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Muscle performance and enzymatic adaptations to sprint interval training

J. DUNCAN MacDOUGALL, AUDREY L. HICKS, JAY R. MacDONALD,
ROBERT S. McKELVIE, HOWARD J. GREEN, AND KELLY M. SMITH

Department of Kinesiology, McMaster University, Hamilton, Ontario, Canada L8S 4K1

MacDougall, J. Duncan, Audrey L. Hicks, Jay R. MacDonald, Robert S. McKelvie, Howard J. Green, and Kelly M. Smith. Muscle performance and enzymatic adaptations to sprint interval training. *J. Appl. Physiol.* 84(6): 2138–2142, 1998.—Our purpose was to examine the effects of sprint interval training on muscle glycolytic and oxidative enzyme activity and exercise performance. Twelve healthy men (22 ± 2 yr of age) underwent intense interval training on a cycle ergometer for 7 wk. Training consisted of 30-s maximum sprint efforts (Wingate protocol) interspersed by 2–4 min of recovery, performed three times per week. The program began with four intervals with 4 min of recovery per session in *week 1* and progressed to 10 intervals with 2.5 min of recovery per session by *week 7*. Peak power output and total work over repeated maximal 30-s efforts and maximal oxygen consumption ($\dot{V}O_{2\max}$) were measured before and after the training program. Needle biopsies were taken from vastus lateralis of nine subjects before and after the program and assayed for the maximal activity of hexokinase, total glycogen phosphorylase, phosphofructokinase, lactate dehydrogenase, citrate synthase, succinate dehydrogenase, malate dehydrogenase, and 3-hydroxyacyl-CoA dehydrogenase. The training program resulted in significant increases in peak power output, total work over 30 s, and $\dot{V}O_{2\max}$. Maximal enzyme activity of hexokinase, phosphofructokinase, citrate synthase, succinate dehydrogenase, and malate dehydrogenase was also significantly ($P < 0.05$) higher after training. It was concluded that relatively brief but intense sprint training can result in an increase in both glycolytic and oxidative enzyme activity, maximum short-term power output, and $\dot{V}O_{2\max}$.

Wingate protocol; muscle biopsy; glycolytic enzymes; oxidative enzymes

IT IS WELL KNOWN that a program of endurance exercise training can result in significant increases in muscle mitochondrial density (14, 15) and oxidative enzyme activity (13, 24) but has minimum effect on glycolytic enzymes (12). In studies with humans and animals, the changes in oxidative enzyme activity are often three- to fivefold greater than the increases observed in maximal oxygen consumption ($\dot{V}O_{2\max}$) (5, 6, 8) and display a close correlation with improvements in endurance exercise capacity (6). The issue as to whether a program of anaerobic or sprint training can result in an increase in the maximal activity of either glycolytic or oxidative enzymes is somewhat more controversial. Whereas the majority of investigators have noted increases in glycolytic enzyme activity after sprint training (2, 4, 16, 25), some have not (9, 11). Moreover, there are reports in the literature that sprint training has either no effect (4, 17) or a lesser effect (27) on mitochondrial enzyme activity than does endurance training. It is possible that many of these disparities (see Table 1) may be due to the differing intensities and durations of exercise

termed “sprint training” as well as to problems in simulating maximal sprinting efforts with certain animal models.

Suggestions that sprint training does not induce increases in muscle enzyme activity are somewhat perplexing, since in most studies in which a performance measure was included such training has been shown to result in an improvement in short-term power output (4, 5, 23, 28). Therefore, we decided to reexamine this issue by investigating glycolytic and oxidative enzyme activity in a group of healthy, physically fit young adults before and after they underwent a program of intense sprint interval training, similar to that previously found to result in enhanced short-term power output (21, 22).

METHODS

Subjects. Twelve healthy young male graduate and senior undergraduate students in kinesiology (age 22.7 ± 2 yr, height 175 ± 6 cm, body mass 73.4 ± 6.2 kg) volunteered to participate in the investigation. All were physically active fitness enthusiasts who engaged in jogging, weight training, and intramural sports, but none were varsity athletes at the time of the study. They were advised of the purposes of the study and associated risks and gave their written informed consent. Subjects were provided remuneration for participating in and completing the study, and training compliance was 100%. The project was approved by the Human Ethics Committee of McMaster University.

Training program. Subjects trained three times per week on alternate days for a total of 7 wk. Training consisted of 30-s maximum-effort intervals on a mechanically braked, pan-loaded Monarch cycle ergometer on which the Wingate protocol was used (1). The program began with four intervals, with 4 min of recovery per session in *week 1*, and with the number of intervals increasing by two each week until *week 4*, whereafter 10 intervals were performed per session. Recovery intervals were 4 min in duration during *weeks 1–4* and were subsequently decreased by 30 s each week for the remaining 3 wk.

During the between-interval recovery periods, subjects were encouraged to maintain some degree of pedal rotation against either no load or a 0.5-kg load. This proved necessary in the early stages of the program to prevent light-headedness and nausea after the exercise intervals. Pedal rotation rates were not recorded during recovery, but in no instance would power output have exceeded 25 W. All training sessions were supervised by a research assistant, who adjusted the resistance, timed the recovery intervals, and provided verbal encouragement during the exercise bouts. Subjects were instructed to do no additional exercise training over the duration of the study, and adherence to this was subsequently confirmed by a poststudy questionnaire.

Measurements. All measurements were made before and after the 7-wk training program.

Anaerobic power. Maximum anaerobic power and capacity were assessed over four repeated 30-s efforts (Wingate test)

Table 1. Effects of sprint training on muscle enzyme activity and sprint performance in men

Reference	Training Regime	Enzymatic Changes	Sprint Performance
Ref. 2; Cadefau et al. (1990)	30–80 sprints and 100- to 500-m runs, 8 mo	Phosphor \uparrow , PFK \uparrow , LDH \rightarrow , SDH \uparrow , CK \rightarrow , PK \uparrow , GS \uparrow , GPh \uparrow	\uparrow
Ref. 4; Costill et al. (1979)	6-s max isokinetic exercise (one-leg) 30-s max isokinetic exercise (other leg) 4 \times /wk for 7 wk	Phosphor \uparrow , PFK \uparrow , CK \uparrow , SDH \uparrow , MDH \uparrow (all in 30-s leg only)	\uparrow
Ref. 8; Henriksson and Reitman (1976)	4-min intervals @ 101% $\dot{V}O_{2max}$ 3 \times /wk for 7–8 wk	PFK \rightarrow , SDH \uparrow	Not reported
Ref. 16; Jacobs et al. (1987)	15- and 30-s sprints 2–3 \times /wk for 6 wk	PFK \uparrow , CS \uparrow	\rightarrow
Ref. 18; Linossier et al. (1993)	5-s sprints, 7 wk	PFK \uparrow , LDH \uparrow , CS \rightarrow	\uparrow
Ref. 22; McKenna et al. (1993)	30-s sprints, 3 \times /wk for 7 wk	Not measured	\uparrow
Ref. 21; McKenna et al. (1997)	30-s sprints, 3 \times /wk for 7 wk	Not measured	\uparrow
Ref. 23; Nevill et al. (1989)	6- and 30-s sprints, 3–4 \times /wk for 8 wk	Not measured	\uparrow
Ref. 25; Roberts et al. (1982)	20- to 30-s sprints, 3 \times /wk for 5 wk	Phosphor \uparrow , PFK \uparrow , GAPDH \uparrow , LDH \uparrow , MDH \uparrow , SDH \rightarrow	\uparrow
Ref. 27; Saltin et al. (1976)	30- to 40-s sprints, 4–5 \times /wk for 4 wk	SDH \uparrow	Not reported
Ref. 28; Slievert et al. (1995)	10-s sprints, 3 \times /wk for 14 wk	Not measured	\uparrow

$\dot{V}O_{2max}$, maximal oxygen consumption; PFK, phosphofructokinase; LDH, lactate dehydrogenase; SDH, succinate dehydrogenase; CK, casein kinase; PK, protein kinase; GS, glycogen synthase; GPh, glycogen phosphorylase; MDH, malate dehydrogenase; CS, citrate synthase, GAPDH, glyceraldehyde-3-phosphate dehydrogenase. \uparrow , Increase; \rightarrow , no change.

on a cycle ergometer with 4-min recovery between each. Resistance was 0.075 kg/kg body mass and was preloaded onto a weight pan for immediate application at the beginning of the test. The subject's feet were firmly strapped to the pedals, and the seat height was adjusted for optimal comfort and pedaling efficiency. Subjects attempted to reach maximum pedaling velocity against only the ergometer's inertial resistance over 2 s, after which the full load was applied and the electronic revolution counter activated. Power output for each second, peak power output, average 30-s power output (total work), and percent fatigue were calculated and displayed for each test (SMI Opto-Sensor and Software System, St. Cloud, MN). The posttraining test was administered 2 days after the last training session.

Brachial arterial and femoral venous, potassium, and lactate concentrations were also monitored continuously from in-dwelling catheters over this period.

Aerobic power. Maximal aerobic power ($\dot{V}O_{2max}$) was measured on an electrically braked cycle ergometer (Jaeger) by using a standard continuous progressive-loading protocol. An open-circuit computerized gas-analysis system was used to calculate oxygen consumption every 20 s during the test until exhaustion. The highest oxygen consumption (averaged over 1 min) achieved during the test was selected as the subject's $\dot{V}O_{2max}$.

Muscle enzyme activity. In 9 of the 12 subjects, percutaneous needle biopsy samples were taken from the vastus lateralis under local anesthesia and with the addition of manual suction. Approximately 100–200 mg of wet tissue were obtained per sample. A portion of each sample was used for the determination of [3H]ouabain binding as an indicator of Na^+K^+ -ATPase concentration and the remainder for muscle enzyme assays.

For enzyme analyses, the tissue was freeze-dried, dissected free of blood and connective tissue, and homogenized in 50% glycerol, 20 mM sodium phosphate buffer (pH = 7.4), 5 mM β -mercaptoethanol, 0.5 mM EDTA, and 2% bovine serum albumin. Activities for total glycogen phosphorylase, hexokinase (Hex), phosphofructokinase (PFK), lactate dehydrogenase (LDH), citrate synthase (CS), succinate dehydrogenase (SDH), malate dehydrogenase (MDH), and 3-hydroxyacyl-CoA dehydrogenase were determined fluorometrically, according to the procedures described by Henriksson and colleagues (7). All assays were performed in duplicate, and, for a given

subject, were completed on the same analytic day. Data are expressed as moles per kilogram protein per hour.

Statistical analysis. All data were analyzed with a one-way repeated-measures ANOVA. Significant main effects were further analyzed by using a Tukey honestly significant difference post hoc test. Values are presented as means \pm SD.

RESULTS

Performance measurements. Before- and after-training measurements of peak and average anaerobic power output (total work) for the four exercise bouts are illustrated in Fig. 1. Although differences in power output were not statistically significant for the first

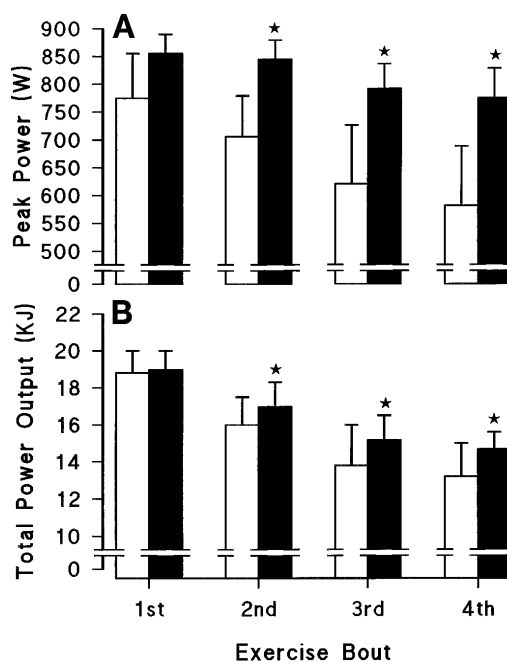


Fig. 1. A: peak power output achieved during 4 successive maximum 30-s efforts (Wingate protocol). Open histograms indicate pretraining and closed histograms posttraining values. B: total work over each 30-s effort. Values are means \pm SD; $n = 12$ men. * $P < 0.05$.

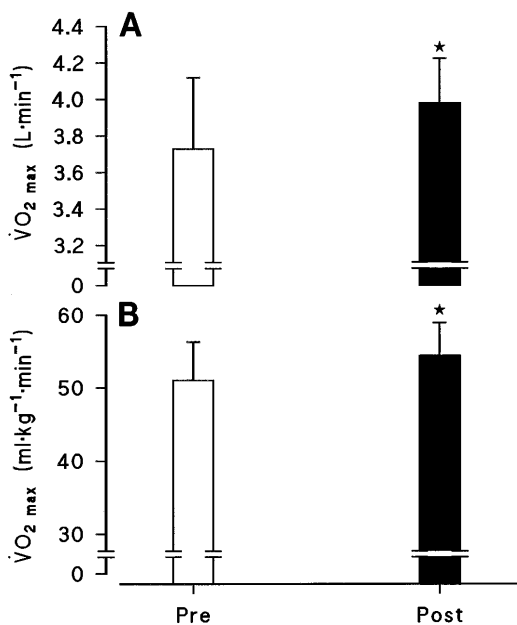


Fig. 2. Maximum oxygen consumption ($\dot{V}O_{2\max}$) before (Pre) and after (Post) training. Values are expressed in absolute units (l/min) in *A* and relative to body mass ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) in *B*. Values are means \pm SD; $n = 12$ men. * $P < 0.05$.

exercise bout, in each of the following three bouts, both peak power output and total work over 30 s were significantly higher after training ($P \leq 0.05$). The effect of the training program on $\dot{V}O_{2\max}$ is illustrated in Fig. 2. $\dot{V}O_{2\max}$ increased from 3.73 ± 0.13 to 4.01 ± 0.08 l/min ($P \leq 0.05$). Because there were no significant changes in body mass over the training program, $\dot{V}O_{2\max}$ relative to body mass also increased significantly from 51.0 ± 1.8 to $54.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$.

Muscle enzyme activity. The effects of the training program on maximal glycolytic enzyme activity are summarized in Fig. 3 and Table 2. After training, the activity of Hex was $\sim 56\%$ higher ($P \leq 0.05$) and that of PFK $\sim 49\%$ higher ($P \leq 0.05$) than before training. The 9% change in total phosphorylase activity and the 7%

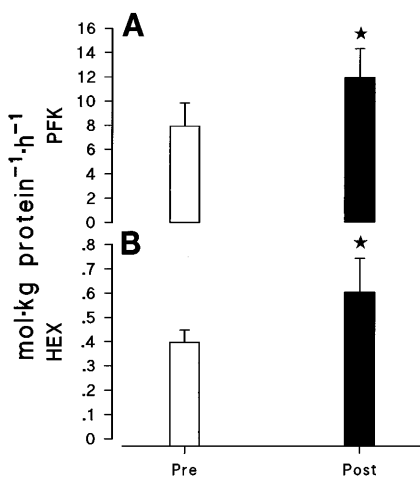


Fig. 3. Maximal enzyme activity for phosphofructokinase (PFK; *A*) and hexokinase (Hex; *B*) before and after training. Values are means \pm SD; $n = 9$ men. * $P < 0.05$.

Table 2. Enzyme activity for TPhos, LDH, and 3-HAD before and after training

Enzyme	Pretraining	Posttraining
TPhos	5.10 ± 1.86	5.58 ± 0.93
LDH	35.17 ± 5.93	37.65 ± 9.15
3-HAD	2.99 ± 1.25	4.15 ± 2.04

Values are means \pm SD, expressed in $\text{mol}\cdot\text{kg protein}^{-1}\cdot\text{h}^{-1}$; $n = 9$ men. TPhos, total glycogen phosphorylase; 3-HAD, 3-hydroxyacyl-CoA dehydrogenase. All changes were not significant.

change in LDH (Table 2) were not statistically significant. Oxidative enzyme activities are summarized in Fig. 4 and Table 2. Training resulted in significant ($P \leq 0.05$) increases in the activities of CS (by $\sim 36\%$), MDH (by $\sim 29\%$), and SDH (by $\sim 65\%$). The 39% change in 3-hydroxyacyl-CoA dehydrogenase (Table 2) was not significant.

DISCUSSION

Over the 7 wk of training, the exercise intervals were all performed at maximum intensity. During the last week of training, fingertip capillary blood samples in a subsample of five subjects indicated lactate concentrations of 29–32 mmol/l after their 10th exercise interval. Over the four test intervals used to measure changes in maximal power output, arterial lactate concentrations reached ~ 25 mmol/l and femoral vein concentrations 27 mmol/l. Although this intensity of training may be considered as being very stressful and uncomfortable, the total duration of the exercise was extremely brief, reaching only 5 min per session during the last week of training. In spite of this relatively brief training stimulus, the program resulted in significant improvements in $\dot{V}O_{2\max}$, maximum short-term power output, and increases in the maximal activity of both glycolytic and oxidative marker enzymes.

Our finding of increased activity for PFK after sprint training is consistent with several previous studies in human subjects (2, 16, 25). Increased activity of this

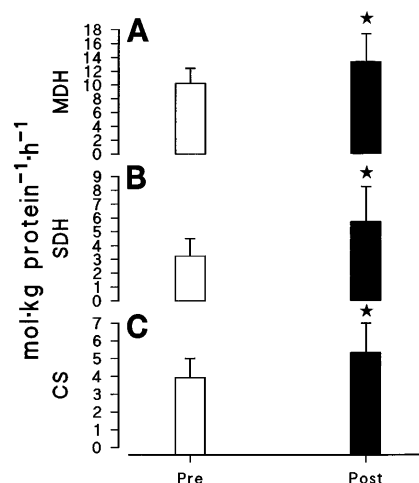


Fig. 4. Maximal enzyme activity for malate dehydrogenase (MDH; *A*), succinate dehydrogenase (SDH; *B*), and citrate synthase (CS; *C*) before and after training. Values are means \pm SD; $n = 9$ men. * $P < 0.05$.

allosteric regulatory enzyme may have resulted in an accelerated glycolytic flux rate during maximum efforts and thus, at least partially, account for both the greater peak and average short-term power outputs that were found in *bouts 2, 3, and 4* after training. Why the increased PFK activity did not result in an increased peak power output in the first exercise bout as was found by McKenna et al. (22) after a similar training protocol is not known, but this result is consistent with findings by Jacobs et al. (16). The inhibitory effect of $[H^+]$ on PFK activity is well known (3), and it is possible that the performance differences associated with higher PFK activity could be expected to become increasingly apparent as the muscle becomes more acidotic. In addition, improved Na^+K^+ -pump capacity and a possible increase in tolerance to H^+ may also have contributed to the increase in total work performed after training. The significance of the large increase in Hex is somewhat more difficult to interpret. It is known, however, that intense interval exercise, as in the present study, is accompanied by large increases in glucose concentration in plasma (20) and, presumably, muscle. Thus an increase in Hex activity would increase the potential for a greater rate of glucose utilization during the exercise and recovery intervals.

The significant increase in $\dot{V}O_{2max}$ and the large increases in muscle oxidative enzyme activity were somewhat unexpected given the nature of the training stimulus and its brevity. Changes of this magnitude are usually associated with training programs involving several hours per week at submaximal exercise intensity (7, 26, 30). Although the mechanism(s) by which such endurance training enhances oxidative enzyme activity is not known, it is generally thought that both the intensity of the exercise (such that there is major involvement of oxidative metabolic pathways) and the duration of the stimulus (volume of training) are important components. In the present study, oxidative metabolism can be considered as having only a minor contribution to energy delivery during each sprint interval. Although this relative contribution probably increased with successive exercise intervals (29), breath-by-breath measurements of oxygen consumption, with similar exercise, have indicated peak values at the end of 30 s to be <60% of $\dot{V}O_{2max}$ and declining rapidly thereafter during the recovery period (17). In addition, the duration of the training stimulus was extremely brief in the present study, amounting to a total of only 6 min/wk in *week 1* and increasing to a total of only 15 min/wk by *week 7*.

Compared with the literature on endurance training, there have been relatively few investigations of the effects of sprint training on mitochondrial enzymes or aerobic power in humans. Among them, however, are reports of increased $\dot{V}O_{2max}$ (8, 27) and increased muscle CS activity (16) after training. Sprint training has also been found to increase CS activity in red vastus muscle of rats (19). Based on this literature and on the findings of the present study, it appears that training at an intensity that exceeds $\dot{V}O_{2max}$ may be a more important component than the volume of training to stimulate an

increase in muscle oxidative potential. Inspection of the individual 30-s power outputs achieved during the sprint training sessions indicates that they typically ranged from that equivalent to $\sim 210\% \dot{V}O_{2max}$ on the first interval to $\sim 140\% \dot{V}O_{2max}$ by the ninth and tenth interval. At such intensities, the production rate for pyruvate may be considered as being almost maximal, and one would expect major increases in the velocity of the catalytic activity of the competing enzymes pyruvate dehydrogenase (PDH) and LDH. Although the 7% increase in LDH activity after training was not statistically significant, we did not measure activity of PDH. One can speculate, however, that had PDH activity increased, this would have resulted in an increased entry rate of pyruvate into the mitochondria and that, perhaps, this was the stimulus for the upregulation of the mitochondrial enzymes.

In summary, we conclude that relatively brief but intense sprint interval training can result in an increase in both glycolytic and oxidative muscle enzyme activity, maximum short-term power output, and $\dot{V}O_{2max}$. The increase in power output may have been a result of the increased maximal glycolytic enzyme activity and Na^+K^+ -pump capacity, whereas the increased mitochondrial enzyme activity may have been a result of increased pyruvate flux rate during such training.

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Address for reprint requests: J. D. MacDougall, Dept. of Kinesiology, McMaster University, Hamilton, Ontario, Canada L8S 4K1.

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