Differential Motor Unit Changes after Endurance or High-Intensity Interval Training

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ABSTRACT

MARTINEZ-VALDES, E., D. FALLA, F. NEGRO, F. MAYER, and D. FARINA. Differential Motor Unit Changes after Endurance or High-Intensity Interval Training. *Med. Sci. Sports Exerc.*, Vol. 49, No. 6, pp. 1126–1136, 2017. **Purpose**: Using a novel technique of high-density surface EMG decomposition and motor unit (MU) tracking, we compared changes in the properties of vastus medialis and vastus lateralis MU after endurance (END) and high-intensity interval training (HIIT). **Methods**: Sixteen men were assigned to the END or the HIIT group (n = 8 each) and performed six training sessions for 14 d. Each session consisted of 8–12 × 60-s intervals at 100% peak power output separated by 75 s of recovery (HIIT) or 90–120 min continuous cycling at ~65% $\dot{V}O_{2peak}$ (END). Pre- and postintervention, participants performed 1) incremental cycling to determine $\dot{V}O_{2peak}$ and peak power output and 2) maximal, submaximal (10%, 30%, 50%, and 70% maximum voluntary contraction [MVC]), and sustained (until task failure at 30% MVC) isometric knee extensions while high-density surface EMG signals were recorded from the vastus medialis and vastus lateralis. EMG signals were decomposed (submaximal contractions) into individual MU by convolutive blind source separation. Finally, MU were tracked across sessions by semiblind source separation. **Results**: After training, END and HIIT improved $\dot{V}O_{2peak}$ similarly (by 5.0% and 6.7%, respectively). The HIIT group showed enhanced maximal knee extension torque by ~7% (P = 0.02) and was accompanied by an increase in discharge rate for high-threshold MU (≥50% knee extension MVC) (P < 0.05). By contrast, the END group increased their time to task failure by ~17% but showed no change in MU discharge rates (P > 0.05). **Conclusions**: HIIT and END induce different adjustments in MU discharge rate despite similar improvements in cardiopulmonary fitness. Moreover, the changes induced by HIIT are specific for high-threshold MU. For the first time, we show that HIIT and END induce specific neuromuscular adaptations, possibly related to differences in exercise load intensity and training volume. **Key Words:** HIGH-DENSITY SURFACE EMG, MOTOR UNIT DECOMPOSITION, MOTOR UNIT TRACKING, MOTOR UNIT DISCHARGE RATE, MOTOR UNIT ADAPTATION, NEUROMUSCULAR ADAPTATION

High-intensity interval training (HIIT) describes physical exercise that is characterized by brief, intermittent bursts of vigorous physical activity, interspersed by periods of rest or low-intensity exercise (15). Subjects perform short periods of training (from 30 s to 1 min) at intensities from 90% of maximum heart rate and above, interspersed with a passive or active rest, achieving a maximum exercise volume of 10 to 20 min per session (30–60 min·wk^{-1}). In comparison to traditional high-volume endurance training (END), HIIT induces similar changes in a range of physiological (e.g., enhanced aerobic metabolism), performance (e.g., faster completion of a certain amount of work), and health-related markers (e.g., increased flow-mediated dilation) (4,16,22,31), but with a much lower time commitment. Therefore, HIIT is typically offered as an alternative to END. However, no study has evaluated the neuromuscular adaptations induced by HIIT. Because neuromuscular adaptations to training are highly specific and vary according to the training regime (35), differences in neuromuscular adaptations to HIIT and END might be expected because the training protocols differ in load intensity and exercise volume.

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Recordings of motor units provide a window to the central nervous system, allowing analysis of the way in which the central nervous system controls muscle force (13). In one of the few studies assessing motor unit adaptations after training, Vila-Chã et al. (35) observed different changes in low-threshold motor unit discharge rates (average discharge rate and discharge rate variability) between END and strength training. These findings suggested a specific adaptation in motor unit discharge rate according to the training regime applied. However, these differences could not be assessed for high-threshold motor units because of previous technical limitations. Indeed, there is a lack of knowledge about changes in discharge rate of high-threshold motor units (9) because classic methods for EMG signal decomposition are limited to the identification of a few motor units concurrently, at low forces (8). Nonetheless, high-density surface EMG (HDEMG) has recently emerged as an alternative to overcome this limitation. The availability of many (tens) observation sites allows for automatic methods of source separation to reliably identify a large number of motor units, for a wide range of forces (close to the maximum voluntary contraction [MVC] force) (10,24,29). Moreover, several observation channels can be used to track the same motor units across different sessions, therefore allowing longitudinal studies of the same motor units in humans over long periods of time (weeks) (25). This achievement has opened new possibilities to study the neuromuscular adaptations to training.

The purpose of the study was to evaluate, for the first time, changes in muscle activity and motor unit properties (discharge rate, discharge rate variability, and recruitment threshold) of synergistic knee extensor muscles after short-term low-volume HIIT and high-volume END training interventions, using a novel technique of HDEMG motor unit tracking. It was hypothesized that, despite similar increases in cardiorespiratory fitness parameters (e.g., peak oxygen uptake [$\dot{V}O_{2peak}$] [26]), these two training protocols will induce different changes in motor output (maximal strength, rate of torque development [RTD], and time to task failure) that will be related to different adjustments in motor unit discharge rates. Moreover, we hypothesized that these adjustments will vary across the motor unit pool, with low-threshold motor units showing different changes compared with high-threshold motor units, given the differences in load intensity and training volume between the two types of training.

METHODS

Participants

Eighteen healthy men (mean ± SD, age = 29 ± 3 yr, height = 178 ± 6 cm, mass = 79 ± 9 kg) participated. All subjects were physically active and took part in some form of recreational exercise at least two to three times per week (e.g., soccer, running, etc.). None of the subjects were engaged in regular training for a particular sporting event or competition. Exclusion criteria included any neuromuscular disorder as well as any current or previous history of knee pain and age <18 or >35 yr. Participants were asked to avoid any strenuous activity 24 h before the measurements. Nine subjects were randomly assigned to an HIIT group, and the other nine were assigned to an END group. A control group was not implemented because we previously reported no changes in motor output and motor unit properties of vasti muscles in control subjects measured in the space of 2 wk (24). The ethics committee of the Universität Potsdam approved the study (approval no. 26/2015), in accordance with the declaration of Helsinki (2004). All participants gave written informed consent.

Experimental Protocol

The experimental protocol consisted of baseline measurements (i.e., isometric knee extension torque, EMG recordings, peak oxygen uptake [$\dot{V}O_{2peak}$] determination), a 2-wk intervention of END or HIIT training, and posttraining measurements.

Baseline measurements (torque and EMG measurements). The participant was seated in an isokinetic dynamometer (CON-TREX MJ; PhysioMed, Regensdorf, Switzerland), with the trunk reclined to 15° in an adjustable chair while the hip and the distal thigh were secured to the chair. The rotational axis of the dynamometer was aligned with the lateral femoral epicondyle of the dominant leg, and the lower leg was secured to the dynamometer lever arm above the lateral malleolus. Maximal and submaximal isometric knee extensions were exerted with the knee flexed to 90°. After the placement of the surface electrodes (as described in the Data Acquisition section), subjects performed three MVC of knee extension each for a period of 5 s. These trials were separated by 2 min of rest. The highest MVC value was used as a reference for the definition of the submaximal torque levels. Five minutes of rest was provided after the MVC measurement. In each of the baseline and postintervention sessions, the submaximal torques were expressed as a percent of the MVC measured during the same session. After the MVC, the participants performed three maximal-ballistic isometric contractions, each separated by 30 s of rest. They were encouraged to exert their maximal torque as fast as possible in response to a visual signal shown on a computer monitor. Then after 5 min of rest, and after a few familiarization trials at low torque levels (10% and 30% MVC), subjects performed submaximal isometric knee extension contractions at 10%, 30%, 50%, and 70% MVC in a randomized order. The contractions at 10%–30% were sustained for 20 s, whereas the contractions at 50% and 70% MVC lasted 15 and 10 s, respectively. In each trial, the subjects received visual feedback of the torque applied by the leg to the dynamometer, which was displayed as a trapezoid (5-s ramps with hold-phase durations as specified previously). Each contraction level was performed twice per session, and 2 min of rest was allowed after each contraction. The randomization order of these contractions was kept the same for each subject in the pre- and postintervention sessions to minimize the possible influence of cumulative fatigue in the results of the motor unit data when studying the training-induced adaptations. Finally, the subjects performed further isometric knee extension contraction at 30% MVC, maintaining the torque level for as

long as possible. Time to task failure was defined as the time instant when the subject exerted a force 10% MVC below the target force for an interval of time of 2 s (5).

Then 24 h after these measurements, the subjects returned to the laboratory to perform an incremental test to exhaustion on an electronically braked cycle ergometer (Lode Excalibur Sport V2.0, Groningen, the Netherlands). $\dot{V}O_{2peak}$ and submaximal ventilation thresholds were determined using a gas analysis system (ZAN 600; Nspire Health, Oberthulba, Germany), which was calibrated before each test with known values of oxygen (O_2), carbon dioxide (CO_2), and volume. After a 3-min warm-up at 30 W, the test began with the workload increasing by 6 W every 12 s until volitional exhaustion. The revolutions per minute were maintained between 80 and 90 throughout the incremental test and training sessions. The value used for $\dot{V}O_{2peak}$ corresponded to the highest value achieved for a 30-s collection period. Peak power output was defined as the maximal power (W) achieved at the end of the ramp $\dot{V}O_{2peak}$ cycle ergometer test. Finally, the first ventilatory threshold (VT1) was identified by the ventilatory equivalent method, where VT1 corresponded to the power output and $\dot{V}O_2$ value at which the ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$) exhibited a systematic increase without a concomitant increase in the ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$) (37). The respiratory compensation point (VT2) was identified by using the criterion of an increase in both $\dot{V}_E/\dot{V}O_2$ and $\dot{V}_E/\dot{V}CO_2$ and by using the first decrease in the end-tidal pressure of CO_2 ($PETCO_2$) as a confirmatory indicator (37).

Training protocols. The training interventions were performed using two protocols that have shown similar improvements in cardiorespiratory fitness ($\dot{V}O_{2peak}$) and aerobic capacity, despite differences in total training volume and intensity (16,22). The training protocol commenced approximately 72 h after the incremental test and consisted of six training sessions for 14 d. Each session was performed on Mondays, Wednesdays, and Fridays. An investigator of the study (E.M.-V.) supervised all training sessions. For the END group, training consisted of 90–120 min of continuous cycling at 65% of $\dot{V}O_{2peak}$ using a protocol described previously (16). The duration of exercise increased from 90 min during sessions 1 and 2 to 105 min during sessions 3 and 4, and finally to 120 min during sessions 5 and 6. For the HIIT group, training consisted of 60-s bouts of high-intensity cycling at 100% peak power output as described previously (22). These bouts were interspersed by 75 s of cycling at 30 W for recovery (22). Participants completed eight high-intensity intervals during sessions 1 and 2, 10 intervals during sessions 3 and 4, and 12 intervals on the final two sessions. A warm-up period of 3 min at 30 W was performed each session before training.

In summary, the HIIT group performed the exercise at an intensity of ~335 W, with a total training commitment of 8–12 min per session (18–27 min including recovery). The total training commitment for HIIT for the 2 wk was 60 min (135 min including recovery), reaching a total exercise volume of ~1205 kJ (~1375 kJ including recovery). By contrast, the END group performed the exercise at an intensity of ~165 W, with a total training commitment of 90–120 min per session. The total training commitment for END during the 2 wk was 630 min, achieving a total exercise volume of ~6250 kJ.

Posttraining measurements. The posttraining sessions (torque, EMG recordings, and incremental test) were identical with the baseline-testing procedures and were performed approximately 72 h posttraining.

Data acquisition. EMG signals were acquired from the vastus medialis (VM), vastus lateralis (VL), and biceps femoris (BF) muscles during maximal and submaximal isometric contractions as described above. For the VM and the VL, surface EMG signals were recorded in monopolar derivation with a two-dimensional adhesive grid (SPES Medica, Salerno, Italy) of 13 × 5 equally spaced electrodes (each of 1 mm diameter, with an interelectrode distance of 8 mm), with one electrode absent from the upper right corner. The electrode grids were positioned as described previously (21,24). EMG signals were initially recorded during a brief voluntary contraction during which a linear nonadhesive electrode array was moved over the skin to detect the location of the innervation zone and tendon regions (23). After skin preparation (shaving, abrasion, and water), the electrode cavities of the grids were filled with conductive paste (SPES Medica), and the grids positioned between the proximal and the distal tendons of the VL and the VM muscles with the electrode columns (comprising 13 electrodes) oriented along the muscle fibers. Reference electrodes were positioned over the malleoli and patella of the dominant leg. Signals from the BF were recorded in bipolar mode with Ag–AgCl electrodes (Ambu Neuroline 720, Ballerup, Denmark; conductive area 28 mm^2) and were positioned according to guidelines (2). The location of the electrodes was marked on the skin of the participants using a surgical pen (subjects were instructed to remark the electrode zone daily). Also, the position of the electrodes was further reported on a transparent sheet by using anatomical landmarks. These procedures allowed a similar electrode positioning across sessions.

Torque and EMG signals were sampled at 2048 Hz, converted to digital data by a 12-bit analog-to-digital converter (EMG-USB 2, 256-channel EMG amplifier; OT Bioelettronica, Torino, Italy; 3 dB, bandwidth 10–500 Hz). EMG signals were amplified by a factor of 2000, 1000, 500, 500, and 500 for the 10%, 30%, 50%, 70%, and 100% MVC contractions, respectively. Data were stored on a computer hard disk and analyzed in Matlab offline (The Mathworks Inc., Natick, MA). Finally, before decomposition, the 64-monopolar EMG channels were re-referenced offline to form 59 bipolar channels using the difference between the adjacent electrodes in the direction of the muscle fibers.

Signal Analysis

Torque. The torque signal was low-pass filtered offline at 15 Hz. The coefficient of variation (CoV) of torque (SD torque/mean torque) was calculated from the stable torque region during the submaximal contractions. RTD was calculated from the ballistic contractions as the maximum slope

of the torque–time curve (Δtorque/Δtime) as presented previously (35). Briefly, for RTD calculation, the torque signal that was originally sampled at 2048 Hz was low-pass filtered at 15 Hz and then resampled at 30 Hz, and the peak slope was detected from the derivative of this torque signal. The onset of torque during the ballistic contractions was defined as the time instant when torque exceeded 7.5 N·m (1).

Interference EMG. The average rectified value (ARV) obtained from submaximal, maximal, and explosive contractions was averaged over all channels of the electrode grid to increase its repeatability between pre- and postintervention trials (14). During the submaximal isometric contractions, the ARV was computed from the HDEMG and bipolar (for BF) signals in intervals of 1 s. These values were extracted from the stable torque region of the contractions (e.g., hold phase of 20 s at 30% MVC). The ARV of the maximal (MVC) contractions was analyzed in a time window of 250 ms centered at the peak EMG activity. During the explosive contractions, ARV was calculated in a 50-ms interval centered at the time instant of the maximal slope in torque (35). Finally, coactivation was quantified as the average of VM and VL ARV divided by the BF ARV (33).

Motor unit analysis. The EMG signals recorded during the submaximal isometric contractions (from 10% to 70% MVC) were decomposed offline with a method that has been extensively validated (29). The signals were decomposed throughout the entire duration of the submaximal contractions, and the discharge times of the identified motor units were converted in binary spike trains (24). The mean discharge rate and the discharge rate variability (CoV of the interspike interval [CoV_{isi}] see below for details) were calculated during the stable plateau torque region. Recruitment thresholds for each motor unit were defined as the knee extension torque (N·m) at the times when the motor unit began discharging action potentials. Discharge times that were separated from the next by >200 ms were excluded from the estimation of recruitment thresholds to avoid aligning the thresholds with noise-generated discharges. Only motor units with a CoV_{isi} <30%, which satisfied the constrains described by Negro et al. (29) during the stable torque portion of the contraction were considered for further analysis. Finally, discharges that were separated from the next by <33.3 or >200 ms (30 and 5 Hz, respectively) were excluded from the mean discharge rate and CoV_{isi} estimates because these discharges are likely due to decomposition errors (24).

Motor unit tracking. A motor unit tracking procedure was applied using a method that has been recently presented (25). The motor unit identification and tracking method is an extension of the convolutive blind source separation technique described by Negro et al. (29), and it was adapted to extract motor units with multichannel action potential shapes maximally similar across sessions. After the full blind HDEMG decomposition was performed on the baseline recording session, we applied a semiblind separation procedure on the posttraining session, focusing on finding only the sources that had dewhitened projection vectors (original multichannel filters or motor unit action potential profiles) similar to the ones extracted from session 1. The normalized cross correlation between the extended projection vectors was used as a measure of similarity. For each motor unit identified in the preintervention trial, we ran the semiblind algorithm on the postintervention trial until a motor unit with normalized cross correlation >0.8 was found. The algorithm maximized the probability to find the matched motor units across different trials (25). In this study, we used an extension factor of 16 for the decomposition iteration and 50 samples for computing the similarity measures between dewhitened projection vectors (motor unit action potential profiles). These parameters have been validated by Martinez-Valdes et al. (25).

Statistical analysis. Before comparisons, all variables were tested for normality using the Shapiro–Wilk test. The assumption of sphericity was checked by the Mauchly test, and, if violated, the Greenhouse–Geisser correction was made to the degrees of freedom. Statistical significance was set at $P < 0.05$. Results are expressed as mean and SD unless stated otherwise.

The effects of the two training programs on peak torque (MVC), RTD, time to task failure, CoV of torque and coactivation, and cardiopulmonary fitness parameters ($\dot{V}O_{2peak}$, peak power output, VT1, and VT2) were assessed with a two-way repeated-measures ANOVA with factors group (END and HIIT) and time (before and after). Changes in ARV parameters during MVC, RTD, and the submaximal contractions as well as mean discharge rate and CoV_{isi} were evaluated with a three-way repeated-measures ANOVA with factors group (END and HIIT), time (before and after), and muscle (VM and VL) at each torque level (10%, 30%, 50%, and 70% MVC) independently. Pairwise comparisons were made with the Student–Newman–Keuls *post hoc* test when ANOVA was significant. A four-way repeated-measures ANOVA was performed (factors: group, time, muscle, and torque level [10%, 30%, 50%, and 70% MVC, respectively]) to check whether the recruitment thresholds (knee extension torque at which motor units began discharging action potentials) of the identified motor units, at each submaximal MVC level, increased with torque and also to evaluate if this parameter changed after the intervention. The intraclass correlation coefficient ($ICC_{2,1}$) was also computed in each of the groups (HIIT and END) at all submaximal torque levels to check the consistency of the recruitment thresholds from the motor units tracked between pre- and posttraining sessions. Finally, the partial eta-squared (η_p^2) for ANOVA was used to examine the effect size of changes in all the aforementioned parameters after the training intervention. An η_p^2 less than 0.06 was classified as "small," 0.07–0.14 as "moderate," and greater than 0.14 as "large" (6).

RESULTS

The two groups initially consisted of nine subjects each; however, one subject from the END group and one subject from the HIIT group did not complete the full training

TABLE 1. Training response for aerobic parameters assessed during incremental cycling in the HIIT and the END training groups.

	HIIT			END		
Parameter	**Pre**	**Post**	***P***	**Pre**	**Post**	***P***
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	44.2 ± 7.1	47.5 ± 8.0*	0.02	44.9 ± 6.3	47.2 ± 4.9*	0.03
Peak power output (W)	334.8 ± 57.8	360.3 ± 53.1*	<0.001	339.6 ± 62.5	361.5 ± 58.3*	<0.001
VT1 (mL·kg^{-1}·min^{-1})	28.0 ± 6.9	32.5 ± 7.8	0.14	28.7 ± 6.6	32.0 ± 4.9	0.17
VT1 (W)	198.5 ± 38.9	222.4 ± 43.6	0.07	196.8 ± 40.5	227.5 ± 36.3	0.05
VT 2 (mL·kg^{-1}·min^{-1})	38.0 ± 6.0	41.2 ± 6.8	0.07	38.4 ± 6.9	41.4 ± 5.9	0.10
VT2 (W)	267.8 ± 39.3	295.0 ± 35.4*	0.03	269.3 ± 53.3	294.0 ± 41.4*	0.01

Values are presented as mean ± SD. VT1, first ventilatory threshold; VT2, second ventilatory threshold or respiratory compensation point. Pre, pre-training; Post, posttraining. There were no significant differences for any variable between HIIT and END (no interaction effects $P > 0.05$). *Significant difference from Pre ($P < 0.05$), according to *post hoc* analysis (Student–Newman–Keuls test).

protocol and were excluded from the analysis. Therefore, results are presented for eight participants in the END group (mean ± SD, age = 29 ± 2 yr, height = 177 ± 6 cm, mass = 77 ± 8 kg) and eight participants in the HIIT group (mean ± SD, age = 29 ± 3 yr, height = 177 ± 7 cm, mass = 79 ± 7 kg). No differences were observed between groups for age, height, and weight ($P > 0.51$). Moreover, there were no differences between the groups for any of the motor output (peak torque, time to task failure, RTD, and CoV of torque), cardiopulmonary fitness ($\dot{V}O_{2peak}$, peak power output, and submaximal ventilation thresholds), or electrophysiological (surface EMG amplitude, vasti–BF coactivation, motor unit discharge rate, CoV_{isi}, and recruitment threshold) parameters assessed during the baseline sessions (before training) ($P > 0.32$ in all cases).

Cardiorespiratory fitness. Table 1 summarizes cardiorespiratory fitness changes assessed pre- and postintervention for the HIIT and the END protocols. Overall, all the variables changed similarly in both groups and none of the parameters showed a between-group interaction effect ($P > 0.56$). $\dot{V}O_{2peak}$ increased after training by 6.7% (4.1%) and 5.0% (7.8%) in HIIT and END group, respectively (main effect for time; $P = 0.001$, $\eta_p^2 = 0.54$). Peak power output also increased by 7.4% (3.3%) in HIIT and by 6.3% (3.0%) in END (main effect for time; $P < 0.001$, $\eta_p^2 = 0.88$). Regarding the submaximal ventilation thresholds, HIIT and END training only induced a significant increase of VT2 exercise intensity (W) of 9.1% (8.3%) and 9.0% (8.2%) in HIIT and END, respectively (main effect for time; $P < 0.001$, $\eta_p^2 = 0.58$). Further results for the cardiorespiratory fitness parameters and *post hoc* tests can be found in Table 1.

Motor output. HIIT and END training induced specific changes in motor performance after the intervention (Fig. 1). Two weeks of HIIT produced a significant increase in peak torque (MVC) of 6.7% (6.6%) that contrasted to the response of END, which showed similar peak torques across pre- and posttesting sessions (time–group interaction; $P = 0.01$, $\eta_p^2 = 0.38$). On the contrary, END showed a significant increase in time to task failure of 16.9% (14.4) that contrasted to the response of HIIT, which showed similar times to task failure across testing sessions (time–group interaction; $P = 0.01$, $\eta_p^2 = 0.33$). Neither HIIT nor END induced any significant change in RTD (time–group interaction; $P = 0.09$, $\eta_p^2 = 0.087$). Finally, the CoV of torque increased significantly from 2.2% (0.4%) to 2.5% (0.6%) after training for the submaximal contractions at 10% MVC in the HIIT group (time–group interaction; $P = 0.033$, $\eta_p^2 = 0.28$). Conversely, the CoV of torque at the other torque levels (30%, 50%, and 70% MVC) showed no significant changes after the intervention for either group ($P > 0.25$) (see Figure, Supplemental Digital Content 1, The CoV of torque for HIIT and END groups across all force levels pre- and post-intervention, http://links.lww.com/MSS/A850).

Surface EMG. Figure 2 shows the EMG amplitude (ARV) of the VM and VL during submaximal (10%, 30%, 50%, and 70% MVC), maximal (MVC), and ballistic isometric knee extension contractions for each testing session (before and after). Overall, both vasti muscles showed similar changes of EMG amplitude over the training period (time–muscle interaction; $P > 0.15$ for all isometric contractions). Regarding submaximal contractions (Figs. 2A and 2B), EMG amplitude

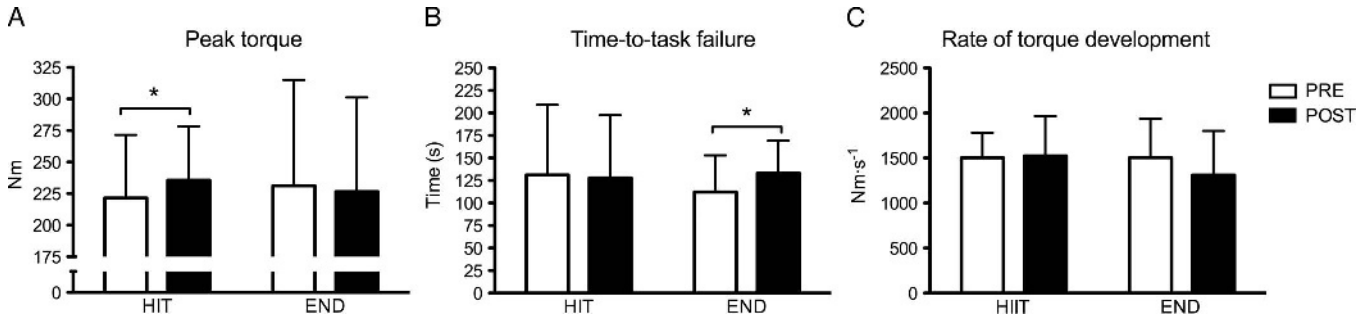

FIGURE 1—Results show changes (mean ± SD) in motor performance across the 2-wk training intervention. A, Peak torque assessed during isometric MVC. B, Time to task failure assessed during sustained isometric contractions at 30% MVC. C, RTD during maximal isometric ballistic contractions (maximum slope). *$P < 0.05$.

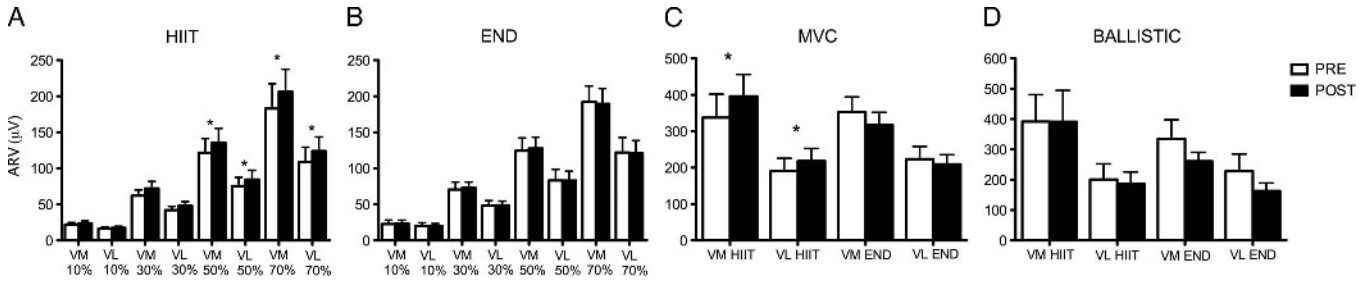

FIGURE 2—Values are presented as means ± SE for the average rectified value (ARV) of the VM and VL obtained during submaximal (10%, 30%, 50%, and 70% MVC), maximal, and ballistic isometric knee extension contractions before and after training (pre and post). A, HIIT submaximal ARV. B, Endurance (END) training submaximal ARV. C, ARV values during MVC for HIIT and END. D, ARV values during explosive contractions for HIIT and END. ARV was assessed during a time interval of 50 ms centered at the time instant of the maximum slope. *$P < 0.05$.

at 10% and 30% MVC did not change after the intervention for any training group or muscle (VM and VL) ($P > 0.14$). However, the ARV of VM and VL during the 50% MVC contractions increased significantly for HIIT (11.4% [7.6%] and 11.3% [5.2%] increase in VM and VL, respectively) but not for END (time–group interaction; $P = 0.007$, $\eta_p^2 = 0.44$). These differences were maintained at 70% MVC (time–group interaction; $P = 0.02$, $\eta_p^2 = 0.35$), where ARV from the HIIT group increased by 13.0% (10.9%) and 14.1% (10.6%) in VM and VL, respectively. A similar result was observed for ARV during

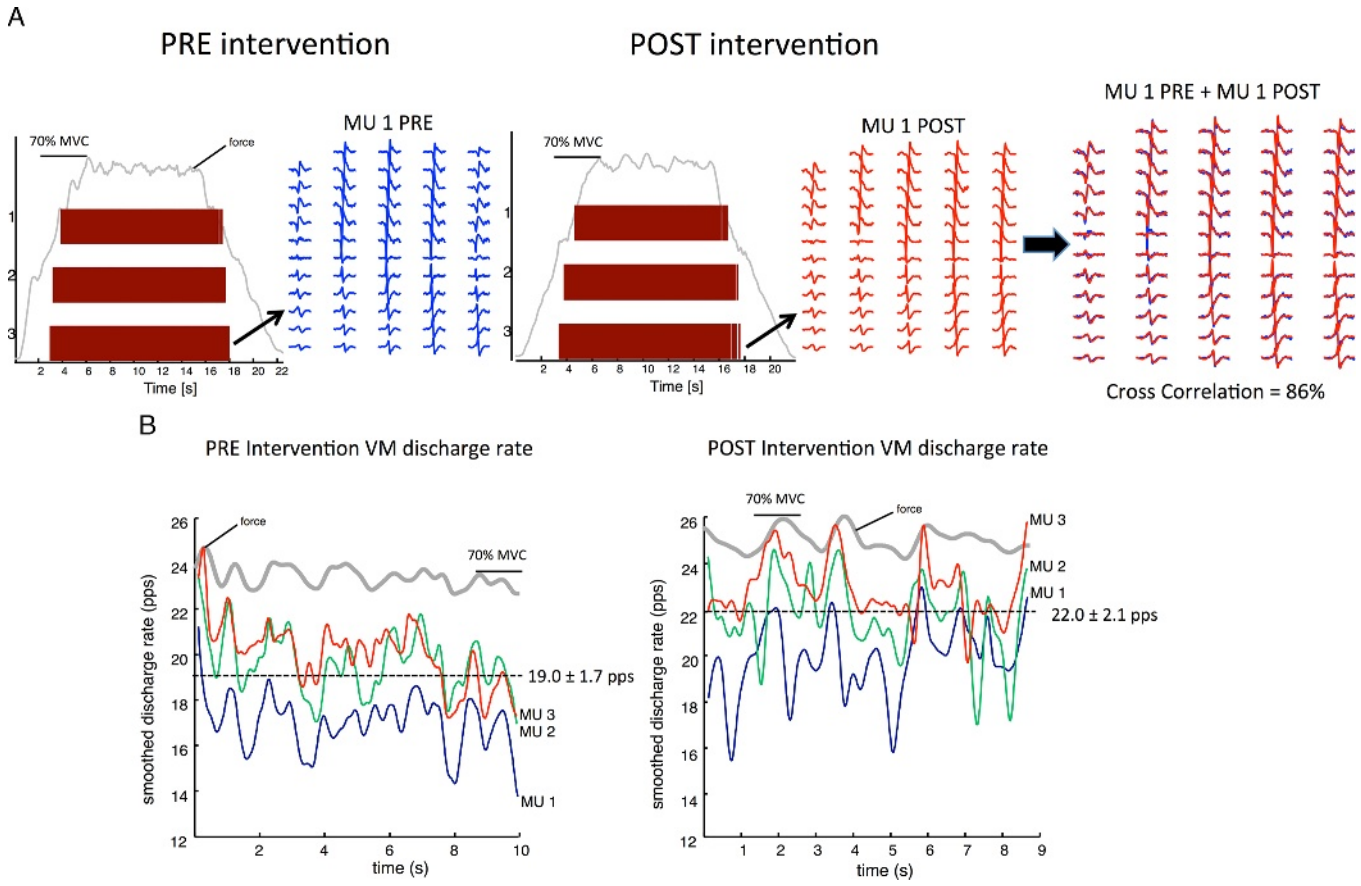

FIGURE 3—Procedure for motor unit tracking from one representative subject in the HIIT group. A, Three VM motor unit spike trains decomposed with convolutive blind source separation at 70% of the MVC before (PRE) the intervention can be seen in the left half of the figure. A dewhitened projection vector (motor unit action potential shapes across the electrode grid in 59 single differential channels) from the first motor unit is shown in blue. Semiblind source separation was applied after the intervention to extract the source that was maximally similar to the projecting vector of motor unit one (center half of the figure, *red*). Finally, these two projecting vectors were compared by cross correlation (right half of the figure) and were regarded as the same motor unit because they had a cross correlation of 86%. This procedure was repeated for motor units 2 and 3 (not shown). B, Instantaneous firing rates (motor unit firings were low-pass filtered at 2 Hz) from the same three motor units presented in panel A, during the stable force region before (PRE, left half of the figure) and after (POST, right half of the figure) the intervention. This subject increased peak toque by 5% (298.0 vs 313.6 N·m) after the intervention. The recruitment thresholds of these units were 192.2 vs 192.2 N·m (64.5% vs 61.3% MVC, MU 1), 175.5 vs 178.5 N·m (58.9% vs 56.9% MVC, motor unit 2), and 168.4 vs 177.0 N·m (56.0% vs 56.4% MVC, motor unit 3), pre- and postintervention, respectively. Note the increase in firing rates from 19 ± 1.7 to 22.0 ± 2.1 pps.

the maximal contractions (Fig. 2C) because VM and VL activity only increased in the HIIT group by 17.3% (12.6%) and 14.1% (10.2%), respectively (time–group interaction; $P = 0.001$, $\eta_p^2 = 0.55$). Neither HIIT nor END training induced any significant change in ARV during the ballistic contractions (Fig. 2D) ($P > 0.16$). Finally, the amount of vasti–BF coactivation did not differ across sessions in either group ($P > 0.50$ for all isometric contractions).

Motor unit decomposition and tracking. The total number of decomposed motor units across the different torque levels and sessions was between 134 (116–154) and 122 (95–141), mean (range), for VM and VL, respectively. An example of the motor unit tracking procedure is reported in Figure 3. Figure 3A shows three motor units of the VM muscle that were identified at 70% MVC (upper left corner). A dewhitened projection vector (motor unit action potential profile) from motor unit 1 (MU 1 PRE, blue) was extracted. This vector was then used to find a source that was maximally similar after the intervention (MU 1 POST, red). Finally, both projection vectors were visually inspected and matched by cross correlation to confirm that the automatic tracking was correct (cross correlation between both projected vectors was 0.86, Fig. 3A, right). This procedure was then repeated for motor units 2 and 3 (not shown in the figure). Figure 3B shows instantaneous discharge rates during the stable force part of the isometric contraction at 70% MVC (motor unit firings were low-pass filtered at 2 Hz) from the same three tracked motor units presented in Figure 3A PRE (left) and POST (right) HIIT. A clear increase from 19.0 ± 1.7 to 22.0 ± 2.1 pulses per second (pps) was observed for these units after the intervention (see Motor Unit Properties section). After this procedure, the number of tracked motor units across pre- and postintervention testing sessions varied between 60 (46–69) and 50 (33–74) for VM and VL, respectively (across all submaximal force levels, in all 16 subjects). Therefore, 44.8% (39.5%–50.9%) and 41.0% (33.7%–49.7%) of motor units from those identified by decomposition could be tracked across sessions (average number of tracked motor units per subject was 4 ± 1 and 3 ± 1 for the VM and VL, respectively). The cross-correlation values from the projecting vectors of the tracked motor units (from VM and VL) ranged between 0.80 and 0.96 (average: 0.86).

Motor unit properties. Figure 4 depicts the mean motor unit discharge rate for the VM and VL during the submaximal contractions at 10%, 30%, 50%, and 70% MVC. No differences in the mean motor unit discharge rate were observed between VM and VL in each testing session (time–muscle interaction; $P > 0.30$ for all submaximal contractions). However, VM showed significantly greater mean motor unit discharge rates at 50% and 70% MVC (effect: muscle; $P = 0.006$, $\eta_p^2 = 0.45$ and $P = 0.016$, $\eta_p^2 = 0.37$, at 50% and 70% MVC, respectively). For the contractions at 10% and 30% MVC (low-threshold motor units, Fig. 4A), the average discharge rate for both vasti muscles was not influenced by either training (time–group interaction; $P = 0.30$ and 0.1, at 10% and 30% MVC, respectively). However, at both 50% and 70% MVC (high-threshold motor units, Fig. 4B), the VM and VL increased their discharge rates (by 8.5% [9.0%] and 9.5% [7.1%] at 50% MVC and by 12.1% [7.6%] and 9.5% [6.6%] at 70% MVC in VM and VL, respectively) in the HIIT group but not in the END group (time–group interaction; $P = 0.036$, $\eta_p^2 = 0.29$ and $P = 0.015$, $\eta_p^2 = 0.38$, at 50% and 70% MVC, respectively). The

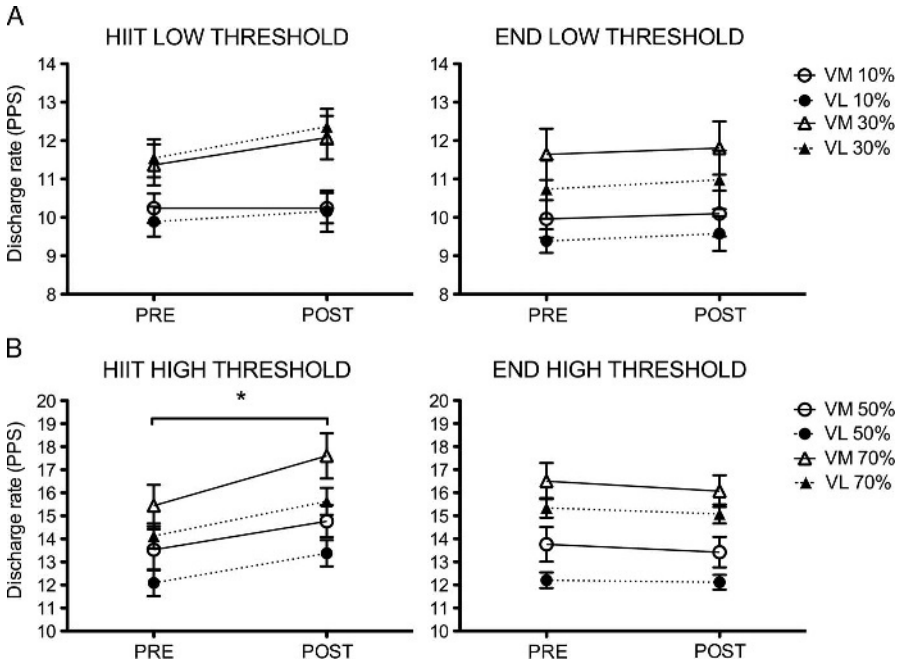

FIGURE 4—Values are presented as means ± SE for motor unit discharge rates (pps) of the VM and VL obtained during submaximal (10%, 30%, 50%, and 70% of the MVC) contractions. A, Low-threshold motor units discharge rate results (10% and 30% MVC) of endurance (END) and HIIT. B, High-threshold motor unit discharge rate results (50% and 70% MVC) of END and HIIT. *$P < 0.05$.

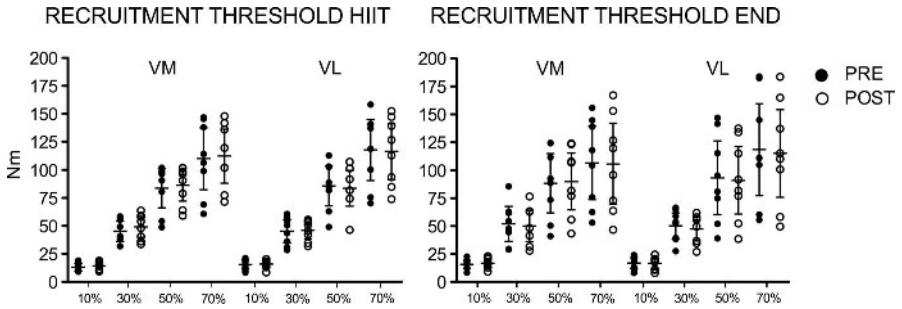

FIGURE 5—Motor unit recruitment threshold individual values (whiskers represent the 95% confidence interval) for VM and VL, before (PRE, *filled circles*) and after (POST, *open circles*) HIIT and endurance training (END) at all force levels (10%, 30%, 50%, and 70% MVC).

recruitment thresholds of the identified motor units increased with torque (effect: torque; $P < 0.001$, $\eta_p^2 = 0.88$), similarly for both muscles (torque–muscle interaction; $P = 0.2$, $\eta_p^2 = 0.12$), and did not change after the intervention (time–group–torque interaction; $P = 0.16$, $\eta_p^2 = 0.14$). These results are confirmed by the high ICC found for the recruitment thresholds pre- and postintervention at all force levels (average ICC of 0.90 and 0.95 for HIIT and END, respectively) (Fig. 5). Finally, neither training induced change in CoV_{isi} ($P > 0.57$) (see Table, Supplemental Digital Content 2, VM and VL CoV_{isi} results for HIIT and END groups across all force levels pre- and postintervention, http://links.lww.com/MSS/A851).

DISCUSSION

This is the first study to show that HIIT and END training elicit distinct adjustments in motor output and motor unit behavior despite similar changes in cardiorespiratory fitness. HIIT determined an increase in MVC peak torque, with an increase in EMG amplitude and motor unit discharge rate at the highest force levels (from 50% MVC and above). Conversely, END induced an increase in time to task failure for a sustained contraction at 30% MVC and no changes in isometric knee extension strength or motor unit discharge rate. Taken together, these findings suggest that HIIT and END induce specific neuromuscular adaptations, which likely relate to their differences in exercise intensity and training volume.

Training protocols and motor output. Previous studies have reported that HIIT can be used as an alternative to endurance training. Studies comparing short-term low-volume HIIT and high-volume END have found similar physiological adaptations in aerobic metabolism (16,22), exercise performance (16,26), and cardiorespiratory fitness (15,26), despite large differences in exercise volume and exercise intensity. Therefore, we used previously validated protocols that differed in both time commitment and intensity but were known to induce similar metabolic and cardiorespiratory fitness adaptations (16,22). These protocols were selected to verify whether similar adaptations were also observed at the neuromuscular level, despite the divergent nature of both training regimes (HIIT: low-volume, high-load vs END: high-volume, low-load). As expected, the two trainings resulted in a similar increase in $\dot{V}O_{2peak}$, peak power output, and submaximal ventilation thresholds (Table 1), in agreement with previous reports (15,26). However, HIIT and END induced different changes in motor performance that can be related to their different training characteristics (Fig. 2). Currently, there are no other studies that have detailed changes in neuromuscular performance after HIIT. In the only study that examined changes in muscle function, the authors did not observe changes in isometric knee extension strength after a 4-wk HIIT intervention (7), in contrast with our results. However, the training consisted of lower loads (average peak power output of 236 vs 335 W, in the current study). Moreover, peak power output was estimated with a stepwise incremental cycling protocol with relatively long steps of 3 min, which is known to underestimate the peak power (38). The current results suggest that HIIT training must be performed at the maximum (or supra maximum) power output achieved during an incremental ramp test to induce a significant increase in knee extensor strength. Indeed, the repetitive muscle activity at high loads was presumably responsible for the increase in MVC peak torque after HIIT.

Previous studies have also reported a significant increase in isometric knee extension endurance time (time to task failure) during low-level submaximal contractions after an END training intervention (34,35). For instance, Vila-Chã et al. (35) observed a 30% increase in time to task failure after a 6-wk END cycling intervention. In the same study, the authors did not find any increase in time to task failure after strength training. These results are comparable to our findings. Again, these different adaptations are presumably due to the differences in training volume and exercise intensity between the two interventions (HIIT: short periods of activity at high intensity vs END: long periods of activity at moderate intensity).

Although HIIT was associated with increased MVC peak torque, no change in RTD was observed (Fig. 1C). Small to moderate increases in knee extensor strength (approximately 7% in the current study) are not typically associated with increased RTD. Both Vila-Chã et al. (35) and Aagaard et al. (1) only observed an increase in RTD after the isometric knee extension strength (after resistance training) increased by 18% and 17%, respectively. Nevertheless, it is possible that more ballistic HIIT protocols, such as the Wingate-based sprint interval training, may induce changes in RTD.

Maximal and submaximal contractions and global EMG parameters. Changes of VM and VL EMG amplitude showed similar behavior in the HIIT and the END groups at the lowest torque levels (10% and 30% MVC), where no significant change in EMG amplitude was observed. However, only HIIT showed a significant increase in EMG amplitude for both vasti muscles in contractions at 50%, 70%, and 100% MVC (Fig. 2). Previous studies have documented that both increases in muscle cross sectional area and neural factors are responsible for increases in maximal muscle strength (9). Because changes in muscle fiber architecture have not been documented after only 2 wk of training, the surface EMG results in the current study strongly suggest that the observed changes in maximal isometric muscle torque after HIIT are mainly of neural origin. Increased agonist muscle activation and decreased antagonist activation have been suggested as important factors influencing increases in muscle strength (9). However, we did not identify changes in vasti–BF coactivation (at all torque levels). Therefore, the increased maximal torque was presumably due to factors that also influenced the EMG–torque relation in the agonist, such as changes in motor unit discharge rates or peripheral factors (e.g., muscle fiber conduction velocity), as also shown in a recent study (35). These early adaptations likely involve changes in supraspinal excitability, spinal pathways, or changes in the membrane properties in the motoneurons (9). Nevertheless, the exact nature of these early neural adaptations is not yet known (9). Regarding the submaximal contractions, the observed changes in surface EMG amplitude in HIIT were markedly greater among the highest contraction levels (Fig. 2A), which ultimately suggest a preferential change in the discharge rates of high-threshold motor units (see Submaximal Contractions and Motor Unit Properties section). Indeed, it is likely that the high loads placed on the subjects during HIIT increased the activity of these units. In support of this observation, Vila-Chã et al. (35) previously reported an increase in EMG amplitude at 30% and 100% MVC, but not at 10% MVC, after 3 wk of resistance training. However, this earlier work also showed an increase of EMG amplitude at 10% and 30% MVC after 3 wk of END training. Because a decrease in motor unit discharge rate was simultaneously observed after END training, this result was interpreted as an increase of motor unit recruitment at these force levels, although EMG amplitude depends on multiple influencing factors (11,12). In this study, we attempted to limit the variability in EMG amplitude estimates by averaging across all electrodes of the grid (14,24).

Submaximal contractions and motor unit properties. In accordance with the surface EMG results, the HIIT and the END groups showed similar motor unit discharge rates pre- and posttraining for VM and VL at 10% and 30% MVC. However, only HIIT induced an increase in motor unit discharge rate at 50% and 70% MVC, which is also in agreement with surface EMG results (Fig. 4). Together, these findings suggest that changes in motor unit discharge rate are specific not only to the training protocol but also to the size (18) and threshold of the motor units recruited during the exercise. Indeed, the main differences between HIIT and END are the volume of training and the loads at which the subjects perform the exercise. Although we did not measure motor unit recruitment during cycling (this is not technically possible), the HIIT protocol that involved short exercise bouts at the maximal power output likely required the recruitment of most motor units (20,36), whereas the END training that was performed at a much lower load likely involved lower threshold units with greater aerobic capacity (20,36). In accordance with size-specific adjustments in motor units, Kamen and Knight (19) previously observed an increase in VL discharge rates at 100% MVC, but not at 10% or 50% MVC, after 6 wk of resistance training involving maximal knee-extension isometric contractions. For END, we did not find training-induced changes in motor unit discharge rates in the torque range investigated (Fig. 4). This observation is in accordance with Mettler et al. (27) but contrasts with the results of Vila-Chã et al. (35). However, the latter study differed with respect to ours for training intensity (50% to 75% of heart rate reserve vs 65% $\dot{V}O_{2peak}$), volume (60–150 vs 285–345 min·wk^{-1}), and duration (3–6 vs 2 wk) (35). Collectively, these findings suggest that END would lead to either maintained or decreased discharge rates because MVC torque is not expected to change after this type of training (17). Maintained or decreased motor unit discharge rates after END training interventions (at the same relative torque level) are thought to be important factors for longer times to task failure during submaximal, isometric fatiguing contractions (27,34,35).

The tracking technique applied in this study allowed for the first time to compare individual motor unit recruitment thresholds before and after training. The recruitment thresholds of the tracked motor units were similar before and after the intervention for both muscles and groups (Fig. 5), suggesting that the observed changes in discharge rate after HIIT were mainly due to an increased neural drive to the muscle, and not to changes in intrinsic motor neuron properties. Previous studies documenting changes in motor unit discharge rates have used unmatched population samples to infer adaptations to a particular motor unit pool (19,27,30,32,35). However, these previous approaches are limited by the possibility of comparing different motor units, with different recruitment thresholds, in the pre- and posttraining sessions. Conversely, we could record and follow the same motor units across sessions, providing an accurate interpretation of changes in discharge rate and recruitment threshold. Finally, no change in discharge rate variability (CoV_{isi}) was observed for any of the groups after the intervention, despite that there was a significant increase in the CoV of torque for the HIIT group at 10% MVC. A recent study showed that 6 wk of resistance training increases force/torque steadiness (reduction in the CoV of force/torque) and reduces motor unit discharge rate variability (CoV_{isi}) in submaximal contractions at 20% and 30% MVC (33). However, an increase in force steadiness after resistance training has not been observed in all studies (3), and the association between enhanced force steadiness and the

reduction of CoV_{isi} is poor (28). Therefore, the increase in the CoV of torque at 10% MVC for the HIIT group in the present study could be related to other factors rather than an increase in CoV_{isi}. Although the high loads performed during HIIT might have induced a reduction in the accuracy to maintain the required steadiness at low torque levels, torque steadiness remained similar at all torque levels after END training despite of the low to moderate loads used for this type of training. Therefore, the observations of training-induced changes in torque steadiness require further investigation.

Methodological implications. In this study, for the very first time, we applied motor unit tracking across sessions to study training interventions (25). With this approach, all differences in motor unit discharge rate between END and HIIT groups had a large effect size and showed a clear intervention effect. Previous investigations of this type but without motor unit tracking have shown contradicting results (19,27,32,35). Some studies have even failed to report an effect in discharge rates despite clear increases in muscle strength and surface EMG amplitude (30). We suggest that these changes could have been masked because of the low number of identified motor units (usually low-threshold) and the unmatched motor units across sessions. Accordingly, we have previously shown that the effect size in longitudinal investigations is substantially increased with our technique (25), which opens new possibilities for further research.

CONCLUSION

Two weeks of HIIT and END showed similar improvement in cardiorespiratory fitness but different adjustments in motor unit behavior. HIIT enhanced maximum torque output and was accompanied by an increase in motor unit discharge rate at the highest torque levels (50% and 70% MVC). By contrast, END increased the time to task failure but did not influence motor unit discharge rates. These findings reveal that HIIT and END induce differential adaptations among low- and high-threshold motor units. The study also shows the first results on training-induced changes in motor unit discharge rate by tracking the same individual units before and after training. This methodology may open new perspectives in the study of neural adaptations to training.

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