Effects of sprint cycle training on human skeletal muscle

CRAIG A. ALLEMEIER, ANDREW C. FRY, PETER JOHNSON, ROBERT S. HIKIDA, FREDRICK C. HAGERMAN, AND ROBERT S. STARON
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Allemeier, Craig A., Andrew C. Fry, Peter Johnson, Robert S. Hikida, Fredrick C. Hagerman, and Robert S. Staron. Effects of sprint cycle training on human skeletal muscle. *J. Appl. Physiol.* 77(5): 2385–2390, 1994.—Eleven men sprint trained two to three times per week for 6 wk to investigate possible exercise-induced slow-to-fast fiber type conversions. Six individuals served as controls. Both groups were tested at the beginning and end of the study to determine anaerobic performance and maximal oxygen consumption. In addition, pre- and postbiopsies were extracted from the vastus lateralis muscle and were analyzed for fiber type composition, cross-sectional area, and myosin heavy chain (MHC) content. No significant changes were found in anaerobic or aerobic performance variables for either group. Although a trend was found for a decrease in the percentage of type IIb fibers, high-intensity sprint cycle training caused no significant changes in the fiber type distribution or cross-sectional area. However, the training protocol did result in a significant decrease in MHC IIb with a concomitant increase in MHC IIa for the training men. These data appear to support previous investigations that have suggested exercise-induced adaptations within the fast fiber population (IIb → IIa) after various types of training (endurance and strength).

fiber types; myosin heavy chains; Wingate test

ON THE BASIS OF MYOSIN HEAVY chain (MHC) content, most adult human skeletal muscle fibers can be classified into one of seven fiber types (I, Ic, IIc, IIac, IIa, IIab, or IIb) (34, 35). The presence of "hybrid" fibers that coexpress either MHC I and MHC IIa (types Ic, IIc, and IIac) or MHC IIa and MHC IIb (type IIab) in varying proportions suggests the possibility of conversions between the fiber types and creates a continuum spanning from the fast type IIb to the slow type I (IIb ↔ IIab ↔ IIa ↔ IIac ↔ IIc ↔ Ic ↔ I) (35). Such interconversions are important because fiber type composition has an important role in athletic performance, and, therefore, it would be advantageous if favorable fiber type changes occurred.

Although the muscles of elite endurance athletes contain a predominance of type I (slow) fibers and the muscles of elite strength/power athletes contain a predominance of type II (fast) fibers (9, 15, 30), it is not known whether these fiber type distributions are primarily the result of genetic or environmental factors. In support of the major influence of genetics, very few human studies have been able to demonstrate a significant training-induced increase in the percentage of either type I or type II fibers (see Ref. 36).

However, as demonstrated by experiments using chronic low-frequency electrical stimulation of animal fast-twitch muscle, muscle fibers do possess the ability to convert from fast type II to slow type I (fast to slow) (29), and cessation of the stimulation can restore their fast characteristics (slow to fast) (10). Thus it is conceivable that long-term endurance training, being a comparatively mild form of chronic stimulation, may cause a limited amount of fast-to-slow conversions (20, 25, 32, 40) and that detraining may cause a return to fast (26).

Exercise-induced transformations in the reverse direction (slow-to-fast) appear much more difficult to achieve. Indeed, high-frequency electrical stimulation of rat soleus muscle causes a slow-to-fast fiber type conversion only after denervation (16). Recently, sprint cycle training has been suggested to cause such transformations in human vastus lateralis muscle (12, 24). This has important implications for strength/power athletes because it has long been thought that "a sprinter is born and not made." The aim of the present investigation was to further investigate the possibility of an exercise-induced slow-to-fast transformation in human skeletal muscle by using one of the training protocols of Jansson et al. (24).

MATERIALS AND METHODS

Subjects. Seventeen healthy men volunteered to participate in the present study. Approval was given by the Ohio University Institutional Review Board, and all subjects signed informed consent documents before beginning the study. The training group consisted of 11 individuals (age 22.7 ± 5.0 yr, height 1.76 ± 0.04 m), with 6 in the control group (24.0 ± 2.3 yr, 1.89 ± 0.10 m). Both groups were assessed at the same time. All of the subjects were untrained and were not currently participating in any regular training program. One of the training men, however, was a cyclist who had not trained for 4 mo before beginning the study.

Anthropometric assessment. Anthropometric measurements (total body mass, estimated fat-free mass, and estimated percent body fat) were determined throughout the study. Skinfold measurements were taken from three sites (anterior thigh, axillary fold, and abdomen) on the same day before extraction of the muscle biopsies and were used in the equation proposed by Jackson et al. (21) for the estimation of percent body fat. Total body mass was determined before every test and training session.

Training protocol. The training subjects participated in a 6-wk high-intensity training program using a Wingate protocol as previously described (24). This involved three consecutive bouts of 30-s supramaximal sprints (20 min of rest between bouts) on a mechanically braked cycle ergometer (Monark-Crescent) using a resistance of 75 g/kg total body mass. During the first 3 wk, the subjects trained twice a week (Tuesday and Thursday). This was increased to three times a week (Monday, Wednesday, and Friday) for the last 3 wk. Familiarization with the equipment and testing procedures took place during a 2-wk orientation before the 6-wk high-intensity training and consisted of one sample Wingate test and one sample maximum O_2 consumption ($\dot{V}O_{2\,max}$) test. Pretraining data were also collected during this period.

Anaerobic and aerobic performance measurements. Power output measurements were taken during each Wingate bout to

TABLE 1. *Anthropometric measurements*

	Training		Control	
	Pre	Post	Pre	Post
%BF	13.5±5.6	12.5±4.1	18.0±8.1	17.4±7.4
FFM, kg	67.3±6.9	67.3±5.9	73.8±6.8	74.2±6.4
TBM	76.7±7.4	77.0±7.4	92.7±14.3	91.8±12.6

Values are means ± SD. %BF, percent body fat; FFM, fat-free mass; TBM, total body mass; pre and post, beginning and end of study, respectively.

determine anaerobic performance. After orientation, the control group performed one Wingate protocol at the beginning and one at the end of the study. For the entire study, the training group performed a pre- and posttraining Wingate and a total of 15 training sessions with three Wingate protocols per session.

Power output was calculated and expressed in watts and watts per kilogram every second for the duration of the Wingate test. At the conclusion of the test, the following anaerobic performance variables were determined: *1*) mean power output (average power during a 30-s test) and *2*) peak power output (highest 1-s power output).

$\dot{V}O_{2\,max}$ and O_2 debt were determined for all subjects at the beginning and end of the study. Metabolic testing was conducted using a Schwinn Velodyne. This apparatus uses an electronically braked computerized revolving drum. After calibration, a ramp protocol was initiated with subjects pedaling at 50 W and was increased 5 W every 12 s. Cessation of the test occurred when the subjects could no longer maintain cadence (desired power output, voluntarily stopped due to exhaustion, O_2 consumption had plateaued or decreased after reaching a maximal level, or an R value of 1.15 was achieved).

O_2 consumption was measured at 30-s intervals throughout the maximal exercise test using a semiautomated computerized open-circuit sampling system. Expired fractional concentrations of O_2 and CO_2 were analyzed from a port on the mixing chamber using a Beckman OM-11 O_2 analyzer and a Beckman LB-2 CO_2 analyzer. Known gases (Micro-Scholander technique) were used for analyzer calibration. Respiratory volumes were measured using a Rayfield RAM-9200 interface connected to a Parkinson-Cowan dry gas meter. All volumes were appropriately corrected to BTPS and STPD. Analytic and volumetric outputs were integrated using an Apple IIe computer. O_2 debt was calculated by subtracting steady-state resting O_2 consumption (sitting position) from the total O_2 consumed during a 30-min sitting recovery period immediately after the incremental maximal exercise test.

Muscle biopsies. Muscle biopsies (80–160 mg) were extracted from the midregion of the vastus lateralis muscle (2) (~16–18 cm proximal to the superior aspect of the patella) and were divided into two pieces (one for histochemistry and the other for electron microscopy). Biopsies were taken from both groups at the beginning (pre) and end (post) of the study. Attempts were made to extract tissue from approximately the same location utilizing the prebiopsy scar. As such, postbiopsies were taken 0.5–1.0 cm lateral to the initial biospy. To ensure adequate sample sizes, large pieces were obtained using a double-chop method (38, 39) combined with suction (13).

A small portion of each biopsy sample was prepared for electron microscopy by fixing the tissue in 1.5% glutaraldehyde and 2% paraformaldehyde in a 0.1 M cacodylate buffer (pH 7.2), postfixed in buffered osmium tetroxide, and embedded in an Epon and Araldite resin mixture. Ultrathin sections were examined with a Zeiss EM109 electron microscope. The largest portion of the biopsy sample was oriented in tragacanth gum, frozen in isopentane cooled by liquid nitrogen to −159°C, and stored at −70°C. The frozen biopsy samples were thawed to −20°C and serially sectioned for the determination of fiber type composition (using 12-μm-thick sections) and MHC content (using 20-μm-thick sections).

Fiber type distribution. Routine myofibrillar adenosinetriphosphatase (mATPase) histochemical analysis was performed using preincubation pH values of 4.3, 4.6, and 10.2 (6) to determine the muscle fiber type distribution. Therefore, a total of six fiber types (I, Ic, IIac, IIa, IIab, and IIb) were distinguished on the basis of their staining intensities (34, 35). Fiber type IIc was included with type IIac because of their extremely small numbers. Cross sections of biopsies from the same individual were placed on one glass coverslip so that they could be assayed simultaneously for mATPase activity.

A composite photomontage of each mATPase preparation (preincubation at pH 4.6) was made using Polaroid micrographs (×56 magnification). These were used in combination with the other histochemical preparations (preincubaton pH values of 4.3 and 10.2) to determine fiber type percentages and total fiber number in each biopsy. The cross-sectional areas of at least 50 fibers per major type (I, IIa, and IIb) per biopsy were determined by the use of direct tracings (×200 magnification) and a digitizing tablet.

MHC analysis. MHC analysis was performed on the 20-μm-thick cross sections using sodium dodecylsulfate (SDS)-polyacrylamide electrophoretic techniques. The protocol for analyzing the muscle samples was based on the procedures of Carraro and Cantani (8) and Perrie and Bumford (28) with modifications recently used for single human muscle fibers (34, 36). Briefly, three to five serial cross sections (20 μm thick) from each biopsy were placed into 0.5 ml of a lysing buffer containing 10% (wt/vol) glycerol, 5% (vol/vol) 2-mercaptoethanol, and 2.3% (wt/vol) SDS in 62.5 mM tris(hydroxymethyl)aminomethane · HCl buffer (pH 6.8) and heated for 10 min at 60°C. Small amounts of the extracts (3–5 μl) were loaded on 4–8% gradient SDS-polyacrylamide gels with 4% stacking gels (1), run overnight (19–21 h) at 120 V, and stained with Coomassie blue. MHC isoforms were identified according to their apparent molecular masses compared with those of marker proteins and migration patterns from single fiber analysis. MHC content was subsequently determined using a laser densitometer.

Statistical analyses. The statistical package for the biomedical sciences was utilized for all statistical analyses. Descriptive statistics were used to derive means ± SD for all variables. Muscle fiber characteristics were grouped into clusters of re-

TABLE 2. *Anaerobic and aerobic performance measurements*

	Training		Control	
	Pre	Post	Pre	Post
Revolutions/min	413.8±31	429.6±36	410±46	419±25
Mean power, W	625±53	656±81	738±113	775±96
Mean power, W/kg	8.2±0.6	8.5±0.7	8.0±0.9	8.3±0.4
Peak power, W	797±92	821±112	924±146	952±200
Peak power, W/kg	10.4±0.8	10.7±0.9	9.9±0.7	10.4±0.8
$\dot{V}O_{2\,max}$, L/min	3.7±0.5	4.2±0.5	3.9±0.2	3.6±0.7
$\dot{V}O_{2\,max}$, $ml \cdot kg^{-1} \cdot min^{-1}$	48.7±6.7	54.8±5.9	42.4±6.9	42.1±9.8
Net O_2 debt; liters	17.4±3.4	26.6±11.8	15.9±4.4	16.3±6.0

Values are means ± SD. Absolute and relative maximal O_2 consumption ($\dot{V}O_{2\,max}$) values are expressed in l/min and $ml \cdot kg^{-1} \cdot min^{-1}$, respectively. Values for net O_2 debt are total liters of O_2 consumed in 30 min minus baseline.

TABLE 3. *Fiber type distribution*

	n	Type I	Type Ic	Type IIac	Type IIa	Type IIab	Type IIb
Training							
Pre	1,163±485	44.4±10.7	0.2±0.4	0.2±0.2	35.7±10.5	6.0±4.1	13.5±7.6
Post	1,026±540	42.2±11.2	0.4±0.3	2.6±5.3	38.0±9.7	9.3±4.8	7.5±5.0
Control							
Pre	927±255	46.4±9.4	0.5±0.4	1.4±1.2	35.7±10.9	5.0±2.6	11.0±7.1
Post	1,035±513	50.1±4.5	0.5±0.4	1.5±2.6	29.0±13.5	4.4±2.3	14.6±8.2

Values are means ± SD given in %; *n*, mean no. of fibers per biopsy.

TABLE 4. *Vastus lateralis muscle fiber characteristics*

	Training		Control	
	Pre	Post	Pre	Post
MHC, %				
Type I	43.9±11.3	40.7±9.9	44.2±9.7	46.5±7.6
Type IIa	42.9±8.3	51.6±7.6*	46.1±7.9	43.8±6.9
Type IIb	13.2±7.7	7.7±4.2*	9.7±7.7	9.7±6.9
Cross-sectional area, μm^2				
Type I	4,991±1,013	5,304±950	5,812±1,282	5,762±799
Type IIa	5,715±1,215	6,382±1,211	6,660±1,760	6,342±680
Type IIb	4,190±1,847	4,697±1,854	5,458±1,694	5,583±1,607

Values are means ± SD. MHC, myosin heavy chain distribution. * Significantly different from respective pre value, $P \leq 0.05$.

lated dependent variables (percent fiber type, cross-sectional area, and MHC content) and analyzed using multivariate analyses of variance (MANOVA) with the Wilks' Lamba criterion for significance ($P \leq 0.05$). After a significant MANOVA *f* value, mixed-model analyses of variance were performed on each dependent variable to identify significant responses for each group of subjects (4). Physical characteristics and performance variables were analyzed with mixed-model analysis of variance. After a significant interaction, subsequent analysis was performed using post hoc-dependent or -independent *t*-tests. Differences were considered significant at $P \leq 0.05$.

RESULTS

Anthropometric measurements. There were no significant differences between pre- and postmeasurements for total body mass, estimated percent body fat, or estimated fat-free mass over the course of the study for either the training or the control group (Table 1).

Anaerobic and aerobic performance measurements. No significant changes, comparing pre- with posttraining values, were found for either group for any of the anaerobic performance measurements (revolutions/min, absolute and relative peak power output, or absolute and relative mean power output; Table 2). Likewise, when comparing pre with post values, no changes occurred in either group for absolute and relative maximal aerobic capacity ($\dot{V}O_{2\,max}$) or the amount of O_2 consumed postexercise (O_2 debt) (Table 2).

Skeletal muscle adaptations. When all six fiber types are considered, no significant changes in fiber type distribution (comparing pre- with posttraining values) were found for either group (Table 3). In addition, no significant changes occurred for either group in cross-sectional area or percent fiber type area (Table 4). However, sprint training caused a significant decrease in the percentage of MHC IIb with a concomitant increase in MHC IIa (Table 4, Fig. 1). No changes in MHC content occurred over time for the control group.

The ultrastructure of each pre- and postbiopsy specimen was examined and interpreted blindly. The prebiopsies from the training subjects and all biopsies (pre and post) from the control subjects showed no abnormalities in their morphological characteristics (Fig. 2*A*). In contrast, signs of muscle damage were observed in the postbiopsies from most of the trained subjects (7 of 11; Fig. 2, *B* and *C*). The damage did not appear to be fiber type specific and was not extensive, consisting of a few fibers containing varying degrees of fiber and myofibrillar disruption in which the contractile material was not dif-

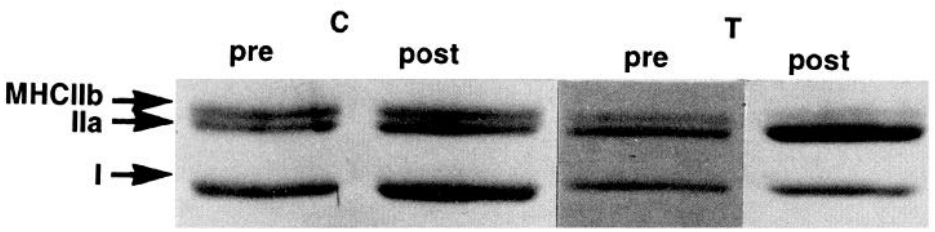
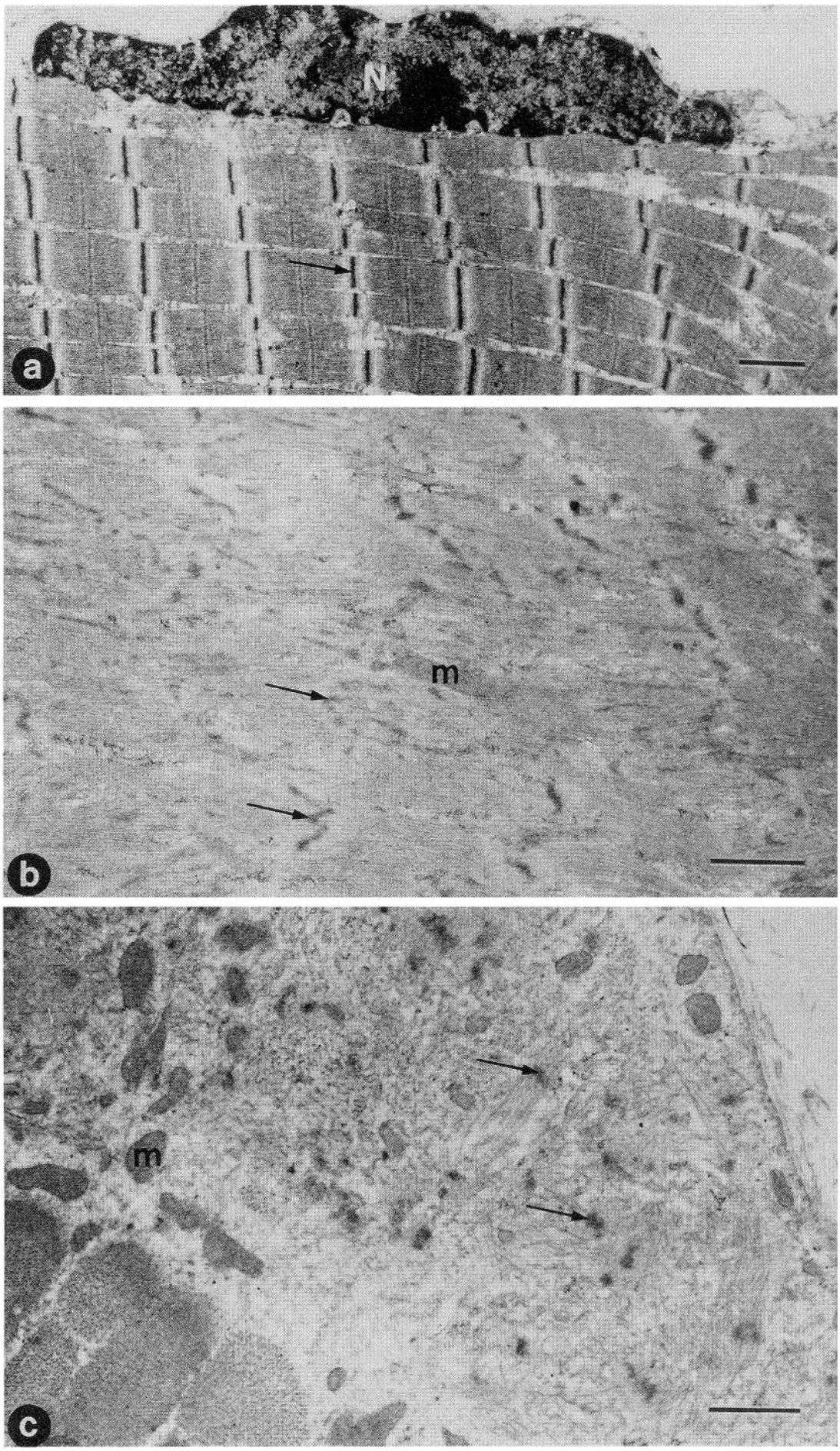


FIG. 1. Myosin heavy chain (MHC) analysis of biopsies obtained from control (C) and training (T) subject at beginning (pre) and end (post) of study. Note change in intensities of bands corresponding to MHC IIb and MHC IIa isoforms from pre to post for training subject. IIb, type IIb; IIa, type IIa; I, type I.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ferentiated into myofibrils or regular striation patterns (Fig. 2, *B* and *C*).

DISCUSSION

If the contribution of genetic factors for the determination of fiber type composition is low, as suggested by Bouchard et al. (5), then the predominance of a particular fiber type within the muscles of elite athletes may be the result of conversions between the major fiber types (fast and slow). However, very few training studies have been able to demonstrate either fast-to-slow or slow-to-fast transformations in human muscle.

Some evidence for fast-to-slow conversions has been presented. A significant increase in the percentage of type I fibers has been shown after various forms of aerobic conditioning: endurance cycle training (20), long-distance running (25), and high-intensity intermittent cycle training (32). Similarly, data from a few cross-sectional studies suggest a fast-to-slow transformation after long-term excessive use (3, 14, 40). Interestingly, an increase in the percentage of type I fibers has also been reported after anaerobic conditioning: strength training (31, 38) and sprint running (7).

Even less information exists on the possibility of transformations in the opposite direction (slow to fast) in healthy adult human muscle. Cases of extreme disuse have reported a significant decrease in the percentage of type I fibers after immobilization (18), long-term endurance training → detraining (26), and hemiplegia (23). In such extreme cases, selective loss of type I fibers and not slow-to-fast conversions may have accounted for the increase in the percentage of type II fibers. We are aware of only two training studies (both from the same laboratory) that have suggested the occurrence of exercise-induced slow-to-fast transitions in human muscle (12, 24).

An attempt to induce slow-to-fast conversions was not successful in the present investigation. Although the protocol in the present investigation followed that of Jansson et al. (24), sprint cycle training caused no significant change in the percentage of the major fiber types (slow and fast). The reason for this descrepancy is not clear. However, caution must be used when interpreting the results from muscle biopsies. Estimation of the fiber composition of, for example, the vastus lateralis muscle is based on a small number of fibers (often <1,000 fibers) from a muscle that contains over 500,000 fibers. In addition, significant variations in fiber composition may occur from biopsy to biopsy, superficial to deep, and proximal to distal (11, 27, 33). To address this problem, care was taken to extract the pre- and postbiopsies from similar locations and depths in the current investigation. In addition, multiple biopsies were taken from each individual using a double-chop method (38, 39). Combined with suction (13), this resulted in an adequate number of fibers for estimation of fiber distribution (27).

Data from the present investigation suggest that sprint training does not cause any changes in the overall percentage of the major fiber types, and, therefore, one must rely on adaptations within and not between these types to improve performance. Indeed, numerous studies have suggested a conversion within the fast fiber population (IIb → IIa) with endurance (see Ref. 36), resistance (19, 37, 38, 39), and sprint (22) training and a conversion in the opposite direction (IIa → IIb) with detraining and disuse (17, 23, 38). Likewise, the present data support the concept of IIb → IIa fiber type conversions with sprint cycle training (Tables 3 and 4, Fig. 1).

As has been previously shown (37), a muscle need not be active for extremely long periods to cause conversions within the fast fiber population (type IIb → IIa). However, the intensity must be high. In the current study, the subjects trained at a supramaximal level for 90 s, two to three times per week. This intensity and duration appear sufficient to induce specific transformations within the fast fiber population. The damage observed ultrastructurally gives further evidence of the high training intensity.

In conclusion, 6 wk of high-intensity sprint cycle training did not cause fiber transformations from type I to type IIa (slow to fast). However, MHC analysis revealed possible conversions within the fast fiber population (IIb → IIa). These data support and extend previous observations of training-induced fast fiber type adaptations in human skeletal muscle after various types of training.

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FIG. 2. Electron micrographs of postbiopsy muscle samples from control (*A*) and trained (*B* and *C*) subjects. Arrows indicate Z lines. Note varying degrees of myofibrillar disruption that are centrally (*B*) and peripherally (*C*) located. Compare orderly arrangement of sacromeres in control vs. trained muscles. N, myonucleus; m, mitochondria. Bar, 1 μm.

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