Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance

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Burgomaster, Kirsten A., George J. F. Heigenhauser, and Martin J. Gibala. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol* 100: 2041–2047, 2006. First published February 9, 2006; doi:10.1152/japplphysiol.01220.2005.— Our laboratory recently showed that six sessions of sprint interval training (SIT) over 2 wk increased muscle oxidative potential and cycle endurance capacity (Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN, and Gibala MJ. *J Appl Physiol* 98: 1895–1900, 2005). The present study tested the hypothesis that short-term SIT would reduce skeletal muscle glycogenolysis and lactate accumulation during exercise and increase the capacity for pyruvate oxidation via pyruvate dehydrogenase (PDH). Eight men [peak oxygen uptake ($\dot{V}_{O_2 \text{ peak}}$) = 3.8 ± 0.2 l/min] performed six sessions of SIT (4–7 × 30-s "all-out" cycling with 4 min of recovery) over 2 wk. Before and after SIT, biopsies (vastus lateralis) were obtained at rest and after each stage of a two-stage cycling test that consisted of 10 min at ~60% followed by 10 min at ~90% of $\dot{V}_{O_2 \text{ peak}}$. Subjects also performed a 250-kJ time trial (TT) before and after SIT to assess changes in cycling performance. SIT increased muscle glycogen content by ~50% (main effect, *P* = 0.04) and the maximal activity of citrate synthase (posttraining: 7.8 ± 0.4 vs. pretraining: 7.0 ± 0.4 mol·kg protein $^{-1}$·h^{-1}; *P* = 0.04), but the maximal activity of 3-hydroxyacyl-CoA dehydrogenase was unchanged (posttraining: 5.1 ± 0.7 vs. pretraining: 4.9 ± 0.6 mol·kg protein $^{-1}$·h^{-1}; *P* = 0.76). The active form of PDH was higher after training (main effect, *P* = 0.04), and net muscle glycogenolysis (posttraining: 100 ± 16 vs. pretraining: 139 ± 11 mmol/kg dry wt; *P* = 0.03) and lactate accumulation (posttraining: 55 ± 2 vs. pretraining: 63 ± 1 mmol/kg dry wt; *P* = 0.03) during exercise were reduced. TT performance improved by 9.6% after training (posttraining: 15.5 ± 0.5 vs. pretraining: 17.2 ± 1.0 min; *P* = 0.006), and a control group (*n* = 8, $\dot{V}_{O_2 \text{ peak}}$ = 3.9 ± 0.2 l/min) showed no change in performance when tested 2 wk apart without SIT (posttraining: 18.8 ± 1.2 vs. pretraining: 18.9 ± 1.2 min; *P* = 0.74). We conclude that short-term SIT improved cycling TT performance and resulted in a closer matching of glycogenolytic flux and pyruvate oxidation during submaximal exercise.

pyruvate dehydrogenase; oxidative metabolism; glycogen

SPRINT INTERVAL TRAINING (SIT), which is characterized by recurring sessions of brief, repeated bouts of very intense exercise, is a potent stimulus for inducing metabolic adaptations in human skeletal muscle (20, 38). With respect to carbohydrate (CHO) metabolism, a wide range of adaptations have been described, including an increase in resting glycogen content (13, 30), increases in the maximal activities of various enzymes involved in glycolytic (18, 24) and oxidative energy

provision (18, 24), and increases in lactate transport capacity (19, 33). Many of these adaptations occur very quickly after a surprisingly small volume of intense exercise training (7, 30, 37); for example, our laboratory recently reported increases in resting glycogen content and citrate synthase (CS) maximal activity after only six sessions of SIT performed over 2 wk (7).

In contrast to the wealth of data regarding adaptations in resting muscle, much less is known about the effect of sprint training on CHO metabolism during an acute bout of exercise. Several investigators have suggested that sprint training either increases or does not change muscle glycogenolytic rate and nonoxidative ATP provision during exercise, based on research designs that incorporated brief "all-out" exercise challenges to exhaustion (e.g., Refs. 23, 27, 42). Although this type of approach is certainly valid, the interpretation of training per se on metabolic control is hampered by the fact that power output differs between the pre- and posttraining tests. Recently, a unique study by Harmer et al. (13) employed a matched-work exercise comparison to investigate the effect of sprint training on metabolic perturbations in human muscle. The authors demonstrated that 7 wk of sprint training reduced nonoxidative ATP generation during intense exercise, as evidenced by lower muscle glycogen degradation and lactate accumulation after 30 s of cycling at 130% of pretraining peak oxygen uptake ($\dot{V}_{O_2 \text{ peak}}$). These findings (13) thus contrasted sharply from other studies that used non-matched-work exercise challenges (23, 27, 42) and implied that the contribution from aerobic metabolism was enhanced during intense exercise after sprint training, as previously suggested by others (25, 26). Harmer et al. (13) did not specifically examine markers of muscle oxidative metabolism, but they hypothesized that sprint training might increase the activity of pyruvate dehydrogenase (PDH) and thus the capacity for mitochondrial pyruvate oxidation. Other investigators have also speculated on the potential importance of PDH in the muscle adaptive response to sprint training (24, 30), but to date no study has directly examined whether sprint training alters PDH activity during exercise.

The primary purpose of the present study was to examine the effect of 2 wk of SIT on skeletal muscle CHO metabolism during submaximal, matched-work exercise. The training protocol was identical to that described in our laboratory's previous study (7) and consisted of six sessions of brief, repeated maximal cycling efforts, performed over 14 days with 1–2 days of recovery between training sessions. We hypothesized that short-term SIT would decrease muscle glycogenolysis and lactate accumulation during exercise and increase the capacity

for CHO oxidation through PDH. In addition to the matched-work exercise test that was used to assess muscle metabolic adaptations, our design included a separate test of volitional exercise performance. Given that many events require athletes to complete a fixed amount of work in as short a time as possible (i.e., a race), we tested the hypothesis that short SIT would improve 250-kJ cycling time-trial performance. As in our laboratory's previous study (7), we included a control group who completed the exercise performance test 2 wk apart with no training intervention, and all subjects were thoroughly familiarized with all experimental procedures before baseline testing.

METHODS

Subjects

Sixteen young healthy men volunteered to participate in the experiment (Table 1). All subjects were drawn from the same subject population, namely young active students at McMaster University who took part in some form of recreational exercise two to three times per week (jogging, cycling, etc.). None of the subjects were specifically engaged in training for a particular sporting event, although one was a varsity runner who was out of season at the time of the experiment. Eight of the subjects served as a training group that performed exercise performance tests before and after a 2-wk sprint training intervention. The other eight subjects served as a control group that completed exercise performance tests ~2 wk apart with no training intervention. The control group was older ($P = 0.03$); however, there were no differences between groups in $\dot{V}O_{2\ peak}$ or any other descriptive characteristic (Table 1). In addition to the exercise performance tests, the training group also performed a separate matched-work exercise test before and after training, and we obtained muscle biopsy samples at rest and during exercise to examine potential adaptations in metabolic regulation. We did not obtain biopsies from the control group for ethical reasons, because other studies have shown no change in resting muscle metabolites or the maximal activities of various enzymes when control subjects are tested several weeks apart with no sprint training intervention (3, 28). After routine medical screening, subjects were advised of the purpose of the study and associated risks, and all provided written, informed consent. The experimental protocol was approved by the McMaster University and Hamilton Health Sciences Research Ethics Board.

Preexperimental Procedures

Before baseline measurements, subjects made several familiarization visits to the laboratory to become oriented with the testing procedures and training devices. During one of these visits, subjects performed an incremental test to exhaustion on an electrically braked cycle ergometer (Excalibur Sport V2.0, Lode, Groningen, The Netherlands) to determine $\dot{V}O_{2\ peak}$ using an online gas-collection system (Moxus modular oxygen uptake system, AEI technologies, Pittsburgh, PA). The initial three stages of the test consisted of 2-min intervals at 50, 100, and 150 W, respectively, and the workload was increased by 25 W every minute until volitional exhaustion. The value

Table 1. Subject characteristics

Variable	Training Group	Control Group
Age, yr	21±1	25±1*
Height, cm	181±2	180±2
Weight, kg	78±5	76±3
$\dot{V}O_{2\ peak}$, l/min	3.8±0.2	3.9±0.2

Values are means ± SE for 8 subjects per group. $\dot{V}O_{2\ peak}$, peak oxygen uptake. *$P < 0.05$ vs. training group.

used for $\dot{V}O_{2\ peak}$ corresponded to the highest value achieved over a 30-s collection period. All subjects also performed a familiarization time trial. Subjects in the training group only also performed a familiarization Wingate test and an incremental exercise test to establish workloads that were used during the main experimental trials. Details regarding all experimental exercise tests are described below.

Details of Experimental Tests

Time trial. Subjects were instructed to complete a 250-kJ self-paced laboratory time trial on an electronically braked cycle ergometer (Lode) as quickly as possible with no temporal, verbal, and physiological feedback. The only feedback that subjects received during the test was work done, which was presented as "distance covered" on a computer monitor (i.e., 250 kJ was equated to 10 km, such that visual feedback at any point during the ride was presented in units of distance rather than work done). Time required to complete the test and average power output were recorded on completion of each test. Method error reproducibility for the time trial [coefficient of variation, determined using the method described by Sale (39)] was 2.6% when eight individuals were tested 1 wk apart with no sprint training intervention (*day 1*: 17.1 ± 1.0 vs. *day 2*: 17.2 ± 0.8 min).

Wingate test. Subjects performed a 30-s all-out effort on a mechanically braked cycle ergometer (model 814E bicycle ergometer, Monark, Stockholm, Sweden) against a resistance equivalent to 0.075 kg/kg body mass. Subjects were instructed to begin pedaling as fast as possible against the ergometer's inertial resistance, and then the appropriate load was manually applied. Subjects were verbally encouraged to continue pedaling as fast as possible throughout the 30-s test. Peak power, mean power, and fatigue index were subsequently determined using an online data-acquisition system.

Exercise metabolism test. Subjects reported to the laboratory and rested in the supine position while the area over one thigh was anesthetized (1% wt/vol lignocaine hydrochloride, AstraZeneca Canada, Mississauga, ON, Canada) and prepared for the extraction of needle biopsy samples from the vastus lateralis muscle (4). Three separate incisions were made through the skin and underlying fascia, ~2 cm apart, and a needle biopsy sample was obtained through one of the incision sites. The three sites were covered with sterile gauze and surgical tape, and the subject climbed onto an electrically braked cycle ergometer (Lode) and commenced cycling at a work intensity that elicited ~60% $\dot{V}O_{2\ peak}$. After 10 min of exercise, the subject stopped cycling, and a second biopsy sample was obtained as quickly as possible through the second incision site while the subject remained seated on the ergometer. The time delay between cessation of exercise and muscle excision was 10–20 s. After the muscle sample was removed from the leg, the area over the biopsy site was recovered with sterile gauze and surgical tape. Sixty seconds after the cessation of exercise at the first workload, subjects resumed cycling at a higher work intensity that elicited ~90% $\dot{V}O_{2\ peak}$. The time delay for the biopsy procedure between workloads was standardized in an effort to reduce potential variability that may have been induced by differing amounts of recovery. After 10 min of exercise at the second workload, the subject stopped cycling, and a third and final biopsy was obtained from the third incision site while the subject remained seated on the ergometer. All biopsy samples were immediately frozen in liquid nitrogen after the needle was removed from the leg. Cardiorespiratory data were collected and averaged over the 6- to 9-min period of exercise at each workload.

Experimental Protocol

After the familiarization procedures, the experimental protocol consisted of *1*) baseline testing; *2*) a 2-wk sprint training intervention or similar period without sprint training (control group); and *3*) posttesting, as described in detail below.

Baseline testing. Baseline measurements for all subjects consisted of a 250-kJ laboratory time trial. Subjects in the training group also

performed a Wingate test and an invasive exercise metabolism test (see above), with at least 2 days of recovery between tests.

Training. The sprint training protocol was identical to that described in our laboratory's previous study (7). Training was initiated 3 days after the exercise metabolism test and consisted of six sessions of sprint interval exercise spread over 14 days. Each training session consisted of repeated 30-s all-out cycling efforts (Wingate tests) with 4 min of recovery between tests. During the 4-min recovery interval, subjects remained on the bike and either rested or cycled at a low cadence (<50 rpm) against a light resistance (<30 W) to reduce venous pooling in the lower extremities and minimize feelings of light-headedness or nausea. The training protocol consisted of exercise performed three times per week on alternate days (i.e., Monday, Wednesday, Friday) for 2 wk. The number of Wingate tests performed on each training day increased from four to seven over the first five training sessions, and on the final session subjects completed four intervals.

Posttesting. All subjects performed a second series of experimental tests that were identical in all respects to the baseline tests. The training group performed the exercise metabolism test (using the same absolute workloads as during the baseline test) 3 days after the final sprint training session, followed 2 days later by a time trial, 1 day later by a $\dot{V}O_{2 peak}$ test, and 1 day later by a Wingate test. Subjects in the control group performed a second time trial ~2 wk after the baseline test with no sprint training intervention.

Physical Activity and Nutritional Controls

All subjects were instructed to continue their normal dietary and physical activity practices throughout the experimental period. Subjects were also specifically instructed to refrain from any exercise aside from activities of daily living for 2 days before all pre- and posttraining exercise tests. To minimize diet-induced variability in muscle metabolism, subjects were instructed to consume the same types and quantities of food for 2 days before the time trial and exercise metabolism test. Subjects were also required to maintain food diaries before the baseline exercise metabolism test, which were then collected, photocopied, and returned to the subjects before the posttraining test. Subjects were asked to replicate their individual pattern of food intake and to highlight any deviations in the types or amounts of food consumed. Subsequent dietary analyses (Nutritionist Five, First Data Bank, San Bruno, CA) revealed no difference (P = 0.2) in total daily energy intake or the relative macronutrient consumption before the experimental trials before (9.36 ± 0.71 MJ; 57 ± 3% CHO, 30 ± 3% fat, 13 ± 1% protein) or after training (8.44 ± 0.70 MJ, 57 ± 3% CHO, 29 ± 3% fat, 14 ± 1% protein).

Muscle Analyses

One piece of frozen wet muscle (~10–15 mg) from all samples was chipped under liquid nitrogen and used for the determination of the active fraction of PDH (PDH$_a$) using the method described by Constantin-Teodosiu et al. (11) as modified by Putman et al. (36). PDH$_a$ values were adjusted to the highest total creatine content for a given subject to account for differences in blood or connective tissue between samples. A second piece of frozen wet muscle (~10–15 mg) from the resting samples only was chipped under liquid nitrogen and homogenized using the method described by Henrikkson and colleagues (16) to a 50 times dilution. The homogenate was subsequently analyzed to determine the maximal activity of CS on a spectrophotometer (Ultrospec 3000 pro UV/Vis) using a method described by Carter et al. (8), and the maximal activity of 3-hydroxyacyl-CoA dehydrogenase (HAD) on a fluorometer (Hitachi F-2500, Hitachi Instruments, Tokyo, Japan) using a method described by Chi and colleagues (10). Protein content of the muscle homogenate was determined by the method of Bradford (6) using a commercial assay kit (Quick Start, Bio-Rad Laboratories, Hercules, CA), and enzyme data are expressed as moles per kilogram of protein per hour.

The remainder of each muscle sample was freeze-dried, powdered, dissected free of all nonmuscle elements, and stored at −80°C. Aliquots of freeze-dried muscle were extracted with 0.5 M perchloric acid; neutralized with 2.2 M KHCO$_3$; and assayed for lactate, ATP, phosphocreatine, and creatine using standard enzymatic methods (14, 31) adapted for fluorometry (Hitachi F-2500, Hitachi Instruments). For glycogen analysis, an ~2-mg aliquot of freeze-dried muscle was incubated in 2.0 N HCl and heated for 2 h at 100°C to hydrolyse the glycogen to glucosyl units. The solution was subsequently neutralized with an equal volume of 2.0 N NaOH and analyzed for glucose using an enzymatic assay adapted for fluorometry (31).

Statistical Analyses

Time-trial performance data were analyzed using a two-factor mixed ANOVA, with the between factor "group" (training, control) and repeated factor "trial" (pretraining, posttraining). Muscle metabolite and PDH$_a$ data were analyzed using a two-factor repeated-measures ANOVA with the factors "trial" (pretraining, posttraining) and "time" (0, 10, and 20 min). Data from the $\dot{V}O_{2 peak}$ and Wingate tests, the maximal activities of CS and HAD, and net changes in muscle glycogen and lactate during exercise were analyzed using paired *t*-tests (pre- vs. posttraining). Significant interactions or main effects were subsequently analyzed using a Tukey's honestly significant difference post hoc test, and the level of significance for all analyses was set at P ≤ 0.05. All data are presented as means ± SE based on eight subjects per group, except for the cardiorespiratory data, which are based on a mean of six subjects because of technical problems during some of the posttraining exercise metabolism tests.

RESULTS

Time-Trial Performance

Time required to complete the 250-kJ time trial decreased (P = 0.004) by 9.6% after training (Fig. 1), and this was reflected by an increase in average power output from 247 ± 37 to 272 ± 24 W (P = 0.004). The effect of training was also evidenced by the fact that peak and mean power output elicited during a 30-s Wingate test increased after training by 5.4% (posttraining: 1,016 ± 97 vs. pretraining: 964 ± 88 W; P = 0.04) and 8.7% (posttraining: 854 ± 86 vs. pretraining: 786 ± 68 W; P = 0.02), respectively, and percent fatigue was reduced by 17.9% (posttraining: 28 ± 2 vs. pretraining: 35 ± 3%; P = 0.002). The control group showed no change in time-trial performance (P = 0.74) when tested 2 wk apart with no
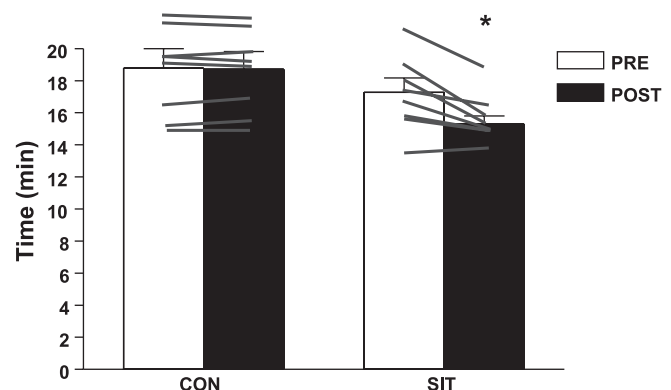
Fig. 1. Time required to complete a 250-kJ cycling time trial (TT) before (Pre) and after 2 wk (Post) of sprint interval training (SIT) or equivalent period without training [control (Con)]. Values are means ± SE for 8 subjects per group. Individual data are plotted for all subjects in each group. *P ≤ 0.05 vs. pretraining.
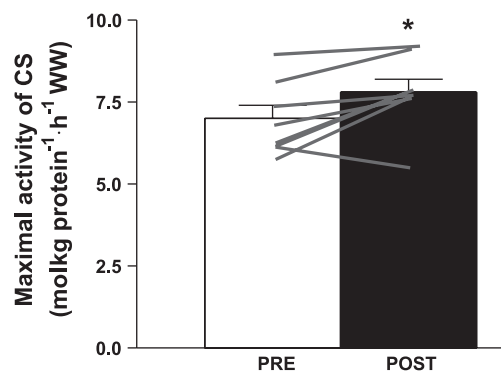
Fig. 2. Maximal activity of citrate synthase (CS) measured in resting muscle biopsy samples before and after 2 wk of sprint interval training. All values are means $\pm$ SE for 8 subjects per group. WW, wet weight. *$P \le 0.05$ vs. pretraining.

Fig. 4. Muscle glycogen content measured at rest and after each stage of a matched-work exercise bout that consisted of 10 min at ~60% $\dot{V}O_{2 \, peak}$ followed by 10 min at ~90% $\dot{V}O_{2 \, peak}$ before and after 2 wk of sprint interval training. Values are means $\pm$ SE for 8 subjects per group. DW, dry weight. *Main effect for trial ($P = 0.03$), such that posttraining > pretraining. There was no condition $\times$ time interaction ($P = 0.06$); however, net muscle glycogenolysis during the exercise bout was lower posttraining vs. pretraining ($P = 0.03$).

training intervention (Fig. 1), and average power output was similarly unchanged (posttraining: 229 ± 14 vs. pretraining: 231 ± 15 W; $P = 0.37$).

Maximal Activities of Mitochondrial Enzymes

The maximal activity of CS increased ($P = 0.04$) by 11% after training (Fig. 2), but the maximal activity of HAD was unchanged (posttraining: 5.1 ± 0.7 vs. pretraining: 4.9 ± 0.6 mol$\cdot$kg protein$^{-1} \cdot$h^{-1}; $P = 0.76$).

Muscle Metabolic and Cardiorespiratory Data During Matched-Work Exercise

PDH$_a$ was higher after training (main effect, $P = 0.04$), but there was no interaction between trials (Fig. 3). Muscle glycogen content was higher after training (main effect, $P = 0.0001$; Fig. 4), and whereas there was no interaction effect ($P = 0.06$), net muscle glycogenolysis during exercise was reduced after training (posttraining: 100 ± 16 vs. pretraining: 139 ± 11 mmol/kg dry wt; $P = 0.03$). Muscle lactate content was lower after training (main effect, $P = 0.02$; Fig. 5), and whereas there

was no interaction effect ($P = 0.07$), net lactate accumulation during exercise was reduced after training (posttraining: 55 ± 2 vs. pretraining: 63 ± 1 mmol/kg dry wt; $P = 0.03$). The muscle contents of creatine and ATP were lower after training (main effects, $P = 0.002$ and $P = 0.007$, respectively), but phosphocreatine content was not different ($P = 0.09$) (Table 2). $\dot{V}O_{2 \, peak}$ was not different after training (posttraining: 51.6 ± 2.1 vs. pretraining: 48.9 ± 2.1 ml$\cdot$kg$^{-1} \cdot$min^{-1}; $P = 0.13$), and there were no training-induced changes in mean exercise oxygen uptake, respiratory exchange ratio, or heart rate during the exercise metabolism test (Table 2).

DISCUSSION

The main finding from the present study was that short-term SIT decreased net muscle glycogenolysis and lactate accumu-
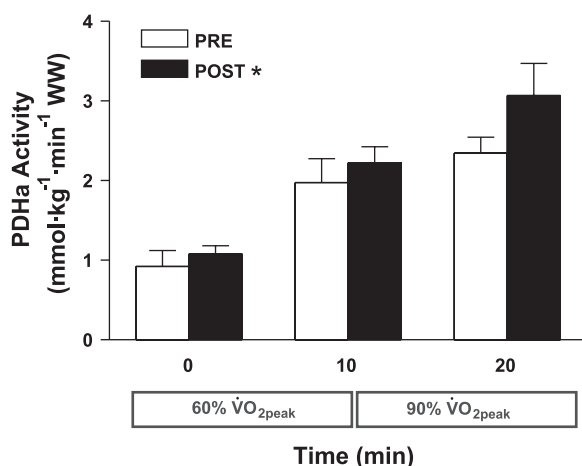
Fig. 3. Muscle pyruvate dehydrogenase activity (PDH$_a$) measured at rest and after each stage of a matched-work exercise bout that consisted of 10 min at ~60% peak oxygen uptake ($\dot{V}O_{2 \, peak}$) followed by 10 min at ~90% $\dot{V}O_{2 \, peak}$ before and after 2 wk of sprint interval training. Values are means $\pm$ SE for 8 subjects per group. *Main effect for trial ($P = 0.04$), such that posttraining > pretraining.
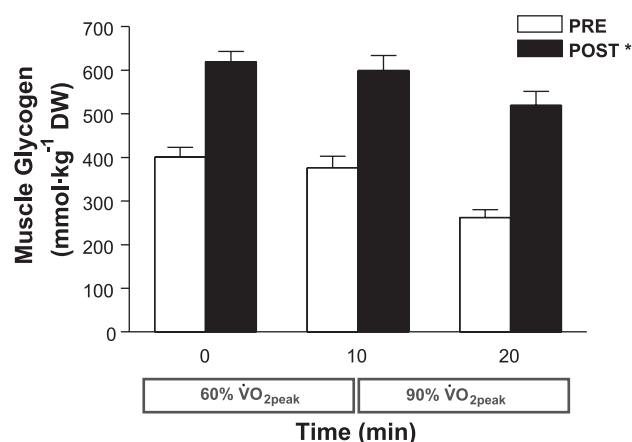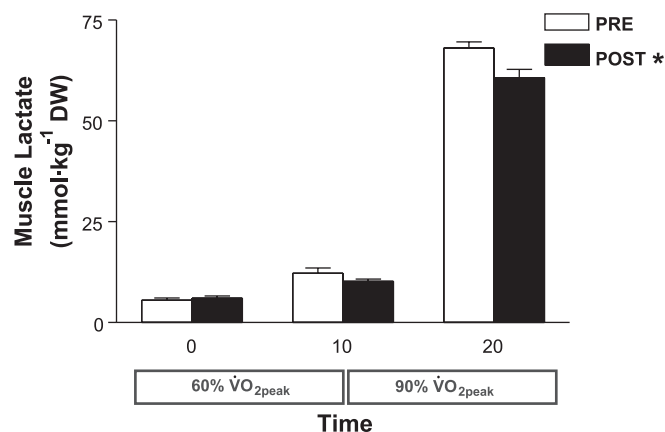
Fig. 5. Muscle lactate content measured at rest and after each stage of a matched-work exercise bout that consisted of 10 min at ~60% $\dot{V}O_{2 \, peak}$ followed by 10 min at ~90% $\dot{V}O_{2 \, peak}$ before and after 2 wk of sprint interval training. Values are means $\pm$ SE for 8 subjects per group. *Main effect for trial ($P \le 0.05$), such that posttraining < pretraining. There was no condition $\times$ time interaction ($P = 0.07$); however, net muscle lactate accumulation during the exercise bout was lower posttraining vs. pretraining ($P = 0.03$).

Table 2. *Cardiorespiratory and muscle metabolite data during a two-stage match-work exercise test before and after training*

Variable	Condition	Rest	60% $\dot{V}_{O_{2peak}}$	90% $\dot{V}_{2peak}$
$\dot{V}_{O_2}$, l/min	Pretraining	ND	2.33±0.09	3.72±0.11
	Posttraining	ND	2.32±0.10	3.66±0.16
Heart rate, beats/min	Pretraining	ND	132±2	178±2
	Posttraining	ND	132±3	175±2
RER	Pretraining	ND	0.96±0.03	1.17±0.05
	Posttraining	ND	0.96±0.03	1.15±0.03
Creatine, mmol/kg dry wt	Pretraining	31±2	52±3	112±3
	Posttraining*	24±2	38±2	104±5
Phosphocreatine, mmol/kg dry wt	Pretraining	98±4	69±3	15±2
	Posttraining	99±2	78±4	20±4
ATP, mmol/kg dry wt	Pretraining	24±1	25±1	23±1
	Posttraining*	20±1	22±2	21±1

Values are means ± SE for 8 subjects for metabolite data and 6 subjects for cardiorespiratory data. ND, not determined; $\dot{V}_{O_2}$, oxygen uptake; RER, respiratory exchange ratio. *Main effect for trial ($P < 0.05$), posttraining vs. pretraining.

lation during matched-work exercise and increased the capacity for pyruvate oxidation through PDH. We also showed that the time required to complete a 250-kJ cycling time trial decreased by 10%, and, to our knowledge, this is the first demonstration that sprint training improves performance during an aerobic-based task that simulates the manner in which athletes typically complete. Finally, the present study confirms our previous finding that muscle oxidative capacity can be enhanced by only a few brief sessions of very intense exercise training in young active individuals (7).

It is well established that as few as 3–10 consecutive days of exercise training for 2 h/day at 60–70% of $\dot{V}_{O_{2\ peak}}$ reduce muscle glycogenolysis and lactate accumulation compared with pretraining at the same absolute workload (9, 12, 35, 41). However, it remains controversial whether these adaptations precede (12, 35) or are attributable to an increase in mitochondrial capacity (9, 41), as judged by changes in the maximal activity of CS. In the present study, we found that six sessions of SIT over 2 wk, or a total of only 16 min of very intense exercise, decreased net muscle glycogenolysis, and lactate accumulation during a matched-work exercise test. These changes were accompanied by an increase in CS maximal activity, although the relative increase was smaller than observed in our laboratory's previous study (7), despite an identical training stimulus. The difference in response magnitude could be related in part to genetic differences in the adaptive response to training (43) or the fact that subjects in the present study were more fit to begin with, as evidenced by a higher mean $\dot{V}_{O_{2\ peak}}$ and CS maximal activity. There was no change in HAD in the present study, which suggests that short-term SIT does not stimulate a coordinated increase in all mitochondrial enzymes. This finding is supported by work from MacDougall et al. (24), who reported large increases in the maximal activities of several tricarboxycylic acid cycle enzymes, including CS, after 7 wk of Wingate-based training but no significant change in HAD. Other studies have suggested that sprint training increases HAD (e.g., Ref. 30), and given these conflicting data, future studies should directly evaluate whether sprint training increases the maximal capacity for lipid oxidation in skeletal muscle, for example, by using stable isotopic tracers or by measuring the maximal activity of the rate-determining enzyme carnitine palmitoyl transferase (1).

PDH_a was higher after training, and thus the present data confirm previous speculations by others (13, 24, 30) that suggested sprint training may increase the maximum capacity to oxidize pyruvate. For example, Harmer et al. (13) proposed that "brief, intense exercise. . .may result in greater PDHa during exercise (and) a slower rate of pyruvate presentation would probably permit a greater proportion to be oxidized, thus constituting a considerable energetic advantage after training." Although the measured active form of PDH has been shown to closely match estimated PDH flux in vivo (41), there were no training-induced changes in whole body gas exchange during exercise in the present study (Table 2). Theoretically, an increase in PDH flux should result in an increase in active muscle oxygen uptake if the pyruvate-derived acetyl units are consumed in the process of oxidative phosphorylation. However, it is unlikely that the small increase in CHO oxidation needed to offset the reduction in nonoxidative ATP provision in the present study could have been detected at the whole body level.

The reason for the higher PDH_a after training is unclear, but given the complex regulation of this multienzyme complex, the adaptive response could involve changes in PDH itself or the associated regulatory enzymes PDH phosphatase (PDP) or PDH kinase (PDK), which serve to activate and inhibit the enzyme complex, respectively (32). Training could have induced either acute changes in intramitochondrial effectors of PDH (i.e., signals that sense muscle contractile state, cellular energy charge, and substrate and/or product availability), or stable changes in total protein content or intrinsic activities of PDH, PDP, and PDK. With respect to acute regulators, it is traditionally believed that calcium stimulation of PDP is the initial and most powerful signal that activates PDH at the start of exercise (41). Ortenblad et al. (28) showed that high-intensity intermittent cycle training enhanced peak sarcoplasmic calcium release, and thus it is possible that the higher PDH_a after training in the present study was due in part to transient alterations in calcium handling that increased PDP. This interpretation is supported by data from Ward and colleagues (44), who showed that resistance training increased PDH_a even though total PDH activity was unchanged. The authors of that study (44) noted that "the type of strength training used in the present study resembles 'sprint' training" and attributed the higher PDH activity to training-induced changes in PDP sensitivity to calcium.

The training-induced increase in PDH_a in the present study may have also been related to an increase in total PDH activity and/or stable changes in PDP or PDK. Two studies (21, 34) have reported no change in total PDH activity after 5–7 consecutive days of aerobic-based exercise and concluded that any regulation of PDH with short-term training would be through acute regulators acting on PDP and PDK. However, given that high-intensity exercise results in rapid, maximal conversion of PDH to its active form (29, 34), it is possible that the time course for changes in total PDH after sprint training may differ from traditional endurance training. Clearly, it is possible to increase the maximal activity of some mitochondrial enzymes such as CS after short-term sprint training (present data and Ref. 7), whereas the effect of short-term endurance training on mitochondrial capacity is equivocal (9,

12, 35, 41). Total PDH activity in skeletal muscle is increased after a longer period of endurance training (8 wk), as recently demonstrated by LeBlanc et al. (22) for the first time in humans. However, endurance training also increased the PDK-2 isoform (22), which the authors proposed would increase metabolic control sensitivity to pyruvate and reduce PDH_a during submaximal exercise, as shown in a separate study (21). Additional work is warranted to clarify the specific factors responsible for changes in PDH_a after short-term sprint training and to determine the effect of long-term sprint training on skeletal muscle fuel metabolism.

A final observation with respect to metabolic changes is that we measured a 20% decrease in muscle ATP content after training in the present study, which differs from the results of our laboratory's previous study (7), but is comparable to the 19% decrease previously reported by Stathis et al. (42) after a 7-wk sprint training program. The discrepancy between studies is likely related in part to individual differences in purine nucleotide metabolism during intense exercise and recovery. During strenuous exercise, AMP produced from ATP hydrolysis can be deaminated by AMP deaminase, resulting in the formation of IMP and ammonia, and subsequent breakdown of IMP to inosine, and hypoxanthine results in a loss of adenine nucleotides from the muscle (15). Replacement of purine nucleotides lost from the muscle is a relatively slow, energy-consuming process and appears to continue for several days after intense exercise (15). Thus the lower ATP content measured after training in the present study may have been due to the stress of chronic training or the acute residual effects of the final training bout, which was performed 72 h before tissue extraction.

With respect to exercise performance, our laboratory recently showed that six sessions of SIT performed over 2 wk dramatically improved cycle endurance capacity, such that the mean time to exhaustion during cycling at $\sim$80% $\dot{V}_{O_2 \, peak}$ increased from 25 to 51 min (7). Because many athletic events require athletes to complete a fixed amount of work in as short a time as possible (i.e., a race), in the present study we incorporated a time trial to evaluate potential changes in volitional exercise capacity. We found that time-trial performance improved by 9.6% after only 2 wk of sprint training, despite no change in $\dot{V}_{O_2 \, peak}$. Thus the physiological adaptations conferred by a short period of sprint training not only increase aerobic endurance capacity (7) but also increase the mean power output than can be sustained during a fixed work bout that is dominated by aerobic metabolism. As in our laboratory's previous study (7), the validity of our performance data is bolstered by the fact that all subjects performed extensive familiarization trials before the experiment, and a control group showed no change in performance when tested 2 wk apart with no training intervention (Fig. 1). The training-induced improvement in time-trial performance is noteworthy considering that our subjects were active individuals who were already quite fit at the start of the study [the mean $\dot{V}_{O_2 \, peak}$ at baseline for the trained group was within the 80th percentile for this age group (2)]. Nonetheless, whereas seven of eight subjects in the trained group showed similar improvements in time-trial performance (Fig. 1), the one nonresponder was the individual who posted the fastest time trial at the beginning of the study. Additional studies are warranted to evaluate the adaptations induced by short-term SIT in very fit subjects, but

other investigators have shown that highly trained athletes can benefit from a period of intensified training that is characterized by short bouts of intense exercise (45).

In summary, the results from the present study demonstrate that six sessions of SIT decreased net muscle glycogenolysis and lactate accumulation during submaximal exercise and increased the activity of PDH_a. The net result was consistent with a closer matching between muscle pyruvate production and oxidation. This is also the first study to show that sprint training improves aerobic exercise performance during a laboratory time trial that closely simulates the way in which athletes typically compete. Finally, the present data confirm the novel results from our laboratory's recent study (7) that showed skeletal muscle oxidative capacity can be enhanced by a brief 2-wk period of sprint training, equivalent to only 16 min of very intense exercise spread over a total time commitment of $\sim$2.5 h.

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