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

Kirsten A. Burgomaster, James 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 (V\textsuperscript{\textcircled{O}2} 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 V\textsuperscript{\textcircled{O}2} 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\textsuperscript{−1}·h\textsuperscript{−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\textsuperscript{−1}·h\textsuperscript{−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, V\textsuperscript{\textcircled{O}2} peak = 3.9 ± 0.2 l/min) showed no change in performance when tested 2 wk after 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 glycolytic flux and pyruvate oxidation during submaximal exercise.

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 (V\textsuperscript{\textcircled{O}2} 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 oxidative energy provision.

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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 event 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 VO$_2$ 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 times. In addition to the exercise at each workload.

**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 electronically braked cycle ergometer (Excalibur Sport V2.0, Lode, Groningen, The Netherlands) to determine VO$_2$ peak using an online gas-collection system (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.

**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 VO_{2peak} 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 > 0.05) of food consumed. Subsequent dietary analyses (Nutritionist Five, training test. Subjects were asked to replicate their individual pattern diaries before the baseline exercise metabolism test, which were then project were also specifically instructed to refrain from any exercise physical activity practices throughout the experimental period. Subjects were also required to maintain food metabolism, subjects were instructed to consume the same

Muscle Analyses

One piece of frozen wet muscle (~10–15 mg) from all samples was shipped 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 shipped 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 (Uitrospec 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 PDHa 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 VO_{2peak} 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
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·kg protein\(^{-1}\)·h\(^{-1}\); \( P = 0.76 \)).

**Muscle Metabolic and Cardiorespiratory Data During Matched-Work Exercise**

PDHa 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·kg\(^{-1}\)·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 accu-
sprint training increases the maximal capacity for lipid oxidation. Other studies have suggested that pretraining at the same absolute workload (9, 12, 35, 41).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Condition</th>
<th>Rest</th>
<th>60% VO2peak</th>
<th>90% VO2peak</th>
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<tbody>
<tr>
<td>VO2, l/min</td>
<td>Pretraining</td>
<td>ND</td>
<td>2.33 ± 0.09</td>
<td>3.72 ± 0.11</td>
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<tr>
<td></td>
<td>Posttraining</td>
<td>ND</td>
<td>2.32 ± 0.10</td>
<td>3.66 ± 0.16</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>Pretraining</td>
<td>ND</td>
<td>123 ± 2</td>
<td>178 ± 2</td>
</tr>
<tr>
<td></td>
<td>Posttraining</td>
<td>ND</td>
<td>123 ± 2</td>
<td>175 ± 2</td>
</tr>
<tr>
<td>RER</td>
<td>Pretraining</td>
<td>ND</td>
<td>0.96 ± 0.03</td>
<td>1.17 ± 0.05</td>
</tr>
<tr>
<td></td>
<td>Posttraining</td>
<td>ND</td>
<td>0.96 ± 0.03</td>
<td>1.15 ± 0.03</td>
</tr>
<tr>
<td>Creatine, mmol/kg dry wt</td>
<td>Pretraining</td>
<td>31 ± 2</td>
<td>52 ± 3</td>
<td>112 ± 3</td>
</tr>
<tr>
<td></td>
<td>Posttraining</td>
<td>24 ± 2</td>
<td>38 ± 2</td>
<td>104 ± 5</td>
</tr>
<tr>
<td>Phosphocreatine, mmol/kg dry wt</td>
<td>Pretraining</td>
<td>98 ± 4</td>
<td>69 ± 3</td>
<td>15 ± 2</td>
</tr>
<tr>
<td></td>
<td>Posttraining</td>
<td>99 ± 2</td>
<td>78 ± 4</td>
<td>20 ± 4</td>
</tr>
<tr>
<td>ATP, mmol/kg dry wt</td>
<td>Pretraining</td>
<td>24 ± 1</td>
<td>25 ± 1</td>
<td>23 ± 1</td>
</tr>
<tr>
<td></td>
<td>Posttraining</td>
<td>20 ± 1</td>
<td>22 ± 2</td>
<td>21 ± 1</td>
</tr>
</tbody>
</table>

Values are means ± SE for 8 subjects for metabolite data and 6 subjects for cardiorespiratory data. ND, not determined; VO2, oxygen uptake; RER, respiratory exchange ratio. *Main effect for trial (P < 0.05), posttraining vs. pretraining.

The reason for the higher PDHα after training is unclear, but given the complex regulation of this multi-enzyme 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 PDHα 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 PDHα 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 PDHα 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,
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 PDHα during submaximal exercise, as shown in a separate study (21). Additional work is warranted to clarify the specific factors responsible for changes in PDHα 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 ~80% VO2peak 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 VO2peak. 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 VO2peak 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 PDHα. 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 ~2.5 h.

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