In contrast, during the final 10 min of the VI work bout in the present study, power outputs exceeded 300 W for only brief periods (see Fig. 1). Indeed, lactate concentrations actually fell during the last 10 min of the VI ride and were only marginally higher than at the end of the constant-load ride (2.1 vs. 2.4 mM). This small difference is unlikely to be of any physiological importance. Taken collectively, the re-

sults of the present study and those of our previous investigation (22) reveal that 9 of 12 riders performed faster in a 20-km TT that followed 140 min of SS compared with VI cycling. These findings strongly suggest that during prolonged (2–3 h) cycling that culminates with a sustained (~30 min) bout of high-intensity exercise, the best riding strategy would be to maintain a SS rather than a VI pace for as long as possible.

In conclusion, this is the first investigation to examine the metabolic and performance responses to prolonged VI or constant-load exercise of the same average intensity. Despite similar whole body responses (i.e.,  $\dot{V}O_2$ , HR, RPE, energy expenditure) to the two different exercise bouts, lactate concentrations tended to be higher throughout the latter stages of the VI compared with during the constant-load exercise. There was also evidence to suggest that VI exercise may result in glycogen sparing in the type I muscle fibers compared with when the same work is performed as constant-load exercise. Further support for this interpretation was the finding that plasma glucose oxidation during VI exercise was significantly greater than during the constant-load work. Such differences, however, did not affect subsequent high-intensity exercise performance.

Thus we conclude that, when well-trained subjects perform prolonged VI exercise or constant-load exercise of the same average intensity, there are only small differences in skeletal muscle CHO metabolism and recruitment and that such differences do not affect the performance of a subsequent bout of high-intensity cycling.

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