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Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans

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Burgomaster, Kirsten A., Scott C. Hughes, George J. F. Heigenhauser, Suzanne N. Bradwell, and Martin J. Gibala. Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J Appl Physiol* 98: 1985–1990, 2005. First published February 10 2005; doi:10.1152/japplphysiol.01095.2004.—Parra et al. (*Acta Physiol. Scand* 169: 157–165, 2000) showed that 2 wk of daily sprint interval training (SIT) increased citrate synthase (CS) maximal activity but did not change "anaerobic" work capacity, possibly because of chronic fatigue induced by daily training. The effect of fewer SIT sessions on muscle oxidative potential is unknown, and aside from changes in peak oxygen uptake ($\dot{V}O_{2\,peak}$), no study has examined the effect of SIT on "aerobic" exercise capacity. We tested the hypothesis that six sessions of SIT, performed over 2 wk with 1–2 days rest between sessions to promote recovery, would increase CS maximal activity and endurance capacity during cycling at ~80% $\dot{V}O_{2\,peak}$. Eight recreationally active subjects [age = 22 ± 1 yr; $\dot{V}O_{2\,peak}$ = 45 ± 3 ml·kg^{-1}·min^{-1} (mean ± SE)] were studied before and 3 days after SIT. Each training session consisted of four to seven "all-out" 30-s Wingate tests with 4 min of recovery. After SIT, CS maximal activity increased by 38% (5.5 ± 1.0 vs. 4.0 ± 0.7 mmol·kg protein^{-1}·h^{-1}) and resting muscle glycogen content increased by 26% (614 ± 39 vs. 489 ± 57 mmol/kg dry wt) (both $P < 0.05$). Most strikingly, cycle endurance capacity increased by 100% after SIT (51 ± 11 vs. 26 ± 5 min; $P < 0.05$), despite no change in $\dot{V}O_{2\,peak}$. The coefficient of variation for the cycle test was 12.0%, and a control group ($n = 8$) showed no change in performance when tested ~2 wk apart without SIT. We conclude that short sprint interval training (~15 min of intense exercise over 2 wk) increased muscle oxidative potential and doubled endurance capacity during intense aerobic cycling in recreationally active individuals.

Wingate test; citrate synthase; muscle glycogen

PERFORMING REPEATED BOUTS of high-intensity "sprint"-type exercise over several weeks or months induces profound changes in skeletal muscle. A wide range of muscle metabolic and morphological adaptations have been described (25, 34); however, the magnitude and direction of change in many variables depend on the nature of the training protocol, i.e., the frequency, intensity, and duration of sprint efforts as well as the recovery between bouts. Given the significant contribution from aerobic energy metabolism during repeated sprinting (3, 26, 29, 40), it is not surprising that an increase in muscle oxidative potential, as indicated by changes in the maximal activities of "marker" enzymes such as citrate synthase, has been reported after 6–8 wk of sprint training (19, 25). Recently, two studies reported large increases in citrate synthase maximal activity, as well as peak oxygen uptake ($\dot{V}O_{2\,peak}$), after only 2 wk of daily sprint training (30, 33). These data suggest that improvements in aerobic energy metabolism can be rapidly stimulated by brief bouts of very intense exercise; however, the effect of fewer sprint training sessions is not known. In addition, aside from changes in $\dot{V}O_{2\,peak}$, we are aware of no data that suggest sprint training leads to an increased ability to perform exercise that is primarily "aerobic" in nature, e.g., an endurance test to fatigue at a fixed submaximal workload.

The primary purpose of the present study, therefore, was to examine the effect of six sessions of sprint interval training on muscle oxidative potential, $\dot{V}O_{2\,peak}$, and endurance time to fatigue during cycling at an intensity equivalent to ~80% $\dot{V}O_{2\,peak}$. On the basis of pilot work in our laboratory that showed modest performance improvements after 6 consecutive days of sprint training, we decided to employ a 2-wk training intervention, such that 1–2 days of rest were permitted between training sessions, in an effort to promote recovery and facilitate performance adaptations. The importance of rest days between sprint training sessions was emphasized in a recent study (30) that showed peak and mean power elicited during a Wingate test were unchanged after 14 consecutive days of sprint training; however, when subjects performed the same number of training sessions over 6 wk (i.e., with 1–2 days of rest between training sessions), power output improved significantly. Although numerous mechanisms could potentially be involved, the importance of rest days between training sessions may be related in part to the fact that strenuous exercise leads to inactivation of muscle cation pumps (23, 36), and it has been speculated that up to several days may be required for normalization of sarcoplasmic reticulum Ca^{2+} pump function (41).

Thus the mode and intensity of sprint efforts in the present study was similar to two recent studies that incorporated 2-wk training interventions (30, 33); however, the overall volume was reduced by approximately two-thirds and in total amounted to only ~15 min of exercise over 2 wk. We hypothesized that our short sprint training protocol would increase muscle oxidative potential and cycle endurance capacity. We also measured resting muscle glycogen concentration because only a few sprint training studies have done so and these have yielded conflicting results (14, 27, 30, 33, 28). Our experimental design included a control group who completed the exercise performance tests ~2 wk apart with no training intervention,

and all subjects performed extensive familiarization trials before baseline testing.

METHODS

Subjects

Sixteen healthy individuals volunteered to take part in the experiment (Table 1). Eight subjects (2 women) were assigned to a training group and performed exercise tests before and after a 2-wk sprint training intervention. Eight other men served as a control group and performed the exercise performance tests ~2 wk apart with no training intervention. We also obtained needle biopsy samples from the training group to examine potential training-induced adaptations in resting skeletal muscle. We did not obtain biopsies from the control group for ethical reasons, because other studies have shown no change in resting muscle metabolite concentrations or the maximal activities of mitochondrial enzymes when control subjects are tested several weeks apart with no sprint training intervention (1, 28). All subjects were recreationally active individuals from the McMaster University student population who participated in some form of exercise two to three times per week (e.g., jogging, cycling, aerobics), but none was engaged in any sort of structured training program. After routine medical screening, the subjects were informed of the procedures to be employed in 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 taking part in any experimental trial (i.e., before baseline measurements), all subjects performed familiarization trials to become oriented with all testing procedures and training devices. Specifically, all subjects performed *1*) a $\dot{V}_{O_2 peak}$ test; *2*) a "practice ride" to establish a workload that elicited ~80% of $\dot{V}_{O_2 peak}$; and *3*) an endurance capacity test that consisted of cycling to volitional fatigue at ~80% of $\dot{V}_{O_2 peak}$ on at least two separate occasions.

Details of Exercise Performance Tests

$\dot{V}_{O_2 peak}$ test. Subjects performed an incremental test to exhaustion on an electronically 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 then increased by 25 W every minute until volitional exhaustion. The value used for $\dot{V}_{O_2 peak}$ corresponded to the highest value achieved over a 30-s collection period.

Cycle endurance capacity test. Subjects cycled to volitional exhaustion on an electronically braked cycle ergometer (Lode) at a workload designed to elicit ~80% of $\dot{V}_{O_2 peak}$. All performance trials were conducted in the absence of temporal, verbal, or physiological feedback. The test was terminated when pedal cadence fell below 40 rpm (according to the manufacturer's specifications, the power output displayed may not have been valid below this cadence), and exercise duration was recorded. Expired breath samples for the determination

Table 1. *Subject characteristics*

	Training Group	Control Group
Age, yr	22±1	25±2
Weight, kg	83±5	79±2
Height, cm	180±4	180±2
$\dot{V}_{O_2 peak}$, ml·kg^{-1}·min^{-1}	44.6±3.2	46.4±1.4

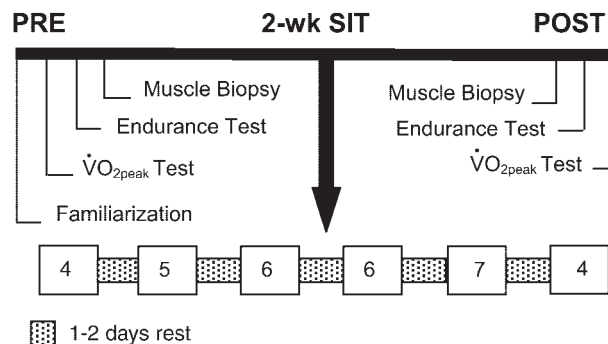Values are means ± SE for 8 subjects. $\dot{V}_{O_2 peak}$, peak oxygen uptake.

Fig. 1. Overview of experimental protocol. $\dot{V}_{O_2 peak}$, peak oxygen uptake; PRE, preexercise; POST, postexercise; SIT, sprint interval training. Numbers in boxes denote number of Wingate tests completed during each of 6 training sessions over a 2-wk period.

of ventilation rate, oxygen uptake, carbon dioxide production, and respiratory exchange ratio were collected and averaged over the 6- to 10-min period of exercise.

Reproducibility of exercise performance tests. Ten individuals who were not subjects in the present study performed a $\dot{V}_{O_2 peak}$ test and cycle endurance capacity test on separate days at least 1 wk apart, and method error reproducibility was calculated as described by Sale (35). The coefficient of variation for the $\dot{V}_{O_2 peak}$ test and cycle endurance capacity test was 3.7 and 12.0%, respectively.

Experimental Protocol

The experimental protocol consisted of *1*) baseline testing (i.e., after familiarization procedures described above); *2*) a 2-wk sprint training intervention or similar period without sprint training (control group); and *3*) posttesting, as described further below.

Baseline testing. Baseline measurements for all subjects consisted of a $\dot{V}_{O_2 peak}$ test and a cycle endurance capacity test. Each baseline test was conducted on a separate day with 24 h between tests. Subjects in the training group also underwent a muscle biopsy procedure 3 days after the baseline cycle endurance capacity test and several days before the start of the training intervention. For the biopsy procedure, the area over the lateral portion of one thigh was anesthetized (2% lidocaine, AstraZeneca Canada, Ontario, Canada), and a small incision was made through the skin and underlying fascia to permit a tissue sample (50–100 mg) to be obtained from the vastus lateralis muscle (1). Details regarding the experimental protocol are summarized in Fig. 1.

Training. Training was initiated 3–5 days after the baseline muscle biopsy procedure and consisted of six sessions of sprint interval training spread over 14 days. Each training session consisted of repeated 30-s "all-out" efforts on an electronically braked cycle ergometer (Lode) against a resistance equivalent to 0.075 kg/kg body mass (i.e., a Wingate test). Subjects were instructed to begin pedaling as fast as possible against the ergometer's inertial resistance, ~2 s before the appropriate load was applied by a computer interfaced with the ergometer and loaded with appropriate software (Wingate software version 1.11, Lode). 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. During the 4-min recovery period between tests, subjects remained on the bike and either rested or were permitted to cycle 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 each day during training increased

from 4 to 7 over the first five training sessions, and on the final session subjects completed four intervals, as summarized in Fig. 1.

Posttesting. A second muscle biopsy sample was obtained 3 days after the final training session to examine training-induced changes in resting muscle, and a second battery of performance tests was initiated 2 days after the biopsy procedure (Fig. 1). The control group performed a second set of tests ~2 wk after the baseline tests. The nature of the posttesting exercise performance measurements were identical in all respects to the baseline tests.

Dietary Controls

In an attempt to minimize any potential diet-induced variability in exercise metabolism and the resting metabolic profile of skeletal muscle, subjects were instructed to consume the same types and quantities of food during the baseline and posttesting phases. The subjects in the training group were particularly encouraged to keep their diet as similar as possible during the 24 h before the pre- and posttraining biopsy procedures. Subjects were asked to record all food intake during these periods, and compliance was assessed by performing dietary analyses on the individual food records maintained by the subjects. Pre- and posttraining food diaries were analyzed for total energy intake and proportion of energy derived from carbohydrates, fats, and protein (Nutritionist Five, First Data Bank, San Bruno, CA). These analyses confirmed that there was no difference between trials in the total amount of energy consumed or macronutrient proportions.

Muscle Analyses

On removal from the leg, each muscle biopsy sample was immediately frozen by plunging the biopsy needle into liquid nitrogen. The samples were subsequently divided into two pieces while still frozen, and one piece was kept in liquid nitrogen for the determination of muscle enzyme activities. The remainder of each sample was freeze-dried, powdered, dissected free of blood and connective tissue, and stored at −86°C before metabolite analyses.

Citrate synthase. Frozen wet muscle samples were initially homogenized using methods described by Henriksson and Reitman (17) to a 50-fold dilution. The maximal activity of citrate synthase was determined on a spectrophotometer (Ultrospec 3000 pro UV/Vis) using a method described by Carter et al. (6). The intra-assay coefficient of variation for the citrate synthase assay, based on 10 repeats of the same sample, was 4.9%. Protein content of the homogenate was determined by the method of Bradford (5) 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.

Metabolites. An aliquot of freeze-dried muscle was extracted on ice using 0.5 M perchloric acid (containing 1 mM EDTA), neutralized with 2.2 M KHCO₃, and the resulting supernatant was used for the determination of all metabolites except glycogen. ATP, phosphocreatine and creatine were measured using enzymatic assays adapted for fluorometry (Hitachi F-2500, Hitachi Instruments, Tokyo, Japan) (15, 31). 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 hydrolyze the glycogen to glucosyl units. The solution was subsequently neutralized with an equal volume of 2.0 N NaOH and analyzed for glucose by using an enzymatic assay adapted for fluorometry (31). The intra-assay coefficient of variation for all muscle metabolite assays, based on 10 repeats of the same sample, ranged from 2 to 3%. All muscle metabolite measurements were corrected to the peak total creatine concentration for a given subject.

Statistical Analyses

All exercise performance data were analyzed by using a two-factor repeated-measures ANOVA. For the single Wingate test, endurance capacity test and $\dot{V}o_{2 peak}$ test, the factors were trial (pretraining, posttraining) and condition (training, control). For the comparison of power output during the first vs. last sprint training session (training group only), the factors were trial (pretraining, posttraining) and sprint bout (*1–4*). All muscle data were analyzed using paired (2-tailed) *t*-tests. The level of significance for analyses was set at $P < 0.05$, and significant interactions and main effects were subsequently analyzed using Tukey's honestly significant difference post hoc test. All data are presented as means ± SE.

RESULTS

Cycle endurance capacity. After training, the individual improvements in cycle endurance capacity ranged from 81 to 169% compared with baseline, with the exception of one subject (16% decrease) who, on completion of the study, disclosed that he had sustained a minor ankle injury (unrelated to the experiment) on the day before his posttraining ride. Even with the inclusion of this subject's data (Fig. 2), the mean increase in cycle endurance time to fatigue for the training group ($n = 8$) was 100% compared with baseline (51 ± 11 vs. 26 ± 5 min; $P < 0.05$), and this was higher ($P < 0.05$) compared with the control group, who showed no change in performance (Fig. 2). Oxygen uptake during exercise was not different between the first and second rides in either group; however, expired ventilation (posttraining: 91 ± 7 vs. pretraining: 104 ± 9 l/min) and respiratory exchange ratio (posttraining: 1.18 vs. pretraining: 1.24) were lower ($P < 0.05$) after training in the sprint-training group ($P < 0.05$). $\dot{V}o_{2 peak}$ did not change in either group over the course of the study.

Anaerobic work capacity. Peak power output during each of the four consecutive Wingate tests performed during the last (sixth) training session was higher ($P < 0.05$) compared with the first training session (Fig. 3). However, fatigue index was also higher ($P < 0.05$) posttraining, and thus there were no differences in mean power output for each of the four Wingate tests during the first compared with the last training session.

Citrate synthase activity and resting muscle metabolite concentrations. The maximal activity of citrate synthase increased ($P < 0.05$) by 38% after training (Fig. 4). Resting muscle glycogen concentration increased ($P < 0.05$) by 26% after training (Fig. 5); however, there were no training-induced changes in the resting muscle concentrations of ATP, phosphocreatine or creatine (Table 2).

DISCUSSION

The primary novel finding from the present study was that six bouts of sprint interval training performed over 14 days
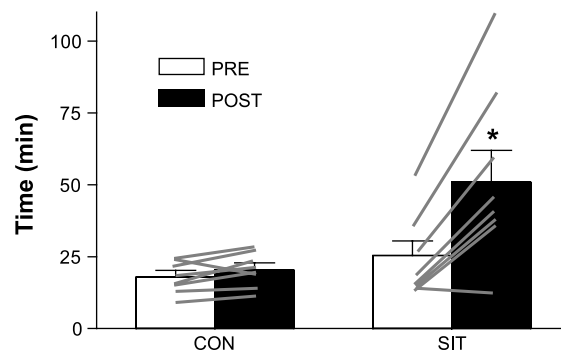

Fig. 2. Cycle endurance time to fatigue before and after a 2-wk sprint training protocol (training group; SIT) or equivalent period without training (control; Con). Values are means ± SE for 8 subjects. Individual data are also plotted for all subjects in each group. *$P < 0.05$.
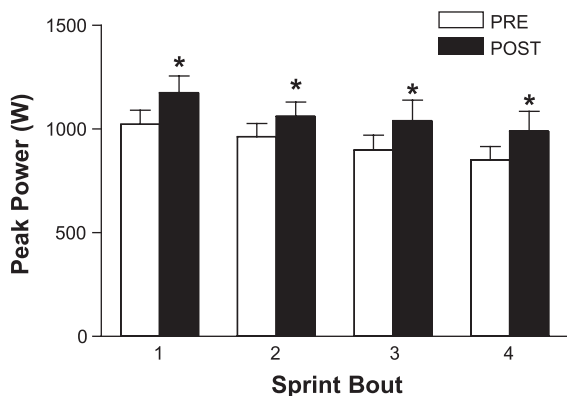
Fig. 3. Peak anaerobic power elicited during 4 consecutive Wingate tests performed during the first and last sprint training session. Values are means ± SE for 8 subjects. *$P < 0.05$.
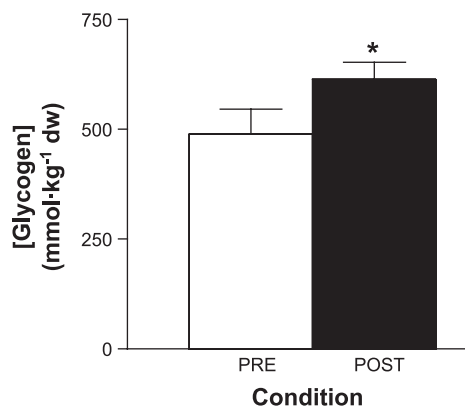

Fig. 5. Muscle glycogen concentration measured in resting biopsy samples obtained before and after a 2-wk sprint training protocol. Values are means ± SE for 8 subjects. dw; Dry weight. *$P < 0.05$.

increased muscle oxidative potential and doubled endurance time to fatigue during cycling at ~80% $\dot{V}_{O_2 peak}$ in recreationally active subjects. The validity of this latter observation is bolstered by the fact that all subjects performed extensive familiarization trials before testing and that a control group showed no change in endurance performance when tested 2 wk apart with no sprint training intervention. We also detected increases in resting muscle glycogen content after sprint training. The present data therefore demonstrate that short, repeated bouts of 30-s all-out cycling efforts, amounting to ~15 min of total exercise over 2 wk, dramatically increased cycle endurance capacity and favorably altered the resting metabolic profile of human skeletal muscle. Although increases in citrate synthase activity and glycogen content have been previously reported after several weeks of sprint interval training (19, 25, 30, 33), the data are equivocal (8, 10, 14, 24, 27), and we show here that the total training volume necessary to stimulate these metabolic adaptations is substantially lower than previously suggested.

Muscle Oxidative Potential and Glycogen Content After Short Sprint Interval Training

We measured the maximal activity of citrate synthase in resting muscle biopsies before and after training, because this is arguably the most commonly used marker of muscle oxida-

tive potential, and other investigators have justified their selection of this enzyme because it exists in constant proportion with other mitochondrial enzymes (e.g., Ref. 13). There are equivocal data regarding the effect of sprint training on the maximal activity of this enzyme; however, studies that have failed to observe an increase in citrate synthase generally used very short sprints lasting <10 s (8, 24) or sprints that were not all-out maximal efforts (10). In contrast, all studies that have reported increases in citrate synthase activity incorporated maximal effort sprint bouts that lasted 15–30 s (19, 25, 30, 33). Another relevant consideration is the fact that acute exercise per se may elevate citrate synthase activity, potentially confounding the interpretation of training-induced effects, and thus the timing of muscle sampling relative to the last exercise session is critical when measuring the activity of this enzyme (22, 39). In the present study, we allowed 72 h of recovery before any biopsy sampling procedure (i.e., after baseline testing and after the final training session) to minimize the potential confounding effects of acute prior exercise on citrate synthase activity (22). Our data clearly show that the maximal activity of citrate synthase was increased after only six sessions of sprint interval training. Notably, the magnitude of the increase was similar to that reported in other studies that incorporated a substantially greater number of sprint training bouts (19, 25, 30, 33). Moreover, the increase in citrate synthase activity in the present study is comparable to that reported by some authors after 6–7 days of traditional endurance exercise training (i.e., 2 h/day at ~65% $\dot{V}_{O_2 peak}$) (7, 38), whereas others have reported no change in muscle oxidative potential after short endurance training (e.g., Ref. 12). The present data do not explain the mechanism for the upregulation of citrate synthase activity, and additional work is warranted in this regard. Finally, although there are limited and equivocal data regarding the effect of sprint interval training on resting
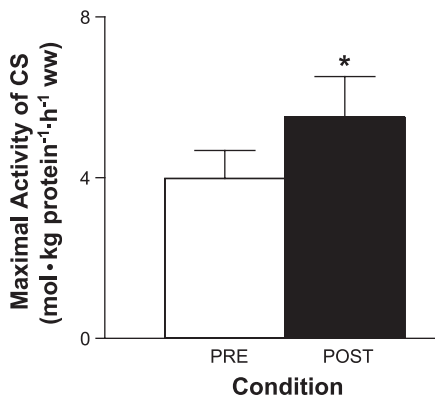

Fig. 4. Maximal activity of citrate synthase (CS) measured in resting muscle biopsy samples obtained before and after a 2-wk sprint training protocol. Values are means ± SE for 8 subjects, ww, Wet wt. *$P < 0.05$.

Table 2. *Muscle metabolites before and after training*

	Pretraining	Posttraining
ATP	24±1	24±2
Phosphocreatine	81±3	84±3
Creatine	30±2	27±3

Values are means ± SE for 8 subjects given in mmol/kg dry wt.

muscle glycogen stores (34), our results are consistent with 2 recent studies that reported increased muscle glycogen content after 14 sessions of sprint interval training (30, 33). One particularly novel aspect of our data is that the magnitude of the increase in muscle glycogen was comparable to what has been reported after five to seven sessions of traditional endurance exercise training (average increase: ~20% range: 13–35%; e.g., Refs. 7, 12, 32).

Effect of Short Sprint Interval Training on Endurance Performance

Several studies have reported increases in $\dot{V}_{O_2 peak}$ after 14–24 sprint interval training sessions performed over 2–8 wk (8, 10, 25, 26). Aside from these observations, however, we are aware of no data that suggest sprint training leads to an increased capacity to perform exercise that is primarily aerobic in nature. Thus, in the present study, we decided to employ an endurance capacity test in the form of cycling at ~80% of $\dot{V}_{O_2 peak}$, a task in which the vast majority of energy is supplied from oxidative metabolism. Our data show that aerobic endurance capacity was dramatically improved after only six sessions of sprint interval training, despite the fact that $\dot{V}_{O_2 peak}$ remained unchanged. Indeed, exercise time to exhaustion more than doubled in six of eight subjects who performed the training intervention (see individual data in Fig. 2) and the mean performance improvement was 100%. It seems unlikely that this finding is a spurious result, given that the mean improvement was substantially higher than the day-to-day variability for this test in our laboratory (coefficient of variation = 12%) and that a control group showed no change in cycle endurance capacity when tested ~2 wk apart with no sprint training intervention. To our knowledge, this is the first study to show that short sprint interval training dramatically improves endurance capacity during a fixed workload test in which the majority of cellular energy is derived from aerobic metabolism.

We can only speculate on potential mechanisms responsible for the dramatic improvement in cycle endurance capacity, but it is plausible that a training-induced increase in mitochondrial potential, as measured by citrate synthase maximal activity, improved respiratory control sensitivity during exercise as classically proposed (18). However, the precise mechanisms that regulate endurance performance are multifactorial and extremely complicated (9), and data from other studies suggest that sprint training can stimulate a range of adaptations that might facilitate performance aside from changes in mitochondrial potential. For example, recent investigations have shown that 5–8 wk of sprint interval training increases skeletal muscle blood flow and vascular conductance (21), lactate transport capacity and H^+ release from active muscle (20), ionic regulation (14), and sarcoplasmic reticulum function (28). Although the time course for these adaptations is unknown, other studies have reported similar adaptations after only 5–7 days of aerobic-based training, including changes in blood flow kinetics (37), lactate extrusion from exercising muscle (4), and cation pump activity (11). We hope that the present observations will stimulate additional research to clarify the precise nature, time course, and significance of the physiological adaptations induced by short sprint interval training.

In conclusion, the results from the present study demonstrate that six bouts of sprint interval training performed over 2 wk (~15 min total of very intense exercise) increased citrate synthase maximal activity and doubled endurance capacity during cycling exercise at ~80% $\dot{V}_{O_2 peak}$ in recreationally active subjects. The validity of this latter observation is bolstered by the fact that all subjects performed extensive familiarization trials before testing and that a control group showed no change in cycle endurance capacity when tested 2 wk apart without any sprint training intervention. To our knowledge, this is the first study to show that sprint training dramatically improves endurance capacity during a fixed-workload test in which the majority of cellular energy is derived from aerobic metabolism. These data demonstrate that brief repeated bouts of very intense exercise can rapidly stimulate improvements in muscle oxidative potential that are comparable to or higher than previously reported aerobic-based training studies of similar duration.

REFERENCES

1. **Barnett C, Carey M, Proietto J, Cerin E, Febbraio MA, and Jenkins D.** Muscle metabolism during sprint exercise in man: influence of sprint training. *J Sci Med Sport* 7: 314–322, 2004.
2. **Bergström J.** Percutaneous needle biopsy of skeletal muscle in physiological and clinical research. *Scand J Clin Lab Invest* 35: 609–616, 1975.
3. **Bogdanis GC, Nevill ME, Boobis LH, and Lakomy HK.** Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *J Appl Physiol* 80: 876–884, 1996.
4. **Bonen A, McCullagh KJ, Putman CT, Hultman E, Jones NL, and Heigenhauser GJ.** Short-term training increases human muscle MCT1 and femoral venous lactate in relation to muscle lactate. *Am J Physiol Endocrinol Metab* 274: E102–E107, 1998.
5. **Bradford MM.** A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. *Anal Biochem* 72: 248–254, 1976.
6. **Carter SL, Rennie CD, Hamilton SJ, and Tarnopolsky MA.** Changes in skeletal muscle in males and females following endurance training. *Can J Physiol Pharmacol* 79: 386–392, 2001.
7. **Chesley A, Heigenhauser GJ, and Spriet LL.** Regulation of muscle glycogen phosphorylase activity following short-term endurance training. *Am J Physiol Endocrinol Metab* 270: E328–E335, 1996.
8. **Dawson B, Fitzsimons M, Green S, Goodman C, Carey M, and Cole K.** Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training. *Eur J Appl Physiol* 78: 163–169, 1998.
9. **Fluck M and Hoppeler H.** Molecular basis of skeletal muscle plasticity-from gene to form and function. *Rev Physiol Biochem Pharmacol* 146: 159–216, 2003.
10. **Gorostiaga EM, Walter CB, Foster C, and Hickson RC.** Uniqueness of interval and continuous training at the same maintained exercise intensity. *Eur J Appl Physiol* 63: 101–107, 1991.
11. **Green HJ, Barr DJ, Fowles JR, Sandiford SD, and Ouyang J.** Malleability of human skeletal muscle Na^+-K^+-ATPase pump with short-term training. *J Appl Physiol* 97: 143–148, 2004.
12. **Green HJ, Cadefau J, Cusso R, Ball-Burnett M, and Jamieson G.** Metabolic adaptations to short-term training are expressed early in submaximal exercise. *Can J Physiol Pharmacol* 73: 474–482, 1995.

13. **Green H, Grant S, Bombardier E, and Ranney D.** Initial aerobic power does not alter muscle metabolic adaptations to short-term training. *Am J Physiol Endocrinol Metab* 277: E39–E48, 1999.

14. **Harmer AR, McKenna MJ, Sutton JR, Snow RJ, Ruell PA, Booth J, Thompson MW, Mackay NA, Stathis CG, Crameri RM, Carey MF, and Eager DM.** Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. *J Appl Physiol* 89: 1793–1803, 2000.

15. **Harris RC, Hultman E, and Nordesjo LO.** Glycogen, glycolytic intermediates and high-energy phosphates determined in biopsy samples of musculus quadriceps femoris of man at rest: methods and variance of values. *Scand J Clin Lab Invest* 33: 109–120, 1974.

16. **Henriksson J, Chi MM, Hintz CS, Young DA, Kaiser KK, Salmons S, and Lowry OH.** Chronic stimulation of mammalian muscle: changes in enzymes of six metabolic pathways. *Am J Physiol Cell Physiol* 251: C614–C632, 1986.

17. **Henriksson J and Reitman JS.** Quantitative measures of enzyme activities in type I and type II muscle fibres of man after training. *Acta Physiol Scand* 97: 392–397, 1976.

18. **Holloszy JO and Coyle EF.** Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol* 56: 831–838, 1984.

19. **Jacobs I, Esbjornsson M, Sylven C, Holm I, and Jansson E.** Sprint training effects on muscle myoglobin, enzymes, fiber types and blood lactate. *Med Sci Sports Exerc* 19: 368–374, 1987.

20. **Juel C, Klarskov C, Nielsen JJ, Krustrup P, Mohr M, and Bangsbo J.** Effect of high-intensity intermittent training on lactate and H^+ release from human skeletal muscle. *Am J Physiol Endocrinol Metab* 286: E245–E251, 2004.

21. **Krustrup P, Hellsten Y, and Bangsbo J.** Intense interval training enhances human skeletal muscle oxygen uptake in the initial phase of dynamic exercise at high but not at low intensities. *J Physiol* 559: 335–345, 2004.

22. **Leek BT, Mudaliar SR, Henry R, Mathieu-Costello O, and Richardson RS.** Effect of acute exercise on citrate synthase activity in untrained and trained human skeletal muscle. *Am J Physiol Regul Integr Comp Physiol* 280: R441–R447, 2001.

23. **Leppik JA, Aughey RJ, Medved I, Fairweather I, Carey MF, and McKenna MJ.** Prolonged exercise to fatigue in humans impairs skeletal muscle Na^+-K^+-ATPase activity, sarcoplasmic reticulum Ca^{2+} release, and Ca^{2+} uptake. *J Appl Physiol* 97: 1414–1423, 2004.

24. **Linossier MT, Denis C, Dormois D, Geyssant A, and Lacour JR.** Ergometric and metabolic adaptation to a 5-s sprint training programme. *Eur J Appl Physiol* 67: 408–414, 1993.

25. **MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, and Smith KM.** Muscle performance and enzymatic adaptations to sprint interval training. *J Appl Physiol* 84: 2138–2142, 1998.

26. **McKenna MJ, Heigenhauser GJ, McKelvie RS, Obminski G, MacDougall JD, and Jones NL.** Enhanced pulmonary and active skeletal muscle gas exchange during intense exercise after sprint training in men. *J Physiol* 501: 703–716, 1997.

27. **Nevill ME, Boobis LH, Brooks S, and Williams C.** Effect of training on muscle metabolism during treadmill sprinting. *J Appl Physiol* 67: 2376–2382, 1989.

28. **Ortenblad N, Lunde PK, Levin K, Andersen JL, and Pedersen PK.** Enhanced sarcoplasmic reticulum Ca^{2+} release following intermittent sprint training. *Am J Physiol Regul Integr Comp Physiol* 279: R152–R160, 2000.

29. **Parolin ML, Chesley A, Matsos MP, Spriet LL, Jones NL, and Heigenhauser GJ.** Regulation of skeletal muscle glycogen phosphorylase and PDH during maximal intermittent exercise. *Am J Physiol Endocrinol Metab* 277: E890–E900, 1999.

30. **Parra J, Cadefau JA, Rodas G, Amigo N, and Cusso R.** The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiol Scand* 169: 157–165, 2000.

31. **Passoneau JV and Lowry OH.** *Enzymatic Analysis: A Practical Guide.* Totowa, NJ: Humana, 1993.

32. **Putman CT, Jones NL, Hultman E, Hollidge-Horvat MG, Bonen A, McConachie DR, and Heigenhauser GJ.** Effects of short-term submaximal training in humans on muscle metabolism in exercise. *Am J Physiol Endocrinol Metab* 275: E132–E139, 1998.

33. **Rodas G, Ventura JL, Cadefau JA, Cusso R, and Parra J.** A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *Eur J Appl Physiol* 82: 480–486, 2000.

34. **Ross A and Leveritt M.** Long-term metabolic and skeletal muscle adaptations to short-sprint training: implications for sprint training and tapering. *Sports Med* 15: 1063–1082, 2001.

35. **Sale DG.** Testing strength and power. In: *Physiological Testing of the High-Performance Athlete* (2nd ed.), edited by MacDougall JD, Wenger HA, and Green HA. Champaign, IL: Human Kinetics, 1991, p. 71–82.

36. **Sandiford SD, Green HJ, Duhamel TA, Perco JG, Schertzer JD, and Ouyang J.** Inactivation of human muscle Na^+-K^+-ATPase in vitro during prolonged exercise is increased with hypoxia. *J Appl Physiol* 96: 1767–1775, 2004.

37. **Shoemaker JK, Phillips SM, Green HJ, and Hughson RL.** Faster femoral artery blood velocity kinetics at the onset of exercise following short-term training. *Cardiovasc Res* 31: 278–286, 1996.

38. **Spina RJ, Chi MM, Hopkins MG, Nemeth PM, Lowry OH, and Holloszy JO.** Mitochondrial enzymes increase in muscle in response to 7–10 days of cycle exercise. *J Appl Physiol* 80: 2250–2254, 1996.

39. **Tonkonogi M, Harris B, and Sahlin K.** Increased activity of citrate synthase in human skeletal muscle after a single bout of prolonged exercise. *Acta Physiol Scand* 161: 435–436, 1997.

40. **Trump ME, Heigenhauser GJ, Putman CT, and Spriet LL.** Importance of muscle phosphocreatine during intermittent maximal cycling. *J Appl Physiol* 80: 1574–1580, 1996.

41. **Tupling AR.** The sarcoplasmic reticulum in muscle fatigue and disease: role of the sarco(endo)plasmic reticulum Ca^{2+}-ATPase. *Can J Appl Physiol* 29: 308–329, 2004.